9

Endodontic Emergencies and Therapeutics

RICHARD WALTON AND NIKITA B. RUPAREL

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Identify causes of emergencies as they occur before treatment, between appointments, and after obturation, differentiating urgency from a true emergency.
2. Identify patients who are at greater risk for experiencing pain after endodontic procedures.
3. Describe the psychological and physiologic factors that affect pain perception and pain reaction and how these are managed.
4. Define the flare-up and describe its management.
5. List the factors that relate to greater frequency of interappointment or postobturation flare-ups.
6. Describe and outline a sequential approach to endodontic emergencies:
   a. Determine the source of pain (pulpal or periapical)
   b. Establish a pulpal and periapical diagnosis
   c. Identify the etiologic factor of the pathosis
   d. Design an emergency (short-term) treatment plan
   e. Design a long-term treatment plan
7. Outline a system of subjective and objective examinations and radiographic findings to identify the source of pain and the pulpal or periapical diagnosis.
8. Describe when pretreatment emergencies might occur and how to manage these emergencies.
9. Outline the steps involved in treatment of painful, irreversible pulpitis.
10. Describe the steps involved in treatment of necrotic pulp with acute apical periodontitis.
11. Describe treatment of acute apical abscess and include the indications and procedure for incision and drainage.
12. Detail pharmacologic supportive therapy (analgesics, anxiolytics, antibiotics, and antiinflammatory agents) used in emergencies and its role in controlling pain and infection.

Introduction

Endodontic emergencies constitute approximately two-thirds of all dental emergencies,[1,2] with many patients seeking emergency care for a painful tooth.[1-3] There are a very large number of hospital emergency room visits for mouth abscess/facial cellulitis.[4] These painful and infectious emergencies pose significant challenges to patients and dental providers. Pain and/or swelling occur in patients before (pretreatment), during (interappointment), and after (postobturation) root canal treatment.[5-10] Understanding the causes of such emergencies results in appropriate diagnosis and treatment protocols for resolution of signs and symptoms.

Classical studies by Stanley, Fitzgerald, and Kakehashi[11] as well as the preponderance of endodontic literature point to a bacterial etiology as the initial causative factor for pulpal pathosis. Both primary and secondary endodontic infections are polymicrobial in nature with an average of 10 to 20 microorganisms in primary[12] and 1 to 3 microbial species in secondary infections.[13] Examples of endodontic pathogens include gram positive (*Enterococcus* species) and gram-negative (*Porphyromonas, Prevotella, Bacteroides* species)

bacteria as well as members of the spirochete family (*Treponema* species). Other pathogens include viruses and fungi. Bacterial byproducts from such a mixed flora therefore constitute a soup of toxins that breakdown host tissues and initiate an inflammatory response. An untimely management attenuating such a response can lead to spread of the inflammatory mediators into the periapical regions, posing greater risks, such as facial abscesses and cellulitis (see Chapter 1).

Bacterial toxins fall into two categories: exotoxins and endotoxins. Exotoxins are secreted and released by the microorganism to degrade host tissues such as the extracellular matrix. Many of these function via enzymatic tissue breakdown and include enzymes belonging to the family of collagenases, proteases, lipases, chondroitinases, hyaluronidases, and cysteine and serine proteinases, among others. On the other hand, endotoxins are typically cell membrane/wall components such as lipoteichoic acid (LTA) and lipopolysaccharide (LPS). Still other specialized toxins include hemolysins and flagellins. Endotoxins play a crucial role in mediating pulpal pain. Because pain is the #1 reason why patients seek dental care, an in-depth understanding of pain detection and transmission is critical for management of a painful emergency.

LPS from a known endodontic pathogen, *Porphyromonas gingivalis,* has been shown in recent years to activate and sensitize trigeminal sensory neurons via Toll-like receptor-4 (TLR-4) expressed on sensory neurons.[14,15] On the other hand, non-TLR mechanisms have also been demonstrated for other bacterial species such as *Staphylococcus aureus* and *Escherichia coli*.[16-18] Moreover, these studies suggest that bacterium-induced pain does not depend on tissue edema or immune cell activation.[18] Collectively, these studies demonstrate that the concentration of the bacterial load dictates hyperalgesic conditions rather than local tissue inflammation (see Chapter 1).

Nonbacterial etiologies can also contribute to pulpal inflammation and emergency situations. These emergency situations include pulpal inflammation caused at the time of caries removal or crown preparations performed without adequate coolant, characteristics of the cavity preparation most importantly, remaining dentin thickness over the pulp, and type of restorative material.[19] It is well known that certain restorative materials promote marginal breakdown due to polymerization shrinkage and promote bacterial microleakage, whereas others release chemical agents such as resin monomers namely, BisGMA, urethane dimethacrylate (UDMA), and triethylene glycol dimethacrylate (TEGDMA), among others. These along with monomers present in dentin bonding agents such as hydroxyethyl methacrylate (HEMA) can be toxic to the underlying pulp if not polymerized fully.[19-22] Biomaterial studies indicate that the hydrophobicity of nonpolymerized monomers displaces dentinal fluid, thereby diffusing through the tubules and into the pulp.[20,23] Collectively, factors that do not directly point to a microbial etiology must be considered in emergencies and appropriate diagnosis must be determined to deliver a suitable treatment.

Both microbial and nonmicrobial etiologies culminate in an immunologic response. Patients with emergencies often report symptoms of spontaneous pain, pain to swallow, referred pain, pain that wakes them up, and pain that lingers after a stimulus. These symptoms are often mediated by an immunologic response and by (1) inflammatory mediators and (2) fluid pressure. Management of such a painful episode cannot be efficiently achieved by pharmacologic means alone.[24-26] Peripheral nerve sprouting,[27-31] increased expression of tetrodotoxin resistant (TTX-R) sodium channels,[32] as well as increased expression of transient receptor potential (TRP) channels,[33,34] all culminate in dramatic reduction of peripheral nerve thresholds of Aδ and C-fibers. Clinical studies consistently implicate preoperative pain as a strong predictor of postoperative pain.[35] Therefore emergency protocols must make every effort to attenuate preoperative pain to minimize development of chronic odontogenic pain conditions.

Endodontic emergencies are therefore a challenge for both diagnosis and management. Knowledge and skill in several aspects of endodontics are required; failure to apply these can result in serious consequences. Incorrect diagnosis and/or treatment can shift an acute condition to a chronic pain or infection-induced life-threatening condition. The clinician must have knowledge of pain mechanisms, patient management, diagnosis, anesthesia, therapeutics, and appropriate treatment measures for both hard and soft tissues. This chapter discusses approaches to the diagnosis and treatment of various categories of emergencies. It includes a review of etiologic factors and details of a systematic approach to identifying and diagnosing the offending cause; then appropriate treatment, including pharmacotherapy, is described.

Definition

By definition, endodontic emergencies are usually associated with pain and/or swelling and require immediate diagnosis and treatment. These emergencies are caused by pathoses in the pulp or periradicular tissues. They also include severe traumatic injuries that result in luxation, avulsion, or fractures of the hard tissues. Management of emergencies related to trauma will not be included in this chapter (see Chapter 11).

Categories

Pretreatment Emergency

These are situations in which the patient is seen initially with severe pain and/or swelling. Problems occur with both diagnosis and treatment.

Interappointment and Postobturation Emergency

Also referred to as a "flare-up," an interappointment and postobturation emergency problem occurs after an endodontic appointment. Although an upsetting event, this problem is easier to manage because the offending tooth has already been identified and a diagnosis has been previously established. Also, the clinician has knowledge of the prior procedure and will be better able to correct the problem.

The Challenge

It is satisfying and rewarding to successfully manage a distraught patient who has an emergency (Fig. 9.1). In contrast, it is very distressing to have a patient with a flare-up after root canal treatment in a previously asymptomatic tooth. The aim is to increase occurrences in the first category and decrease those in the second.

Differentiation of Emergency and Urgency

Whether a pretreatment, interappointment, or postobturation problem, it is important to differentiate between a *true emergency* and the less critical *urgency.* A *true emergency* is a condition requiring an unscheduled office visit with diagnosis and treatment. The

• **Fig. 9.1** Patient is distraught from severe pain of irreversible pulpitis. This patient will be a challenge to diagnose and treat.

visit cannot be rescheduled because of the severity of the problem. *Urgency* indicates a less severe problem; a visit may be scheduled for mutual convenience of the patient and the dentist. Key questions, which may be asked by telephone, to determine severity include the following:

1. Does the problem disturb your sleeping, eating, working, concentrating, or other daily activities? (A true emergency disrupts the patient's activities or quality of life.)
2. How long has this problem been bothering you? (A true emergency has rarely been severe for more than a few hours to 2 days.)
3. Have you taken any pain medication? Was the medication ineffective? (Analgesics do not relieve the pain of a true emergency.)

