From Orifice
To Apex

Reading club

No:00000001
Fanta Dental

ART AND SCIENCE DIAGNOSIS
EXAMINATION AND TESTING
CLINICAL CLASSIFICATION OF PULPAL AND PERIAPICAL DISEASES

CONTENT

1.Cohens

Diagnosis

LOUIS H. BERMAN | ILAN ROTSTEIN

CHAPTER OUTLINE

ART AND SCIENCE OF DIAGNOSIS

Diagnosis is the art and science of detecting and distinguishing deviations from health and the cause and nature thereof.[6] The purpose of a diagnosis is to determine what problem the patient is having and why the patient is having that problem. Ultimately, this will directly relate to what treatment, if any, will be necessary. No appropriate treatment recommendation can be made until all of the *whys* are answered. Therefore, careful data gathering as well as a planned, methodical, and systematic approach to this investigatory process is crucial.

Gathering objective data and obtaining subjective findings are not enough to formulate an accurate clinical diagnosis. The data must be interpreted and processed to determine what information is significant, and what information might be questionable. The facts need to be collected with an active dialogue between the clinician and the patient, with the clinician asking the right questions and carefully interpreting the answers. In essence, the process of determining the existence of an oral pathosis is the culmination of the art and science of making an accurate diagnosis.

The process of making a diagnosis can be divided into five stages:

1. The patient tells the clinician the reasons for seeking advice.
2. The clinician questions the patient about the symptoms and history that led to the visit.
3. The clinician performs objective clinical tests.
4. The clinician correlates the objective findings with the subjective details and creates a tentative list of differential diagnoses.
5. The clinician formulates a definitive diagnosis.

This information is accumulated by means of an organized and systematic approach that requires considerable clinical judgment. The clinician must be able to approach the problem by crafting what questions to ask the patient and how to ask these pertinent questions. Careful listening is paramount to begin painting the picture that details the patient's complaint. These subjective findings combined with results of diagnostic tests provide the critical information needed to establish the diagnosis.

Neither the art nor the science is effective alone. Establishing a differential diagnosis in endodontics requires a unique blend of knowledge, skills, and ability to interpret and interact with a patient in real time. Questioning, listening, testing, interpreting, and finally answering the ultimate question of *why* will lead to an accurate diagnosis and in turn result in a more successful treatment plan.

Chief Complaint

On arrival for a dental consultation, the patient should complete a thorough registration that includes information pertaining to medical and dental history (Figs. 1-1 and 1-2). This should be signed and dated by the patient, as well as initialed by the clinician as verification that all of the submitted information has been reviewed (see Chapter 29 for more information).

The reasons patients give for consulting with a clinician are often as important as the diagnostic tests performed. Their remarks serve as initial important clues that will help the clinician to formulate a correct diagnosis. Without these direct and unbiased comments, objective findings may lead to an incorrect diagnosis. The clinician may find a dental pathosis, but it may not contribute to the pathologic condition that mediates the patient's chief complaint. Investigating these complaints may indicate that the patient's concerns are related to a medical condition or to recent dental treatment. Certain patients may

TELL US ABOUT YOUR SYMPTOMS

LAST NAME_____ **FIRST NAME** _____

1. Are you experiencing any pain at this time? If not, please go to question 6. Yes _____ No _____

2. If yes, can you locate the tooth that is causing the pain? Yes _____ No _____

3. When did you first notice the symptoms? _____

4. Did your symptoms occur suddenly or gradually? _____

5. Please check the frequency and quality of the discomfort, and the number that most closely reflects the intensity of your pain:

LEVEL OF INTENSITY (On a scale of 1 to 10) 1 = Mild 10 = Severe	FREQUENCY	QUALITY
1 2 3 4 5 6 7 8 9 10	_____ Constant	_____ Sharp
	_____ Intermittent	_____ Dull
	_____ Momentary	_____ Throbbing
	_____ Occasional	

Is there anything you can do to relieve the pain? Yes _____ No _____

If yes, what? _____

Is there anything you can do to cause the pain to increase? Yes _____ No _____

If yes, what? _____

When eating or drinking, is your tooth sensitive to: Heat _____ Cold _____ Sweets _____

Does your tooth hurt when you bite down or chew? Yes _____ No _____

Does it hurt if you press the gum tissue around this tooth? Yes _____ No _____

Does a change in posture (lying down or bending over) cause your tooth to hurt? Yes _____ No _____

6. Do you grind or clench your teeth? Yes _____ No _____

7. If yes, do you wear a night guard? Yes _____ No _____

8. Has a restoration (filling or crown) been placed on this tooth recently? Yes _____ No _____

9. Prior to this appointment, has root canal therapy been initiated on this tooth? Yes _____ No _____

10. Is there anything else we should know about your teeth, gums, or sinuses that would assist us in our diagnosis? _____

Signed: Patient or Parent_____ **Date** _____

FIG. 1-1 Dental history form that also allows the patient to record pain experience in an organized and descrip- tive manner.

TELL US ABOUT YOUR HEALTH

LAST NAME _____ **FIRST NAME** _____

How would you rate your health? **Please circle one.** **Excellent Good Fair Poor**

When did you have your last physical exam? _____

If you are under the care of a physician, please give reason(s) for treatment.

_____

Physician's Name, Address, and Telephone Number:

Name _____ **Address** _ **City** _____

State _____ **Zip** _____ **Telephone** _____

Have you ever had any kind of surgery? **Yes** _____ **No** _____

If yes, what kind? _____ **Date** _____

_____ **Date** _____

Have you ever had any trouble with prolonged bleeding after surgery? **Yes**

Do you wear a pacemaker or any other kind of prosthetic device? **No**

Are you taking any kind of medication or drugs at this time?

If yes, please give name(s) of the medicine(s) and reason(s) for taking them:

Name _____ **Reason** _____

_____ _____

Have you ever had an unusual reaction to an anesthetic or drug (like penicillin)? **Yes** _____ **No** _____

If yes, please explain: _____

Please circle any past or present illness you have had:

Alcoholism	Blood pressure	Epilepsy Glaucoma	Hepatitis Herpes	Kidney or liver	Rheumatic fever
Allergies	Cancer			Mental	Sinusitis
Anemia	Diabetes	Head/Neck injuries	Immunodeficiency	Migraine	Ulcers
Asthma	Drug dependency	Heart disease	Infectious diseases	Respiratory	Venereal disease

Are you allergic to Latex or any other substances or materials? **Yes** _____ **No** _____

If so, please explain _____

If female, are you pregnant? **Yes** _____ **No** _____

Is there any other information that should be known about your health? _____

_____

Signed: Patient or Parent _____ **Date:** _____

FIG. 1-2 Succinct, comprehensive medical history form designed to provide insight into systemic conditions that could produce or affect the patient's symptoms, mandate alterations in treatment modality, or change the treatment plan.

even receive initial emergency treatment for pulpal or periapical symptoms in a general hospital.[93] On occasion, the chief complaint is simply that another clinician correctly or incorrectly advised the patient that he or she had a dental problem, with the patient not necessarily having any symptoms or any objective pathosis. Therefore, the clinician must pay close attention to the actual expressed complaint, determine the chronology of events that led to this complaint, and question the patient about other pertinent issues, including medical and dental history. For future reference and in order to ascertain a correct diagnosis, the patient's chief complaint should be properly documented, using *the patient's own words.*

Medical History

The clinician is responsible for taking a proper medical history from every patient who presents for treatment. Numerous examples of medical history forms are available from a variety of sources, or clinicians may choose to customize their own forms. After the form is completed by the patient, or by the parent or guardian in the case of a minor, the clinician should review the responses with the patient, parent, or guardian and then initial the medical history form to indicate that this review has been done. The patient "of record" should be questioned at each treatment visit to determine whether there have been any changes in the patient's medical history or medications. A more thorough and complete update of the patient's medical history should be taken if the patient has not been seen for over a year.[51,52]

Baseline blood pressure and pulse should be recorded for the patient at each treatment visit. Elevation in blood pressure or a rapid pulse rate may indicate an anxious patient who may require a stress reduction protocol, or it may indicate that the patient has hypertension or other cardiovascular health problems. Referral to a physician or medical facility may be indicated. It is imperative that vital signs be gathered at each treatment visit for any patient with a history of major medical problems. The temperature of patients presenting with subjective fever or any signs or symptoms of a dental infection should be taken.[57,80,105]

The clinician should evaluate a patient's response to the health questionnaire from two perspectives: (1) those medical conditions and current medications that will necessitate altering the manner in which dental care will be provided and (2) those medical conditions that may have oral manifestations or mimic dental pathosis.

Patients with serious medical conditions may require either a modification in the manner in which the dental care will be delivered or a modification in the dental treatment plan (Box 1-1). In addition, the clinician should be aware if the patient has any drug allergies or interactions, allergies to dental products, an artificial joint prosthesis, organ transplants, or is taking medications that may negatively interact with common local anesthetics, analgesics, sedatives, and antibiotics.[80] This may seem overwhelming, but it emphasizes the importance of obtaining a thorough and accurate medical history while considering the various medical conditions and dental treatment modifications that may be necessary before dental treatment is provided.

Several medical conditions have oral manifestations, which must be carefully considered when attempting to arrive at an accurate dental diagnosis. Many of the oral soft tissue changes that occur are more related to the medications used to treat the medical condition rather than to the condition itself. More common examples of medication side effects are stomatitis, xerostomia, petechiae, ecchymoses, lichenoid mucosal lesions, and bleeding of the oral soft tissues.[80]

When developing a dental diagnosis, a clinician must also be aware that some medical conditions can have clinical presentations that mimic oral pathologic lesions.[13,28,32,74,80,102,107,133] For example, tuberculosis involvement of the cervical and submandibular lymph nodes can lead to a misdiagnosis of lymph node enlargement secondary to an odontogenic infection. Lymphomas can involve these same lymph nodes.[80] Immunocompromised patients and patients with uncontrolled diabetes mellitus respond poorly to dental treatment and may exhibit recurring abscesses in the oral cavity that must be differentiated from abscesses of dental origin.[43,76,80,83] Patients with iron deficiency anemia, pernicious anemia, and leukemia frequently exhibit paresthesia of the oral soft tissues. This finding may complicate making a diagnosis when other dental pathosis is present in the same area of the oral cavity. Sickle cell anemia has the complicating factor of bone pain, which mimics odontogenic pain, and loss of trabecular bone pattern on radiographs, which can be confused with radiographic lesions of endodontic origin. Multiple myeloma can result in unexplained mobility of teeth. Radiation therapy to the head and neck region can result in increased sensitivity of the teeth and osteoradionecrosis.[80] Trigeminal neuralgia, referred pain from cardiac angina, and multiple sclerosis can also mimic dental pain (see also Chapter 17). Acute maxillary sinusitis is a common condition that may create diagnostic confusion because it may mimic tooth pain in the maxillary posterior quadrant. In this situation the teeth in the quadrant may be extremely sensitive to cold and percussion, thus mimicking the signs and symptoms of pulpitis. This is certainly not a complete list of all the medical entities that can mimic dental disease, but it should alert the clinician that a medical problem could confuse and complicate

BOX 1-1

Medical Conditions That Warrant Modification of Dental Care or Treatment

Cardiovascular: High- and moderate-risk categories of endocarditis, pathologic heart murmurs, hypertension, unstable angina pectoris, recent myocardial infarction, cardiac arrhythmias, poorly managed congestive heart failure[57,80,105]

Pulmonary: Chronic obstructive pulmonary disease, asthma, tuberculosis[80,129]

Gastrointestinal and renal: End-stage renal disease; hemodialysis; viral hepatitis (types B, C, D, and E); alcoholic liver disease; peptic ulcer disease; inflammatory bowel disease; pseudomembranous colitis[25,34,48,80]

Hematologic: Sexually transmitted diseases, HIV and AIDS, diabetes mellitus, adrenal insufficiency, hyperthyroidism and hypothyroidism, pregnancy, bleeding disorders, cancer and leukemia, osteoarthritis and rheumatoid arthritis, systemic lupus erythematosus[35,43,76,80,83,88,100,135]

Neurologic: Cerebrovascular accident, seizure disorders, anxiety, depression and bipolar disorders, presence or history of drug or alcohol abuse, Alzheimer disease, schizophrenia, eating disorders, neuralgias, multiple sclerosis, Parkinson disease[36,44,80]

the diagnosis of dental pathosis; this issue is discussed in more detail in subsequent chapters.

If, at the completion of a thorough dental examination, the subjective, objective, clinical testing and radiographic findings do not result in a diagnosis with an obvious dental origin, then the clinician must consider that an existing medical problem could be the true source of the pathosis. In such instances, a consultation with the patient's physician is always appropriate.

Dental History

The chronology of events that lead up to the chief complaint is recorded as the *dental history*. This information will help guide the clinician as to which diagnostic tests are to be performed. The history should include any past and present symptoms, as well as any procedures or trauma that might have evoked the chief complaint. Proper documentation is imperative. It may be helpful to use a premade form to record the pertinent information obtained during the dental history interview and diagnostic examination. Often a SOAP format is used, with the history and findings documented under the categories of *S*ubjective, *O*bjective, *A*ppraisal, and *P*lan. There are also built-in features within some practice management software packages that allow digital entries into the patient's electronic file for the diagnostic workup (Figs. 1-3 and 1-4).

History of Present Dental Problem

The dialogue between the patient and the clinician should encompass all of the details pertinent to the events that led to the chief complaint. The clinician should direct the conversation in a manner that produces a clear and concise narrative that chronologically depicts all of the necessary information about the patient's symptoms and the development of these symptoms. To help elucidate this information, the patient is first instructed to fill out a dental history form as a part of the patient's office registration. This information will help the clinician decide which approach to use when asking the patient questions. The interview first determines *what is going on* in an effort to determine *why is it going on* for the purpose of eventually determining *what is necessary to resolve the chief complaint.*

Dental History Interview

After starting the interview and determining the nature of the chief complaint, the clinician continues the conversation by documenting the sequence of events that initiated the request for an evaluation. The dental history is divided into five basic directions of questioning: localization, commencement, intensity, provocation and attenuation, and duration.

Localization. "Can you point to the offending tooth?" Often the patient can point to or tap the offending tooth. This is the most fortunate scenario for the clinician because it helps direct the interview toward the events that might have caused any particular pathosis in this tooth. In addition, localization allows subsequent diagnostic tests to focus more on this particular tooth. When the symptoms are not well localized, the diagnosis is a greater challenge.

Commencement. "When did the symptoms first occur?" A patient who is having symptoms often remembers when these symptoms started. Sometimes the patient will even remember the initiating event: it may have been spontaneous in nature; it may have begun after a dental visit for a restoration; trauma may be the etiology, biting on a hard object may have initially produced the symptoms, or the initiating event may have occurred concurrently with other symptoms (sinusitis, headache, chest pain, etc.). However, the clinician should resist the tendency to make a premature diagnosis based on these circumstances. The clinician should not simply assume "guilt by association" but instead should use this information to enhance the overall diagnostic process.

Intensity. "How intense is the pain?" It often helps to quantify how much pain the patient is actually having. The clinician might ask, "On a scale from 1 to 10, with 10 the most severe, how would you rate your symptoms?" Hypothetically, a patient could present with "an uncomfortable sensitivity to cold" or "an annoying pain when chewing" but might rate this "pain" only as a 2 or a 3. These symptoms certainly contrast with the type of symptoms that prevent a patient from sleeping at night. Often the intensity can be subjectively measured by what is necessary for the diminution of pain—for example, acetaminophen versus anarcotic pain reliever. This intensity level may affect the decision to treat or not to treat with endodontic therapy. Pain is now considered a standard vital sign, and documenting pain intensity (scale of 0 to 10) provides a baseline for comparison after treatment.

Provocation and attenuation. "What produces or reduces the symptoms?" Mastication and locally applied temperature changes account for the majority of initiating factors that cause dental pain. The patient may relate that drinking something cold causes the pain or possibly that chewing or biting is the only stimulus that "makes it hurt." The patient might say that the pain is only reproduced on "release from biting." On occasion, a patient may present to the dental office with a cold drink in hand and state that the symptoms can only be *reduced* by bathing the tooth in cold water. Nonprescription pain relievers may relieve some symptoms, whereas narcotic medication may be required to reduce others (see Chapter 4 for more information). Note that patients who are using narcotic as well as non-narcotic (e.g., ibuprofen) analgesics may respond differently to questions and diagnostic tests, thereby altering the validity of diagnostic results. Thus, it is important to know what drugs patients have taken in the previous 4 to 6 hours. These provoking and relieving factors may help the clinician to determine which diagnostic tests should be performed to establish a more objective diagnosis.

Duration. "Do the symptoms subside shortly, or do they linger after they are provoked?" The difference between a cold sensitivity that subsides in a few seconds and one that subsides in minutes may determine whether a clinician repairs a defective restoration or provides endodontic treatment. The duration of symptoms after a stimulating event should be recorded to establish how long the patient felt the sensation in terms of seconds or minutes. Clinicians often first test control teeth (possibly including a contralateral "normal" tooth) to define a "normal" response for the patient; thus, "lingering" pain is apparent when comparing the duration between the control teeth and the suspected tooth.

With the dental history interview complete, the clinician has a better understanding of the patient's chief complaint and can concentrate on making an objective diagnostic evaluation,

Name: (Last) _____ (First) _____ **Date:** _____ **Tooth:** _____

S. (SUBJECTIVE)
Chief Complaint:
History of Present Illness:

Nature of Pain: None Mild Moderate Severe
Quality: Dull Sharp Throbbing Constant
Onset: Stim Required Intermittent Spontaneous
Location: Localized Diffuse Referred Radiating to:
Duration: Seconds Minutes Hours Constant
Initiated by: Cold Heat Sweet Spontaneous Palpation Mastication Supination Keeps awake at night
Relieved by: Cold Heat OTC-Meds Narc-Meds

O. (OBJECTIVE)

Extraoral:
Facial swelling: Yes No
L Nodes swollen: Yes No

Intraoral:
Soft tissues: WNL
Swelling: Yes No Mild Moderate Severe Location:
Sinus tract: Yes No Closed
Clinical crown: Restn Caries Exposure Fracture

#	Cold	Heat	EPT	Perc	Palp	Mob	Bite Stick	Dis-color	Periodontal Exam								
									MB	B	DB	DL	L	ML	Recessn	Furcation	Bleed - Probing

(Normal: N No Response: 0 Mild: + Moderate: ++ Severe: +++ Lingered: L Delayed: D)

Radiographic Findings:
Alveolar Bone: WNL Apical lucency Lateral lucency Ap / Lat opacity Crestal bone loss
Lamina Dura: WNL Obscure Broken Widened
Roots: WNL Curvature Resorption Perforation Dilaceration Fracture Long Sinus / IAN
Pulp Chamber: WNL Calcification Pulp Stone Exposure Resorption Perforation
Pulp Canal: WNL Calcification Bifurcated Resorption Prior RCT Furcation Involvement Perforation
Crown: WNL Caries Restoration Crown Dens in dente
Sinus Tract: Traces to:

A. (Assessment)
Diagnosis: Pulpal: WNL Rev Pulpitis Irrev Pulpitis Necrosis Prior RCT / Non-healing Pulpless
Periapical: WNL APP CPP APA CPA Cond Osteitis
Etiology: Caries Restoration Prior RCT Iatrogenic Coronal leakage Trauma Perio Elective Resorptn VRF
Prognosis: Good Fair Poor

P. (PLAN)
Endodontic: Caries control RCT ReTx I&D Apico Apexification/genesis Perf / Resorption Repair

Periodontal: S/RP Crown lengthen Root amp Hemisection Extraction

Restorative: Temp Post space B/U P&C Onlay / Crown Bleach

FIG. 1-3 When taking a dental history and *performing* a diagnostic examination, often a premade form can facilitate complete and accurate documentation. (Courtesy Dr. Ravi Koka, San Francisco, CA.)

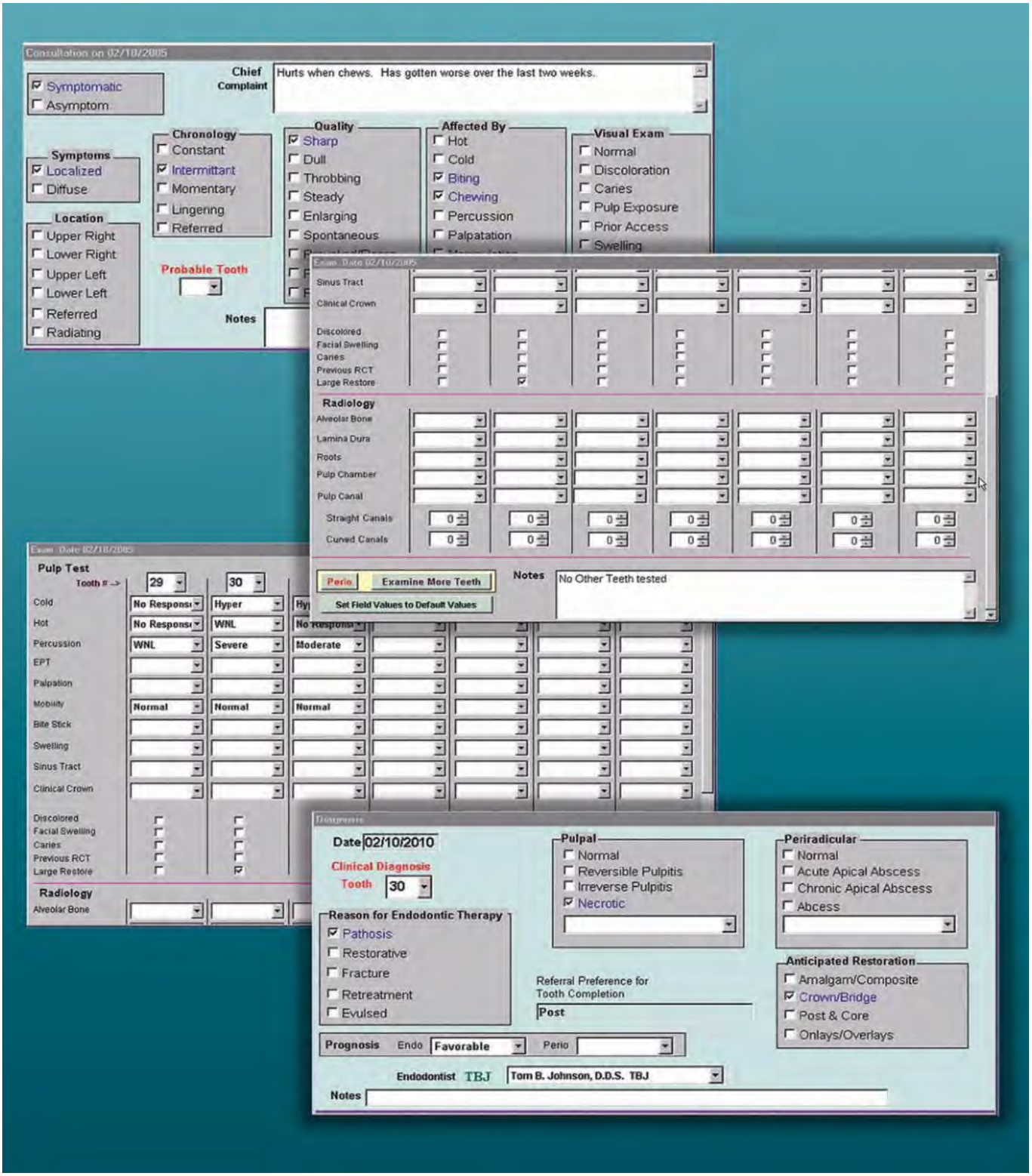


FIG. 1-4 Several practice management software packages have features for charting endodontic diagnoses using user-defined drop-down menus and areas for specific notations. Note that for legal purposes, it is desirable that all recorded documentation have the ability to be locked, or if any modifications are made after 24 hours, the transaction should be recorded with an automated time/date stamp. This is necessary so that the data cannot be fraudulently manipulated. (Courtesy PBS Endo, Cedar Park, TX.)

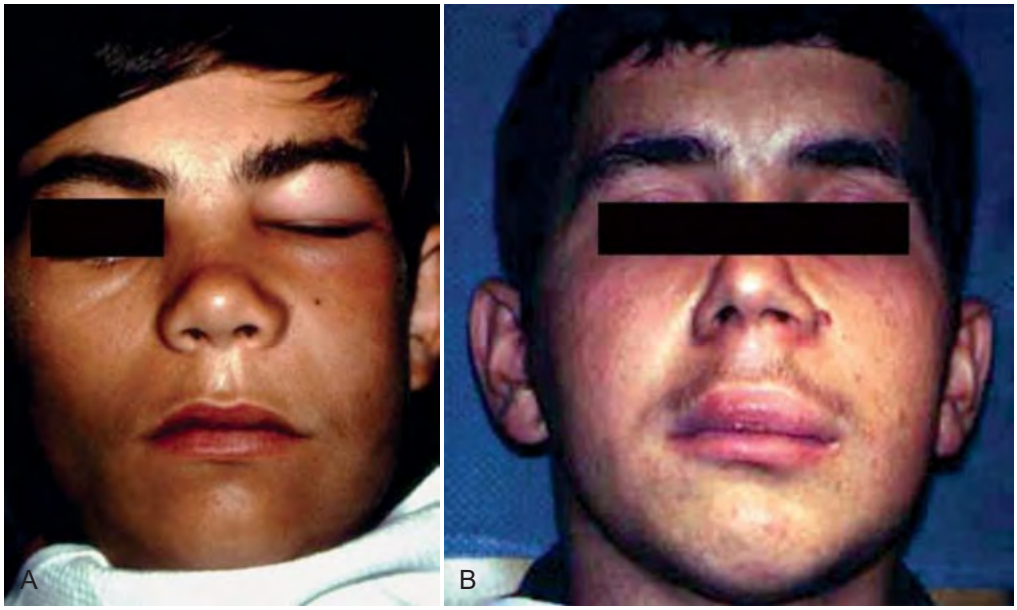


FIG. 1-5 A, Canine space swelling of the left side of the face extending into and involving the left eye. B, Swelling of the upper lip and the loss of definition of the nasolabial fold on the patient's left side, which indicates an early canine space infection.

although the subjective (and artistic) phase of making a diagnosis is not yet complete and will continue after the more objective testing and scientific phase of the investigatory process.

EXAMINATION AND TESTING

Extraoral Examination

Basic diagnostic protocol suggests that a clinician observe patients as they enter the operatory. Signs of physical limitations may be present, as well as signs of facial asymmetry that result from facial swelling. Visual and palpation examinations of the face and neck are warranted to determine whether swelling is present. Many times a facial swelling can be determined only by palpation when a unilateral "lump or bump" is present. The presence of bilateral swellings may be a normal finding for any given patient; however, it may also be a sign of a systemic disease or the consequence of a developmental event. Palpation allows the clinician to determine whether the swelling is localized or diffuse, firm or fluctuant. These latter findings will play a significant role in determining the appropriate treatment.

Palpation of the cervical and submandibular lymph nodes is an integral part of the examination protocol. If the nodes are found to be firm and tender along with facial swelling and an elevated temperature, there is a high probability that an infection is present. The disease process has moved from a localized area immediately adjacent to the offending tooth to a more widespread systemic involvement.

Extraoral facial swelling of odontogenic origin typically is the result of endodontic etiology because diffuse facial swelling resulting from a periodontal abscess is rare. Swellings of nonodontogenic origin must always be considered in the differential diagnosis, especially if an obvious dental pathosis is not found.[77] This situation is discussed in subsequent chapters.

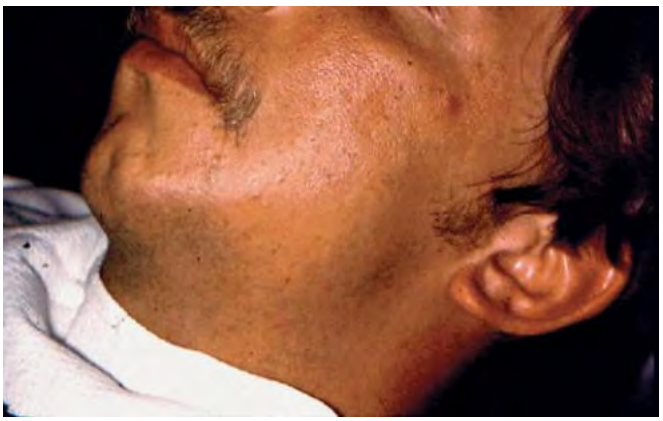


FIG. 1-6 Buccal space swelling associated with an acute periradicular abscess from the mandibular left second molar.

A subtle visual change such as loss of definition of the nasolabial fold on one side of the nose may be the earliest sign of a canine space infection (Fig. 1-5). Pulpal necrosis and periradicular disease associated with a maxillary canine should be suspected as the source of the problem. Extremely long maxillary central incisors may also be associated with a canine space infection, but most extraoral swellings associated with the maxillary centrals express themselves as a swelling of the upper lip and base of the nose.

If the buccal space becomes involved, the swelling will be extraoral in the area of the posterior cheek (Fig. 1-6). These swellings are generally associated with infections originating from the buccal root apices of the maxillary premolar and molar teeth and the mandibular premolar (Fig. 1-7) and first molar teeth. The mandibular second and third molars may also be involved, but infections associated with these two teeth are just as likely to exit to the lingual where other spaces

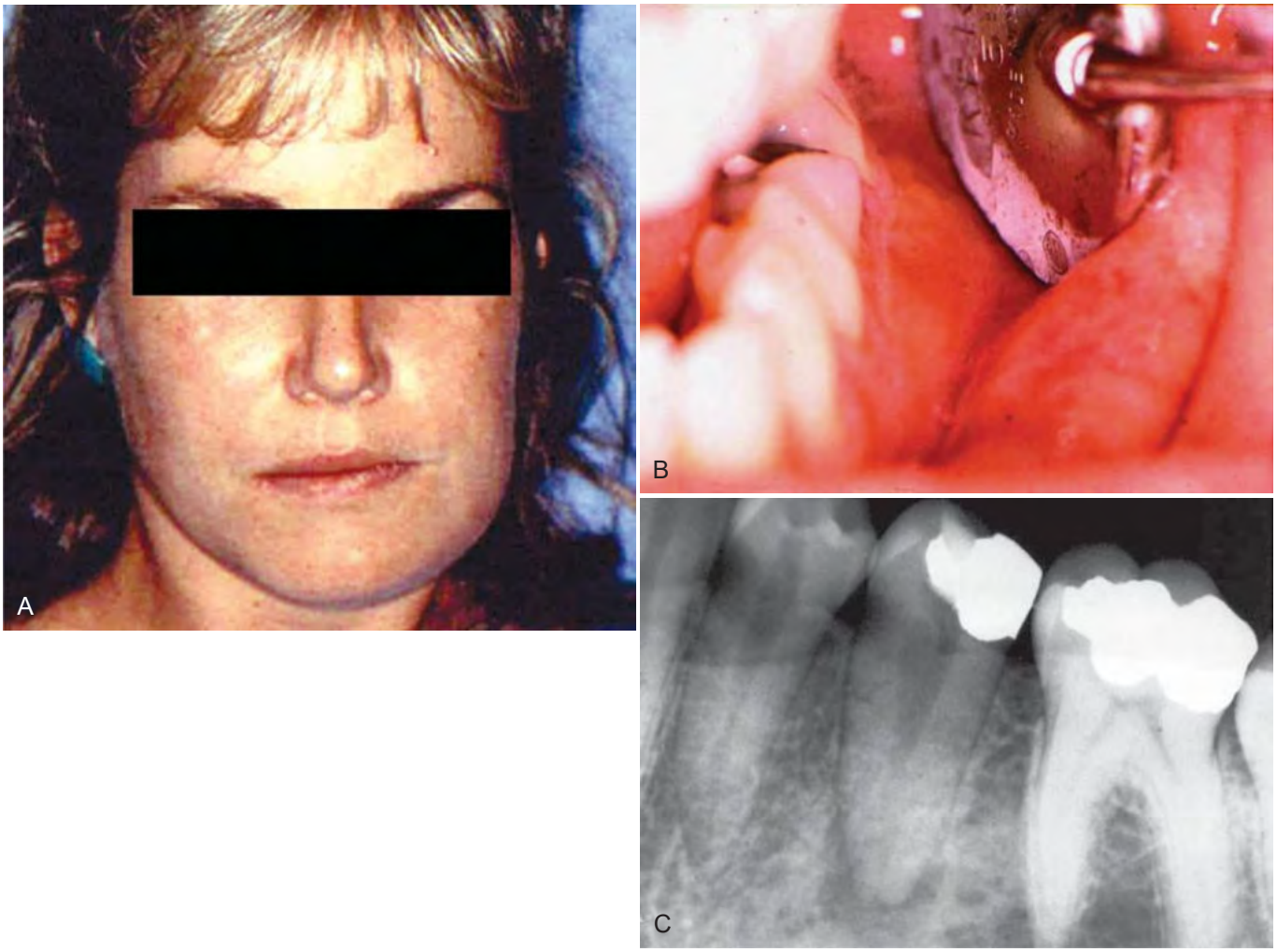


FIG. 1-7 A, Buccal space swelling of the left side of the patient's face. Note the asymmetry of the left side of the face. B, Intraoral view shows swelling present in the left posterior mucobuccal fold. C, This buccal space infection was associated with periradicular disease from the mandibular left second premolar. Note on the radiograph the periradicular radiolucency and large restoration associated with this tooth.

would be involved. For infections associated with these teeth, the root apices of the maxillary teeth must lie superior to the attachment of the buccinator muscle to the maxilla, and the apices of the mandibular teeth must be inferior to the buccinator muscle attachment to the mandible.[77]

Extraoral swelling associated with mandibular incisors will generally exhibit itself in the submental (Fig. 1-8) or submandibular space. Infections associated with any mandibular teeth, which exit the alveolar bone on the lingual and are inferior to the mylohyoid muscle attachment, will be noted as swelling in the submandibular space. Further discussions of fascial space infections may be found in Chapter 14.

Sinus tracts of odontogenic origin may also open through the skin of the face (Figs. 1-9 and 1-10).[2,56,64] These openings in the skin will generally close once the offending tooth is treated and healing occurs. A scar is more likely to be visible on the skin surface in the area of the sinus tract stoma than on the oral mucosal tissues (Fig. 1-10, *C* and *D*). Many patients with extraoral sinus tracts give a history of being treated by general physicians, dermatologists, or plastic surgeons with systemic or topical antibiotics or surgical procedures in

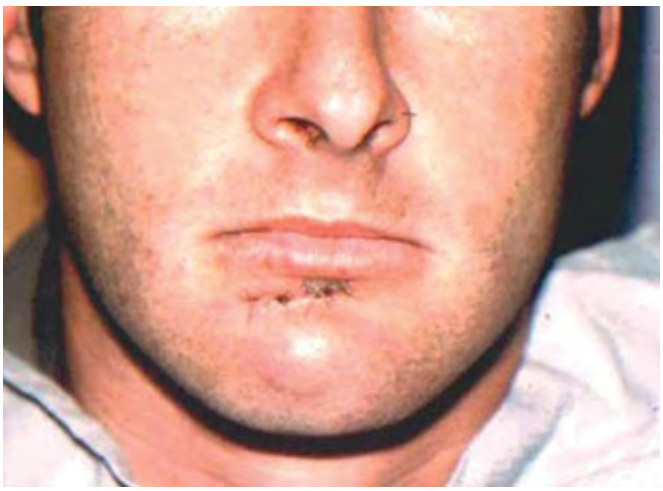


FIG. 1-8 Swelling of the submental space associated with periradicular disease from the mandibular incisors.

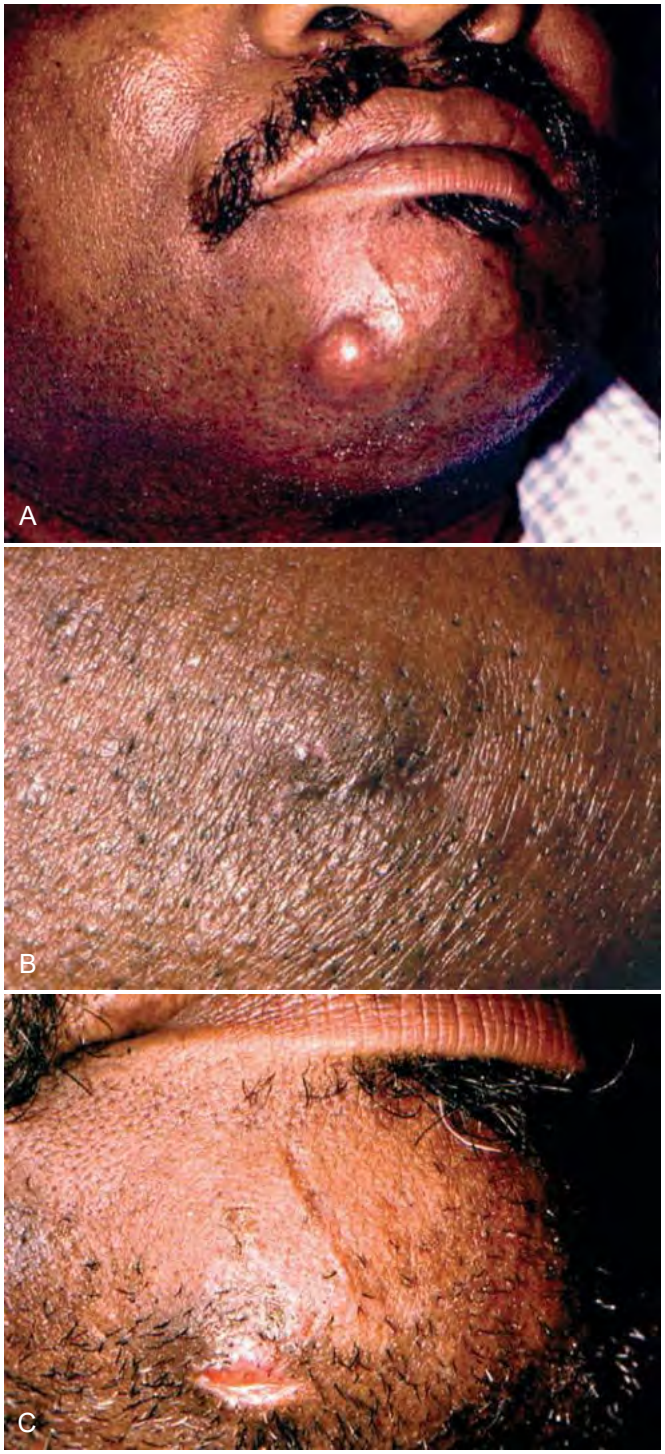


FIG. 1-9 A, Extraoral drainage associated with periradicular disease from the mandibular right canine. Note the parulis on the right anterior side of the face. B, Initial scar associated with the extraoral drainage incision after the parulis was drained and root canal therapy performed on the canine. C, Three-month follow-up shows healing of the incision area. Note the slight inversion of the scar tissue.

attempts to heal the extraoral stoma. In these particular cases, after multiple treatment failures, the patients may finally be referred to a dental clinician to determine whether there is a dental cause. Raising the awareness of physicians to such cases will aid in more accurate diagnosis and faster referral to the dentist or endodontist.

Intraoral Examination

The intraoral examination may give the clinician insight as to which intraoral areas may need a more focused evaluation. Any abnormality should be carefully examined for either prevention or early treatment of associated pathosis.[4,30,75,113,110,126] Swelling, localized lymphadenopathy, or a sinus tract should provoke a more detailed assessment of related and proximal intraoral structures.

Soft Tissue Examination

As with any dental examination, there should be a routine evaluation of the intraoral soft tissues. The gingiva and mucosa should be dried with either a low-pressure air syringe or a 2-by-2-inch gauze pad. By retracting the tongue and cheek, all of the soft tissue should be examined for abnormalities in color or texture. Any raised lesions or ulcerations should be documented and, when necessary, evaluated with a biopsy or referral.[82]

Intraoral Swelling

Intraoral swellings should be visualized and palpated to determine whether they are diffuse or localized and whether they are firm or fluctuant. These swellings may be present in the attached gingiva, alveolar mucosa, mucobuccal fold, palate, or sublingual tissues. Other testing methods are required to determine whether the origin is endodontic, periodontic, or a combination of these two or whether it is of nonodontogenic origin.

Swelling in the anterior part of the palate (Fig. 1-11) is most frequently associated with an infection present at the apex of the maxillary lateral incisor or the palatal root of the maxillary first premolar. More than 50% of the maxillary lateral incisor root apices deviate in the distal or palatal directions. A swelling in the posterior palate (Fig. 1-12) is most likely associated with the palatal root of one of the maxillary molars.[77]

Intraoral swelling present in the mucobuccal fold (Fig. 1-13) can result from an infection associated with the apex of the root of any maxillary tooth that exits the alveolar bone on the facial aspect and is inferior to the muscle attachment present in that area of the maxilla (see also Chapter 14). The same is true with the mandibular teeth if the root apices are superior to the level of the muscle attachments and the infection exits the bone on the facial. Intraoral swelling can also occur in the sublingual space if the infection from the root apex spreads to the lingual and exits the alveolar bone superior to the attachment for the mylohyoid muscle. The tongue will be elevated and the swelling will be bilateral because the sublingual space is contiguous with no midline separation. If the infection exits the alveolar bone to the lingual with mandibular molars and is inferior to the attachment of the mylohyoid muscle, the swelling will be noted in the submandibular space. Severe infections involving the maxillary and mandibular molars can extend into the parapharyngeal space, resulting in intraoral swelling of the tonsillar and pharyngeal areas. This can be life threatening if the patient's airway becomes obstructed.[77,80]

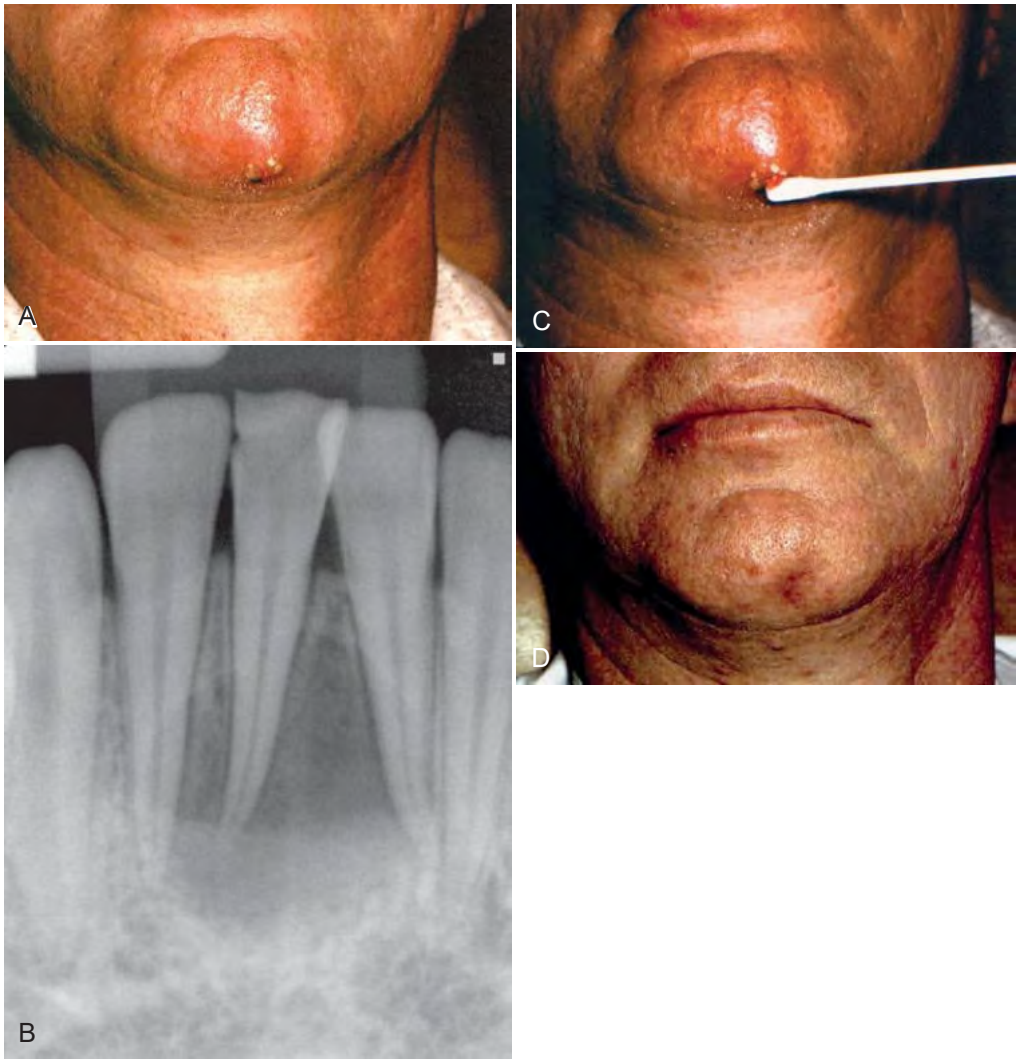


FIG. 1-10 A, Extraoral sinus tract opening onto the skin in the central chin area. B, Radiograph showing large radiolucency associated with the mandibular incisors. C, A culture is obtained from the drainage of the extraoral sinus tract. D, The healed opening of the extraoral sinus tract 1 month after root canal therapy was completed. Note the slight skin concavity in the area of the healed sinus tract.

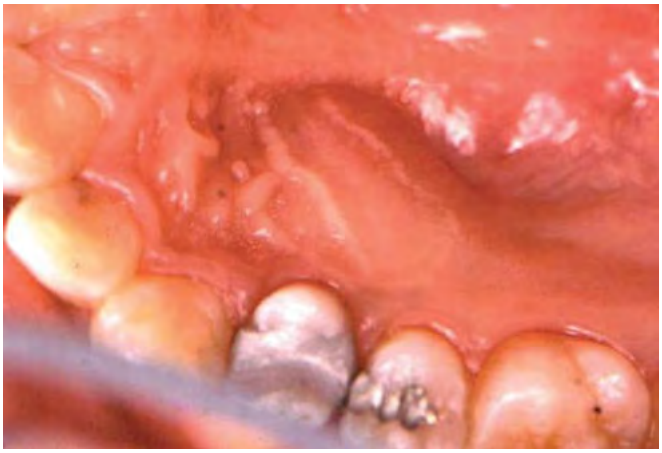


FIG. 1-11 Fluctuant swelling in the anterior palate associated with periradicular disease from the palatal root of the maxillary first premolar.

Intraoral Sinus Tracts

On occasion, a chronic endodontic infection will drain through an intraoral communication to the gingival surface and is known as a *sinus tract*.[12] This pathway, which is sometimes lined with epithelium, extends directly from the source of the infection to a surface opening, or *stoma,* on the attached gingival surface. As previously described, it can also extend extraorally. The term *fistula* is often inappropriately used to describe this type of drainage. The fistula, by definition, is actually an abnormal communication pathway between two internal organs or from one epithelium-lined surface to another epithelium-lined surface.[6]

Histologic studies have found that most sinus tracts are not lined with epithelium throughout their entire length. One study found that only 1 out of the 10 sinus tracts examined were lined with epithelium, whereas the other nine specimens were lined with granulation tissue.[55] Another study, with a larger sample size, found that two thirds of the specimens did not have epithelium extending beyond the level of the surface mucosa rete ridges.[12] The remaining specimens had some

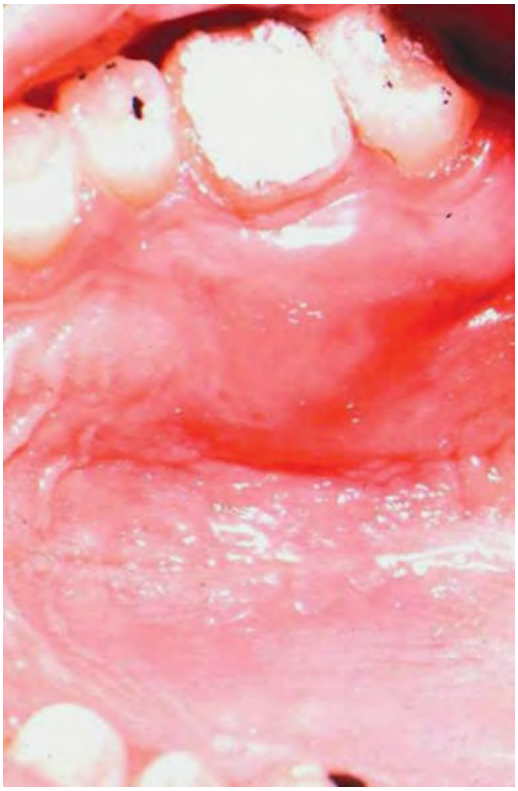


FIG. 1-12 Fluctuant swelling in the posterior palate associated with periradicular disease from the palatal root of the maxillary first molar.

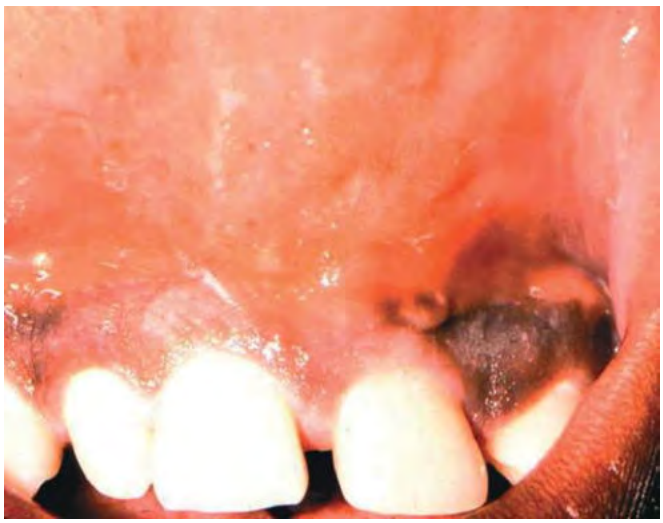


FIG. 1-13 Fluctuant swelling in the mucobuccal fold associated with periradicular disease from the maxillary central incisor.

epithelium that extended from the oral mucosa surface to the periradicular lesion.[12] The presence or absence of an epithelial lining does not seem to prevent closure of the tract as long as the source of the problem is properly diagnosed and adequately treated and the endodontic lesion has healed. Failure of a sinus tract to heal after treatment will necessitate further diagnostic procedures to determine whether other sources of infection are present or whether a misdiagnosis occurred.

In general, a periapical infection that has an associated sinus tract is not painful, although often there is a history of varying magnitudes of discomfort before sinus tract development. Besides providing a conduit for the release of infectious exudate and the subsequent relief of pain, the sinus tract can also provide a useful aid in determining the source of a given infection. Sometimes objective evidence as to the origin of an odontogenic infection is lacking. The stoma of the sinus tract may be located directly adjacent to or at a distant site from the infection. Tracing the sinus tract will provide objectivity in diagnosing the location of the problematic tooth. To trace the sinus tract, a size #25 or #30 gutta-percha cone is threaded into the opening of the sinus tract. Although this may be slightly uncomfortable to the patient, the cone should be inserted until resistance is felt. After a periapical radiograph is exposed, the origin of the sinus tract is determined by following the path taken by the gutta-percha cone (Fig. 1-14). This will direct the clinician to the tooth involved and, more specifically, to the root of the root of the tooth that is the source of the pathosis. Once the causative factors related to the formation of the sinus tract are removed, the stoma and the sinus tract will close within several days.

The stomata of intraoral sinus tracts may open in the alveolar mucosa, in the attached gingiva, or through the furcation or gingival crevice. They may exit through either the facial or the lingual tissues depending on the proximity of the root apices to the cortical bone. If the opening is in the gingival crevice, it is normally present as a narrow defect in one or two isolated areas along the root surface. When a narrow defect is present, the differential diagnosis must include the opening of a periradicular endodontic lesion, a vertical root fracture, or the presence of a developmental groove on the root surface. This type of sinus tract can be differentiated from a primary periodontal lesion because the latter generally presents as a pocket with a broad coronal opening and more generalized alveolar bone loss around the root. Other pulp testing methods may assist in verifying the source of infection.[111,112,121]

Palpation

In the course of the soft tissue examination, the alveolar hard tissues should also be palpated. Emphasis should be placed on detecting any soft tissue swelling or bony expansion, especially noting how it compares with and relates to the adjacent and contralateral tissues. In addition to objective findings, the clinician should question the patient about any areas that feel unusually sensitive during this palpation part of the examination.