Affirmative answers to these questions require an immediate office visit for management and constitute a true emergency. Obviously, the patient's emotional and mental status must also be determined. To some patients, even a minor problem has major proportions and is disruptive.

Development of a System

Because a misdiagnosis will probably result in improper treatment and an exacerbation of the problem, a systematic approach is mandatory. The emotional status of the patient, pressures of time, and stress on dentist and staff should not affect such an orderly approach.

Pain Perception and Pain Reaction

Pain is a complex physiologic and psychological phenomenon. Pain perception levels are not constant; pain thresholds as well as reactions to pain change significantly in various circumstances.[36] Psychological components of pain perception and pain reaction comprise cognitive, emotional, and symbolic factors. The pain reaction threshold is significantly altered by past experiences and by present anxiety levels and emotional status. Anxiety decreases levels of both pain perception and pain reaction.[37]

To reduce anxiety and consequently obtain reliable information about the chief complaint and to receive cooperation during treatment, the clinician should (1) establish and maintain control of the situation, (2) gain the confidence of the patient, (3) provide attention and sympathy, and (4) treat the patient as an important individual.[38] Providing positive written information about pain control during root canal therapy can also reduce the fear associated with an emergency endodontic procedure.[39] By managing these pain components, pain perception and reaction thresholds are raised significantly, greatly facilitating the procedure. Psychological management of the patient is the most important factor in emergency treatment!

Adjunctive pharmacotherapy may also be required in the management of patient anxiety during emergency treatment.[36] Reducing anxiety at this stage will not only reduce the response to potentially painful stimuli during treatment, but also will decrease the tendency for the patient to recall the endodontic procedure as unpleasant.[37] Mild anxiety may be managed with nitrous oxide[40]; however, the apparatus may be a bit cumbersome when obtaining treatment radiographs. Oral benzodiazepines can be very effective in managing more significant anxiety. Triazolam has a fast onset and a relatively short half-life and, because of its lipophilic nature, can be administered sublingually for rapid absorption.[41] Thus this anxiolytic medication is quite convenient for sedation in the dental office. One quarter of a milligram of oral triazolam has been shown to be as effective as intravenous diazepam.[42] Of course, patients who have taken or are given an oral sedative in the dental office must have transportation provided. Importantly, the potential drug interactions with other centrally acting agents must also be considered.

System of Diagnosis

Patients in pain often provide information and responses that are exaggerated and inaccurate. They tend to be confused as well as apprehensive. Clinicians may find it easy (and tempting) to rush through the diagnosis to institute treatment for a suffering patient. After pertinent information regarding the medical and dental histories is obtained, both subjective questioning and an objective examination are performed carefully and completely (See Box 9.1).[43,44]

A rule of the true emergency is that *one tooth is the offender,* that is, the source of pain. In the excitement of the moment, the patient might believe that the severe pain is emanating from more than a single tooth. The clinician may become convinced also, leading to overtreatment.

Medical and Dental Histories

Medical and dental histories should be reviewed first. If the patient is the dentist's own patient, the medical history is briefly reviewed and updated. If the patient is new, a standard, complete history is obtained. An important medical complication may be easily overlooked in an emergency situation. Certainly, the need for antibiotic prophylaxis must be determined even before initiating any portion of the oral examination that might induce a bacteremia, such as periodontal probing. Either a short or a complete dental history is recorded. This process includes recollection of dental procedures, recording a chronology of symptoms, or discussing an earlier relevant comment by a dentist.

Subjective Examination

When the patient is in pain, the subjective examination comprises careful questioning and is the most important aspect of diagnosis. Questions relate to the history, location, severity, duration, character, and eliciting stimuli of pain. Questions relating to the cause or stimulus that elicits or relieves the pain help select appropriate objective tests to arrive at a final diagnosis.

Pain that is elicited by thermal stimuli and/or pain that is referred is likely to originate from the pulp. Pain that occurs on mastication or tooth contact and is well localized is probably apical.

The three important factors constituting the quality and magnitude of pain are its spontaneity, intensity, and duration. If the patient reports any of these symptoms (and assuming that the patient is not exaggerating), significant pathosis is likely to be present. Careful questioning provides important information about the source of the pain and whether it is pulpal or periradicular. In fact, a perceptive, clever clinician should be able to arrive at a tentative diagnosis by means of a thorough subjective examination; objective tests and radiographic findings are then used for confirmation. For example, a reported complaint of severe, continuous (lingering) pain when the patient drinks cold beverages and marked tenderness on mastication indicates irreversible pulpitis and symptomatic apical periodontitis. These stimuli are then repeated in an objective examination to confirm the patient's response.

Objective Examination

An endodontic diagnosis consists of two parts: pulpal and periapical diagnoses. Therefore objective examination is a comprehensive evaluation of the health of the pulp and periapical tissues. The clinician's first clue to identifying the offending tooth is to carefully listen to the patient's chief complaint and reproduce it using all the available tools. Objective examination includes the following clinical tests.

Physical Condition/Extraoral Examination

It is imperative to not miss signs of an infectious spread systemically. Signs include extraoral swelling (unilateral or bilateral), facial cellulitis, lymphadenopathy, trismus, and eye shut. Such signs are commonly also involved with elevated temperature. In addition to the emotional factors that complicate the diagnosis of endodontic emergencies, physical conditions induced as a result of these situations also contribute to the problems. Pain or swelling may limit mouth opening, thereby hampering diagnostic procedures as well as treatment (Fig. 9.2, *A–D*). In addition, hypersensitivity to thermal stimuli or pressure influences diagnosis and treatment. Therefore the most severe aspect of the emergency is treated first to facilitate diagnosis.

Intraoral Examination

Included under this examination is observation for intraoral swelling or sinus tracts, as well as mirror and explorer examination to note the presence of defective restorations, discolored crowns, recurrent caries, and fractures.

Pulp Vitality Tests

Pulp vitality tests are the most commonly used objective tests for diagnosing a painful or offending tooth. Although cold, hot, and electric pulp tests (EPTs) truly test only the function of nerves rather than pulpal inflammation or vitality, they are the most convenient tests available. Among these assessments, cold testing is the most accurate test[45] and a combination of cold with EPT increases accuracy.

Again, it is important in identifying the offending tooth to repeat tests that mimic what the patient reports subjectively. In other words, the best test is to repeat the stimulus that reportedly causes the pain. This is especially true for pulpal disease that has not extended to the periradicular tissues (e.g., irreversible pulpitis with asymptomatic apical periodontitis). It is often difficult for the patient to localize the pain to a particular tooth due to the paucity of proprioceptive neurons in the dental pulp. As in the previous example, applying cold should reproduce pain of basically the same type and magnitude as that related by the patient. If similar subjective symptoms are not reproduced, this situation may not be a true emergency; the patient may be "overreporting" (exaggerating the problem), or the pain may be referred from a source other than that perceived by the patient.

Periapical Tests

Periapical inflammation occurs as early as 1 to 3 days after pulp exposure.[46-48] Conceivably, periapical symptoms such as tenderness to biting, chewing, and pain to palpation or pressure can ensue shortly after.[49,50] These symptoms often occur despite absence of periapical bone resorption on radiographic examination, and therefore clinical tests that can localize pain to the offending tooth are essential tools for diagnosis of the periapical inflammatory status. These include (1) palpation over the apex; (2) digital pressure on, or wiggling of, teeth (preferred if the patient reports severe pain on mastication); (3) light percussion with the end of the mirror handle; and (4) selective biting on an object such as a cotton swab or Tooth Slooth.