A palpation test is performed by applying firm digital pressure to the mucosa covering the roots and apices. The index finger is used to press the mucosa against the underlying cortical bone. This will detect the presence of periradicular abnormalities or specific areas that produce painful response to digital pressure. A positive response to palpation may indicate an active periradicular inflammatory process. This test does not indicate, however, whether the inflammatory process is of endodontic or periodontal origin.

Percussion

Referring back to the patient's chief complaint may indicate the importance of percussion testing for this particular case. If the patient is experiencing acute sensitivity or pain on mastication, this response can typically be duplicated by individually percussing the teeth, which often isolates the symptoms to a particular tooth. Pain to percussion does not indicate that the

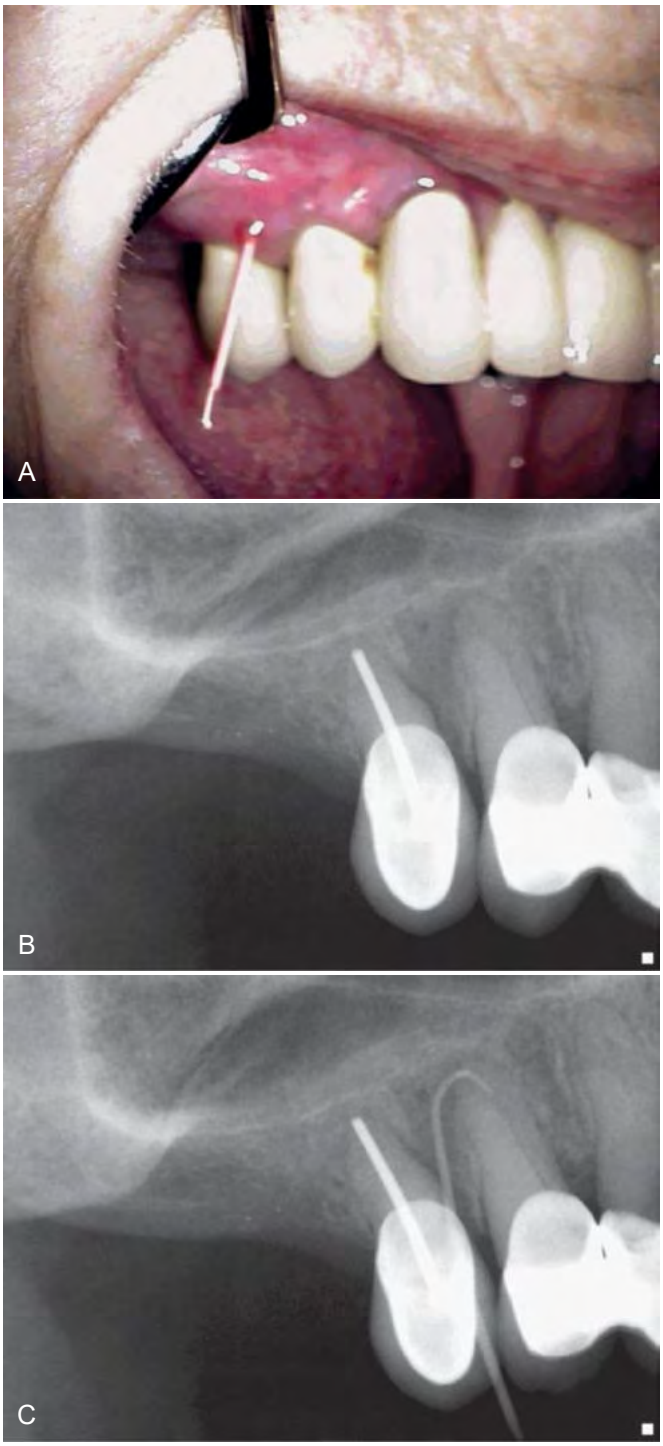


FIG. 1-14 A, To locate the source of an infection, the sinus tract can be traced by threading the stoma with a gutta-percha point. B, Radiograph of the area shows an old root canal in a maxillary second premolar and a questionable radiolucent area associated with the first premolar, with no clear indication of the etiology of the sinus tract. C, After tracing the sinus tract, the gutta-percha is seen to be directed to the source of pathosis, the apex of the maxillary first premolar.

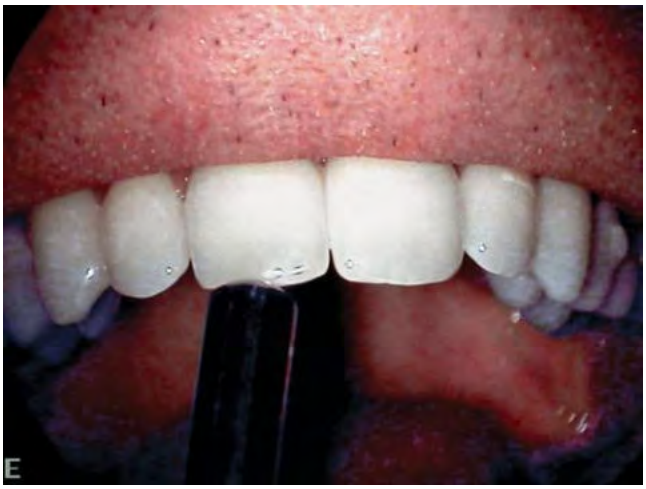


FIG. 1-15 Percussion testing of a tooth, using the back end of a mirror handle.

tooth is vital or nonvital but is rather an indication of inflammation in the periodontal ligament (i.e., symptomatic apical periodontitis). This inflammation may be secondary to physical trauma, occlusal prematurities, periodontal disease, or the extension of pulpal disease into the periodontal ligament space. The indication of where the pain originates is interpreted by the mesencephalic nucleus, receiving its information from proprioceptive nerve receptors. Although subject to debate, the general consensus is that there are relatively few proprioceptors in the dental pulp; however, they are prevalent in the periodontal ligament spaces.[24] This is why it may be difficult for the patient to discriminate the location of dental pain in the earlier stages of pathosis, when only the C fibers are stimulated. Once the disease state extends into the periodontal ligament space, the pain may become more localized for the patient; therefore, the affected tooth will be more identifiable with percussion and mastication testing.

Before percussing any teeth, the clinician should tell the patient what will transpire during this test. Because the presence of acute symptoms may create anxiety and possibly alter the patient's response, properly preparing the patient will lead to more accurate results. The contralateral tooth should first be tested as a control, as should several adjacent teeth that are certain to respond normally. The clinician should advise the patient that the sensation from this tooth is normal and ask to be advised of any tenderness or pain from subsequent teeth.

Percussion is performed by tapping on the incisal or occlusal surfaces of the teeth either with the finger or with a blunt instrument. The testing should initially be done gently, with light pressure being applied digitally with a gloved finger tapping. If the patient cannot detect significant difference between any of the teeth, the test should be repeated using the blunt end of an instrument, like the back end of a mirror handle (Fig. 1-15). The tooth crown is tapped vertically and horizontally. The tooth should first be percussed occlusally, and if the patient discerns no difference, the test should be repeated, percussing the buccal and lingual aspects of the teeth. For any heightened responses, the test should be repeated as necessary to determine that it is accurate and reproducible, and the information should be documented.

Although this test does not disclose the condition of the pulp, it indicates the presence of a periradicular inflammation.

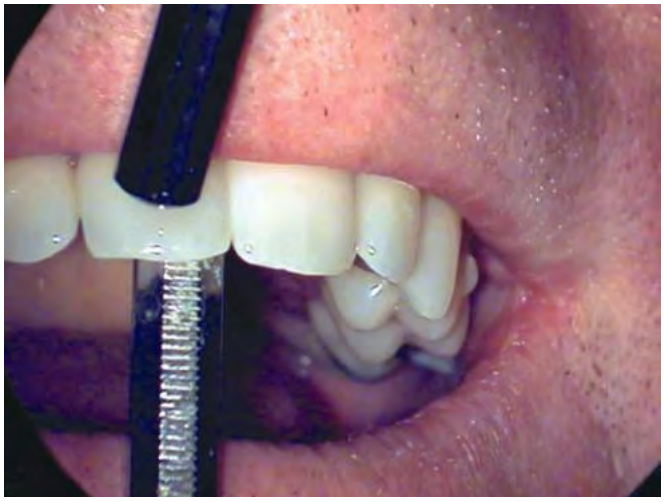


FIG. 1-16 Mobility testing of a tooth, using the back ends of two mirror handles.

BOX 1-2

Recording Tooth Mobility

+1 mobility: The first distinguishable sign of movement greater than normal
+2 mobility: Horizontal tooth movement no greater than 1 mm
+3 mobility: Horizontal tooth movement greater than 1 mm, with or without the visualization of rotation or vertical depressability

BOX 1-3

Recording Furcation Defects

Class I furcation defect: The furcation can be probed but not to a significant depth.
Class II furcation defect: The furcation can be entered into but cannot be probed completely through to the opposite side.
Class III furcation defect: The furcation can be probed completely through to the opposite side.

An abnormal positive response indicates inflammation of the periodontal ligament that may be of either pulpal or periodontal origin. The sensitivity of the proprioceptive fibers in an inflamed periodontal ligament will help identify the location of the pain. This test should be done gently, especially in highly sensitive teeth. It should be repeated several times and compared with control teeth.

Mobility

Like percussion testing, an increase in tooth mobility is not an indication of pulp vitality. It is merely an indication of a compromised periodontal attachment apparatus. This compromise could be the result of acute or chronic physical trauma, occlusal trauma, parafunctional habits, periodontal disease, root fractures, rapid orthodontic movement, or the extension of pulpal disease, specifically an infection, into the periodontal ligament space. Tooth mobility is directly proportional to the integrity of the attachment apparatus or to the extent of inflammation in the periodontal ligament. Often the mobility reverses to normal after the initiating factors are repaired or eliminated. Because determining mobility by simple finger pressure can be visually subjective, the back ends of two mirror handles should be used, one on the buccal aspect and one on the lingual aspect of the tooth (Fig. 1-16). Pressure is applied in a facial-lingual direction as well as in a vertical direction and the tooth mobility is scored (Box 1-2). Any mobility that exceeds +1 should be considered abnormal. However, the teeth should be evaluated on the basis of how mobile they are relative to the adjacent and contralateral teeth.

Periodontal Examination

Periodontal probing is an important part of any intraoral diagnosis. The measurement of periodontal pocket depth is an indication of the depth of the gingival sulcus, which corresponds to the distance between the height of the free gingival margin and the height of the attachment apparatus below. Using a calibrated periodontal probe, the clinician should record the periodontal pocket depths on the mesial, middle, and distal aspects of both the buccal and lingual sides of the tooth, noting the depths in millimeters. The periodontal probe is "stepped" around the long axis of the tooth, progressing in 1-mm increments. Periodontal bone loss that is wide, as determined by a wide span of deep periodontal probings, is generally considered to be of periodontal origin and is typically more generalized in other areas of the mouth. However, isolated areas of vertical bone loss may be of an endodontic origin, specifically from a nonvital tooth whose infection has extended from the periapex to the gingival sulcus. Again, proper pulp testing is imperative, not just for the determination of a diagnosis but also for the development of an accurate prognosis assessment. For example, a periodontal pocket of endodontic origin may resolve after endodontic treatment, but if the tooth was originally vital with an associated deep periodontal pocket, endodontic treatment will not improve the periodontal condition. In addition, as discussed in Chapter 21, a vertical root fracture may often cause a localized narrow periodontal pocket that extends deep down the root surface. Characteristically, the adjacent periodontium is usually within normal limits.

Furcation bone loss can be secondary to periodontal or pulpal disease. The amount of furcation bone loss, as observed both clinically and radiographically, should be documented (Box 1-3). Results of pulp tests (described later) will aid in diagnosis.

Pulp Tests

Pulp testing involves attempting to make a determination of the responsiveness of pulpal sensory neurons.[62,63] The tests involve thermal or electrical stimulation of a tooth in order to obtain a subjective response from the patient (i.e., to determine whether the pulpal nerves are functional), or the tests may involve a more objective approach using devices that detect the integrity of the pulpal vasculature. Unfortunately, the quantitative evaluation of the status of pulp tissue can only be determined histologically, as it has been shown that there is not necessarily a good correlation between the objective clinical signs and symptoms and the pulpal histology.[122,123]

Thermal

Various methods and materials have been used to test the pulp's response to thermal stimuli. The baseline or normal response

to either cold or hot is a patient's report that a sensation is felt but disappears immediately upon removal of the thermal stimulus. Abnormal responses include a lack of response to the stimulus, a lingering or intensification of a painful sensation after the stimulus is removed, or an immediate, excruciatingly painful sensation as soon as the stimulus is placed on the tooth.

Cold testing is the primary pulp testing method used by many clinicians today. It is especially useful for patients presenting with porcelain jacket crowns or porcelain-fused-to-metal crowns where no natural tooth surface (or much metal) is accessible. If a clinician chooses to perform this test with sticks of ice, then the use of a rubber dam is recommended, because melting ice will run onto adjacent teeth and gingiva, yielding potentially false-positive responses.

Frozen carbon dioxide (CO_2), also known as *dry ice* or *carbon dioxide snow*, or *CO_2 stick*, has been found to be reliable in eliciting a positive response if vital pulp tissue is present in the tooth.[46,98,99] One study found that vital teeth would respond to both frozen CO_2 and skin refrigerant, with skin refrigerant producing a slightly quicker response.[66] Frozen carbon dioxide has also been found to be effective in evaluating the pulpal response in teeth with full coverage crowns for which other tests such as electric pulp testing is not possible.[11] For testing purposes, a solid stick of CO_2 is prepared by delivering CO_2 gas into a specially designed plastic cylinder (Fig. 1-17). The resulting CO_2 stick is applied to the facial surface of either the natural tooth structure or crown. Several teeth can be tested with a single CO_2 stick. The teeth should be isolated and the oral soft tissues should be protected with a 2-by-2-inch gauze or cotton roll so the frozen CO_2 will not come into contact with these structures. Because of the extremely cold temperature of the frozen CO_2 (−69°F to −119°F; −56°C to −98°C), burns of the soft tissues can occur. It has been demonstrated on extracted teeth that frozen CO_2 application has resulted in a significantly greater intrapulpal temperature decrease than either skin refrigerant or ice.[11] Also, it appears that the application of CO_2 to teeth does not result in any irreversible damage to the pulp tissues or cause any significant enamel crazing.[61,104]

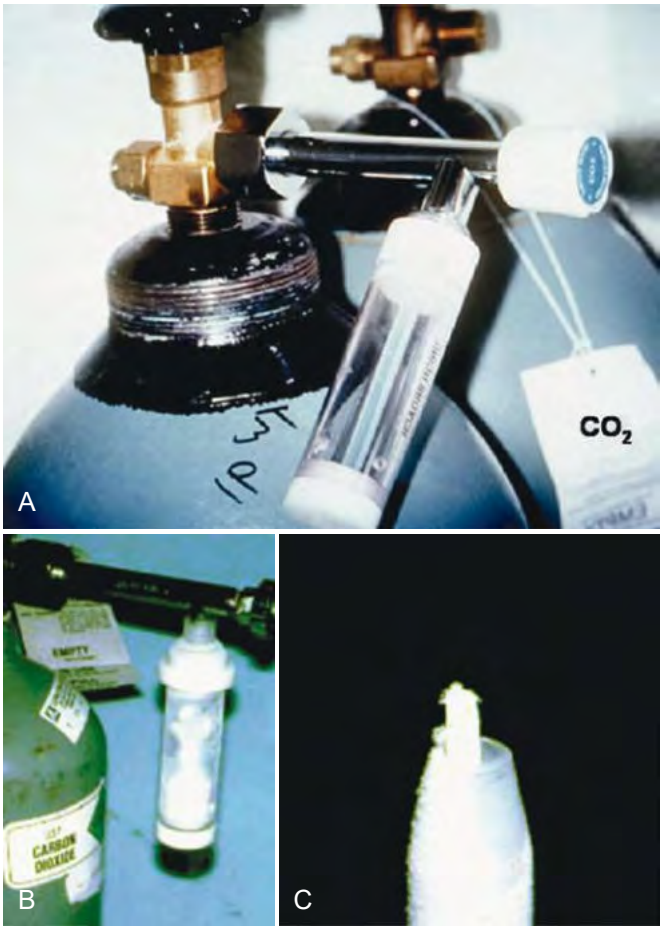


FIG. 1-17 A, Carbon dioxide tank with apparatus attached to form solid CO_2 stick/pencil. B, CO_2 gas being transformed into a solid stick/pencil. C, CO_2 stick/pencil extruded from end of a plastic carrier and ready for use.

The most popular method of performing cold testing is with a refrigerant spray. It is readily available, easy to use, and provides test results that are reproducible, reliable, and equivalent to that of frozen CO_2.[46,66,96,141] One of the current products contains 1,1,1,2-tetrafluoroethane, which has zero ozone depletion potential and is environmentally safe. It has a temperature of −26.2°C.[66] The spray is most effective for testing purposes when it is applied to the tooth on a large #2 cotton pellet (Fig. 1-18). In one study,[65] a significantly lower intrapulpal temperature was achieved when a #2 cotton pellet was dipped or sprayed with the refrigerant compared with the result when a small #4 cotton pellet or cotton applicator was used. The sprayed cotton pellet should be applied to the midfacial area of the tooth or crown. As with any other pulp testing method, adjacent or contralateral "normal" teeth should also be tested to establish a baseline response. It appears that frozen CO_2 and refrigerant spray are superior to other cold testing methods and equivalent or superior to the electric pulp tester for assessing pulp vitality.[11,46] However, one study found that periodontal attachment loss and gingival recession may influence the reported pain response with cold stimuli.[116]

To be most reliable, cold testing should be used in conjunction with an electric pulp tester (described later in this chapter) so that the results from one test will verify the findings of the other test. If a mature, nontraumatized tooth does not respond to both cold testing and electric pulp testing, then the pulp should be considered necrotic.[23,98,141] However, a multirooted tooth, with at least one root containing vital pulp tissue, may respond to a cold test and electric pulp test even if one or more of the roots contain necrotic pulp tissue.[98]

Another thermal testing method involves the use of heat. Heat testing is most useful when a patient's chief complaint is intense dental pain on contact with any hot liquid or food. When a patient is unable to identify which tooth is sensitive, a heat test is appropriate. Starting with the most posterior tooth in that area of the mouth, each tooth is individually isolated with a rubber dam. An irrigating syringe is filled with a liquid (most commonly plain water) that has a temperature similar to that which would cause the painful sensation. The liquid is then expressed from the syringe onto the isolated tooth to determine whether the response is normal or abnormal. The clinician moves forward in the quadrant, isolating each individual tooth until the offending tooth is located. That tooth will exhibit an immediate, intense painful response to the heat. With heat testing, a delayed response may occur, so waiting 10 seconds between each heat test will allow sufficient time for the onset of symptoms. This method can also be used to apply cold water to the entire crown for cases in which cold is the precipitating stimulus.

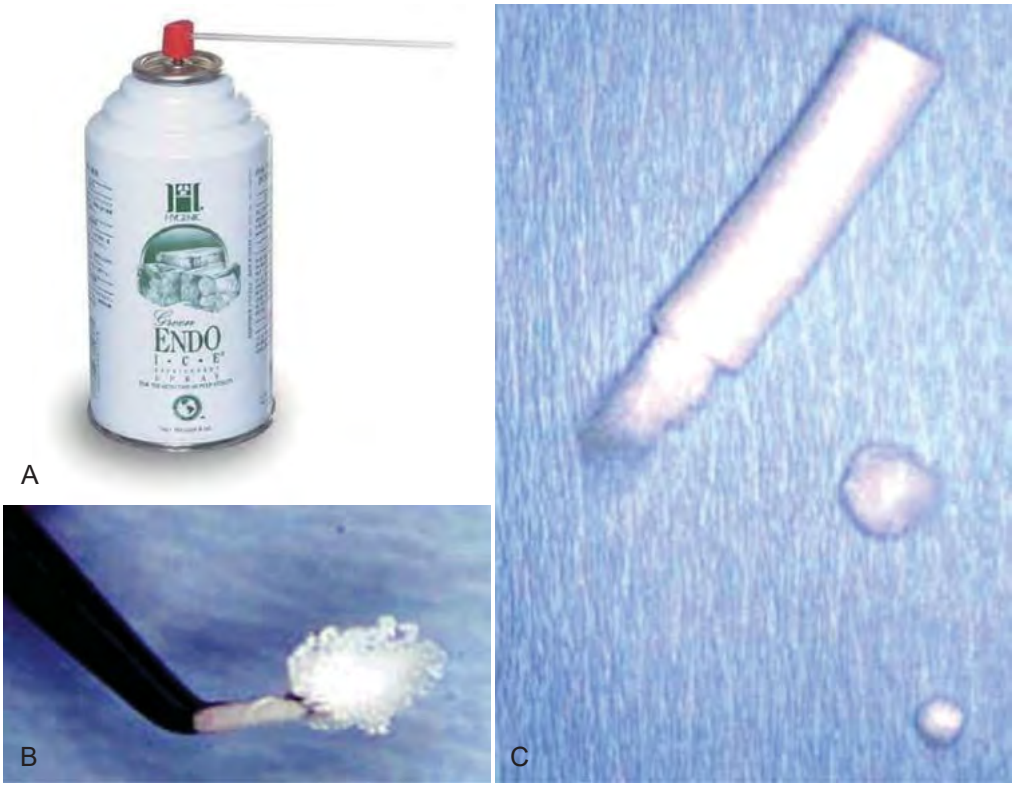


FIG. 1-18 A, Refrigerant spray container. B, A large cotton pellet made of a cotton roll or a ready-made size #2 (large) cotton pellet can be used to apply the refrigerant spray to the tooth surface. The small #4 cotton pellet does not provide as much surface area as the #2 cotton pellet, and therefore should not be used to deliver the refrigerant to the tooth surface. C, A large cotton pellet sprayed with the refrigerant and ready to be applied to the tooth surface. (*A*, Courtesy Coltène/Whaledent, Cuyahoga Falls, OH.)

Another method for heat testing is to apply heated gutta- perch
tooth. If this method is used, a light layer of lubricant should be pl
the heated material to prevent the hot gutta-percha or compoun
Heat can also be generated by the friction created when a dry r
speed against the dry surface of a tooth. However, this latter m
recommended. Another approach is the use of electronic heat-testi

If the heat test confirms the results of other pulp testing proced
Often a tooth that is sensitive to heat may also be responsible for
present with cold liquids in hand just to minimize the pain (Fig
cold to a specific tooth may eliminate the pain and greatly assi
responds to heat and then is relieved by cold is found to be necr

FIG. 1-19 Irreversible pulpitis associated with the mandibular right second molar. Patient has found that the only way to alleviate the pain is to place a jar filled with ice water against the right side of his face.

Electric

Assessment of pulp neural responses (*vitality*) can also be accomplished by electric pulp testing.[79] Electric pulp testers of different designs and manufacturers have been used for this purpose. Electric pulp testers should be an integral part of any dental practice. It should be noted that the vitality of the pulp is determined by the intactness and health of the vascular supply, not by the status of the pulpal nerve fibers. Even though

BOX 1-4

Potential Common Interpretation Errors of Responses Obtained from Electric Pulp Testing

False-Positive Responses Partial pulp necrosis Patient's high anxiety Ineffective tooth isolation
Contact with metal restorations

False-Negative Responses
Calcific obliterations in the root canals Recently traumatized teeth
Immature apex
Drugs that increase patient's threshold for pain
Poor contact of pulp tester to tooth

advances are being made with regard to determining the vitality of the pulp on the basis of the blood supply, this technology has not been perfected enough at this time to be used on a routine basis in a clinical setting.

The electric pulp tester has some limitations in providing predictable information about the vitality of the pulp. The response of the pulp to electric testing does not reflect the histologic health or disease status of the pulp.[122,123] A response by the pulp to the electric current only denotes that some viable nerve fibers are present in the pulp and are capable of responding. Numeric readings on the pulp tester have significance only if the number differs significantly from the readings obtained from a control tooth tested on the same patient with the electrode positioned at a similar area on both teeth. However, in most cases, the response is scored as either present or absent. Studies[122,123] have shown that electric pulp test results are most accurate when no response is obtained to any amount of electric current. This lack of response has been found most frequently when a necrotic pulp is present. In addition, false-positive and false-negative responses can occur (Box 1-4), and the clinician must take it into account when formulating the final diagnosis.

The electric pulp tester will not work unless the probe can be placed in contact with or be bridged to the natural tooth structure.[95] With the advent of universal precautions for infection control, the use of rubber gloves prevents the clinician from completing the circuit.[7] Some pulp testers may require the patient to place a finger, or fingers, on the tester probe to complete the electric circuit; however, the use of lip clips is an alternative to having patients hold the tester. Proper use of the electric pulp tester requires the evaluated teeth to be carefully isolated and dried. A control tooth of similar tooth type and location in the arch should be tested first in order to establish a baseline response and to inform the patient as to what a "normal" sensation is. The suspected tooth should be tested at least twice to confirm the results. The tip of the testing probe that will be placed in contact with the tooth structure must be coated with a water- or petroleum-based medium.[86] The most commonly used medium is toothpaste. The coated probe tip is placed in the incisal third of the facial or buccal area of the tooth to be tested.[15] Once the probe is in contact with the tooth, the patient is asked to touch or grasp the tester probe, unless a lip clip is used (Fig. 1-20, *A*). This completes the circuit and initiates the delivery of electric current to the tooth.

FIG. 1-20 A, Electric pulp tester with probe. The probe tip will be coated with a conducive medium such as toothpaste and placed in contact with the tooth surface. The patient will activate the unit by placing a finger on the metal shaft of the probe. B, View of the electric pulp tester control panel; the knob on the front right of the unit controls the rate at which the electric current is delivered to the tooth. The plastic panel on the left front displays the digital numerical reading obtained from the pulp test. The digital scale runs from 0 to 80. (Courtesy SybronEndo, Orange, CA.)

The patient is instructed to remove his or her finger(s) from the probe when a "tingling" or "warming" sensation is felt in the tooth. The readings from the pulp tester are recorded (Fig. 1-20, *B*) and will be evaluated once all the appropriate teeth have been tested by the electric pulp tester and the other pulp testing methods.