Periodontal Examination

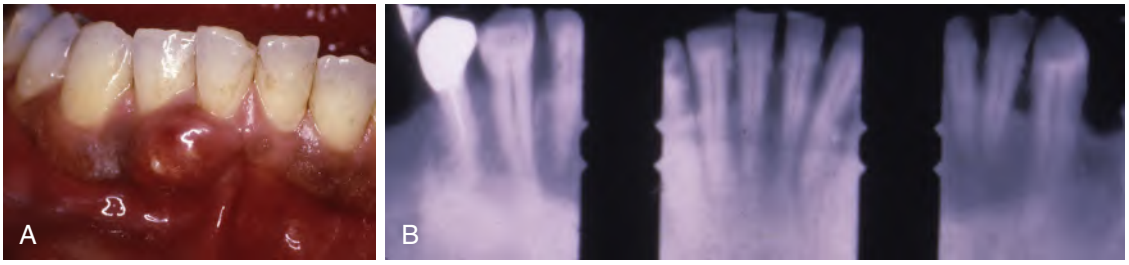
A periodontal examination is always necessary. Probing helps in differentiating endodontic from periodontal disease. For example, a periodontal abscess can simulate an acute apical abscess (Fig. 9.3); however, with a localized periodontal abscess, the pulp is usually vital (see Chapter 7). In contrast, an acute apical abscess is related to an unresponsive (necrotic) pulp. These abscesses occasionally communicate with the sulcus and have a deep probing defect. In addition to these tests, when the differential diagnosis is difficult, a test cavity may identify the pulp status and isolate the offending tooth. A narrow-walled, isolated probing defect may also indicate a coronal fracture that has extended beyond the level of sulcular attachment, or a vertical root fracture (see Chapter 8).

Radiographic Examination

Radiographic examination is a crucial tool in diagnosing the offending tooth. As stated before, patients often have difficulty localizing pulpal pain. Additionally, studies have demonstrated that approximately half the teeth with periapical pathosis are asymptomatic to periapical tests.[3] Vitality tests certainly aid in narrowing down the source of pain to one to two teeth. When vitality tests cannot confirm the true diagnosis due to presence of crowns, multiple teeth involvement, an anxious patient, or a patient with heightened responses to clinical tests, radiographic examination can provide several key clues that point to the offender. Recurrent caries, possible pulpal exposure, internal or external resorption, unusual appearance of the lamina dura, periapical pathosis, and traceable sinus tracts are some very important identifiers that help confirm diagnosis.

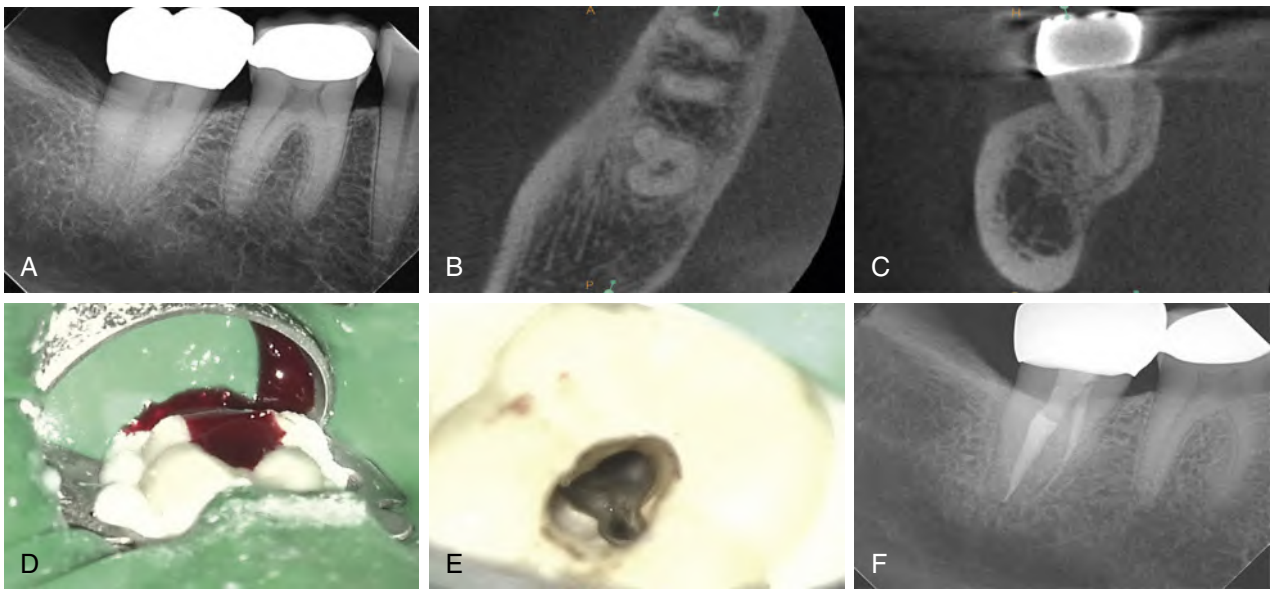
Both periapical and bitewing radiographs must be exposed during initial evaluation, as clinicians must never miss an opportunity to determine restorability of a tooth in addition to identifying the cause of the emergency. Additionally, three-dimensional

• **Fig. 9.2** Examples of **A,** mandibular swelling; **B**, trismus; **C** and **D**, eye shut. (Courtesy Dr. Daniel Perez, UTHSCSA, San Antonio, TX, USA.)



• **Fig. 9.3** Periodontal abscess with vital mandibular anterior teeth. **A,** Clinical photo obuccal swelling; and **B,** Periapical radiograph of periodontal bone loss. (Courtesy Dr. Brian Mealey, UTHSCSA, San Antonio.)

(3D) cone beam computed tomography (CBCT) renders diagnosis more predictable than two-dimensional (2D) imaging alone. Some of the most diagnostically challenging cases are ones with tooth fractures. A recent meta-analysis suggests that CBCT has greater accuracy in confirming tooth fractures in teeth with clinically suspected but periapical-radiography–undetected tooth fractures.[51] CBCT imaging therefore not only elevates a clinician's ability to accurately diagnose, but also to appropriately treat an emergency case. To this end, knowing the offending tooth's anatomy, the 3D relationship of critical anatomic structures such



• **Fig. 9.4** A difficult management case of symptomatic irreversible pulpitis with symptomatic apical periodontitis for a 60-year-old female patient with pain at 10/10 on visual analog scale (VAS). **A**, Preoperative radiograph of #31. **B**, Axial view of #31 showing mesiobuccal (MB) canal and C-shaped canal. **C,** Sagittal view of #31 showing anatomy of the C-shaped canal. **D**, Photograph of #31 with profuse hemorrhage upon access. **E**, MB and C-shaped canal anatomy at orifice level. **F**, Postoperative radiograph of completed obturation and access restoration at second visit. (Courtesy Dr. Anibal Diogenes, UTHSCSA San Antonio, TX, USA.)

as the inferior alveolar nerve (IAN), and the extent of resorptive defects, are some unique advantages of 3D imaging.[52]

Collectively, a systematic approach to diagnosis must be followed, and a combination of subjective and objective findings are carefully collected before attempting treatment. More specifics of diagnosis are included in Chapter 4.

Diagnostic Outcome

After carefully working through the sequence described in the previous sections, the offending tooth and the tissue (pulpal or periapical) that is the source of pain should have been identified and a pulpal and periradicular diagnosis recorded. For many reasons, all or none of these conclusions may be clear. This circumstance may not be a true emergency, or the problem may be beyond the capability of the general dentist, and the patient should be referred (Fig. 9.4). However, if the diagnosis is clear, treatment planning follows.

Treatment Planning

As previously discussed, inflammation and its consequences, i.e., increased tissue pressure and release of chemical mediators in the inflamed pulp or periapical tissues, are the major causes of painful dental emergencies.[53] Therefore reducing the irritant, reduction of pressure, or removal of the inflamed pulp or periapical tissue should be the immediate goal; this approach usually results in pain relief. Of the two, pressure release is the most effective.

Pretreatment Emergencies

These emergencies require a diagnosis and treatment sequencing. Each of these steps is important: (1) categorizing the problem, (2) taking a medical history, (3) identifying the source, (4) making the diagnosis, (5) planning the treatment, and (6) treating the patient.

Patient Management

Patient management is always the most critical factor. The frightened patient in pain must have confidence that his or her problem is being properly managed.

Profound Anesthesia

Obtaining profound anesthesia of inflamed painful tissues is a challenge. Adequate anesthesia, however, will instill confidence and cooperation and influence the patient's desire to save the offending tooth. Maxillary anesthesia is usually obtained by infiltration, or block injections in the buccal and palatal regions. With mandibular teeth, in addition to an IAN block with lidocaine, a long buccal injection for soft tissue anesthesia as well as infiltration of articaine on the facial may be necessary. Often (particularly with mandibular molars), although all "classic" signs of profound anesthesia are present (such as lip numbness), access into the dentin or pulp is painful, presumably due to sensitized pulpal nociceptors. It is therefore prudent to retest the tooth with a cold stimulus to assess pulpal anesthesia before initiating access into the pulp space. For those patients who still respond with pain, periodontal, intrapulpal, or intraosseous injection techniques are indicated.[54,55] These supplemental injections are often administered prophylactically, particularly with painful irreversible pulpitis.[54] Other conditions (for example, acute apical abscess) require other approaches. Chapter 8 contains details.