If a complete coverage crown or extensive restoration is present, a bridging technique can be attempted to deliver the electric current to any exposed natural tooth structure.[95] The tip of an endodontic explorer is coated with toothpaste or other appropriate medium and placed in contact with the natural tooth structure. The tip of the electric pulp tester probe is coated with a small amount of toothpaste and placed in contact with the side of the explorer. The patient completes the circuit and the testing proceeds as described previously. If no natural tooth structure is available, then an alternative pulp testing method, such as cold, should be used.

One study compared the ability of thermal and electric pulp testing methods to register the presence of vital pulp tissue.[99] The *sensitivity,* which is the ability of a test to identify teeth that are diseased, was 0.83 for the cold test, 0.86 for heat test,

and 0.72 for the electric test. This means the cold test correctly identified 83% of the teeth that had a necrotic pulp, whereas heat tests were correct 86% of the time and electric pulp tests were correct only 72% of the time. This same study evaluated the *specificity* of these three tests. Specificity relates to the ability of a test to identify teeth without disease. Ninety-three percent of teeth with healthy pulps were correctly identified by both the cold and electric pulp tests, whereas only 41% of the teeth with healthy pulps were identified correctly by the heat test. From the results of the testing, it was found that the cold test had an accuracy of 86%, the electric pulp test 81%, and the heat test 71%.

Some studies have indicated there might not be a significant difference between pulp testing results obtained by electric pulp tester and those obtained by the thermal methods.[46,98,99] Cold tests, however, have been shown to be more reliable than electric pulp tests in younger patients with less developed root apices.[5,42,98] This is the reason to verify the results obtained by one testing method and compare them with results obtained by other methods. Until such time that the testing methods used to assess the vascular supply of the pulp become less time consuming and technique sensitive, thermal and electric pulp testing will continue to be the primary methods for determining pulp vitality.

Laser Doppler Flowmetry

Laser Doppler flowmetry (LDF) is a method used to assess blood flow in microvascular systems. Attempts are being made to adapt this technology to assess pulpal blood flow. A diode is used to project an infrared light beam through the crown and pulp chamber of a tooth. The infrared light beam is scattered as it passes through the pulp tissue. The Doppler principle states that the light beam's frequency will shift when hitting moving red blood cells but will remain unshifted as it passes through static tissue. The average Doppler frequency shift will measure the velocity at which the red blood cells are moving.[114]

Several studies[40,60,69,84,114,115,117] have found LDF to be an accurate, reliable, and reproducible method of assessing pulpal blood flow. One of the great advantages of pulp testing with devices such as the LDF is that the collected data are based on objective findings rather than subjective patient responses. As is discussed in Chapter 20, certain luxation injuries will cause inaccuracies in the results of electric and thermal pulp testing. LDF has been shown to be a great indicator for pulpal vitality in these cases.[130] This technology, however, is not being used routinely in the dental practice.

Pulse Oximetry

The pulse oximeter is another noninvasive device (Fig. 1-21). Widely used in medicine, it is designed to measure the oxygen concentration in the blood and the pulse rate. A pulse oximeter works by transmitting two wavelengths of light, red and infrared, through a translucent portion of a patient's body (e.g., a finger, earlobe, or tooth). Some of the light is absorbed as it passes through the tissue; the amount absorbed depends on the ratio of oxygenated to deoxygenated hemoglobin in the blood. On the opposite side of the targeted tissue, a sensor detects the absorbed light. On the basis of the difference between the light emitted and the light received, a microprocessor calculates the pulse rate and oxygen concentration in the blood.[118] The transmission of light to the sensor requires that there be no

FIG. 1-21 Nellcor OxiMax N-600x pulse oximeter. (Courtesy Nellcor Puritan Bennett, Boulder, CO; now part of Covidien.)

obstruction from restorations, which can sometimes limit the usefulness of pulse oximetry to test pulp vitality.

Custom-made sensors have been developed and were found to be more accurate than electric and thermal pulp tests.[31,54] This sensor has been especially useful in evaluating teeth that have been subjected to traumatic injuries, as such teeth tend to present, especially in the short term, with questionable vitality using conventional pulp testing methods.[8,31,53]

Studies regarding the ability of pulse oximetry to diagnose pulp vitality draw various conclusions. Several studies have found pulse oximetry to be a reliable method for assessing pulp vitality.[69,70,118,125,140] Others have stated that in its present form the pulse oximeter may not be predictable in diagnosing pulp vitality.[140] Most of the problems appear to be related to the currently available technology. Some investigators have concluded that the devices used for pulp testing are too cumbersome and complicated to be used on a routine basis in a dental practice.[68,118,140]

Special Tests

Bite Test

Percussion and bite tests are indicated when a patient presents with pain while biting. On occasion, the patient may not know which tooth is sensitive to biting pressure, and percussion and bite tests may help to localize the tooth involved. The tooth may be sensitive to biting when the pulpal pathosis has extended into the periodontal ligament space, creating a *symptomatic apical periodontitis,* or the sensitivity may be present secondary to a crack in the tooth. The clinician can often differentiate between periradicular periodontitis and a cracked tooth or fractured cusp. If periradicular periodontitis is present, the tooth will respond with pain to percussion and biting tests regardless of where the pressure is applied to the coronal part of the tooth. A cracked tooth or fractured cusp will typically elicit pain only when the percussion or bite test is applied in a certain direction to one cusp or section of the tooth.[22,108]

For the bite test to be meaningful, a device should be used that will allow the clinician to apply pressure to individual cusps or areas of the tooth. A variety of devices have been used for bite tests, including cotton tip applicators, toothpicks, orangewood sticks, and rubber polishing wheels. There are several devices specifically designed to perform this test. The Tooth Slooth (Professional Results, Laguna Niguel, CA) (Fig. 1-22) and FracFinder (Hu-Friedy, Oakbrook, IL) are just two of the commercially available devices used for the bite test. As with all pulp tests, adjacent and contralateral teeth should

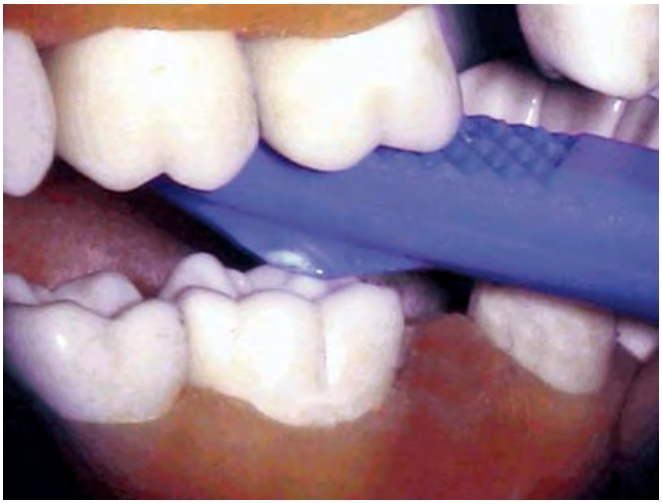


FIG. 1-22 To determine which tooth, or tooth part, is sensitive to mastication, having the patient bite on a specially designed bite stick is often helpful.

be used as controls so that the patient is aware of the "normal" response to these tests. The small cupped-out area on these instruments is placed in contact with the cusp to be tested. The patient is then asked to apply biting pressure with the opposing teeth to the flat surface on the opposite side of the device. The biting pressure should be applied slowly until full closure is achieved. The firm pressure should be applied for a few seconds; the patient is then asked to release the pressure quickly. Each individual cusp on a tooth can be tested in a like manner. The clinician should note whether the pain is elicited during the pressure phase or on quick release of the pressure. A common finding with a fractured cusp or cracked tooth is the frequent presence of pain upon release of biting pressure.

Test Cavity

The test cavity method for assessing pulp vitality is not routinely used since, by definition, it is an invasive irreversible test. This method is used only when all other test methods are deemed impossible or the results of the other tests are inconclusive. An example of a situation in which this method can be used is when the tooth suspected of having pulpal disease has a full coverage crown. If no sound tooth structure is available to use a bridging technique with the electric pulp tester and cold test results are inconclusive, a small class I cavity preparation is made through the occlusal surface of the crown. This is accomplished with a high-speed #1 or #2 round bur with proper air and water coolant. The patient is not anesthetized while this procedure is performed, and the patient is asked to respond if any painful sensation is felt during the drilling procedure. If the patient feels pain once the bur contacts sound dentin, the procedure is terminated and the class I cavity preparation is restored. This sensation signifies only that there is some viable nerve tissue remaining in the pulp, not that the pulp is totally healthy. If the patient fails to feel any sensation when the bur reaches the dentin, this is a good indication that the pulp is necrotic and root canal therapy is indicated.

Staining and Transillumination

To determine the presence of a crack in the surface of a tooth, the application of a stain to the area is often of great assistance. It may be necessary to remove the restoration in the tooth to better visualize a crack or fracture. Methylene blue dye, when painted on the tooth surface with a cotton tip applicator, will penetrate into cracked areas. The excess dye may be removed with a moist application of 70% isopropyl alcohol. The dye will indicate the possible location of the crack.

Transillumination using a bright fiberoptic light probe to the surface of the tooth may be very helpful (Fig. 1-23). Directing a high-intensity light directly on the exterior surface of the tooth at the cementum-enamel junction (CEJ) may reveal the extent of the fracture. Teeth with fractures block transilluminated light. The part of the tooth that is proximal to the light source will absorb this light and glow, whereas the area beyond this fracture will not have light transmitted to it and will show as gray by comparison.[101] Although the presence of a fracture may be evident using dyes and transillumination, the depth of the fracture cannot always be determined.

Selective Anesthesia

When symptoms are not localized or referred, the diagnosis may be challenging. Sometimes the patient may not even be able to specify whether the symptoms are emanating from the maxillary or mandibular arch. In these instances, when pulp testing is inconclusive, *selective anesthesia* may be helpful.

If the patient cannot determine which arch the pain is coming from, then the clinician should first selectively anesthetize the maxillary arch. This should be accomplished by using a periodontal ligament (intraligamentary) injection. The injection is administered to the most posterior tooth in the quadrant of the arch that may be suspected, starting from the distal sulcus. The anesthesia is subsequently administered in an anterior direction, one tooth at a time, until the pain is eliminated. If the pain is not eliminated after an appropriate period of time, then the clinician should similarly repeat this technique on the mandibular teeth below. It should be understood that periodontal ligament injections may anesthetize an adjacent tooth and thus are more useful for identifying the arch rather than the specific tooth.

Radiographic Examination and Interpretation

Intraoral Radiographs

The radiographic interpretation of a potential endodontic pathosis is an integral part of endodontic diagnosis and prognosis assessment. Few diagnostic tests provide as much useful information as dental radiography. For this reason, the clinician is sometimes tempted to prematurely make a definitive diagnosis based solely on radiographic interpretation. However, the image should be used only as one sign, providing important clues in the diagnostic investigation. When not coupled with a proper history and clinical examination and testing, the radiograph alone can lead to a misinterpretation of normality and pathosis (Fig. 1-24). Because treatment planning will ultimately be based on the diagnosis, the potential for inappropriate treatment may frequently exist if the radiograph alone is used for making final diagnosis. The clinician should not subject the patient to unnecessary multiple radiation exposures; two pretreatment images from different angulations are often sufficient. Under extenuating circumstances, however, especially when the diagnosis is difficult, additional exposures may be necessary to determine the presence of multiple roots,

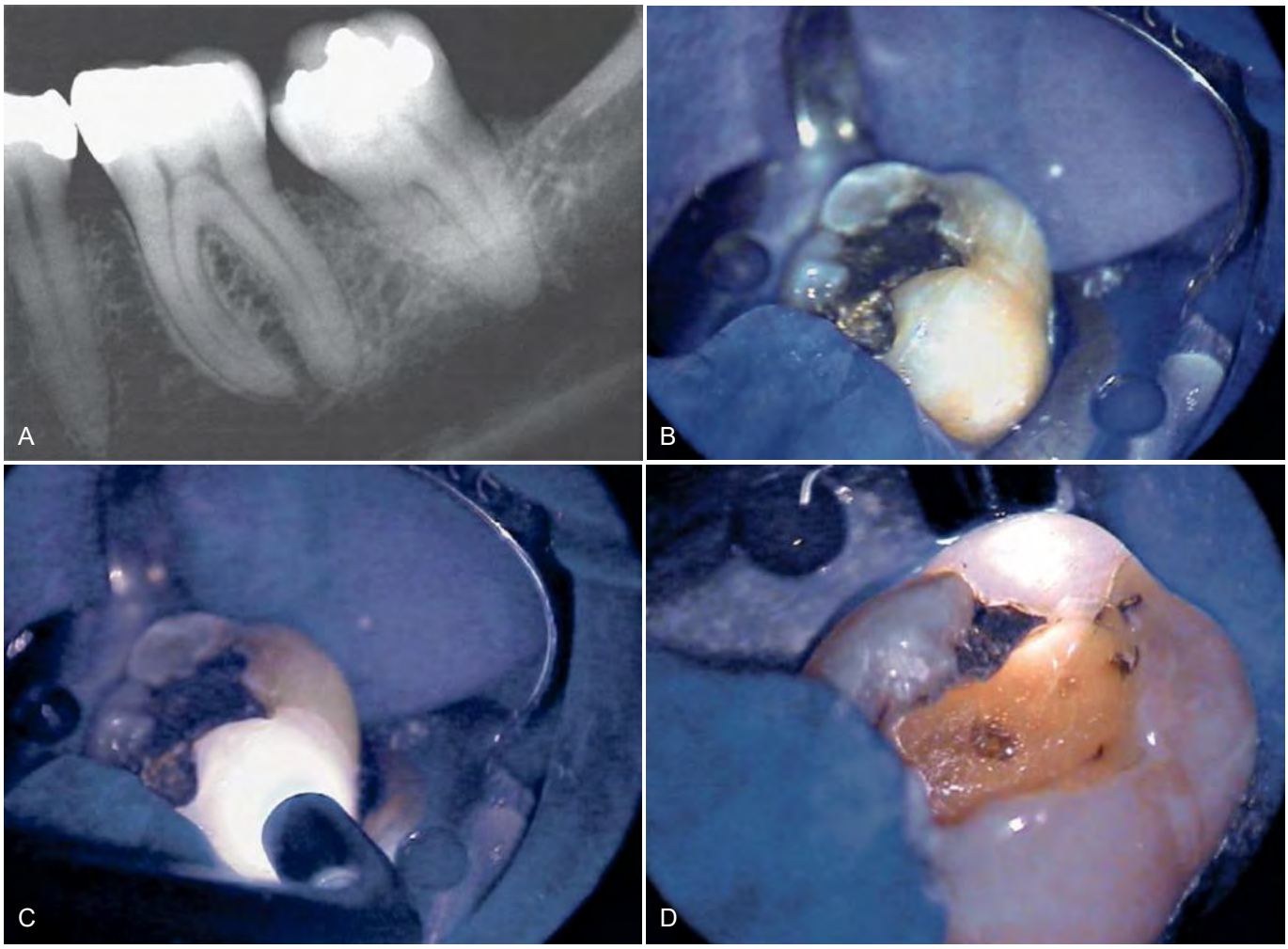


FIG. 1-23 Sometimes there is no clear indication of why a tooth is symptomatic. This radiograph shows a mandibular second molar with a moderately deep restoration (A); the pulp tests nonvital. Without any transillumination, a fracture cannot be detected (B). However, by placing a high-intensity light source on the tooth surface, a root fracture can be observed on the buccal surface (C) and the distal-lingual surface (D).

multiple canals, resorptive defects, caries, restoration defects, root fractures, and the extent of root maturation and apical development.

The radiographic appearance of endodontic pathosis can sometimes be highly subjective. In a study by Goldman and colleagues, there was only 50% agreement among interpreters for the radiographic presence of pathosis.[49] When the cases were reevaluated several months later, the same evaluators agreed with their own original diagnosis less than 85% of the time.[50] This further emphasizes the necessity for additional objective diagnostic tests, as well as the importance of obtaining and comparing older radiographs.

For standard two-dimensional radiography, clinicians basically project x-radiation through an object and capture the image on a recording medium, either x-ray film or a digital sensor. Much like casting a shadow from a light source, the image appearance may vary greatly depending on how the radiographic source is directed. Thus, the three-dimensional interpretation of the resulting two-dimensional image requires not only knowledge of normality and pathosis but also advanced knowledge of how the radiograph was exposed. By virtue of "casting a shadow," the anatomic features that are closest to the film (or sensor) will move the least when there is a change in the horizontal or vertical angulation of the radiation source (Fig. 1-25). This may be helpful in determining the existence of additional roots, the location of pathosis, and the unmasking of anatomic structures. Changes in the horizontal or vertical angulation may help elucidate valuable anatomic and pathologic information; it also has the potential to hide important information. An incorrect vertical angulation may cause the buccal roots of a maxillary molar to be masked by the zygomatic arch. An incorrect horizontal angulation may cause roots to overlap with the roots of adjacent teeth, or it may incorrectly create the appearance of a one-rooted tooth, when two roots are actually present.

In general, when endodontic pathosis appears radiographically, it appears as bone loss in the area of the periapex. The pathosis may present merely as a widening or break in the lamina dura—the most consistent radiographic finding when a tooth is nonvital[67]—or it may present as a radiolucent area at the apex of the root or in the alveolar bone adjacent to the exit of a lateral or furcation accessory canal. On occasion there may be no radiographic change at all, even in the presence of a disease process in the alveolar bone.

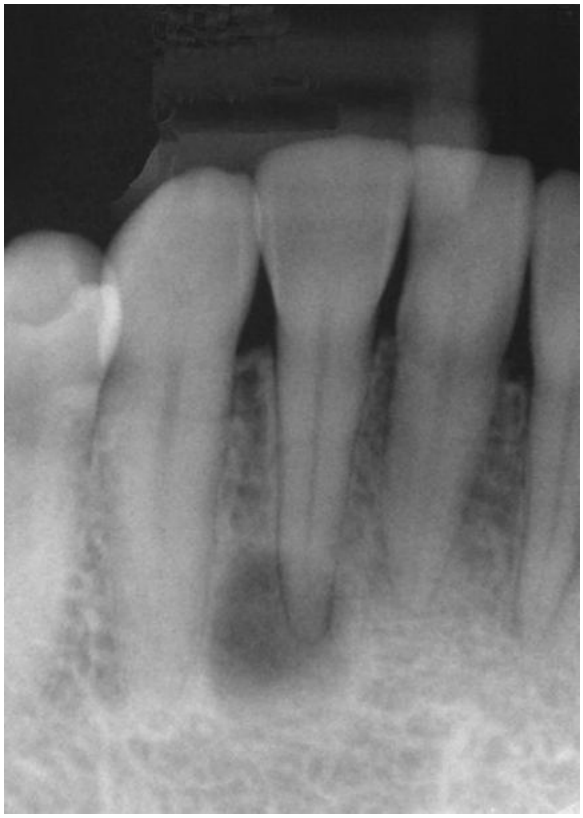


FIG. 1-24 Radiograph showing what appears to be a mandibular lateral incisor associated with periapical lesion of a nonvital tooth. Although pulp necrosis can be suspected, the tooth tested vital. In this case, the appearance of apical bone loss is secondary to a cementoma.

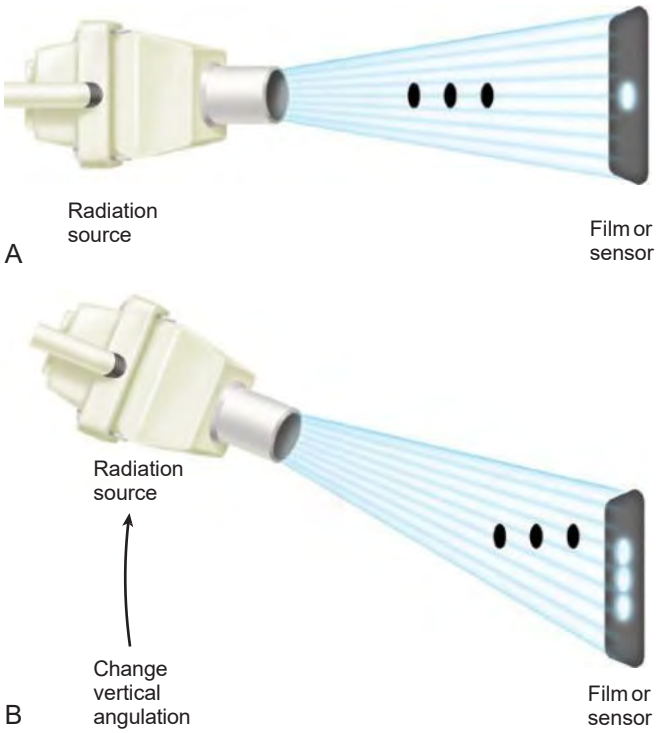


FIG. 1-25 Radiographic images are only two-dimensional, and often it is difficult to discriminate the relative location of overlapping objects. A, When the source of radiation is directly perpendicular to overlapping objects, the image is captured without much separation of the objects. However, when the radiation source is at an angle to offset the overlapping objects, the image is captured with the objects being viewed as separated. B, The object that is closest to the film (or sensor) will move the least, with the object closest to the radiation source appearing farthest away.

Two-dimensional dental radiography has two basic shortcomings: the lack of early detection of pathosis in the cancellous bone, because of the density of the cortical plates, and the influence of the superimposition of anatomic structures. Variability in the radiographic expression of an osseous pathosis has much to do with the relative location of the root of the tooth and how it is oriented with respect to the cortical and cancellous bone. Radiographic changes from bone loss will not be detected if the loss is only in cancellous bone.[16] However, the radiographic evidence of pathosis will be observed once this bone loss extends to the junction of the cortical and cancellous bone. In addition, certain teeth are more prone to exhibit radiographic changes than others, depending on their anatomic location.[17] The radiographic appearance of endodontic pathosis is correlated with the relationship of the periapex of the tooth and its juxtaposition to the cortical-cancellous bone junction. The apices of most anterior and premolar teeth are located close to the cortical-cancellous bone junction. Therefore, periapical pathosis from these teeth is exhibited sooner on the radiograph. By comparison, the distal roots of mandibular first molars and both roots of mandibular second molars are generally positioned more centrally within the cancellous bone, as are maxillary molars, especially the palatal roots. Periapical lesions from these roots must expand more before they reach the cortical-cancellous bone junction and are recognized as radiographic pathosis. For these reasons, it is important not to exclude the possibility of pulpal pathosis in situations in which there are no radiographic changes.

Many factors can influence the quality of the radiographic interpretation, including the ability of the person exposing the radiograph, the quality of the radiographic film, the quality of the exposure source, the quality of the film processing, and the skill with which the film is viewed. Controlling all of these variables can be a difficult challenge but is paramount for obtaining an accurate radiographic interpretation.

Digital Radiography

Digital radiography has been available since the late 1980s and has recently been refined with better hardware and more user-friendly software. It has the ability to capture, view, magnify, enhance, and store radiographic images in an easily reproducible format that does not degrade over time. Significant advantages of digital radiographs over conventional radiographs include lower radiation doses, instant viewing, convenient manipulation, efficient transmission of an image via the Internet, simple duplication; and easy archiving.

Digital radiography uses no x-ray film and requires no chemical processing. Instead, a *sensor* is used to capture the image created by the radiation source. This sensor is either directly or wirelessly attached to a local computer, which interprets this signal and, using specialized software, translates the signal into a two-dimensional digital image that can be displayed, enhanced, and analyzed. The image is stored in the patient's file, typically in a dedicated network server, and can be recalled as needed. Further information about digital radiography may be found in Chapter 2.

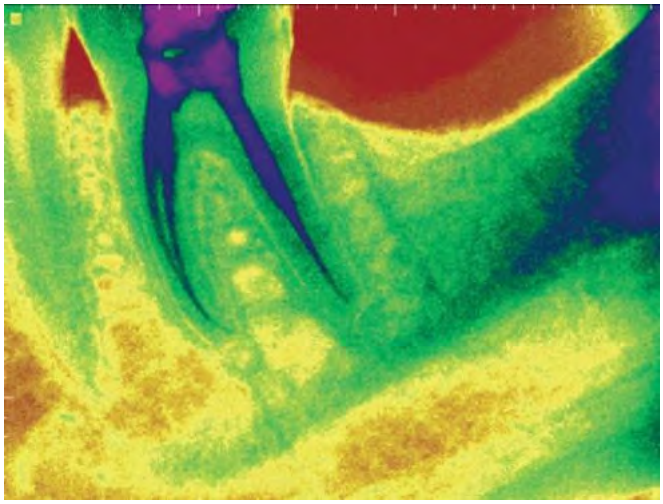


FIG. 1-26 Digital radiography has an advantage over conventional film in that the image can be enhanced and colorized, a useful tool for patient education.

The viewing of a digital radiographic image on a high-resolution monitor allows for rapid and easy interpretation for both the clinician and the patient. The image appears almost instantly, with no potential for image distortion from improper chemical processing. The clinician can magnify different areas on the radiograph and then digitally enhance the image in order to better visualize certain anatomic structures; in some cases the image can even be colorized, a useful tool for patient education (Fig. 1-26).

In the past, x-ray film has had a slightly better resolution than most digital radiography images, at about 16 line pairs per millimeter (lp/mm).[87] Some sensor manufacturers, however, now claim to offer resolutions beyond that of conventional film. Under the best of circumstances, the human eye can see only about 10 lp/mm, which is the lowest resolution for most dental digital radiography systems. Digital sensors are much more sensitive to radiation than conventional x-ray film and thus require 50% to 90% less radiation in order to acquire an image, an important feature for generating greater patient acceptance of dental radiographs.