Management of Symptomatic Irreversible Pulpitis

Because pain is the result of inflammation, primarily in the coronal pulp, removal of the inflamed tissue will usually reduce the pain.

With or Without Symptomatic Apical Periodontitis

Teeth with caries, large restorations, cracked teeth, or trauma are some etiologies of symptomatic irreversible pulpitis**.** Complete cleaning and shaping of the root canals are the preferred treatments if time permits. Access to contemporary aids such as the electronic apex locator (EAL), surgical operating microscope (SOM), ultrasonic instruments, and CBCT facilitates complete instrumentation. However, during times when time- or patient-related factors prevent complete instrumentation, pulpotomy or a partial pulpectomy on the largest canals (palatal or distal root of molars) is performed. Both procedures have demonstrated greater than 90% success rate in reducing postoperative pain from moderate to severe to mild to no pain.[25,35,56-59] On the other hand, partial pulpectomy, but not pulpotomy of severely inflamed teeth, upregulates inflammatory mediators that promote further nerve sprouting, leading to greater postoperative pain, and has been strongly discouraged.[56,60] When there is a vital, inflamed pulp, other procedures such as trephination (artificial fistulation) by creating an opening through mucosa and bone are not useful and are contraindicated.[59,61]

An old but still popular idea is that chemical medicaments sealed in chambers help control or prevent additional pain; this idea is not true. A dry cotton pellet alone is as effective in relieving pain as a pellet moistened with camphorated monochlorophenol (CMCP), formocresol, Cresatin, eugenol, or saline.[58,62] Therefore after irrigation of the chamber or canals with sodium hypochlorite (NaOCl), a dry cotton pellet is placed, and the access is sealed temporarily. These cases can be completed in a single visit; however, results of a recent meta-analysis suggest that cases completed in one visit are more likely to have postoperative pain medication consumption.[63] Moreover, as stated before, preoperative pain is a strong predictor of postoperative pain. Additionally, calcium hydroxide [$Ca(OH)_2$] has been shown to significantly reduce inflammatory mediators such as cytokines and neuropeptides[64] commonly known to activate and sensitize nociceptors.[65,66] Therefore allowing further reduction in the inflammatory load with intracanal medicament placement may reduce the probability of postoperative pain associated with one-visit cases. Lastly, reducing the occlusion to eliminate contact has been shown to aid in relief of symptoms[67] but does not prevent symptoms.[68]

Postoperative Pharmacologic Management

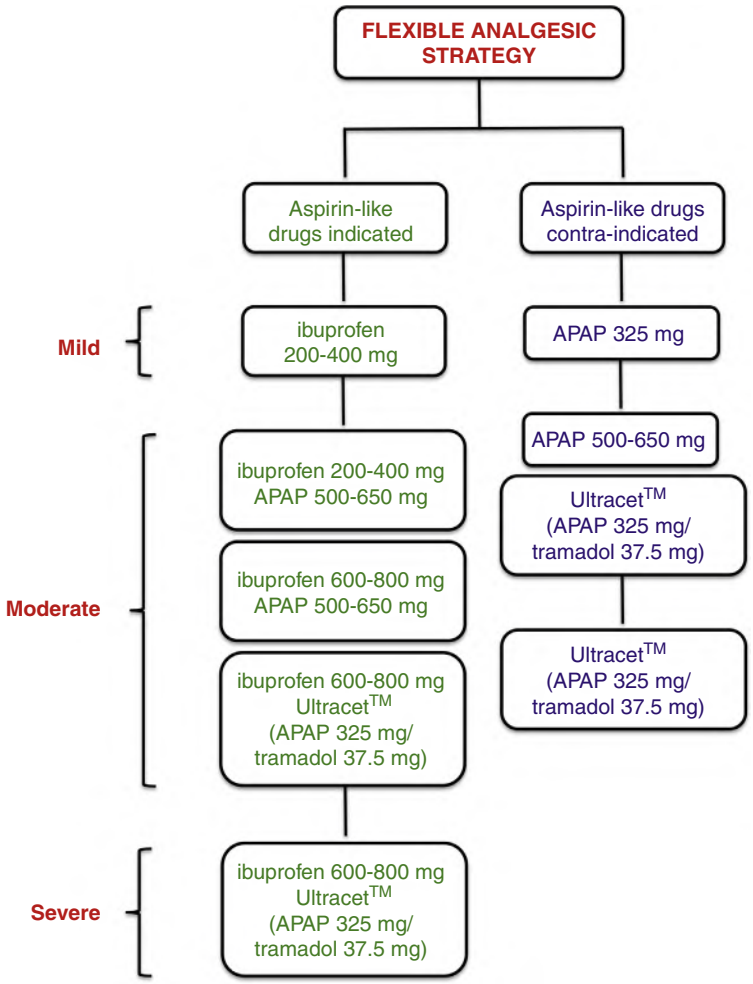
Pain Management: Recent systematic reviews and meta-analysis demonstrate that 600 mg ibuprofen or 600 mg ibuprofen with N-acetyl-p-aminophenol (APAP) 1000 mg is most effective in attenuating postoperative endodontic pain.[69,70] To prevent the build-up of the arachidonic acid metabolites that contribute a large portion of the inflammatory pain stimulus, the patient should take the first dose before the loss of local anesthesia and then take the nonsteroidal antiinflammatory drug (NSAID) "by the clock," rather than "as needed" (PRN). Administering ibuprofen to the patient while in the chair has been shown to reduce initial postoperative pain.[71] Moreover, a newer ibuprofen formulation, ibuprofen sodium dihydrate at a 512-mg dose has been shown to have a faster onset of action than ibuprofen acid producing a greater reduction in spontaneous pain and mechanical allodynia.[26] It is noteworthy that the U.S. Food and Drug Administration (FDA)-recommended maximum daily dose of APAP is 4 gm per day, owing to the increasing evidence of APAP-induced hepatoxicity.[72-74] However, it is well known that patients often underreport their use of over-the-counter (OTC) medications, such as NyQuil, Theraflu, and so on, that contain as high as 500 to 1000 mg APAP.[75] Therefore a lower dose of 325 to 500 mg APAP in combination with 600 mg ibuprofen will have a safer drug profile compared with 1000 mg APAP, and will avoid "therapeutic misadventures."

For patients with severe postoperative pain, other drug classes such as opioids maybe considered. However, the clinician must be fully aware of the recent opioid crisis that prevails; opioid overdose-related deaths have increased five-fold since 1999 (https://www.cdc.gov/drugoverdose/epidemic/index.html). An even more striking statistic shows that prescription opioid use by adolescents between the ages of 19 to 23 has a 33% increased risk of opioid misuse at a later stage in life.[76] Moreover, the Practitioners Engaged in Applied Research and Learning (PEARL) Network findings suggest that endodontists are second after oral surgeons in prescribing opioid-like drugs to patients.[77] Clinicians must significantly curtail contributing to the opioid epidemic. For patients with persistent, severe pain after an endodontic procedure, opioids with less abuse potential must be considered. Tramadol, a mu-opioid agonist, at varying doses has shown to have fewer opioid-like central effects compared with morphine[78] (RA, 2018 #924); however, it is not devoid of addiction and abuse potential and must be used judiciously for severe postoperative pain. Ultracet (325 mg APAP, 37.5 mg tramadol) is a combination drug, which is a viable option for patients with severe postoperative pain. See Fig. 9.5 for a flexible analgesic strategy.

Postoperative pain is attributed in large part to a process called *central sensitization.* Constant input from primary afferent nerve fibers can increase activity in the second-order neuron located in the trigeminal nucleus and cause signal amplification. Randomized clinical trials have elegantly demonstrated that administration of 0.5% bupivacaine with 1:200,000 epinephrine preoperatively significantly reduces postoperative pain at 6 and 12 hours compared with 2% lidocaine with 1:80,000 epinephrine after treatment of teeth with symptomatic irreversible pulpitis.[79,80]

Other pharmacologic drugs targeted at minimizing postendodontic pain include corticosteroids. Steroids inhibit phospholipase A2 enzyme upon membrane release of arachidonic acid, thereby inhibiting effects mediated by both lipoxygenases and cyclooxygenases. Several studies have evaluated the effects of corticosteroids in the prevention of postoperative endodontic pain and have demonstrated that drugs such as prednisolone and dexamethasone significantly attenuate the incidence of pain at 6, 12, and 24 hours posttreatment.[81,82] Collectively, several strategies are available to the clinician to reduce postoperative pain in patients with symptomatic irreversible pulpitis.