The diagnostic quality of this expensive technology has been shown to be comparable to, but not necessarily superior to, perfectly exposed and perfectly processed conventional film-based radiography.[39,73,97] Furthermore, it was found that the interpretation of a digital radiograph can be subjective, similar to that of the conventional film.[134] Factors that appear to have the most impact on the interpretation of the image are the years of experience of the examiner and familiarity of the operator with the given digital system.[134]

Cone-Beam Computerized Tomography

Limitations in conventional two-dimensional radiography promulgated a need for three-dimensional imaging, known as *cone-beam computerized tomography* (CBCT) (also known as *cone-beam volumetric tomography* [CBVT]) or as *cone-beam volumetric imaging* [CBVI]. Although a form of this technology has existed since the early 1980s,[106] specific devices for dental use first appeared almost two decades later.[90] Most of these machines are similar to a dental panoramic radiographic device, whereby the patient stands or sits as a cone-shaped

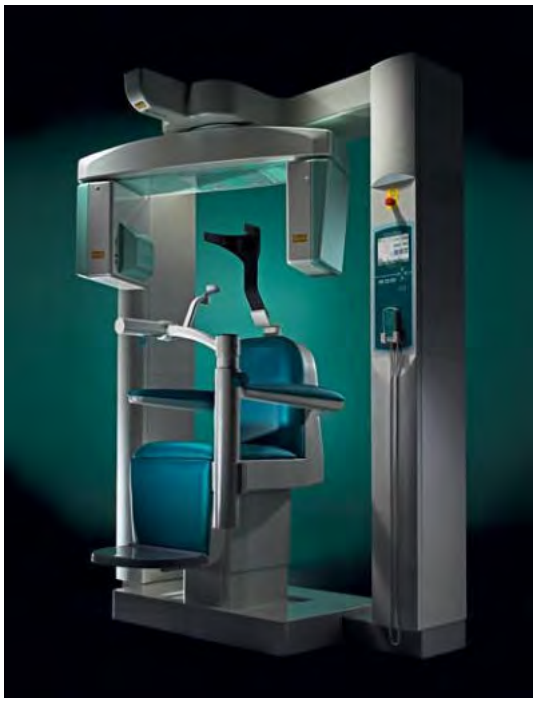


FIG. 1-27 Cone-beam volumetric tomography, using the 3D Accuitomo 80. (Courtesy J. Morita USA, Irvine, CA.)

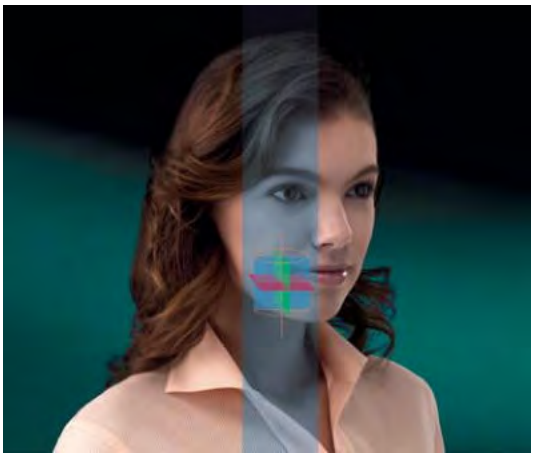


FIG. 1-28 Cone-beam volumetric tomography has the ability to capture, store, and present radiographic images in various horizontal and vertical planes. (Courtesy J. Morita USA, Irvine, CA.)

radiographic beam is directed to the target area with a reciprocating capturing sensor on the opposite side (Fig. 1-27). The resulting information is digitally reconstructed and interpreted to create an interface whereby the clinician can three-dimensionally interpret "slices" of the patient's tissues in a multitude of planes (Figs. 1-28 and 1-29).[37,33] The survey of the scans can be interpreted immediately after the scan. Various software applications have been used to enable the images to be sent to other clinicians. This is accomplished either in printed format or with portable and transferable software that can be used interactively by another clinician.

In general, many dental applications only require a limited field of vision, confining the study to the maxilla and mandible.

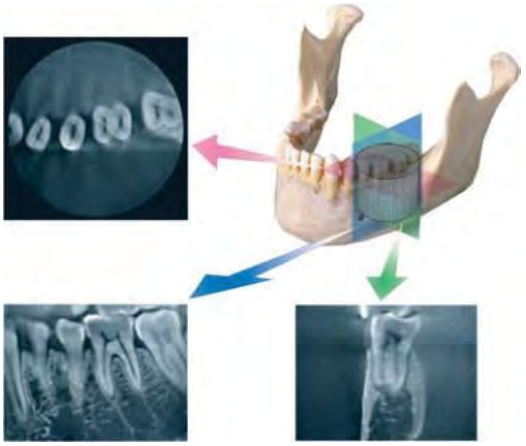


FIG. 1-29 Cone-beam volumetric tomography has the advantage of being able to detect pathosis in the bone or associated with the teeth without the obstruction of anatomic structures. The planes of vision may be axial, sagittal, or coronal. (Courtesy J. Morita USA, Irvine, CA.)

However, many devices have the ability to provide a full field of vision for viewing more regional structures. Clinicians should thoroughly understand the ethical and medical-legal ramifications of doing scans with full fields of view. Incidental nondental findings have been seen from these scans, such as intracranial aneurysms, that when undetected could be life threatening.[91]

The radiation source of CBCT is different from that of conventional two-dimensional dental imaging in that the radiation beam created is conical in shape. Also, conventional digital dental radiography is captured and interpreted as *pixels,* a series of dots that collectively produces an image of the scanned structure. For CBCT, the image is instead captured as a series of three-dimensional pixels, known as *voxels.* Combining these voxels gives a three-dimensional image that can be "sliced" into various planes, allowing for specific evaluations never before possible without a necropsy (Fig. 1-30). One of the advantages of using a device that has a limited field of vision is that the voxel size can be less than half that of a device using a full field of vision, thereby increasing the resolution of the resulting image and providing for a more accurate interpretation of anatomic structures and pathologic conditions. The development of limited field of vision devices has also contributed to decreasing the costs of these relatively expensive machines, making them more practical for dental office use.[41]

Compared with two-dimensional radiographs, CBCT can clearly visualize the interior of the cancellous bone without the superimposition of the cortical bone. Studies show that CBCT is much more predictable and efficient in demonstrating anatomic landmarks, bone density, bone loss, periapical lesions, root fractures, root perforations and root resorptions.[1,21,26,27,38,47,71,78,81,85,92,94,128,131,142]

The superimposition of anatomic structures can also mask the interpretation of alveolar defects. Specifically, the maxillary sinus, zygoma, incisive canal and foramen, nasal bone, orbit, mandibular oblique ridge, mental foramen, mandibular mentalis, sublingual salivary glands, tori, and the overlap of adjacent roots may either obscure bone loss or mimic bone loss, making an accurate interpretation of conventional radiography sometimes difficult or impossible. Several studies have

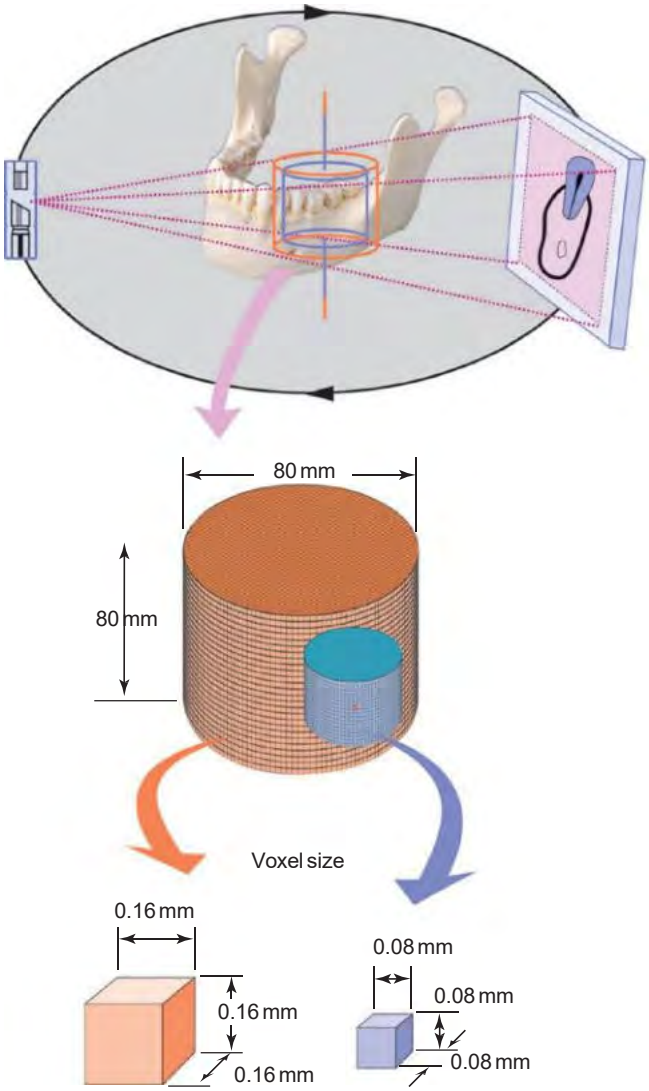


FIG. 1-30 The radiation source in cone-beam volumetric tomography is conical. The receiving sensor captures the image as "voxels," or three-dimensional pixels of information, allowing digital interpretation.

demonstrated the advantages of CBCT in the differential diagnosis of such structures from pathologic conditions.[21,29,71,137]

Cone-beam computerized tomography should not be seen as a replacement for conventional dental radiography, but rather as a diagnostic adjunct. The advantage of conventional dental radiography is that it can visualize most of the structures in one image. CBCT can show great detail in many planes of vision but can also leave out important details if the "slice" is not in the area of existing pathosis (Fig. 1-31). There is a promising future for the use of CBCT for endodontic diagnosis and treatment. It has already proven invaluable in the detection of dental and nondental pathoses (Fig. 1-32). For a further review of CBCT and radiography, see Chapter 2.

Magnetic Resonance Imaging (MRI)

MRI has also been suggested for dental diagnosis. It may offer simultaneous three-dimensional hard- and soft-tissue imaging of teeth without ionizing radiation.[58] The use of MRI in endodontics is still limited.

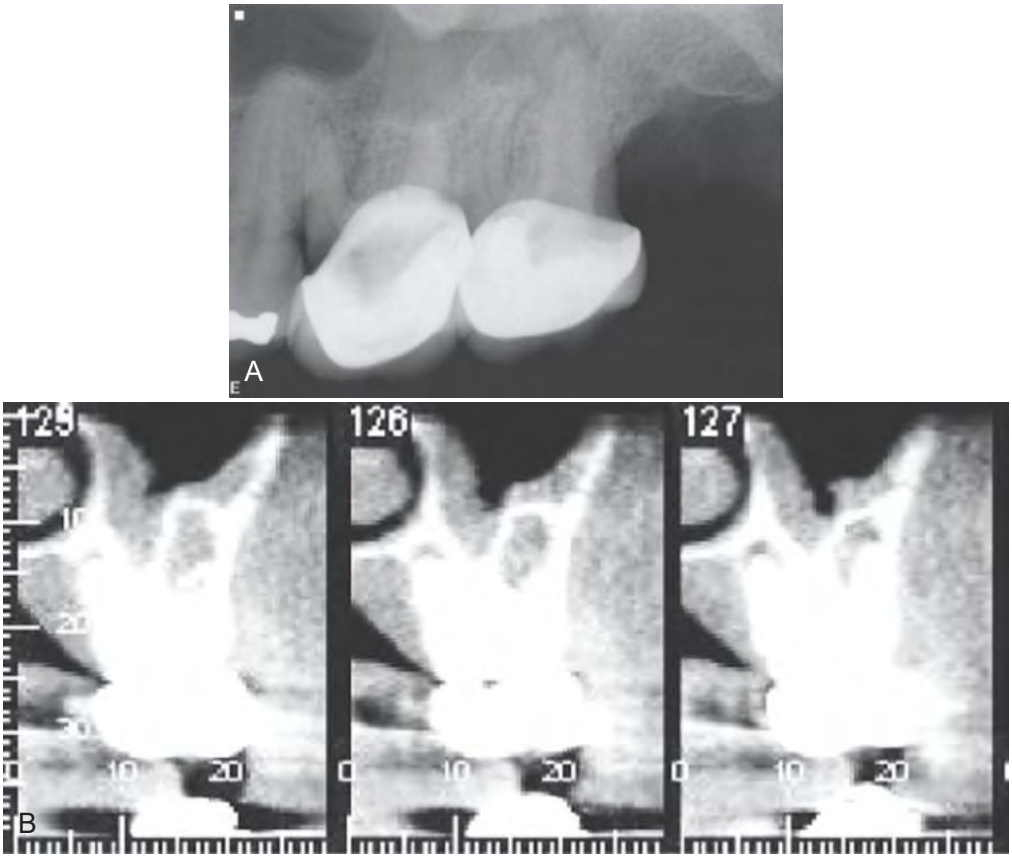


FIG. 1-31 A, This standard two-dimensional radiographic image reveals recurrent caries under the mesial margin of the maxillary first molar. However, the patient localized pain to mastication on the maxillary second molar. B, Cone-beam volumetric tomography revealed an apical radiolucency associated with the maxillary second molar. The bone loss was obscured on the two-dimensional radiograph by the maxillary sinus, zygoma, and cortical bone.

Cracks and Fractures

The wide variety of types of cracks and fractures in teeth and their associated signs and symptoms often make their diagnosis difficult. The extensiveness of the crack or fracture line may directly alter the prognosis assessment for a given tooth and should be examined before treatment decision making. Certain types of cracks may be as innocent as a superficial enamel craze line, or they may be as prominent as a fractured cusp. The crack may progress into the root system to involve the pulp, or it may split the entire tooth into two separate segments. The crack may be oblique, extending cervically, such that once the coronal segment is removed the tooth may or may not be restorable. Any of these situations may present with mild, moderate, or severe symptoms or possibly no symptoms at all.

Crack Types

There have been many suggestions in the literature of was to classify cracks in teeth. By defining the type of crack present, an assessment of the prognosis may be determined and treatment alternatives can be planned (see Chapter 21). Unfortunately, it is often extremely difficult to determine how extensive a crack is until the tooth is extracted.

Cracks in teeth can be divided into three basic categories:

- Craze lines
- Fractures (also referred to as *cracks*)
- Split tooth/roots

Craze lines are merely cracks in the enamel that do not extend into the dentin and either occur naturally or develop after trauma. They are more prevalent in adult teeth and usually occur more in the posterior teeth. If light is transilluminated through the crown of such a tooth, these craze lines may show up as fine lines in the enamel with light being able to transmit through them, indicating that the crack is only superficial. The use of optical coherence tomography (OCT) has also been suggested for detection of enamel cracks.[59] Craze lines typically will not manifest with symptoms. No treatment is necessary for craze lines unless they create a cosmetic issue.

Fractures extend deeper into the dentin than superficial craze lines and primarily extend mesially to distally, involving the marginal ridges. Dyes and transillumination are helpful for visualizing potential root fractures.

Symptoms from a fractured tooth range from none to severe pain. A fracture in the tooth does not necessarily dictate that the tooth has split into two pieces; however, left alone or especially with provocations such as occlusal prematurities, the fracture may progress into a split root. A fractured tooth may be treated by a simple restoration, endodontics (nonsurgical or surgical), or even extraction, depending on the extent and orientation of the fracture, the degree of symptoms, and whether the symptoms can be eliminated. This makes the clinical management of fractured teeth difficult and sometimes unpredictable.

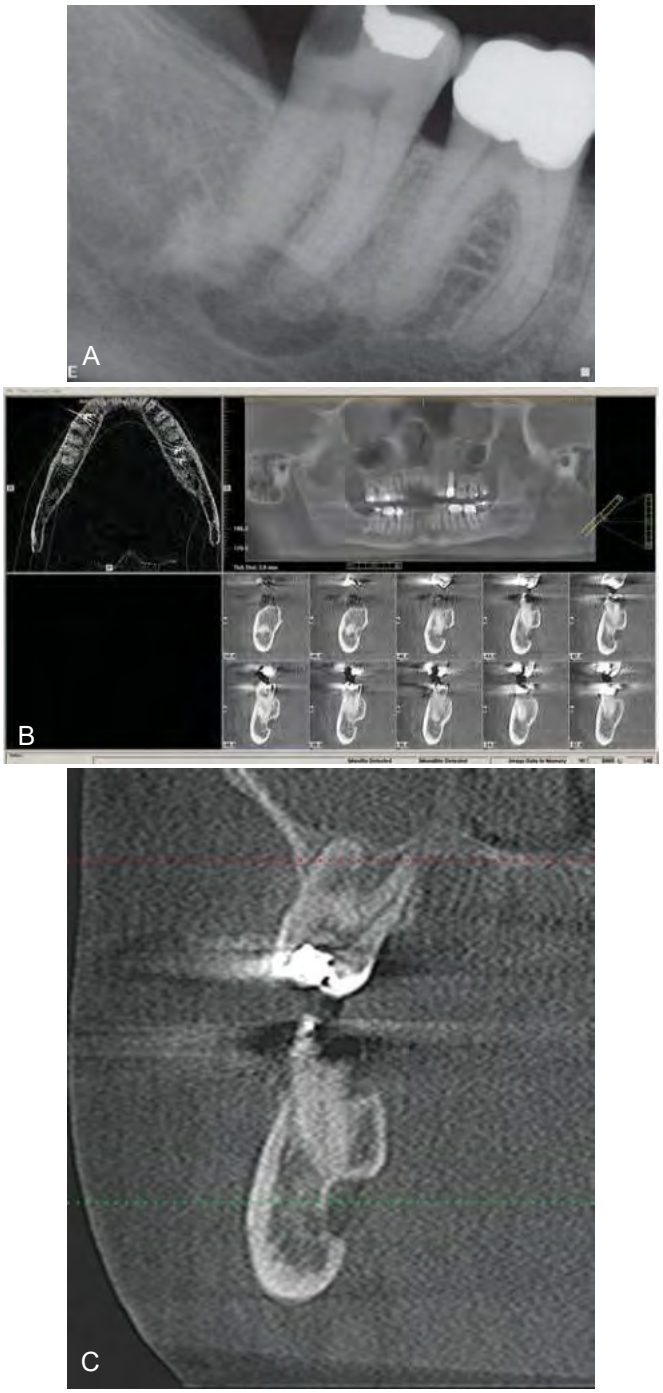


FIG. 1-32 A, Periapical radiograph showing a large apical radiolucency associated with the mandibular second molar. Apical pathosis should be ruled out. B, Cone-beam volumetric tomography revealed salivary indentation of the mandible in the area apical and lingual to the mandibular second molar, consistent with a Stafne defect. C, Enlargement of coronal section in the area of the mandibular second molar and the Stafne defect located on the lingual aspect of the mandible.

A definitive combination of factors, signs, and symptoms that, when collectively observed, allows the clinician to conclude the existence of a specific disease state is termed a *syndrome*. However, given the multitude of signs and symptoms that fractured roots can present with, it is often difficult to achieve an objective definitive diagnosis. For this reason, the terminology of *cracked tooth syndrome*[22,108] should be avoided.[6] The subjective and objective factors seen in cases of fractured teeth will generally be diverse; therefore, a tentative diagnosis of a fractured tooth will most likely be more of a prediction. Once this prediction is made, the patient must be properly informed as to any potential decrease in prognosis of the pending dental treatment. Because treatment options for repairing fractured teeth have only a limited degree of success, early detection and prevention, and proper informed consent, are crucial.[9,10,72,119,120,124,132]

Split tooth/roots occur when a fracture extends from one surface of the tooth to another surface of the tooth, with the tooth separating into two segments. If the split is more oblique, it is possible that once the smaller separated segment is removed, the tooth might still be restorable—for example, a fractured cusp. However, if the split extends below the osseous level, the tooth may not be restorable and endodontic treatment may not result in a favorable prognosis.

Proper prognosis assessment is imperative before any dental treatment but is often difficult in cases of cracked teeth. Because of the questionable long-term success from treating cases of suspected or known fractures, the clinician should be cautious in making the decision to continue with treatment and should avoid endodontic treatment in cases of a definitive diagnosis of split roots.

Vertical Root Fractures

One of the more common reasons for recurrent endodontic pathosis is the *vertical root fracture,* a severe crack in the tooth that extends longitudinally down the long axis of the root (Figs. 1-33 and 1-34). Often it extends through the pulp and to the periodontium. It tends to be more centrally located within the tooth, as opposed to being more oblique, and typically traverses through the marginal ridges. These fractures may be present before endodontic treatment, secondary to endodontic treatment, or may develop after endodontic treatment has been completed. Because diagnosing these vertical root fractures may be difficult, they often go unrecognized. Therefore, diagnosing the existence and extent of a vertical root fracture is imperative before any restorative or endodontic treatment is done, as it can dramatically affect the overall success of treatment.

A patient who consents to endodontic treatment must be informed if the tooth has a questionable prognosis. The clinician must be able to interpret the subjective and objective findings that suggest a vertical root fracture or split tooth, be able to make a prediction as to the eventual potential of healing, and convey this information to the patient. A more detailed discussion on vertical root fractures is described in Chapter 21.

Perforations

Root perforations are clinical complications that may lead to treatment failure. When root perforation occurs, communications between the root canal system and either periradicular tissues or the oral cavity may reduce the prognosis of treatment. Root perforations may result from extensive carious lesions, resorption, or operator error occurring during root canal instrumentation or post preparation.

The treatment prognosis of root perforations depends on the size, location, time of diagnosis and treatment, degree of periodontal damage, as well as the sealing ability and biocompatibility of the repair material.[45] It has been recognized that

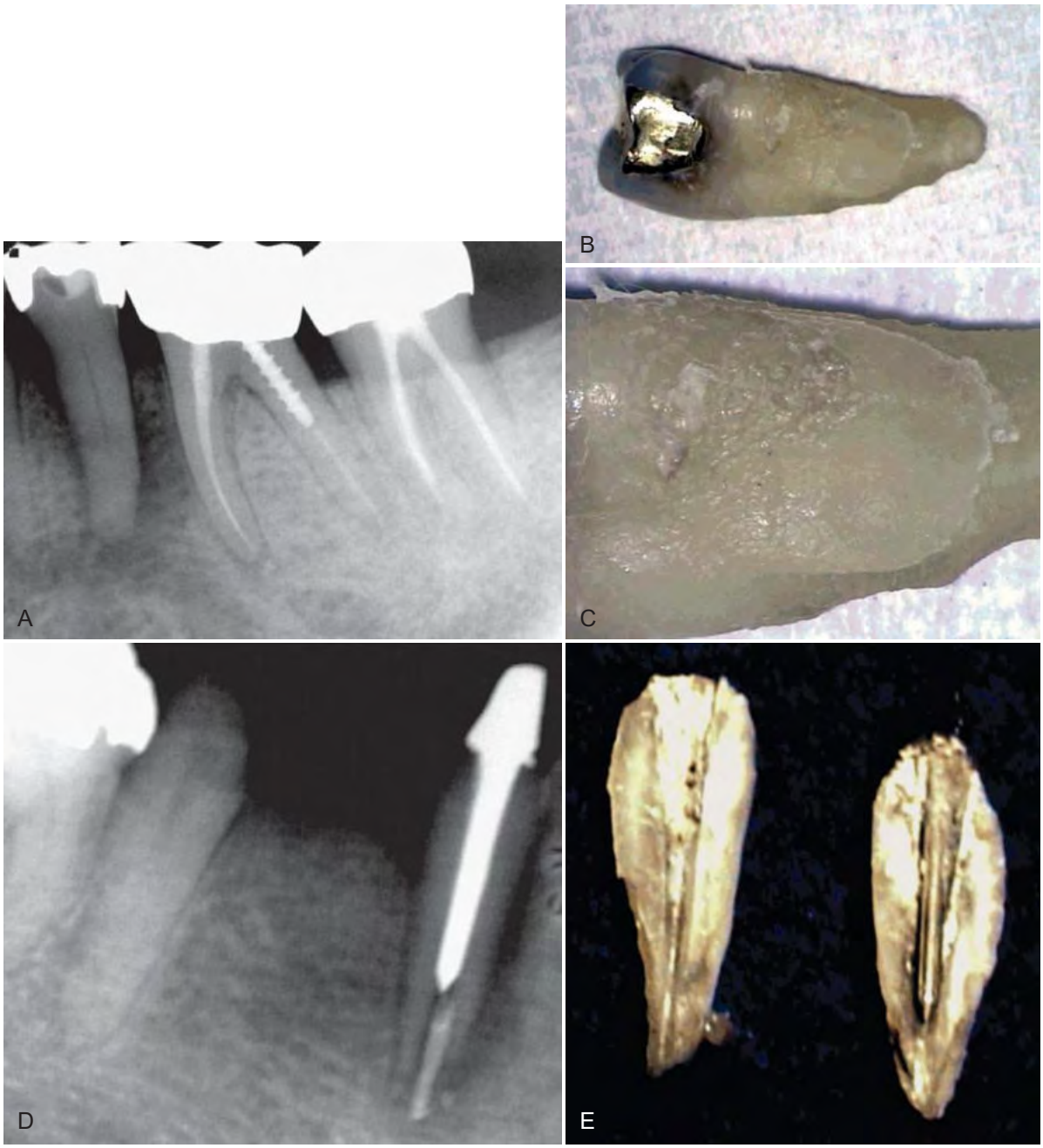


FIG. 1-33 Poorly fitting intracoronal restorations can place stresses within the tooth that can cause a vertical root fracture. A, This radiograph of a mandibular second premolar (with a gold inlay) reveals extensive periapical and periradicular bone loss, especially on the distal aspect. B, The tooth pulp tested nonvital, and there was an associated 12-mm-deep, narrow, isolated periodontal pocket on the buccal aspect of the tooth. After the tooth was extracted, the distal aspect was examined. C, On magnification (×16) the distal aspect of the root revealed an oblique vertical root fracture. Similarly, the placement of an ill-fitting post may exert intraradicular stresses on a root that can cause a fracture to occur vertically. D, This radiograph depicts a symmetrical space between the obturation and the canal wall, suggesting a vertical root fracture. E, After the tooth is extracted, the root fracture can be easily observed.

treatment success depends mainly on immediate sealing of the perforation and appropriate infection control. Among the materials that are commonly used to seal root perforations are mineral trioxide aggregate (MTA), Super EBA, intermediate restorative material (IRM), glass ionomer cements, and composites. The topic of perforations is further discussed in Chapter 19.

CLINICAL CLASSIFICATION OF PULPAL AND PERIAPICAL DISEASES

Many attempts have been made over the years to develop classifications of pulpal and periapical disease. However, studies have shown that making a correlation between clinical signs and symptoms and the histopathology of a given clinical

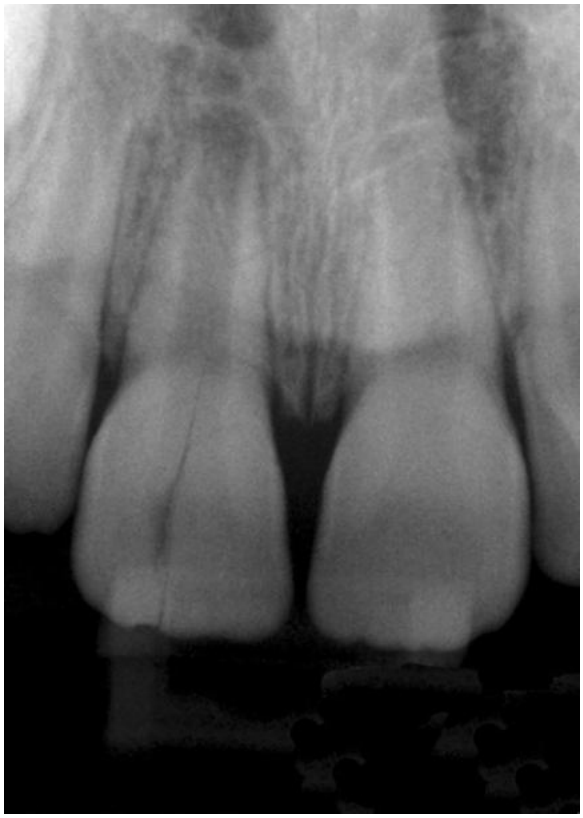


FIG. 1-34 Physical trauma from sports-related injuries or seizure-induced trauma, if directed accordingly, may cause a vertical root fracture in a tooth. This fracture occurred in a 7-year-old child secondary to trauma from a grand mal seizure.

condition is challenging.[122,123] Therefore, *clinical* classifications have been developed in order to formulate treatment plan options. In the most general terms, the objective and subjective findings are used to classify the suspected pathosis, with the assigned designations merely representing the presence of healthy or diseased tissue.

The terminology and classifications that follow are based on those suggested by the American Association of Endodontists in 2012.[6]

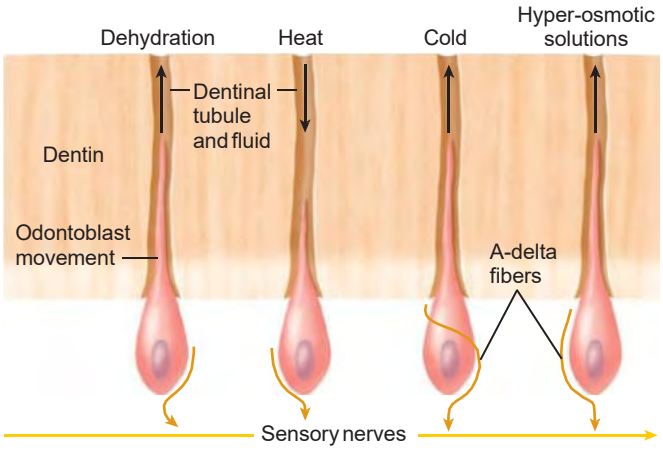
Pulpal Disease

Normal Pulp

This is a clinical diagnostic category in which the pulp is symptom-free and normally responsive to pulp testing.[6] Teeth with normal pulp do not usually exhibit any spontaneous symptoms. The symptoms produced from pulp tests are mild, do not cause the patient distress, and result in a transient sensation that resolves in seconds. Radiographically, there may be varying degrees of pulpal calcification but no evidence of resorption, caries, or mechanical pulp exposure. No endodontic treatment is indicated for these teeth.

Pulpitis

This is a clinical and histologic term denoting inflammation of the dental pulp, clinically described as reversible or irreversible and histologically described as acute, chronic, or hyperplastic.[6]



Dentin tubule fluid movement

FIG. 1-35 Dentinal tubules are filled with fluid that, when stimulated, will cause sensation. Temperature changes, air, and osmotic changes can provoke the odontoblastic process to induce the stimulation of underlying A-delta fibers.

Reversible Pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the inflammation should resolve and the pulp return to normal.[6] When the pulp within the tooth is irritated so that the stimulation is uncomfortable to the patient but reverses quickly after irritation, it is classified as *reversible pulpitis*. Causative factors include caries, exposed dentin, recent dental treatment, and defective restorations. Conservative removal of the irritant will resolve the symptoms. Confusion can occur when there is exposed dentin, without evidence of pulp pathosis, which can sometimes respond with sharp, quickly reversible pain when subjected to thermal, evaporative, tactile, mechanical, osmotic, or chemical stimuli. This is known as *dentin* (or *dentinal*) *sensitivity* (or *hypersensitivity*). Exposed dentin in the cervical area of the tooth accounts for most of the cases diagnosed as dentin sensitivity.[103]

As described in Chapter 12, fluid movement within dentinal tubules can stimulate the odontoblasts and associated fast-conducting A-delta nerve fibers in the pulp, which in turn produce sharp, quickly reversible dental pain (Fig. 1-35). The more open these tubules are (e.g., from a newly exposed preparation, dentin decalcification, periodontal scaling, tooth-bleaching materials, or coronal tooth fractures), the more the tubule fluid will move and, subsequently, the more the tooth will display dentin sensitivity when stimulated. When making a diagnosis, it is important to discriminate this dentin sensitivity sensation from that of reversible pulpitis, which would be secondary to caries, trauma, or new or defective restorations. Detailed questioning about recent dental treatment and a thorough clinical and radiographic examination will help to separate dentin sensitivity from other pulpal pathosis, as the treatment modalities for each are completely different.[18]

Irreversible Pulpitis

As the disease state of the pulp progresses, the inflammatory condition of the pulp can change to *irreversible pulpitis*. At this stage, treatment to remove the diseased pulp will be necessary. This condition can be divided into the subcategories of *symptomatic* and *asymptomatic* irreversible pulpitis.

Symptomatic Irreversible Pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing.[6] Teeth that are classified as having *symptomatic irreversible pulpitis* exhibit intermittent or spontaneous pain. Rapid exposure to dramatic temperature changes (especially to cold stimuli) will elicit heightened and prolonged episodes of pain even after the thermal stimulus has been removed. The pain in these cases may be sharp or dull, localized, diffuse, or referred. Typically, there are minimal or no changes in the radiographic appearance of the periradicular bone. With advanced irreversible pulpitis, a thickening of the periodontal ligament may become apparent on the radiograph, and there may be some evidence of pulpal irritation by virtue of extensive pulp chamber or root canal space calcification. Deep restorations, caries, pulp exposure, or any other direct or indirect insult to the pulp, recently or historically, may be present. It may be seen radiographically or clinically or may be suggested from a complete dental history. Patients who present with symptomatic anterior teeth for which there are no obvious etiologic factors should be also questioned regarding past general anesthesia or endotracheal intubation procedures.[3,127,138] In addition, patients should be questioned about a history of orthodontic treatment. Typically, when symptomatic irreversible pulpitis remains untreated, the pulp will eventually become necrotic.[109,139]

Asymptomatic Irreversible Pulpitis

This is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing.[6] The patient, however, does not complain of any symptoms. On occasion, deep caries will not produce any symptoms, even though clinically or radiographically the caries may extend well into the pulp. Left untreated, the tooth may become symptomatic or the pulp will become necrotic. In cases of *asymptomatic irreversible pulpitis,* endodontic treatment should be performed as soon as possible so that symptomatic irreversible pulpitis or necrosis does not develop and cause the patient severe pain and distress.

Pulp Necrosis

This is a clinical diagnostic category indicating death of the dental pulp. The pulp is usually nonresponsive to pulp testing.[6] When pulpal *necrosis* (or *nonvital pulp*) occurs, the pulpal blood supply is nonexistent and the pulpal nerves are nonfunctional. It is the only clinical classification that directly attempts to describe the histologic status of the pulp (or lack thereof). This condition is subsequent to symptomatic or asymptomatic irreversible pulpitis. After the pulp becomes completely necrotic, the tooth will typically become asymptomatic until such time when there is an extension of the disease process into the periradicular tissues. With pulp necrosis, the tooth will usually not respond to electric pulp tests or to cold stimulation. However, if heat is applied for an extended period of time, the tooth may respond to this stimulus. This response could possibly be related to remnants of fluid or gases in the pulp canal space expanding and extending into the periapical tissues.

Pulpal necrosis may be partial or complete and it may not involve all of the canals in a multirooted tooth. For this reason, the tooth may present with confusing symptoms. Pulp testing over one root may give no response, whereas over another root it may give a positive response. The tooth may also exhibit symptoms of symptomatic irreversible pulpitis. Pulp necrosis, in the absence of restorations, caries, or luxation injuries, is likely caused by a longitudinal fracture extending from the occlusal surface and into the pulp.[19]

After the pulp becomes necrotic, bacterial growth can be sustained within the canal. When this infection (or its bacterial byproducts) extends into the periodontal ligament space, the tooth may become symptomatic to percussion or exhibit spontaneous pain. Radiographic changes may occur, ranging from a thickening of the periodontal ligament space to the appearance of a periapical radiolucent lesion. The tooth may become hypersensitive to heat, even to the warmth of the oral cavity, and is often relieved by applications of cold. As previously discussed, this may be helpful in attempting to localize a necrotic tooth (i.e., by the application of cold one tooth at a time) when the pain is referred or not well localized.

Previously Treated

This is a clinical diagnostic category indicating that the tooth has been endodontically treated and the canals are obturated with various filling materials other than intracanal medicaments.[6] In this situation, the tooth may or may not present with signs or symptoms but will require additional nonsurgical or surgical endodontic procedures to retain the tooth. In most such situations, there will no longer be any vital or necrotic pulp tissue present to respond to pulp testing procedures.

Previously Initiated Therapy

This is a clinical diagnostic category indicating that the tooth has been previously treated by partial endodontic therapy (e.g., pulpotomy, pulpectomy).[6] In most instances, the partial endodontic therapy was performed as an emergency procedure for symptomatic or asymptomatic irreversible pulpitis cases. In other situations, these procedures may have been performed as part of vital pulp therapy procedures, traumatic tooth injuries, apexification, or apexogenesis therapy. At the time these cases present for root canal therapy it would not be possible to make an accurate pulpal diagnosis because all, or part, of the pulp tissue has already been removed.

Apical (Periapical) Disease

Normal Apical Tissues

This classification is the standard against which all of the other apical disease processes are compared. In this category the patient is asymptomatic and the tooth responds normally to percussion and palpation testing. The radiograph reveals an intact lamina dura and periodontal ligament space around all the root apices.

Periodontitis

This classification refers to an inflammation of the periodontium.[6] When located in the periapical tissues it is referred to as apical periodontitis. Apical periodontitis can be subclassified to symptomatic apical periodontitis and asymptomatic apical periodontitis.

Symptomatic Apical Periodontitis

This condition is defined as an inflammation, usually of the apical periodontium, producing clinical symptoms including a painful response to biting or percussion or palpation. It might or might not be associated with an apical radiolucent area.[6]

This tooth may or may not respond to pulp vitality tests, and the radiograph or image of the tooth will typically exhibit at least a widened periodontal ligament space and may or may not show an apical radiolucency associated with one or all of the roots.

Asymptomatic Apical Periodontitis

This condition is defined as inflammation and destruction of apical periodontium that is of pulpal origin, appears as an apical radiolucent area, and does not produce clinical symptoms.[6] This tooth does not usually respond to pulp vitality tests, and the radiograph or image of the tooth will exhibit an apical radiolucency. The tooth is generally not sensitive to biting pressure but may "feel different" to the patient on percussion. Manifestation of persistent apical periodontitis may vary among patients.[89]

Acute Apical Abscess

This condition is defined as an inflammatory reaction to pulpal infection and necrosis characterized by *rapid onset*, spontaneous pain, tenderness of the tooth to pressure, pus formation, and swelling of associated tissues.[6] A tooth with an *acute apical abscess* will be acutely painful to biting pressure, percussion, and palpation. This tooth will not respond to any pulp vitality tests and will exhibit varying degrees of mobility. The radiograph or image can exhibit anything from a widened periodontal ligament space to an apical radiolucency. Swelling will be present intraorally and the facial tissues adjacent to the tooth will almost always present with some degree of swelling. The patient will frequently be febrile, and the cervical and submandibular lymph nodes may exhibit tenderness to palpation.

Chronic Apical Abscess

This condition is defined as an inflammatory reaction to pulpal infection and necrosis characterized by *gradual onset*, little or no discomfort, and the intermittent discharge of pus through an associated sinus tract.[6] In general, a tooth with a *chronic apical abscess* will not present with clinical symptoms. The tooth will not respond to pulp vitality tests, and the radiograph or image will exhibit an apical radiolucency. Usually the tooth is not sensitive to biting pressure but can "feel different" to the patient on percussion. This entity is distinguished from asymptomatic apical periodontitis because it will exhibit intermittent drainage through an associated sinus tract.

REFERRED PAIN

The perception of pain in one part of the body that is distant from the actual source of the pain is known as *referred pain*. Whereas pain of nonodontogenic origin can refer pain to the teeth, teeth may also refer pain to other teeth as well as to other anatomic areas of the head and neck (see Chapters 4 and 17). This may create a diagnostic challenge, in that the patient may insist that the pain is from a certain tooth or even from an ear when, in fact, it is originating from a distant tooth with pulpal pathosis. Using electronic pulp testers, investigators found that patients could localize *which* tooth was being stimulated only 37.2% of the time and could narrow the location to three teeth only 79.5% of the time, illustrating that patients may have a difficult time discriminating the exact location of pulpal pain.[44]

Referred pain from a tooth is usually provoked by an intense stimulation of pulpal C fibers, the slow conducting nerves that when stimulated cause an intense, slow, dull pain. Anterior teeth seldom refer pain to other teeth or to opposite arches, whereas posterior teeth may refer pain to the opposite arch or to the periauricular area but seldom to the anterior teeth.[14] Mandibular posterior teeth tend to transmit referred pain to the periauricular area more often than maxillary posterior teeth. One study showed that when second molars were stimulated with an electric pulp tester, patients could discriminate accurately which arch the sensation was coming from only 85% of the time, compared with an accuracy level of 95% with first molars and 100% with anterior teeth.[136] The investigators also pointed out that when patients first feel the sensation of pain, they are more likely to accurately discriminate the origin of the pain. With higher levels of discomfort, patients have less ability to accurately determine the source of the pain. Therefore, in cases of diffuse or referred pain, the history of where the patient first felt the pain may be significant.

Because referred pain can complicate a dental diagnosis, the clinician must be sure to make an accurate diagnosis to protect the patient from unnecessary dental or medical treatment. If after all the testing procedures are complete and it is determined that the pain is not of odontogenic origin, then the patient should be referred to an orofacial pain clinic for further testing. For further information on pain of nonodontogenic origin, see Chapter 17.

SUMMARY

Endodontics is a multifaceted specialty, with much emphasis on how cases are clinically treated. Clinicians have increased their ability to more accurately perform endodontic procedures by way of increased visualization using the operating microscope, precise apical foramen detection using electronic apex locators, enhanced imaging techniques using digital radiography, and more. Practices have incorporated more refined canal cleaning and shaping techniques by using ultrasonics and rotary-driven nickel titanium files facilitated with computer-assisted electronic handpieces. Many other advancements have also been introduced with the objective of achieving an optimal result during endodontic treatment. However, these advancements are useless if an incorrect diagnosis is made. Before the clinician ever considers performing any endodontic treatment, the following questions must be answered:

- Is the existing problem of dental origin?
- Are the pulpal tissues within the tooth pathologically involved?
- Why is the pulpal pathosis present?
- What is the prognosis?
- What is the appropriate form of treatment?

Testing, questioning, and reasoning are combined to achieve an accurate diagnosis and to ultimately form an appropriate treatment plan. The art and science of making this diagnosis are the first steps that must be taken before initiating any endodontic treatment.

REFERENCES

1. Abella F, Patel S, Duran-Sindreu F, et al: Evaluating the periapical status of teeth with irreversible pulpitis by using cone-beam computed tomography scanning and periapical radiographs, *J Endod* 38:1588, 2012.
2. Abuabara A, Zielak JC, Schramm CA, Baratto-Filho F: Dental infection simulating skin lesion, *An Bras Dermatol* 87:619, 2012.
3. Adolphs N, Kessler B, von Heymann C, et al: Dentoalveolar injury related to general anaesthesia: a 14 years review and a statement from the surgical point of view based on a retrospective analysis of the documentation of a university hospital, *Dent Traumatol* 27:10, 2011.
4. Al-Hezaimi K, Naghshbandi J, Simon JH, Rotstein I: Successful treatment of a radicular groove by intentional replantation and Emdogain therapy: four years follow-up, *Oral Surg Oral Med Oral Pathol Oral Radiol Endodon* 107:e82, 2009.
5. Alomari FA, Al-Habahbeh R, Alsakarna BK: Responses of pulp sensibility tests during orthodontic treatment and retention, *Int Endod J* 44:635, 2011.
6. American Association of Endodontists: *Glossary of endodontic terms*, ed 8, Chicago, 2012, American Association of Endodontists.
7. Anderson RW, Pantera EA: Influence of a barrier technique on electric pulp testing, *J Endod* 14:179, 1988.
8. Andreasen J, Andreasen F, Andreasen L, editors: *Textbook and color atlas of traumatic injuries to the teeth*, ed 4, Philadelphia, 2008, Wiley Blackwell.
9. Andreasen JO, Ahrensburg SS, Tsilingaridis G: Root fractures: the influence of type of healing and location of fracture on tooth survival rates: an analysis of 492 cases, *Dent Traumatol* 28:404, 2012.
10. Arakawa S, Cobb CM, Rapley JW, Killoy WJ, et al: Treatment of root fracture by CO_2 and Nd:YAG lasers: an in vitro study, *J Endod* 22:662, 1996.
11. Augsburger RA, Peters DD: In vitro effects of ice, skin refrigerant, and CO_2 snow on intrapulpal temperature, *J Endod* 7:110, 1981.
12. Baumgartner JC, Picket AB, Muller JT: Microscopic examination of oral sinus tracts and their associated periapical lesions, *J Endod* 10:146, 1984.
13. Beltes C, Zachou E: Endodontic management in a patient with vitamin D-resistant Rickets, *J Endod* 38:255, 2012.
14. Bender IB: Pulpal pain diagnosis: a review, *J Endod* 26:175, 2000.
15. Bender IB, Landau MA, Fonsecca S, Trowbridge HO: The optimum placement-site of the electrode in electric pulp testing of the 12 anterior teeth, *J Am Dent Assoc* 118:305, 1989.
16. Bender IB, Seltzer S: Roentgenographic and direct observation of experimental lesions in bone. Part I, *J Am Dent Assoc* 62:152, 1961.
17. Bender IB, Seltzer S: Roentgenographic and direct observation of experimental lesions in bone. Part II, *J Am Dent Assoc* 62:708, 1961.
18. Berman LH: Dentinal sensation and hypersensitivity: a review of mechanisms and treatment alternatives, *J Periodontol* 56:216, 1984.
19. Berman LH, Kuttler S: Fracture necrosis: diagnosis, prognosis assessment, and treatment recommendations, *J Endod* 36:442, 2010.
20. Bierma MK, McClanahan S, Baisden MK, Bowles WR: Comparison of heat-testing methodology, *J Endod* 38:1106, 2012.
21. Bornstein MM, Lauber R, Sendi P, von Arx T: Comparison of periapical radiography and limited cone-beam computed tomography in mandibular molars for analysis of anatomical landmarks before apical surgery, *J Endod* 37:151, 2011.
22. Cameron CE: The cracked tooth syndrome: additional findings, *J Am Dent Assoc* 93:971, 1981.
15. Chen E, Abbottt PV: Evaluation of accuracy, reliability, and repeatability of five dental pulp tests, *J Endod* 37:1619, 2011.
16. Chiego DJ, Cox CF, Avery JK: H-3 HRP analysis of the nerve supply to primate teeth, *Dent Res* 59:736, 1980.
17. Cleveland JL, Gooch BF, Shearer BG, Lyerla RL: Risk and prevention of hepatitis C virus infection, *J Am Dent Assoc* 130:641, 1999.
18. Costa FF, Gaia BF, Umetsubo OS, Cavalcanti MGP: Detection of horizontal root fracture with small-volume cone-beam computed tomography in the presence and absence of intracanal metallic post, *J Endod* 37:1456, 2011.
19. Costa FF, Gaia BF, Umetsubo OS, et al: Use of large-volume cone-beam computed tomography in identification and localization of horizontal root fracture in the presence and absence of intracanal metallic post, *J Endod* 38:856, 2012.
28. Costa FWG, Rodrigues RR, Batista ACB: Multiple radiopaque mandibular lesions in a patient with Apert syndrome, *J Endod* 38:1639, 2012.
29. Cotton TP, Geisler TM, Holden DT, Schwartz SA, et al: Endodontic applications of cone-beam volumetric tomography, *J Endod* 33:1121, 2007.
30. Dankner E, Harari D, Rotstein I: Dens evaginatus of anterior teeth: literature review and radiographic survey of 15,000 teeth, *Oral Surg Oral Med Oral Pathol* 81:472, 1996.
31. Dastmalchi N, Jafarzadeh H, Moradi S: Comparison of the efficacy of a custom-made pulse oximeter probe with digital electric pulp tester, cold spray, and rubber cup for assessing pulp vitality, *J Endod* 38:1182, 2012.
32. Davido N, Rigolet A, Kerner S, et al: Case of Ewing's sarcoma misdiagnosed as a periapical lesion of maxillary incisor, *J Endod* 37:259, 2011.
33. Deepak BS, Subash TS, Narmatha VJ, et al: Imaging techniques in endodontics: an overview, *J Clin Imaging Sci* 2:13, 2012.
34. DeRossi SS, Glick M: Dental considerations for the patient with renal disease receiving hemodialysis, *J Am Dent Assoc* 127:211, 1996.
35. DeRossi SS, Glick M: Lupus erythematosus: considerations for dentistry, *J Am Dent Assoc* 129:330, 1998.
36. Dirks SJ, Paunovich ED, Terezhalmy GT, Chiodo LK: The patient with Parkinson's disease, *Quint Int* 34:379, 2003.
37. Durack C, Patel S: Cone beam computed tomography in endodontics, *Braz Dent J* 23:179, 2012.
38. Edlund M, Nair MK, Nair UP: Detection of vertical root fractures by using cone-beam computed tomography: a clinical study, *J Endod* 37:768, 2011.
39. Eikenerg S, Vandre R: Comparison of digital dental x-ray systems with self-developing film and manual processing for endodontic file length determination, *J Endod* 26:65, 2000.
40. Evans D, Reid J, Strang R, Stirrups D: A comparison of laser Doppler flowmetry with other methods of assessing the vitality of traumatized anterior teeth, *Endod Dent Traumatol* 15:284, 1999.
41. Farman AG, Levato CM, Scarfe WC: A primer on cone beam CT. *Inside Dentistry* 1:90, 2007.
42. Filippatos CG, Tsatsoulis IN, Floratos S, Kontakiotis EG: A variability of electric pulp response threshold in premolars: a clinical study, *J Endod* 38:144, 2012.
43. Fouad AF: Diabetes mellitus as a modulating factor of endodontic infections, *J Dent Educ* 67:459, 2003.
44. Friend LA, Glenwright HD: An experimental investigation into the localization of pain from the dental pulp, *Oral Surg Oral Med Oral Pathol* 25:765, 1968.
45. Fuss Z, Trope M. Root perforations: classification and treatment choices based on prognostic factors, *Endod Dent Traumatol* 12:255, 1996.
28. Fuss Z, Trowbridge H, Bender IB, Rickoff B, Sorin S: Assessment of reliability of electrical and thermal pulp testing agents, *J Endod* 12:301, 1986.
29. Ganz SD: Cone beam computed tomography-assisted treatment planning concepts, *Dent Clin North Am* 55:515, 2011.
30. Gillcrist JA: Hepatitis viruses A, B, C, D, E and G: implications for dental personnel, *J Am Dent Assoc* 130:509, 1999.
31. Goldman M, Pearson A, Darzenta N: Endodontic success: who is reading the radiograph? *Oral Surg Oral Med Oral Pathol* 33:432, 1972.
32. Goldman M, Pearson A, Darzenta N: Reliability of radiographic interpretations, *Oral Surg Oral Med Oral Pathol* 38:287, 1974.
33. Goodchild JH, Glick M: A different approach to medical risk assessment, *Endod Topics* 4:1, 2003.
34. Goon WW, Jacobsen PL: Prodromal odontalgia and multiple devitalized teeth caused by a herpes zoster infection of the trigeminal nerve: report of case, *J Am Dent Assoc* 116:500, 1988.
35. Gopikrishna V, Tinagupta K, Kandaswamy D: Comparison of electrical, thermal and pulse oximetry methods for assessing pulp vitality in recently traumatized teeth, *J Endod* 33:531, 2007.
54. Gopikrishna V, Tinagupta K, Kandaswamy D: Evaluation of efficacy of a new custom-made pulse oximeter dental probe in comparison with electrical and thermal tests for assessing pulp vitality, *J Endod* 33:411, 2007.
55. Harrison JW, Larson WJ: The epithelized oral sinus tract, *Oral Surg Oral Med Oral Pathol* 42:511, 1976.
56. Heling I, Rotstein I: Apersistent oronasal sinus tract of endodontic origin, *J Endod* 15:132, 1989.
57. Herman WW, Konzelman JL, Prisant LM: New national guidelines on hypertension, *J Am Dent Assoc* 135:576, 2004.
58. Idiyatullin D, Corum C, Moeller S, et al: Dental magnetic resonance imaging: making the invisible visible, *J Endod* 37:745, 2011.
59. Imai K, Shimada Y, Sadr A, et al: Nonivasive cross-sectional visualization of enamel cracks by optical coherence tomography *in vitro*, *J Endod* 38:1269, 2012.
60. Ingolfsson AER, Tronstad L, Riva CE: Reliability of laser Doppler flowmetry in testing vitality of human teeth, *Endod Dent Traumatol* 10:185, 1994.
61. Ingram TA, Peters DD: Evaluation of the effects of carbon dioxide used as a pulp test. Part 2: in vivo effect on canine enamel and pulpal tissues, *J Endod* 9:296, 1983.
62. Jafarzadeh H, Abbott PV: Review of pulp sensibility tests. Part I: general information and thermal tests, *Int Endod J* 43:738, 2010.
63. Jafarzadeh H, Abbott PV: Review of pulp sensibility tests. Part II: electric pulp tests and test cavities, *Int Endod J* 43:945, 2010.
64. Johnson BR, Remeikis NA, Van Cura JE: Diagnosis and treatment of cutaneous facial sinus tracts of dental origin, *J Am Dent Assoc* 130:832, 1999.
65. Jones DM: Effect of the type carrier used on the results of dichlorodifluoromethane application to teeth, *J Endod* 25:692, 1999.
66. Jones VR, Rivera EM, Walton RE: Comparison of carbon dioxide versus refrigerant spray to determine pulpal responsiveness, *J Endod* 28:531, 2002.
67. Kaffe I, Gratt BM: Variations in the radiographic interpretation of the periapical dental region, *J Endod* 14:330, 1988.
68. Kahan RS, Gulabivala K, Snook M, Setchell DJ: Evaluation of a pulse oximeter and customized probe for pulp vitality testing, *J Endod* 22:105, 1996.
69. Karayilmaz H, Kirzioglu Z: Comparison of the reliability of laser Doppler flowmetry, pulse oximetry and electric pulp tester in assessing the pulp vitality of human teeth, *J Oral Rehabil* 38:340, 2011.