Use of Antibiotics: The irreversibly inflamed pulp is still vital and immunocompetent, with the ability to resist bacterial infection. Antibiotics, therefore, are definitely not indicated in cases of irreversible pulpitis without swelling.[83] Moreover, antibiotics are not analgesics and have no role in inhibition of nociceptors.[5,35] Unfortunately, old habits die hard, and there are still practitioners who prescribe antibiotics inappropriately, as in the case of irreversible pulpitis.[84] Antibiotic administration has the potential to result in at least two very serious sequelae: adverse reaction to the antibiotic, as well as increasing antibiotic-resistant microbial strains. The former is a local problem in which injudicious use of an antibiotic could lead to a life-threatening situation for an allergic patient. The latter is a global problem. Resistant microbial strains are emerging faster than pharmaceutical companies



• **Fig. 9.5** Simplified analgesic strategy to guide drug selection based on patient history and level of present or anticipated posttreatment pain.

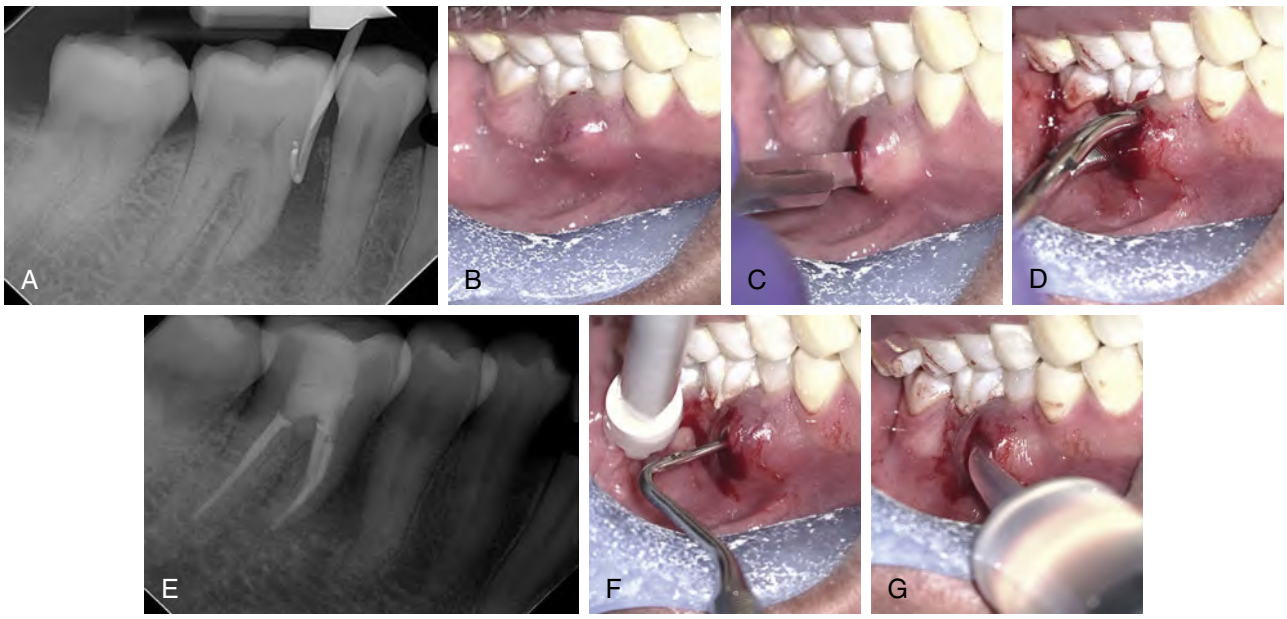
are developing new antibiotics; therefore it is critical that health care providers practice good stewardship with existing antibiotics.[85] Otherwise, in the near future there may not be a pharmacologic option in treating severe odontogenic or systemic infections.

Management of Pulp Necrosis with Apical Pathosis

The pain is related to periradicular inflammation, which results from potent irritants in the necrotic tissue in the pulp space. Treatment now is biphasic: (1) remove or reduce the pulp irritants and (2) relieve the apical fluid pressure (when possible). Therefore with pulp necrosis and pain from periradicular tissues there may be (1) symptomatic apical periodontitis without swelling, (2) acute apical abscess with localized intraoral swelling, or (3) acute apical abscess with diffuse extraoral swelling. Each circumstance is managed differently.

Pulp Necrosis/Symptomatic Apical Periodontitis Without Swelling

The microbiota is much more developed and stable compared with severely inflamed pulps. These teeth harbor not just planktonic bacteria but also well-established biofilms that release their toxins and byproducts into the canal system as well as into the periapical tissues. An inflammatory response consisting of activation of innate and adaptive immune cells and release of inflammatory mediators such as interleukin-1 (IL-1), prostaglandin E2 (PGE2), and tumor necrosis factor-α (TNF-α)[53,86] activate osteoclasts, leading to bone resorption. Some of these lesions expand and form an abscess that is confined to bone. These abscesses are often painful, primarily because of fluid pressure in a noncompliant environment. The two-fold aim is to reduce the canal irritants and to try to encourage some drainage through the tooth. Complete canal débridement, after determining the correct working length, is the treatment of choice. If time is limited, partial débridement at the estimated working length is performed with light instrumentation with a passive step-back or crown-down technique to reduce or remove irritating debris. Canals are not enlarged without knowledge of the working length. During cleaning, canals are flooded and flushed with copious amounts of full-strength NaOCl (6% or 8%). Finally, canals are irrigated with 17% ethylene diaminetetraacetic acid (17% ethylenediaminetetraacetic acid [EDTA]) followed by NaOCl, dried with paper points, filled with $Ca(OH)_2$ paste (if the preparation is large enough), and sealed with a dry cotton pellet and a temporary restoration. The access must never be left open for drainage.



• **Fig. 9.6** Management of pulp necrosis with acute apical abscess with localized intraoral swelling for a 60-year-old female patient with pain at 5/5 on visual analog scale (VAS). **A,** Preoperative radiograph of #30 with sulcular sinus tract. **B**, Preoperative photograph of intraoral swelling. **C**, Photograph of incision. **D**, Blunt dissection. **E**, Postoperative radiograph of completed obturation and access restoration at second visit. **F**, Curettage. **G,** Sterile saline irrigation. (Courtesy Dr. Saeed Bayat, UTHSCSA, San Antonio, TX, USA.)

Postoperative Pharmacologic Management

Pain Management: Pain-management protocols follow guidelines similar to those stated previously. Antibiotics are not indicated due to lack of systemic involvement.[87] The patient is informed that there will still be some pain (the inflamed, sensitive periapical tissues are still present), but that the pain usually subsides during the next 2 or 3 days, as the inflammation decreases.

Management of Infections: These patients seldom have elevated temperatures or other systemic signs.[88] Therefore in acute apical abscess with localized swelling, the use of systemic antibiotics is not necessary, having been shown to be of no benefit.[87,89,90]

Pulp Necrosis/Acute Apical Abscess with Localized Intraoral Swelling

In these situations, an abscess has invaded regional soft tissues, and, at times, there is purulent exudate in the canal. Radiographic findings range from no periapical change (seldom) to a large radiolucency. Again, treatment is biphasic. *First and most important* is débridement (complete cleaning and shaping if time permits) of the canal or canals. *Second in urgency* is drainage. Localized swelling (whether fluctuant or nonfluctuant) should be incised (Fig. 9.6). Drainage accomplishes three things: (1) relief of pressure and pain, (2) removal of potent irritants (purulence and inflammatory mediators), and (3) prevention of the spread of infection to fascial spaces.