70. Kataoka SH, Setzer FC, Gondim-Junior E, et al: Pulp vitality in patients with intraoral and oropharyngeal malignant tumors undergoing radiation therapy assessed by pulse oximetry, *J Endod* 37:1197, 2011.
71. Katz J, Chaushu G, Rotstein I: Stafne's bone cavity in the anterior mandible: a possible diagnosis challenge, *J Endod* 27:304, 2001.
72. Kawai K, Masaka N: Vertical root fracture treated by bonding fragments and rotational replantation, *Dent Traumatol* 18:42, 2002.
73. Khocht A, Janal M, Harasty L, Chang K: Comparison of direct digital and conventional intraoral radiographs in detecting alveolar bone loss, *J Am Dent Assoc* 134:1468, 2003.
74. Koivisto T, Bowles WR, Rohrer M: Frequency and distribution of radiolucent jaw lesions: a retrospective analysis of 9,723 cases, *J Endod* 38:729, 2012.
75. Kusgoz A, Yildirim T, Kayipmaz S, Saricaoglu S: Nonsurgical endodontic treatment of type III dens invaginatus in maxillary canine: an 18-month follow up, *Oral Surg Oral Med Oral Pathol Oral Radiol Endodon* 107:e103, 2009.
76. Lalla RV, D'Ambrosio JA: Dental management considerations for the patient with diabetes mellitus, *J Am Dent Assoc* 132:1425, 2001.
77. Laskin DM: Anatomic considerations in diagnosis and treatment of odontogenic infections, *J Am Dent Assoc* 69:308, 1964.
78. Liang YH, Li G, Wesselink PR, Wu MK: Endodontic outcome predictors identified with periapical radiographs and cone-beam computed tomography scans, *J Endod* 37:326, 2011.
79. Lin J, Chandler NP: Electric pulp testing: a review, *Int Endod J* 41:365, 2008.
80. Little JW, Falace DA, Miller CS, Rhodus NL: *Dental management of the medically compromised patient*, ed 8, St. Louis, 2013, Elsevier Mosby.
81. Lofthag-Hansen S, Huumonen S, Gröndahl K, Gröndahl HG: Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 103:114, 2007.
82. Marder MZ: The standard of care for oral diagnosis as it relates to oral cancer, *Compend Contin Educ Dent* 19:569, 1998.
83. Mattson JS, Cerutis DR: Diabetes mellitus: a review of the literature and dental implications, *Comp Cont Educ Dent* 22:757, 2001.
84. Mesaros S, Trope M, Maixner W, Burkes EJ: Comparison of two laser Doppler systems on the measurement of blood flow of premolar teeth under different pulpal conditions, *Int Endod J* 30:167, 1997.
85. Metska ME, Aartman IHA, Wesselink PR, Özcor AR: Detection of vertical root fractures *in vivo* in endodontically treated teeth by cone-beam computed tomography scans, *J Endod* 38:1344, 2012.
86. Michaelson RE, Seidberg BH, Guttuso J: An in vivo evaluation of interface media used with the electric pulp tester, *J Am Dent Assoc* 91:118, 1975.
87. Miles DA, VanDis ML: Advances in dental imaging, *Dent Clin North Am* 37:531, 1993.
88. Miller CS, Little JW, Falace DA: Supplemental corticosteroids for dental patients with adrenal insufficiency: reconsideration of the problem, *J Am Dent Assoc* 132:1570, 2001.
89. Morsani JM, Aminoshariae A, Han YW: Genetic predisposition to persistent apical periodontitis, *J Endod* 37:455, 2011.
90. Mozzo P, Proccaci A, et al: A new volumetric CT machine for dental imaging based on the cone-beam technique: preliminary results, *Eur Radiol* 8:1558, 1998.
91. Nair M, Pettigrew J, Mancuso A: Intracranial aneurysm as an incidental finding, *Dentomaxillofac Radiol* 36:107, 2007.
92. Nakata K, Naitob M, Izumi M, et al: Effectiveness of dental computed tomography in diagnostic imaging of periradicular lesion of each root of a multirooted tooth: a case report, *J Endod* 32:583, 2007.
70. Nalliah RP, Allareddy V, Elangovan S, et al: Hospital emergency department visits attributed to pulpal and periapical disease in the United States in 2006, *J Endod* 37:6, 2011.
71. Özer SY: Detection of vertical root fractures by using cone beam computed tomography with variable voxel sizes in an *in vitro* model, *J Endod* 37:75: 2011.
72. Pantera EA, Anderson RW, Pantera CT: Use of dental instruments for bridging during electric pulp testing, *J Endod* 18:37, 1992.
73. Pantera EA, Anderson RW, Pantera CT: Reliability of electric pulp testing after pulpal testing with dichlorodifluoromethane, *J Endod* 19:312, 1993.
74. Paurazas SM, Geist JR, Pink FE: Comparison of diagnostic accuracy of digital imaging using CCD and CMOS-APS sensors with E-speed film in the detection of periapical bony lesions, *Oral Surg Oral Med Oral Pathol Oral Radiology Endodon* 44:249, 2000.
75. Peters DD, Baumgartner JC, Lorton L: Adult pulpal diagnosis. 1. Evaluation of the positive and negative responses to cold and electric pulp tests, *J Endod* 20:506, 1994.
76. Petersson K, Soderstrom C, Kiani-Anaraki M, Levy G: Evaluation of the ability of thermal and electric tests to register pulp vitality, *Endod Dent Traumatol* 15:127, 1999.
77. Pinto A, Glick M: Management of patients with thyroid disease: oral health considerations, *J Am Dent Assoc* 133:849, 2002.
78. Pitts DL, Natkin E: Diagnosis and treatment of vertical root fractures, *J Endod* 9:338, 1983.
79. Poeschl PW, Crepaz V, Russmueller G, et al: Endodontic pathogens causing deep neck space infections: clinical impact of different sampling techniques and antibiotic susceptibility, *J Endod* 37:1201, 2011.
80. Rees JS, Addy M: A cross-sectional study of dentine hypersensitivity, *J Clin Periodontol* 29:997, 2002.
81. Rickoff B, Trowbridge H, Baker J, Fuss Z, et al: Effects of thermal vitality tests on human dental pulp, *J Endod* 14:482, 1988.
82. Riley CK, Terezhalmy GT: The patient with hypertension, *Quint Int* 32:671, 2001.
106. Robb RA, Sinak LJ, Hoffman EA, et al: Dynamic volume imaging of moving organs, *J Med Syst* 6:539, 1982.
107. Rodrigues CD, Villar-Neto MJC, Sobral APV, et al: Lymphangioma mimicking apical periodontitis, *J Endod* 37:91, 2011.
108. Rosen H: Cracked tooth syndrome, *J Prosthet Dent* 47:36, 1982.
109. Rotstein I, Engel G: Conservative management of a combined endodontic-orthodontic lesion, *Endod Dent Traumatol* 7:266, 1991.
110. Rotstein I, Moshonov J, Cohenca N: Endodontic therapy for a fused mandibular molar, *Endod Dent Traumatol*, 13:149, 1997.
111. Rotstein I, Simon HS: Diagnosis, prognosis and decision-making in the treatment of combined periodontal-endodontic lesions, *Periodontol 2000* 34:165, 2004.
112. Rotstein I, Simon HS: The endo-perio lesion: a critical appraisal of the disease condition, *Endodon Topics* 13:34, 2006.
113. Rotstein I, Stabholz A, Heling I, Friedman S: Clinical considerations in the treatment of dens invaginatus, *Endod Dent Traumatol* 3:249, 1987.
114. Roykens H, Van Maele G, DeMoor R, Martens L: Reliability of laser Doppler flowmetry in a 2-probe assessment of pulpal blood flow, *Oral Surg Oral Med Oral Pathol Oral Radiol Endodon* 87:742, 1999.
115. Rud J, Omnell KA: Root fractures due to corrosion: diagnostic aspects, *Scand J Dent Res* 78:397, 1970.
116. Rutsatz C, Baumhardt SG, Feldens CA, et al: Response of pulp sensibility test is strongly influenced by periodontal attachment loss and gingival recession, *J Endod* 38:580, 2012.
117. Sasano T, Nakajima I, Shohi N, et al: Possible application of transmitted laser light for the assessment of human pulpal vitality, *Endod Dent Traumatol* 13:88, 1997.
106. Schnettler JM, Wallace JA: Pulse oximetry as a diagnostic tool of pulp vitality, *J Endod* 17:488, 1991.
107. Schwartz RS: Mineral trioxide aggregate: a new material for endodontics, *J Am Dent Assoc* 130:967, 1999.
108. Selden HS: Repair of incomplete vertical root fractures in endodontically treated teeth: in vivo trials, *J Endod* 22:426, 1996.
109. Seltzer S, Bender IB, Nazimov H: Differential diagnosis of pulp conditions, *Oral Surg Oral Med Oral Pathol* 19:383, 1965.
110. Seltzer S, Bender IB, Ziontz M: The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. Part I, *Oral Surg Oral Med Oral Pathol* 16:846, 1963.
111. Seltzer S, Bender IB, Ziontz M: The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. Part II, *Oral Surg Oral Med Oral Pathol* 16:969, 1963.
112. Seo DG, Yi YA, Shin AJ, Park JW: Analysis of factors associated with cracked teeth, *J Endod* 38:288, 2012.
113. Setzer FC, Kataoka SH, Natrielli F, et al: Clinical diagnosis of pulp inflammation based on pulp oxygenation rates measured by pulse oximetry, *J Endod* 38:880, 2012.
114. Simon JHS, Dogan H, Ceresa LM, Silver GK: The radicular groove: it's potential clinical significance, *J Endod* 26:295, 2000.
115. Simon JHS, Lies J: Silent trauma, *Endod Dent Traumatol* 15:145, 1999.
128. Shemesh H, Cristescu RC, Wesselink PR, Wu MK: The use of cone-beam computed tomography and digital periapical radiographs to diagnose root perforations, *J Endod* 37:513, 2011.
129. Steinbacher DM, Glick M: The dental patient with asthma: an update and oral health considerations, *J Am Dent Assoc* 132:1229, 2001.
130. Stroblitt H, Gojer G, Norer B, Emshoff R: Assessing revascularization of avulsed permanent maxillary incisors by laser Doppler flowmetry, *J Am Dent Assoc* 134:1597, 2003.
131. Suebnukarn S, Rhienmora P, Haddawy P: The use of cone-beam computed tomography and virtual reality simulation for pre-surgical practice in endodontic microsurgery, *Int Endod J* 45:627, 2012.
132. Sugaya T, Kawanami M, Noguchi H, et al: Periodontal healing after bonding treatment of vertical root fracture, *Dent Traumatol* 17:174, 2001.
133. Tatlidil R, Gözübüyük MM: Mucinous adenocarcinoma of lung presenting as oral metastases: a case report and literature review, *J Endod* 37:110, 2011.
134. Tewary S, Luzzo J, Hartwell G: Endodontic radiography: who is reading the digital radiograph, *J Endod* 37:919, 2011.
135. Treister N, Glick M: Rheumatoid arthritis: a review and suggested dental care considerations, *J Am Dent Assoc* 130:689, 1999.
136. Van Hassel HJ, Harrington GW: Localization of pulpal sensation, *Oral Surg Oral Med Oral Pathol* 28:753, 1969.
137. Velvart P, Hecker H, Tillinger G: Detection of the apical lesion and the mandibular canal in conventional radiography and computed tomography, *Oral Surg Oral Med Oral Pathol Oral Radiol Endodon* 92:682, 2001.
138. Vogel J, Stubinger S, Kaufmann M: Dental injuries resulting from tracheal intubation: a retrospective study, *Dent Traumatol* 25:73, 2009.
139. Von Böhl M, Ren Y, Fudalej PS, Kuijpers-Jagtman AM: Pulpal reactions to orthodontic force application in humans: a systematic review, *J Endod* 38:1463, 2012.
140. Wallace JA, Schnettler JM: Pulse oximetry as a diagnostic tool of pulpal vitality, *J Endod* 17:488, 1993.
141. Weisleder R, Yamauchi S, Caplan DJ, et al: The validity of pulp testing: a clinical study, *J Am Dent Assoc* 140:1013, 2009.
142. Zou X, Liu D, Yue L, Wu M: The ability of cone-beam computerized tomography to detect vertical root fractures in endodontically treated and nonendodontically treated teeth: a report of 3 cases, *Oral Surg Oral Med Oral Pathol Oral Radiol Endodon* 111:797, 2011.

CLINICAL RESEARCH

Ultrasound Examination to Visualize and Trace Sinus Tracts of Endodontic Origin

Elisabetta Cotti, DDS, MS, Davide Musu, DDS,* Alessandro Goddi, DDS,* Claudia Dettori, DDS,* Girolamo Campisi, MD,* and Hagay Shemesh, DDS, PhD†*

Ultrasound real-time examination is a promising tool to detect and trace noninvasively sinus tracts of endodontic origin. It produces a direct image rather than one obtained with a gutta-percha cone inserted into the tract. The vascular reaction within and around the tract can be also observed by the 3D rendering and the color and power Doppler implementation.

ABSTRACT

Introduction: The detection of a tooth with a sinus tract (ST) of endodontic origin and its pathway are conventionally assessed with a periapical radiograph and a gutta-percha cone introduced into its stoma. The aim of this study was to evaluate the possibility to detect STs and trace their route using ultrasound real-time examination. **Methods:** Two calibrated examiners performed echography on 10 patients who had a lesion of apical periodontitis (AP) and ST and 10 patients in the control group with AP without an ST recruited in 2 endodontic practices. They also traced the pattern of the STs with a computer program. The images were then submitted to 2 calibrated and blinded observers who were asked to describe the presence of AP and ST and to trace it with the same program. The data obtained were compared with the clinical and radiographic diagnosis of ST. For sensitivity, specificity, accuracy, and positive and negative predictive values, the receiver operating characteristic curve and Fisher exact test were used ($P < .05$). **Results:** Interobserver agreement was high as was the diagnostic accuracy of the ultrasound examination of STs (mean value = 97.5%), and the Fisher exact test showed statistical significance ($P < .05$). High sensitivity and a negative predictive value and 100% specificity and a positive predictive value were also obtained. The application of the 3-dimensional mode further enabled the reconstructions of the more complex paths, and the implementation with color power Doppler disclosed the vascularity surrounding the STs. **Conclusions:** The ultrasound examination is a technique feasible to describe and trace the STs of endodontic origin. *(J Endod 2019;45:1184–1191.)*

KEY WORDS

Apical periodontitis; oral radiology; sinus tract; ultrasound examination

A sinus tract (ST) of endodontic origin is a pathway from an enclosed area of infection (eg, a root canal) to an epithelial surface through an opening (or stoma), which can be intraoral or extraoral[1]. The presence of apical periodontitis (AP) associated with an ST is classified as a chronic apical abscess[2,3], and the prevalence of this condition ranges from 7.4%–30.75%[4–7]. The degree of epithelialization of the tract is still debated because of its tortuous nature[8]. Patients presenting with an ST are usually asymptomatic, with the draining fistulous opening as the only chief complaint[9]. The ST is expected to heal when the source of the endodontic infection is eliminated after the root canal treatment or the extraction of the offending tooth[10,11]. The identification of the tooth responsible for the ST can be complicated by its opening at a distant site or by the presence of multiple stomas[6], and when STs open in the skin of the face and neck, they can be easily misdiagnosed as dermatologic diseases[9,12,13]. The detection of the infected tooth and the pathway of the ST from the root canal to the stoma are conventionally assessed by taking a periapical radiograph after introducing, in its orifice, a gutta-percha cone[14,15] or a fine stainless steel orthodontic wire[6,16]. Ultrasound (US) real-time examination is a noninvasive imaging technique extensively used in medicine based on the propagation and reflection of acoustic waves across the different tissues of the body[17,18]. In recent times, US has found a wide application in the examination of the head and neck district[19] and in the assessment, differential diagnosis, and follow-up of AP after endodontic treatment[20–23]. The aim of this study was to evaluate the possibility to detect STs of endodontic origin and trace their route using US real-time examination, also called echography.

From the *Department of Conservative Dentistry and Endodontics, University of Cagliari, Cagliari, Italy; and †Department of Endodontology, Academic Centre for Dentistry Amsterdam, Amsterdam, The Netherlands

Address requests for reprints to Dr Elisabetta Cotti, Department of Conservative Dentistry and Endodontics, University of Cagliari, Via Roma 149, Cagliari 09124, Italy.
E-mail address: cottiend@tin.it
0099-2399/$ - see front matter

https://doi.org/10.1016/j.joen.2019.07.009

MATERIALS AND METHODS

The present protocol was conducted in accordance with the institutional ethics committee and the Declaration of Helsinki 1975 (as revised in 2000, PG/2017/9991). The subjects participating in this study were selected among the patients referred to 2 local endodontic practices. After approval for participating in the present study, the patients were asked to sign an informed consent form and were recruited in the research protocol according to the inclusion/exclusion criteria.

Inclusion Criteria

Healthy patients not taking any medication with at least 1 tooth with AP[24] with a periapical index score (PAI)[25] of 3 or more with or without an ST were included in this study.

Exclusion Criteria

Pregnant women, patients with systemic diseases taking medications, patients with a fracture or periodontitis diagnosed on the tooth with the ST, and patients not willing to participate to the study were excluded.

Diagnosis of AP

After assessment of the medical and dental history, the patients received a thorough dental examination. Endodontic clinical evaluation was performed along with the following tests: palpation, percussion, occlusal evaluation, tooth mobility, periodontal probing, bite test, and pulp sensitivity tests for each tooth. Two or more intraoral periapical radiographs were also performed[26]. The diagnosis of ST was done through direct observation of a stoma (draining or not draining), and a periapical radiograph was performed positioning a gutta-percha cone within the tract[12,24].

The assessment of PAI was performed by the postgraduate endodontic students following the tables of Ørstavik et al[25] at the Department of Conservative Dentistry and Endodontics, University of Cagliari, Cagliari, Italy. The decision had to be confirmed by the head of the department. For the aim of the present study, the PAI score was used as a diagnostic decisional tree where the patients were dichotomized in PAI >3 and PAI <3.

Subject Material

Twenty white patients (20 teeth) affected by AP (8 men and 12 women, mean age = 47.2) were divided into the following 2 groups:

1. The cases (APST): 10 teeth with 1 lesion of AP with an ST
2. The controls (CT): 10 teeth with 1 lesion of AP without an ST

The cases and controls were selected by 2 examiners (an expert sonologist and a trained expert endodontist) and examined by 2 blinded observers.

US Examination

The 2 examiners performed calibration with the images of STs collected from a previous pilot study (A. Goddi, 2017), and these samples were not included in the main experiment. The level of agreement among the observers was calculated through kappa statistics. The 2 blinded observers performed calibration exercises with the images of cases of AP, with and without an ST, from the previous pilot study until they obtained a good agreement (Cohen kappa of 0.3, 0.67, and 1.00, at the first, second, and third examination, respectively). Two weeks later, the same images were submitted for further examination to calculate the intraexaminer agreement. All US examinations were performed using either an Elegra (Siemens, Erlangen, Germany) or an Aplio XG (Toshiba Medical Systems, Crawley, UK) apparatus with regular and small-size, linear, high-definition, multifrequency US probes. To have the availability of the 2 units during the main experiment and to not produce confounding data, the 2 linear multifrequency probes of 7–9 MHz and 8–12 MHz, were respectively coupled with the Elegra Siemens and the Toshiba Aplio XG to generate equivalent output scans during the calibration. After the application of an echographic gel, the probe was placed and moved both extraorally on the skin of the upper and lower jaw and intraorally in the area of the alveolar mucosa, corresponding to the roots of the involved teeth. The position of the probe was changed several times to obtain an adequate and representative number of scans. In addition, the color power Doppler (CPD) was applied to all APST cases to assess the presence, amount, and features of the vascular supply within or outside the tract. Once several scans were acquired from each case, the data were converted into 3-dimensional (3D) images. The representative images were saved as TIFF files, organized on a PowerPoint presentation (Microsoft Office 2013; Microsoft Corporation, Redmond, WA) on the screen of a Dell Inspiron 15 laptop computer (Dell Inc, Round Rock, TX), and submitted to the blinded observers separately and without the radiograph where the ST was traced. The blinded observers were then asked to diagnose independently the presence or absence of an ST associated with AP and to fill a dedicated chart. In addition, they were asked to draw the route of the ST on the images using the same computer program. The diagnosis of ST in this study was considered valid only if the fistulous pathway was traced by both observers and if it was possible to superimpose the 2 images. The observers repeated the same examination 2 weeks later.

Data Analysis

The data from the ultrasonic examinations were then compared with the clinical and radiographic diagnosis of ST to assess sensitivity, specificity, accuracy, positive predictive value, and negative predictive value and to establish the reliability of the technique. The accuracy of the procedure was also evaluated with the receiver operating characteristic curve (area under the curve). Given the small number of samples observed in this study, the Fisher exact test was used coherently with the sample size to calculate the significance of agreement on the echographic analysis according to the clinical and radiographic diagnoses ($P < .05$). The Fisher exact test requires 2×2 contingency tables, a number of observations <30, and cells with expected frequencies <5.

RESULTS

All 20 patients did not experience any discomfort during the US examination. The examiners detected AP in all US examinations (APST and CT groups) and traced all 10 STs in the APST group (Table 1). The US images of AP in the CT group were anechoic to hypoechoic areas, developing deeply under a well-defined hyperechoic band corresponding to the buccal cortical bone plate. In the presence of AP, the buccal plate was either perforated or thinned (depending on the scan), creating an acoustic window for the US waves to access the lesions. A posterior acoustic reinforcement (white rim) where the UT could not penetrate any further was the common additional feature at the deepest part of the lesion[27] (Fig. 1*A* and *B*). In the echographic images of all APST cases, there was a detectable interruption of the cortical bone plate (Fig. 2*A–F*) necessary for the discharge of exudation from the lesion to the soft tissues. In the B-mode US images, the STs appeared as dishomogeneous, hypoechoic pathways lined by echogenic and reinforced walls, connecting the lesion to the intra- or extraoral tissues

TABLE 1 - Data Collection of the Apical Periodontitis (AP) Lesions Investigated by Ultrasound (US) Real-time Examination

	Patient	Sex	Age	Teeth	US diagnosis	Diagnostic group	Doppler signal	3D modality
1		F	50	3.4	AP with ST	Case	Absent	Not performed
2		F	25	1.2, 1.1	AP with ST	Case	Present	Performed
3		F	56	3.6	AP with ST	Case	Absent	Not performed
4		M	57	3.6	AP with ST	Case	Present	Performed
5		F	35	1.6	AP with ST	Case	Present	Performed
6		M	52	1.2	AP with ST	Case	Present	Performed
7		F	54	3.7	AP with ST	Case	Present	Performed
8		M	57	1.6	AP with ST	Case	Absent	Performed
9		F	50	3.4, 3.5	AP with ST	Case	Present	Performed
10		F	42	1.6	AP with ST	Case	Absent	Not performed
11		F	24	3.6	AP	Control	Absent	Not performed
12		M	53	1.1, 1.2	AP	Control	Absent	Not performed
13		F	73	1.2	AP	Control	Present	Performed
14		M	68	4.3	AP	Control	Present	Performed
15		M	37	3.6	AP	Control	Absent	Performed
16		M	24	4.7	AP	Control	Present	Not performed
17		F	70	23	AP	Control	Absent	Not performed
18		M	50	2.1	AP	Control	Absent	Not performed
19		F	25	2.6	AP	Control	Absent	Not performed
20		F	42	2.5	AP	Control	Absent	Not performed

3D, 3-dimensional; F, female; M, male; ST, sinus tract.

where the stoma opened (9 cases and 1 case, respectively). When these tracts reached the alveolar mucosa, they were framed by an echogenic area at the interface between the buccal oral tissues and the cortical plate (Fig. 2). In the scans of the extraoral ST, which generated from tooth #19 and opened at the skin surface of the right mandible, the hypoechoic band crossed the muscular layer of the buccinator to reach the surface (Fig. 3*A–F*). The observers diagnosed the absence of an ST in all the images of the CT group and highlighted the presence and route of an ST in 9 out of 10 images in the APST group. In 1 case, there was a mismatch between the observers in the tracing of the ST path, and the diagnosis of ST could not be validated. After a consensus with the nonblinded examiners was reached, 1 of the pathways was considered correct. In 6 of the APST cases, the whole ST could be entirely framed within a single scan. However, in 4 cases (patients #4, 6, 7, and 8), the pathway of the ST was particularly tortuous and required more scans to be traced. In these cases, a 3D image of the entire tract was obtained with the 3D elaboration mode (3D rendering multislice view) (Fig. 4*A–F*). The results from the CPD showed no vascular signal within the lumen of all the tracts; however, in 5 patients[2,4,7], small vessels were identified along the echogenic walls of the ST, revealing a possible inflammatory reaction (Fig. 4). At the first examination, the level of interobserver agreement was very high (Cohen $\kappa = 0.9$). The 2 observers confirmed the results 2 weeks later (Cohen $\kappa = 0.9$). In addition, the

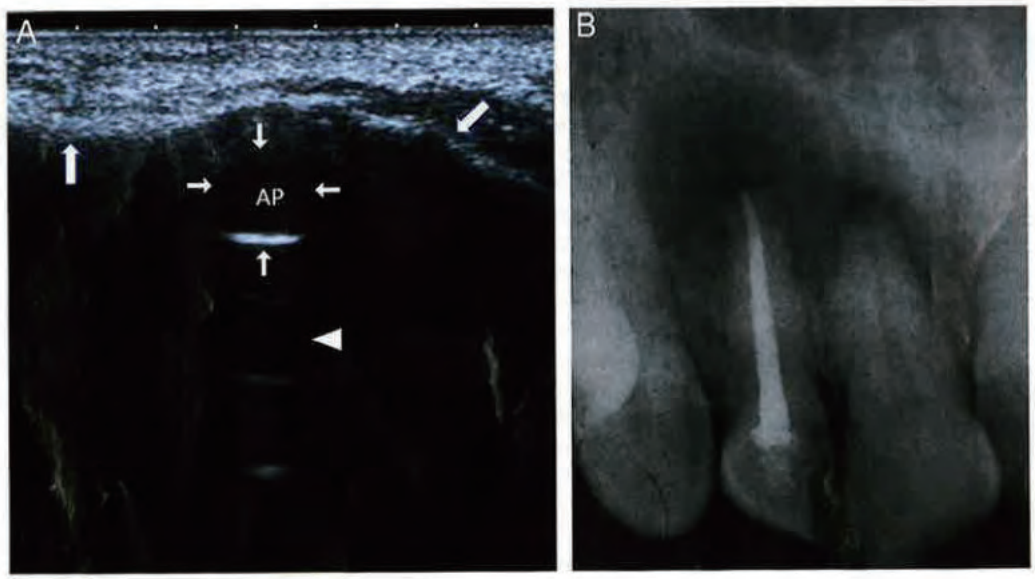


FIGURE 1 – (*A*) An echographic image of AP showing an intact cortical bone plate (*arrows*) and the posterior acoustic reinforcement (*arrowhead*). (*B*) A periapical radiograph depicting the same lesion (tooth #7).