In teeth that drain readily after opening, instrumentation should be confined to the root canal system (Fig. 9.7). In patients with a periapical abscess but no drainage through the canal, penetration of the apical foramen with small files (up to #25) may initiate drainage and release of pressure. This release often does not occur because the abscess cavity does not communicate directly with the apical foramen. Copious irrigation with NaOCl reduces amounts of necrotic tissue and bacteria. The canals are then dried with paper points and filled with $Ca(OH)_2$ paste. Occasionally, purulence will continue to fill the canal during the preparation (the so-called "weeping" canal). If this occurs, the patient should sit for some time. Usually, the flow will cease, and the access may be closed. After placement of a dry cotton pellet, the access is sealed temporarily. These teeth should not be left open to drain. A canal exposed to the oral cavity is a potential home for introduced bacteria, food debris, and even viruses and leads to increased exacerbation from an activated immune response (Video 9.1).[91]

After débridement, an incision and drainage (I&D) procedure must be performed in cases with more than one abscess (see Fig. 9.7): one that communicates with the apex when another separate abscess is found in the vestibule. Because they do not communicate, drainage must occur through both the tooth and a mucosal incision. Steps of an I&D procedure typically involve a vertical incision followed by blunt dissection of the incised area, thorough curettage and copious irrigation with sterile saline and/or 0.12% chlorhexidine (see Fig. 9.7). A drain maybe placed if cessation of drainage does not occur during the appointment. Postoperative pain may be associated with the I&D procedure; however, this pain typically resolves within 2 to 3 days.[92]

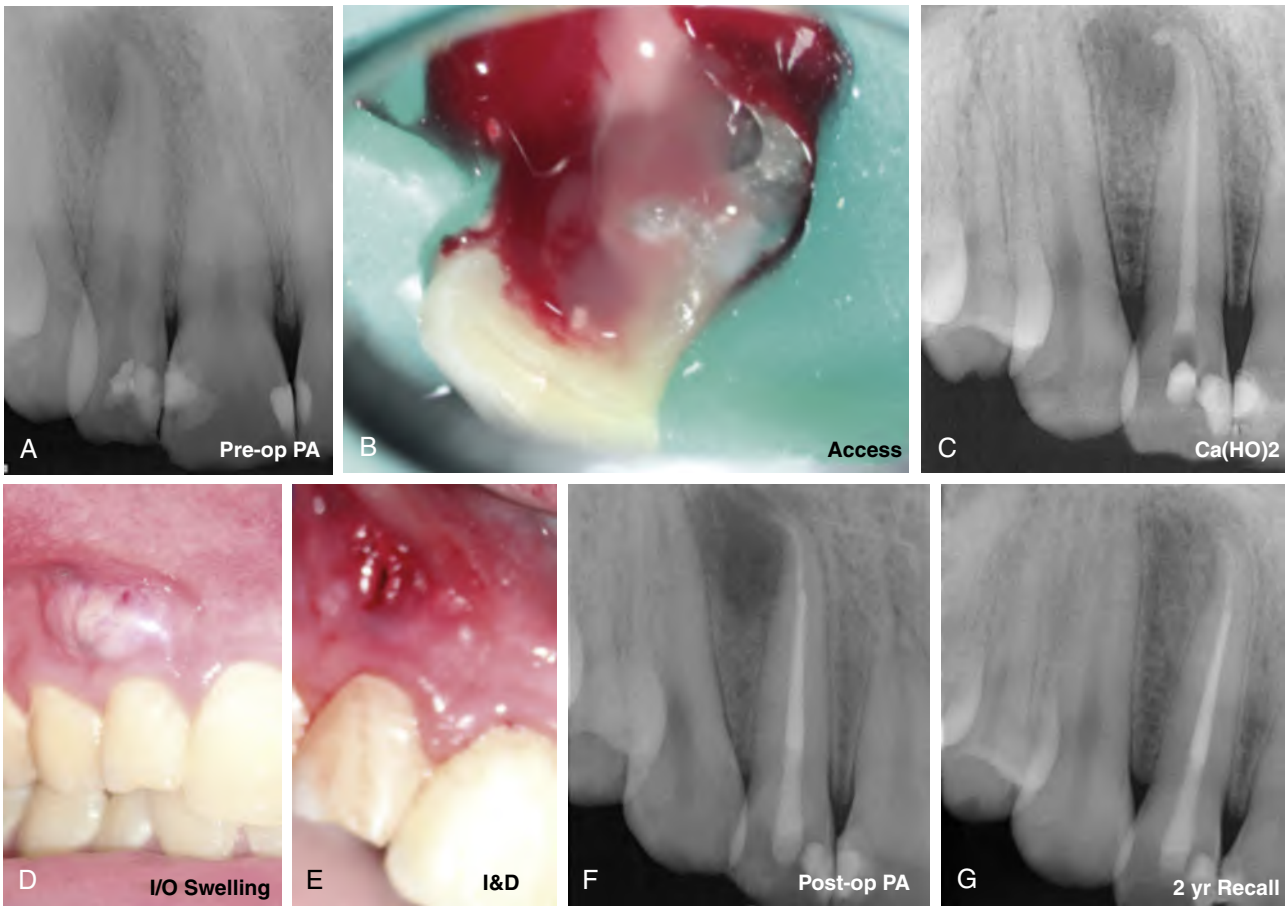
Postoperative Pharmacologic Management

Pain Management: An analgesic regimen appropriate for mild to moderate pain should be followed as described previously; relief of pressure is most important in pain control for these patients.

Management of Infections: These patients seldom experience an elevated temperature or other systemic signs. Therefore in acute apical abscess with localized swelling, the use of systemic antibiotics is not necessary, having been shown to be of no benefit.[88]

Pulp Necrosis with Diffuse Swelling

Endodontic abscesses that perforate through the cortical bone can spread to nearby fascial spaces; these occurrences are also referred to as cellulitis. The muscle attachment and arrangement



• **Fig. 9.7** Management of pulp necrosis with acute apical abscess with localized intraoral swelling for a 60-year-old female patient with pain at 5/5 on visual analog scale (VAS). **A,** Preoperative radiograph of #7. **B**, Drainage through tooth upon access. **C**, Radiograph of #7 with calcium hydroxide [$Ca(OH)_2$]. **D**, Preoperative photograph of intraoral swelling. **E**, Photograph post–incision and drainage. **F**, Postoperative radiograph of completed obturation and access restoration at second visit. **G**, Postoperative radiograph at 2 years post treatment. (Courtesy Dr. Obadah Austah, UTHSCSA, San Antonio, TX, USA.)

of fasciae will determine the pathway of spread and the possible site of localization. These rapidly progressive and spreading swellings are not localized and may have dissected into the fascial spaces (Fig. 9.2A-D). Limited opening, pain and difficulty in swallowing, and occasionally bilateral spread may also occur. Fortunately, these serious infections seldom occur. There may be elevated temperature or other systemic signs indicating a potentially serious infection. These patients should be referred to an oral surgeon for extraoral drainage, intravenous antibiotics, and pain management. Often, the patient is hospitalized for this aggressive treatment.

Once stable, the patient may return for initiation of canal débridement. Most important is removal of the irritant by canal débridement (cleaning and shaping is completed, if possible) or by extraction. The apical foramen may be gently penetrated with a file to permit any possible flow of exudate, although drainage often does not occur. After placing $Ca(OH)_2$ paste and a dry pellet, the access is closed with a temporary restoration.

Postoperative Pharmacologic Management

Pain Management: An analgesic regimen appropriate for moderate-severe pain should be followed as described previously; resolution of extraoral swelling will provide for maximum pain relief for these patients.

Management of Infections: Systemic antibiotics are indicated for diffuse, rapidly spreading swelling (cellulitis).[93] Culturing and molecular identification techniques used to study odontogenic abscesses reveal a polymicrobial infection, with anaerobic species predominating.[94-96] Antibiotic sensitivity testing confirms that the majority of isolates are susceptible to penicillin VK (Pen VK).[95] However, its bactericidal efficacy is significantly lower than that of other drugs such as amoxicillin due to inferior penetration and absorption properties. Moreover, it is associated with gastrointestinal side effects due to depletion of gut commensal flora.[93] Amoxicillin with clavulanic acid has shown a 100% efficacy against pathogens found in endodontic abscesses followed by amoxicillin with metronidazole at 99%. Amoxicillin achieves higher serum levels, maintained over a longer period of time than Pen VK. A loading dose of amoxicillin 1-g load followed by 500 mg every 6 hours for 2 to 3 days must be prescribed.

For patients with penicillin allergy, clindamycin is an alternative that demonstrates 96% antibacterial efficacy in vitro. It has superior oral absorption with excellent distribution in bone. It has both bacteriostatic and bactericidal effects and is effective against gram-positive aerobes and gram-positive and negative anaerobes, thereby satisfying a broad spectrum of microbes. A loading dose of 300 to 600 mg followed by 150 to 300 mg every 6 hours for 2

to 3 days must be prescribed. Note: Shorter prescription duration has been recommended based on emerging evidence and recommendations. Recent evidence suggests that shorter prescription duration of 2 to 3 days until resolution of symptoms is equally efficacious as longer prescription duration of 7 to 10 days. Moreover, the longer the commensal microbes in our bodies are exposed to antibiotics, the greater their ability to select for resistance. Therefore our threat increases more from the commensal bacteria than from pathogenic bacteria.[93,97-100]

Because of the reduction in normal gut flora, patients on oral antibiotics occasionally develop signs of colitis due to the overgrowth of *Clostridium difficile* and a potentially fatal condition of pseudomembranous colitis. This problem can occur with nearly all classes of antibiotics, especially clindamycin, which has an eight-fold higher risk of *C. difficile* infection compared with penicillin; the patient should be warned to watch for the development of watery diarrhea, cramping abdominal pain, and low-grade fever. Patients taking oral contraceptives for whom antibiotics are prescribed should also be warned to use alternative methods during, and for 1 week after, the course of antibiotic therapy. There is some clinical, albeit empirical, evidence that probiotics may minimize the superinfections.

Speed of recovery (whether the swelling is localized or diffuse) depends primarily on canal débridement and drainage. Because edema (fluid) has spread through the tissues, diffuse swelling decreases slowly over several days.

Postoperative Instructions

With all emergencies, patients must be informed of their responsibilities and of what to expect. Pain and swelling will take time to resolve. Proper nutrition and adequate intake of fluids are important, and medications must be taken as prescribed. The problem may recur or worsen (flare-up), requiring another emergency visit.[5] Communication is very important; calling the patient the day after the appointment reduces pain perception and analgesic needs[101] and allows for closer monitoring of progress.

Interappointment Emergencies

The interappointment flare-up is a true emergency that occurs after an endodontic appointment and is so severe that an unscheduled patient visit and treatment are required. Despite judicious and careful treatment procedures, complications such as pain, swelling, or both may occur. Regional temporary paresthesia has even been reported.[57] As with emergencies occurring before root canal therapy, these flare-ups are undesirable and disruptive events and must be resolved quickly. Occasionally flare-ups are unexpected, although they can often be better predicted according to certain patient presenting factors.

Incidence

The reported incidence of flare-ups in endodontics varies widely due to variations in study parameters. Properly controlled prospective studies show an incidence of approximately 3%.[5,102,103] Even though the occurrence is low, interappointment flare-ups represent such a stressful situation to the patient (most postoperative discomfort is in the mild to moderate range), that it behooves the prudent clinician to consider the likely related factors and to try to prepare the patient for the possibility of such an event. It is especially distressing for the patient who had minimal preoperative discomfort to experience pain and/or swelling after treatment.

Causative Factors

Assessing causality is difficult when reviewing the literature on flare-ups; however, certain risk factors have emerged. These factors generally can be categorized as related to the patient (including pulpal or periapical diagnosis). Treatment procedures are unrelated to flare-ups, although this is a popular belief. Patient factors include gender (more flare-ups are reported to occur in females, although this circumstance may represent a greater tendency for females to seek medical care for painful symptoms)[104] and preoperative diagnosis. Flare-ups are uncommon in teeth with vital pulps.[5,105] More often, flare-ups occur in teeth with necrotic pulps, and especially in those with a periapical diagnosis of symptomatic periapical periodontitis or acute apical abscess.[5,102,105,106] The presence of a periapical radiolucency has also been shown to be a risk factor.[5,102,103,107] Clearly, the patient who experiences a flare-up is more likely to have presented with significant preoperative pain and/or swelling.

Treatment factors have also been examined for the potential to cause flare-ups. Although it would seem intuitive that flare-ups would be related to certain procedures, such as overinstrumentation, pushing debris beyond the apex, or completing the endodontic therapy in one visit, no definitive treatment risk factors have been identified.

Prevention

Procedures

Use of long-acting anesthetic solutions, complete cleaning and shaping of the root canal system (possibly), analgesics, and psychological preparation of patients (particularly those with preoperative pain) will decrease interappointment symptoms in the mild to moderate levels.[35] There are, however, no demonstrated treatment or therapeutic measures that will reduce the number of interappointment flare-ups. In other words, no particular relationship has been shown between flare-ups and specific treatment procedures.

Verbal Instructions

Most important is the preparation of patients for what to expect after the appointment. They should be told that discomfort ("soreness") is likely; the discomfort should subside within a day or two. Increases in pain, noticeable swelling, or other adverse signs necessitate a call and sometimes a visit. This explanation reduces the number of calls from unnecessarily concerned patients.

Therapeutic Prophylaxis

A popular preventive approach has been the prescribing of antibiotics to minimize postoperative symptoms. This practice has been demonstrated to be not useful and needlessly exposes the patient to expensive, potentially dangerous drugs, as described previously.[108-110] In contrast, certain NSAIDs have been shown to reduce postendodontic treatment pain.[71,111] For patients at risk for a flare-up, 400-600 mg of ibuprofen should be given while the patient is in the chair, and then taken by the clock for the first 24 to 48 hours postoperatively. Although this medication will reduce postoperative symptoms, it is uncertain whether it will reduce the incidence of flare-ups.

Diagnosis

The same basic procedure is followed as outlined earlier in this chapter for pretreatment emergencies, although with modifications. The problem has already been diagnosed initially; the clinician has an advantage. However, a step-by-step approach to diagnosing the

existing condition reduces confusion and error; most important, it calms a patient who has been frightened by the episode of pain or swelling. After the underlying complications are identified, treatment is initiated.

Treatment of Flare-Ups

Reassurance (the "Big R") is the most important aspect of treatment. The patient is generally frightened and upset and may even assume that extraction is necessary. The explanation is that the flare-up is neither unusual nor irrevocable and will be managed. Next in importance are restoring the patient's comfort and breaking the pain cycle. For extended anesthesia and analgesia, administration of bupivacaine hydrochloride is recommended.[112]

Interappointment emergencies are divided into patients with an initial diagnosis of a vital or a necrotic pulp, and patients with or without swelling.

Previously Vital Pulps with Complete Débridement

If complete removal of the inflamed vital pulp tissues was accomplished at the first visit, this situation is unlikely to be a true flare-up, and patient reassurance and the prescription of a mild to moderate analgesic (9.5) often will suffice. Generally, nothing is to be gained by opening these teeth; the pain will usually regress spontaneously, but it is important to check that the temporary restoration is not in traumatic occlusion. Placing corticosteroids in the canal or administering an intraoral or intramuscular injection of these medications after cleaning and shaping reduces inflammation and somewhat lowers the level of moderate pain.[113-115] Flare-ups, however, have not been shown to be prevented by steroids, whether administered intracanal or systemically.[116]

Previously Vital Pulps with Incomplete Débridement

In previously vital pulps with incomplete débridement, it is likely that tissue remnants have become inflamed and have become a major irritant. The working length should be rechecked, and the canal(s) should be carefully cleaned with copious irrigation with NaOCl. A dry cotton pellet is then placed, followed by a temporary restoration, and a mild to moderate analgesic is prescribed (see Fig. 9.5). Occasionally, a previously vital pulp (with or without complete débridement) will develop into an acute apical abscess. This problem will occur sometime after the appointment and indicates that pulpal remnants have become necrotic and are invaded by bacteria.

Previously Necrotic Pulps with No Swelling

Occasionally teeth with previously necrotic pulps but no swelling develop an acute apical abscess (flare-up) after the appointment (1). The abscess is confined to bone and can be very painful. The tooth is opened, and the canal is gently recleaned and irrigated with NaOCl. Drainage should be established if possible (see Fig. 9.7). If there is active drainage from the tooth after opening, the canal should be recleaned (or débridement completed) and irrigated with NaOCl. The rubber dam is left in place after the tooth is opened; the patient is allowed to rest pain-free for at least 30 minutes or until drainage stops. Then, the canals are dried, $Ca(OH)_2$ paste is placed, and the access is sealed. The tooth should not be left open! If there is no drainage, the tooth should also be lightly instrumented and gently irrigated, medicated with $Ca(OH)_2$ paste, and then closed. The symptoms usually subside but do so more slowly than if drainage were present. Again, patient education and reassurance are critical. A long-acting anesthetic and an analgesic regimen for moderate to severe pain are helpful; antibiotics are not indicated.[87,89]

Previously Necrotic Pulps with Swelling

These cases are best managed with I&D (see Fig. 9.6). In addition, it is most important that the canals have been débrided. If not, they should be opened and débrided, medicated with $Ca(OH)_2$ paste, and sealed. Then I&D with placement of a drain (if there is continuous drainage) are completed. Occasionally, but rarely, a flare-up or a presenting acute apical abscess may be serious or even life-threatening (Fig. 9.8). These situations may require hospitalization and aggressive therapy with the cooperation of an oral surgeon.

Follow-Up Care

With flare-ups, the patient should be contacted daily until the symptoms abate. Communication may be made by telephone; patients with more serious problems or those that are not resolving (many may not and require additional measures) should return to the dentist for treatment as described previously, depending on findings. When symptoms recur or cannot be controlled, these patients should be considered for referral. Ultimate treatment by a specialist may include extra measures, such as apical surgery, or even hospitalization.

Postobturation Emergencies

True emergencies (flare-ups) postobturation are infrequent, although pain at the mild level is common. Therefore active intervention is seldom necessary; usually symptoms will resolve spontaneously.

Causative Factors

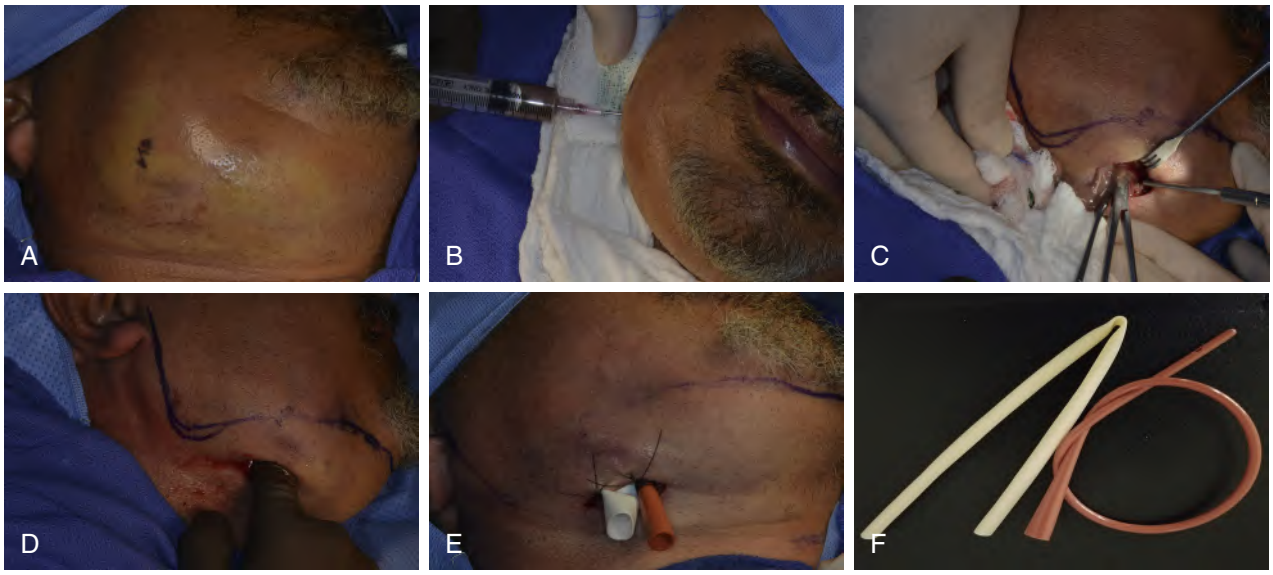
Little is known about the etiologic factors involved in postoperative pain after obturation. Reports of the incidence of postobturation pain vary; however, most reports show that the pain tends to occur in the first 24 hours.[35,117] A correlation has been found between the level of obturation and pain incidence, with overextension associated with the highest incidence of discomfort.[35,117] Postobturation pain also relates to preobturation pain; levels of pain reported after obturation tend to correlate to levels of pain before the appointment.[117,118]

Treatment

Information about possible discomfort for the first few days (especially in patients who had higher levels of preoperative pain), reassurance about the availability of emergency services, and administration of analgesics for mild pain (see Fig. 9.5) significantly control the patient's anxiety and prevent overreaction. This support, in turn, decreases the incidence of postobturation frantic telephone calls or "emergency" visits. Some patients, however, do develop serious complications and require follow-up treatment.

Retreatment is indicated when prior treatment obviously has been inadequate. Apical surgery is often required when an acute apical abscess develops, and there is uncorrectable, inadequate root canal treatment. If root canal treatment was acceptable, I&D of swelling after obturation (an occasional occurrence) should be performed; usually the swelling resolves without further treatment. At times, the patient reports severe pain, but there is no evidence of acute apical abscess, and the root canal treatment has been well done. These patients are treated with reassurance and appropriate analgesics (see Fig. 9.5); again, the symptoms usually subside spontaneously.

Patients with postobturation emergencies that do not respond to therapy should be referred to an endodontist for other treatment modalities, such as surgery.



• **Fig. 9.8** Management of pulp necrosis with acute apical abscess with diffused extraoral swelling for a 43-year-old male patient hospitalized for aggressive therapy with nasal intubation. Pain at 5/5 on visual analog scale (VAS). **A**, Preoperative photograph of extraoral swelling. **B**, Photograph of syringe aspiration. **C** and **D**, Drainage. **E**, Postoperative photograph of two drains placed and sutured. **F**, Types of extraoral drains: Covidien Dover Rob-Nel Urethral Catheter and Penrose drain. (Courtesy Dr. Daniel Perez, UTH-SCSA, San Antonio, TX, USA.)

Study Questions

1. What effect does anxiety have on pain threshold levels?
   a. Increases
   b. Decreases
   c. No effect
2. The most important aspect of diagnosis is
   a. Subjective examination
   b. Pulp testing with cold
   c. Pulp testing with electrical pulp test device
   d. Percussion test
   e. Radiographic examination
3. Anesthesia of mandibular teeth is enhanced by
   a. Infiltration of articaine on the facial
   b. Injecting buffers in the region of the mandibular foramen
   c. Increasing the concentration of epinephrine
   d. Repeating the injection while itipositioning the patient upright
   e. Injecting on the lingual to block the mylohyoid nerve
4. Most important in managing symptomatic irreversible pulpitis is:
   a. Selecting the appropriate intracanal medicament
   b. Removing the inflamed tissue
   c. Reducing the occlusion
   d. Prescribing opiate/NSAID combinations
   e. Prescribing the appropriate antibiotic
5. Antibiotic administration is recommended for:
   a. Prophylaxis to minimize flare-ups
   b. Severe pain with symptomatic irreversible pulpitis
   c. Localized swelling with a large radiolucency
   d. Retreatment of a failed root canal treatment
   e. Cellulitis
   f. None of the above
6. An analgesic choice for severe pain is
   a. Ibuprofen 400 to 600 mg
   b. Combination: Ibuprofen 200 to 400 mg and APAP 500 mg
   c. Combination: Ibuprofen 200 to 400 mg and APAP 325 mg/tramadol 37.5 mg
   d. Combination: Ibuprofen 600 to 800 mg and APAP/tramadol 37.5 mg
   e. Combination: APAP 325 mg and tramadol 37.5 mg
7. The incidence of flare-ups is
   a. Approximately 3%
   b. Approximately 15%
   c. Decreased with prophylactic antibiotics
   d. Higher in inflamed versus necrotic pulps
   e. Higher in preoperative asymptomatic versus symptomatic conditions
8. Most important for resolution of pulp necrosis/acute apical abscess is
   a. Administering appropriate medications (antibiotics and analgesics)
   b. Obtaining drainage through the tooth
   c. Removing irritants from the canal space
   d. Modifying the immune response with regional steroid injections
9. In an emergency situation, the most important objective test is
   a. Cold
   b. Heat
   c. Test cavity
   d. Percussion
   e. The test that reproduces the painful stimulus
10. The best way to differentiate an endodontic from a periodontal abscess swelling is
    a. Endodontic is over the apex, periodontal is more cervical
    b. Endodontic swelling is more painful
    c. Periodontal swelling is associated with probing defects
    d. Periodontal swelling is associated with cervical bone loss
    e. Determining pulp status; endodontic swelling results from pulp necrosis

• BOX 9.1

Diagnosis Sequence

1. Obtain pertinent information about the patient's medical and dental histories.
2. Ask pointed subjective questions about the patient's pain: history, location, severity, duration, character, and eliciting stimuli.
3. Perform an extraoral examination.
4. Perform an intraoral examination.
5. Perform pulp testing as appropriate.
6. Use palpation and percussion sensitivity tests to determine periapical status.
7. Interpret appropriate radiographs.
8. Identify the offending tooth and tissue (pulp or periapex).
9. Establish a pulpal and periapical diagnosis.
10. Design a treatment plan (both emergency and definitive).

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