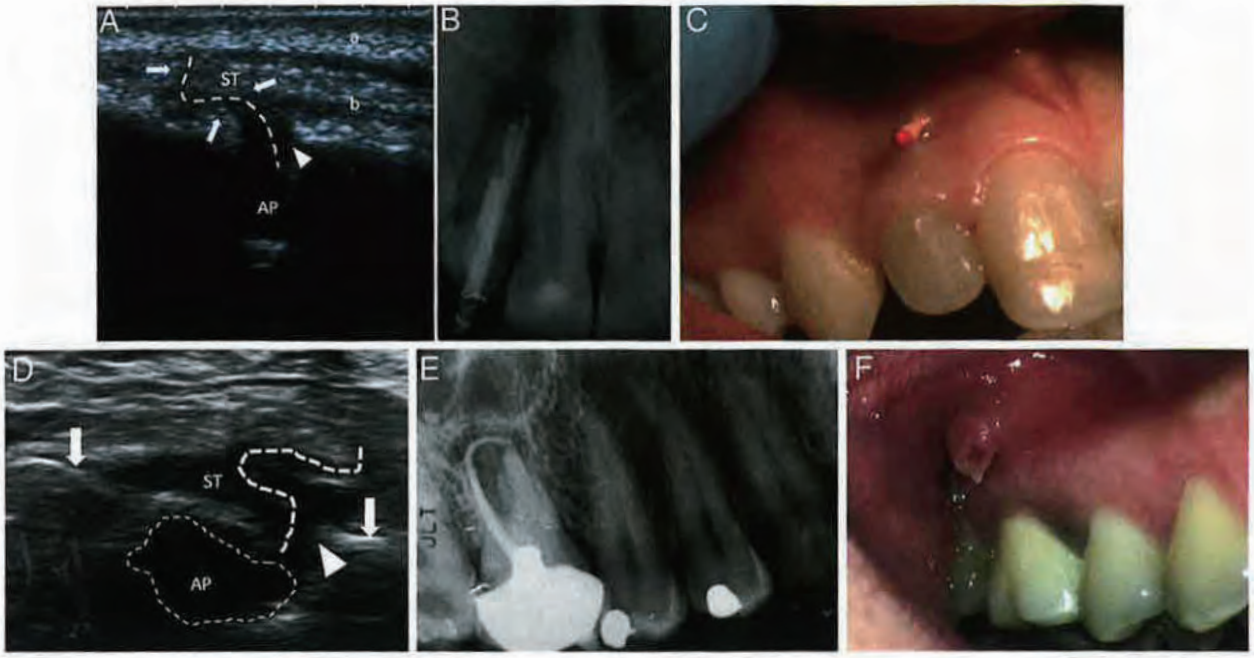


FIGURE 2 – (*A*) An ecographic representation of an ST interrupting the cortical bone plate (*arrowhead*) depicted as a dishomogeneous, hypoechoic pathway (*interrupted line*) lined by echogenic and reinforced walls (*arrows*). (*B*) The same ST traced with a periapical radiograph (tooth #7). (*C*) The corresponding clinical image. (*D*) Ecographic tracing of another ST interrupting the cortical bone plate (*arrows*) through a breach (*arrowhead*). (*E*) The same ST traced with a periapical radiograph (tooth #3). (*F*) A clinical image of the corresponding hypertrophic stoma.

intraobserver agreement calculated 2 weeks later showed a kappa value of 1.00. This technique showed high sensitivity and a negative predictive value and 100% specificity and a positive predictive value (Table 2). According to the receiver operating characteristic curve analysis (Fig. 5), the diagnostic accuracy of the US examination to detect STs had a mean value of 97.5% in the blinded observers. In addition, the Fisher exact test showed a statistical significance for both observers ($P < .05$, Table 2).

DISCUSSION

Achieving new and more complete information on STs is of primary interest in clinical endodontics because the presence of a preoperative ST has been reported to reduce the odds of success of periapical

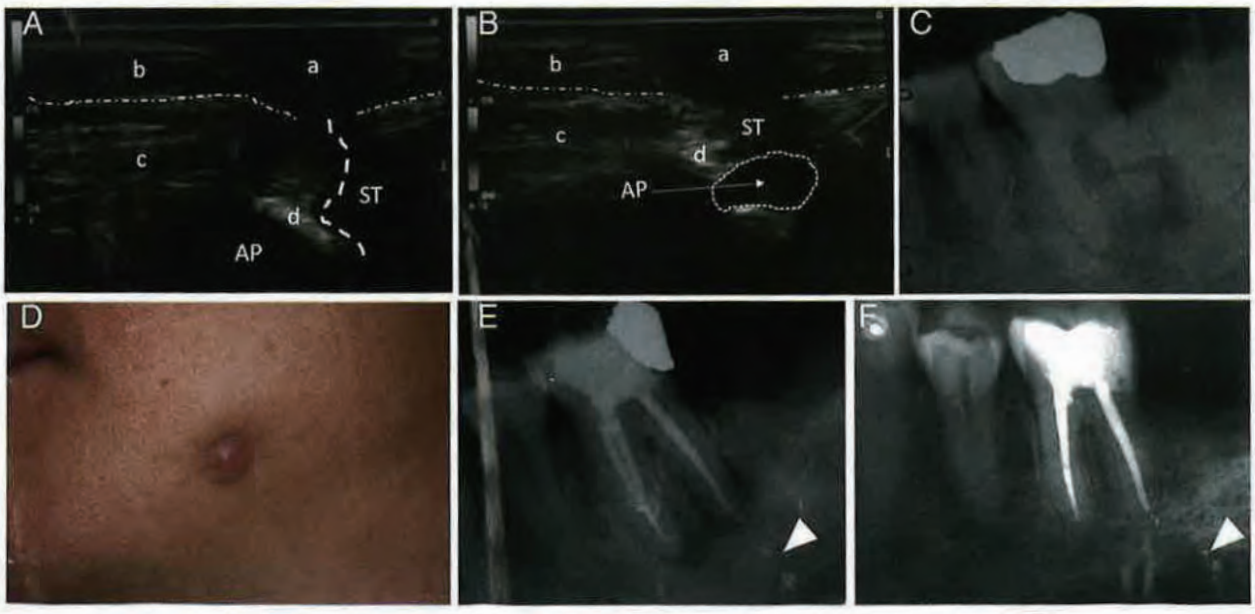


FIGURE 3 – (*A*) An ecographic image of an extraoral ST (*interrupted line*) representing the (*a*) opening at the (*b*) skin surface (*fragmented contour*), the (*c*) muscular layer, and the (*d*) mandibular bone. (*B*) Another perspective of the same lesion showing the whole AP (*interrupted contour*). (*C*) A periapical radiograph of the same lesion on tooth #18. (*D*) Clinical detail of the cutaneous stoma. (*E*) A postoperative ecographic image showing the extruded root canal sealer following the ST (*arrowhead*). (*F*) A 3-month postoperative radiograph showing the progressive movement of the sealer (*arrowhead*) and healing of the lesion.

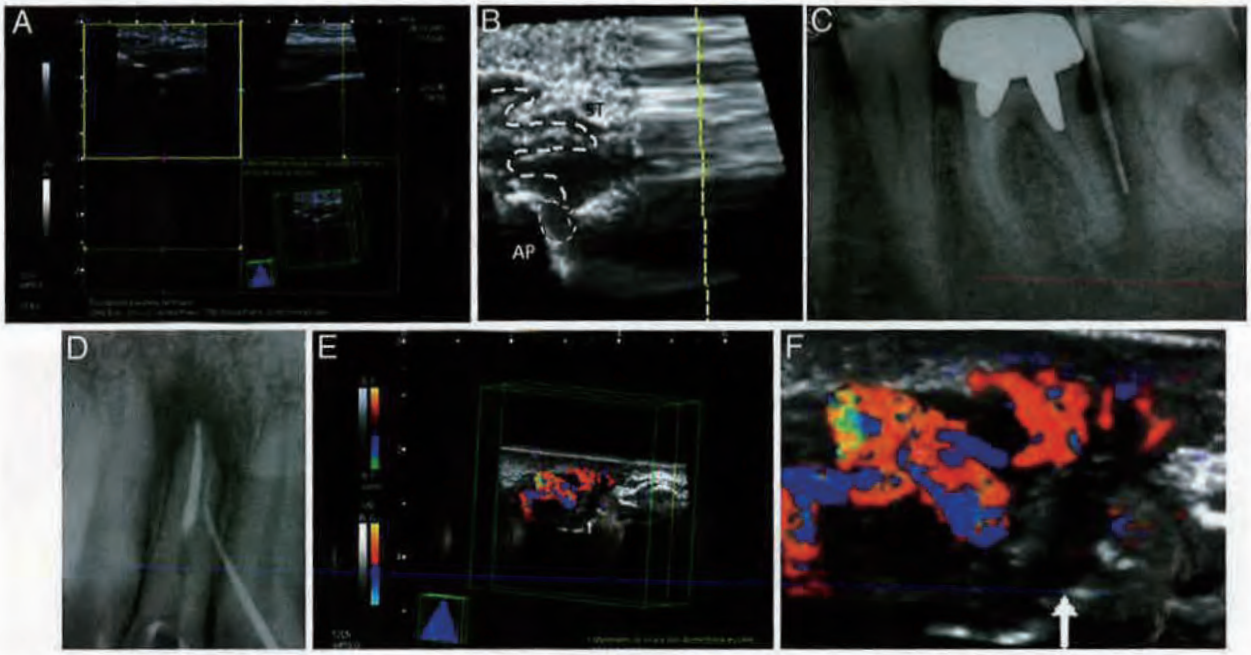


FIGURE 4 – (*A*) An APST case visualized in the 3D elaboration mode. (*B*) Tridimensional reconstruction of AP (*interrupted contour*) and the ST (*interrupted line*). (*C*) A periapical radiograph of the same case and tracing of the tract (tooth #18). (*D*) An APST case traced with a gutta-percha cone and a periapical radiograph (tooth #7). (*E*) Evaluation of the vascularity in the same case using CPD. (*F*) CPD detail showing the ST (*arrow*) and identifying the presence of small vessels at the walls of the ST lumen.

healing by 48%[28] and to increase the hazard of tooth loss by 120%[29]. The difficulty in the healing of STs has been explained in the past with the presence of an epithelial lining throughout the tract[8,30], which would hamper the closure of the pathologic pathway despite the adequate treatment of the root canal infection. Epithelialization as the cause of failure has been dispelled by the evidence that the persistence of infection was a more important reason for the lack of resolution of the ST[11,31]. Concentrations above the average of specific pathogens in intraradicular infection have been considered responsible for the poorer prognosis of teeth with chronic apical abscesses; yet, no significant differences were found between cases with and without STs in terms of total bacterial counts[32]. Finally, the persistence of STs has been attributed to the presence of extraradicular infections such as actinomycosis[33], but there is still no definite evidence that an extraradicular infection alone can sustain refractory periapical inflammation[34,35]. Indeed, in a recent histobacteriologic study on 24 biopsy specimens from roots of teeth associated with AP and an ST[36], the authors highlighted the complexity of the infection that characterized those cases. They reported that 83% of the samples had a compound infectious pattern that involved the canal system, the periapical lesion, and the extraradicular fraction of the root, the latter always derived from the intraradicular biofilm present in all canals. To our knowledge, this is the first experimental study designed to assess the potential of US to scan STs, track their pathways, and evaluate their vascularity. Only 1 article in the field of dermatology has reported the application of US in 3 cases of cutaneous STs of endodontic origin at first misdiagnosed and overtreated with invasive and unnecessary interventions. In the mentioned study, a linear high-frequency US probe was used to investigate the lesions in the skin; the tracts were described as hypoechoic bands connecting the skin to the alveolar bone with a distinguished CPD signal at the periphery of 2 lesions, as observed in some of our cases, yet they did not highlight the pathway of the STs[37]. The results of this study showed that US examination was feasible to visualize and track the path of an ST of endodontic origin. The common procedure used to trace a chronic apical abscess contemplated the insertion of a gutta-percha cone through the opening of the ST followed by a periapical radiograph of the area to achieve an indirect image of the tract (Fig. 2*B* and *E*)[14,16]. With US imaging, the route of drainage of the fluid can be completely imaged from the periapical lesion to the stoma of the ST (Figs. 2 and 3). The reconstruction of the images with the 3D mode can also disclose 3-dimensionally the pathway of the tract and illustrate its irregularities and curves. The implementation of the examination with CPD further allows the assessment of the vascular pattern and, consequently, the inflammatory response of the tissues surrounding the ST. Moreover, the technique has the serious advantage of not using ionizing radiations[38]. The choice of a linear multifrequency probe in this experiment

TABLE 2 – Data Analysis of the Results Obtained by the Blinded Observers during the Different Examinations

Observer	Examination	Sensibility	Specificity	PPV	NPV	ROC	Significance ($P < .05$)
Observer 1	1	100	100	100	100	100	$P = .0$
Observer 2	1	90	100	100	91	95	$P = .001$
Observer 1	2	100	100	100	100	100	$P = .0$
Observer 2	2	90	100	100	91	95	$P = .001$

NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristic.

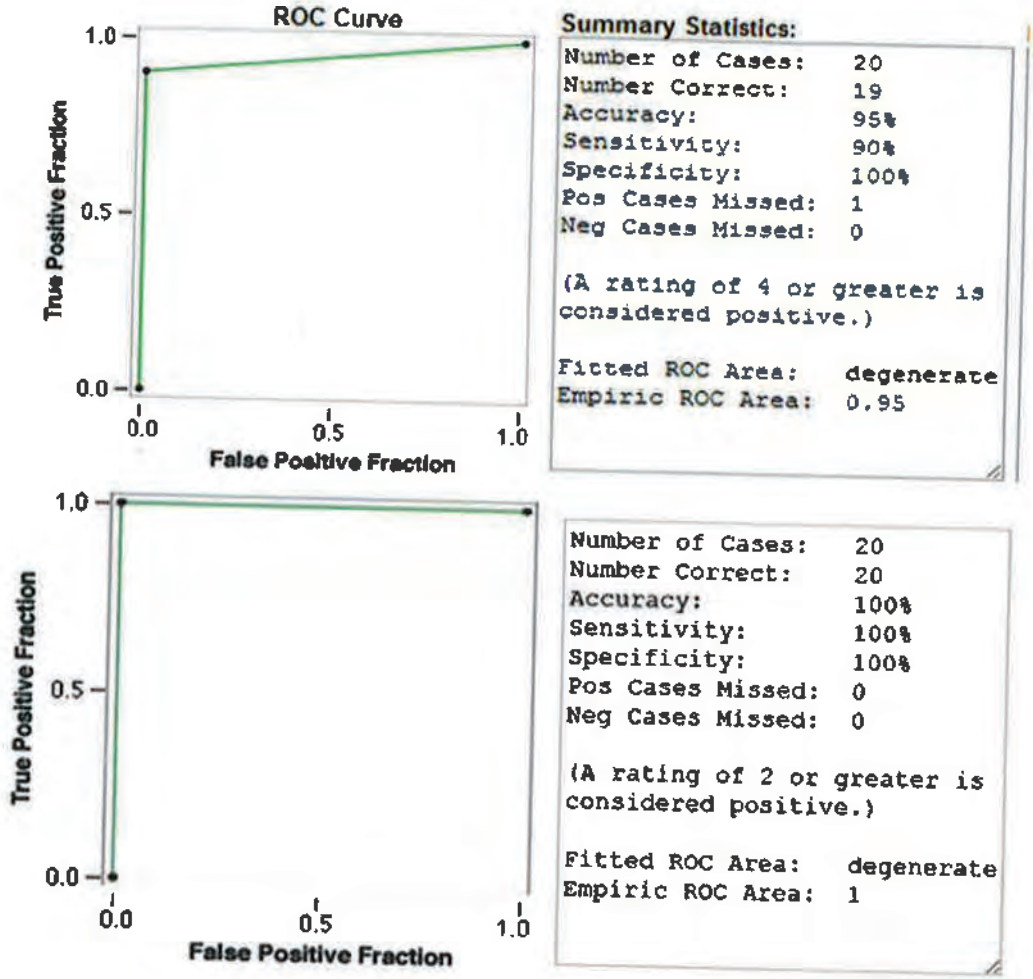


FIGURE 5 – Receiver operating characteristic curve (area under the curve) analysis showing the diagnostic accuracy of the technique for the blinded observers.

is justified because lower frequencies can penetrate deeper within the bone defect, whereas higher frequencies permit visualization of the soft tissues and the STs with a better definition[39]. Although the examination should be performed in real time to achieve the most from its diagnostic power, the analysis of the images performed from the blinded examiners after their acquisition showed high values of sensibility, specificity, and accuracy (Table 2). The time necessary to obtain the US image of the ST on a selected area is longer than making a radiograph; on the other hand, once the image is obtained, using the CPD or the 3D extension is relatively fast.

The small number of cases constitutes 1 of the main limitations of the statistical significance of the present study, and in order to determine the power of the associations of the observations, we have performed a statistical test of significance. One of the reported limitations of US examination is that it requires a learning curve and that the interpretation of the images necessitates training and a certain level of experience[23,27]. Nevertheless, good training may enable most clinicians to perform and analyze the images because the anatomic layers that need to be crossed by the US waves to reach the lesions and the STs are the skin, the alveolar mucosa, and the cortical bone plate.

CONCLUSION

US real-time examination can be successfully used to detect the STs of endodontic origin and to trace their route of drainage from the periapical lesion to the opening within the oral mucosa or the skin. Future studies are needed to standardize the technique. To date, the importance of this achievement may be limited to the descriptive diagnostic field.

ACKNOWLEDGMENTS

The authors deny any conflicts of interest related to this study.

REFERENCES

1. American Association of Endodontists. Glossary of Endodontic Terms. 9th ed. American Associations of Endodontists (AAE); 2016. Available at: http://www.nxtbook.com/nxtbooks/aae/endodonticglossary2016/index.php. Accessed October 9, 2018.
2. Abbott PV. Classification, diagnosis and clinical manifestations of apical periodontitis. Endod Topics 2004;8:36–54.

3. Torabinejad M, Shabahang S. Pulp and periapical pathosis. In: Torabinejad M, Walton RE, Fouad AF, editors. Endodontics. Principles and Practice. 5th ed. St Louis, MO: Saunders/ Elsevier; 2015. p. 48–67.

4. Gupta R, Hasselgren G. Prevalence of odontogenic sinus tracts in patients referred for endodontic therapy. J Endod 2003;12:798–800.

5. Miri SS, Atashbar O, Atashbar F. Prevalence of sinus tract in the patients visiting department of endodontics, Kermanshah school of dentistry. Glob J Health Sci 2015;6:271–5.

6. Slutzky-Goldberg I, Tsesis I, Slutzky H, Heling I. Odontogenic sinus tracts: a cohort study. Quintessence Int 2009;40:13–8.

7. Soğur E, Sen HB, Baksı GB, Mert A. Prevalence of odontogenic sinus tracts in patients referred for endodontic therapy. Cumhuriyet Dent J 2013;16:282–8.

8. Vire DE, Stalker WH, Kessler HP. Epithelium-lined oral sinus tract. Oral Surg 1982;53:209–11.

9. Mittal N, Gupta P. Management of extra oral sinus cases: a clinical dilemma. J Endod 2004;30:541–7.

10. McWalter GM, Alexander JB, del Rio CE, Knott JW. Cutaneous sinus tracts of dental etiology. Oral Surg 1988;66:608–14.

11. Spear KL, Sheridan PJ, Perry HO. Sinus tracts to the chin and jaw of dental origin. J Am Acad Dermatol 1983;8:486–92.

12. Gupta M, Das D, Kapur R, Sibal N. A clinical predicament—diagnosis and differential diagnosis of cutaneous facial sinus tracts of dental origin: a series of case reports. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112:e132–6.

13. Heling I, Rotstein I. A persistent oronasal sinus tract of endodontic origin. J Endod 1989;15:132–4.

14. Baumgartner JC, Picket AB, Muller JT. Microscopic examination of oral sinus tracts and their associated periapical lesions. J Endod 1984;10:146–52.

15. Johnson BR, Remeikis NA, Van Cura JE. Diagnosis and treatment of cutaneous facial sinus tracts of dental origin. J Am Dent Assoc 1999;130:832–6.

16. Bender IB, Seltzer S. The oral fistula: its diagnosis and treatment. Oral Surg Oral Med Oral Pathol 1961;64:139–48.

17. Moore CL, Copel JA. Point-of-care ultrasonography. N Engl J Med 2011;364:749–57.

18. Miller DL. Safety assurance in obstetrical ultrasound. Semin Ultrasound CT MR 2008;29:156–64.

19. Mohan KR, Koteswara RN, Leela KG, et al. Role of ultrasonography in oral and maxillofacial surgery: a review of literature. J Maxillofac Oral Surg 2015;14:162–70.

20. Cotti E, Simbola V, Dettori C, Campisi G. Echographic evaluation of bone lesions of endodontic origin: report of two cases in the same patient. J Endod 2006;32:901–5.

21. Rajendran N, Sundaresan B. Efficacy of ultrasound and color power Doppler as a monitoring tool in the healing of endodontic periapical lesions. J Endod 2007;33:181–6.

22. Cotti E, Esposito SA, Musu D, et al. Ultrasound examination with color power Doppler to assess the early response of apical periodontitis to the endodontic treatment. Clin Oral Investig 2018;22:131–40.

23. Curvers F, Meschi N, Vanhoenacker A, et al. Ultrasound assessment of bone healing after root-end surgery: echoes back to patient's safety. J Endod 2017;44:32–7.

24. Hargraves K, Berman LH. Cohen's Pathways of the Pulp Expert Consult. 11th ed. Philadelphia: Elsevier Health Sciences; 2016. p. 2–32.

25. Ørstavik D, Kerekes K, Eriksen HM. The periapical index: a scoring system for radiographic assessment of apical periodontitis. Endod Dent Traumatol 1986;2:20–34.

26. European Society of Endodontology. Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology. Int Endod J 2006;39:921–30.

27. Cotti E, Campisi G, Garau V, Puddu G. A new technique for the study of periapical bone lesion; ultrasound real time imaging. Int Endod J 2002;35:148–52.

28. Ng YL, Mann V, Gulabivala K. A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 1: periapical health. Int Endod J 2011;44:583–609.

29. Ng YL, Mann V, Gulabivala K. A prospective study of the factors affecting outcomes of nonsurgical root canal treatment: part 2: tooth survival. Int Endod J 2011;44:610–25.

30. Sommer RF, Ostrander FD, Crowley MC, editors. Clinical Endodontics. Philadelphia: WB Saunders; 1956. p. 267–70.

31. Harrison JW, Larson WJ. The epithelized oral sinus tract. Oral Surg Oral Med Oral Pathol 1976;42:511–7.

32. Sassone LM, Fidel R, Faveri M, et al. Microbiological evaluation of primary endodontic infections in teeth with and without sinus tract. Int Endod J 2008;41:508–15.

33. Happonen RP. Periapical actinomycosis: a follow-up study of 16 surgically treated cases. Endod Dent Traumatol 1986;2:205–9.

34. Ricucci D, Siqueira Jr JF. Apical actinomycosis as a continuum of intraradicular and extraradicular infection: case report and critical review on its involvement with treatment failure. J Endod 2008;34:1124–9.

35. Siqueira JF Jr, Rôças IN, Ricucci D, Hulsmann M. Causes and management of posttreatment apical periodontitis. Br Dent J 2014;216:305–12.

36. Ricucci D, Loghin S, Gonçalves LS, et al. Histobacteriologic conditions of the apical root canal system and periapical tissues in teeth associated with sinus tracts. J Endod 2018;44:405–13.

37. Shobatake C, Miyagawa F, Fukumoto T, et al. Usefulness of ultrasonography for rapidly diagnosing cutaneous sinus tracts of dental origin. Eur J Dermatol 2014;24:683–7.

38. Kurtz AB. American institute of ultrasound in medicine. The AIUM celebrates 50 years of excellence. Available at: https://www.aium.org/aboutUs/history/articles/jum1.pdf. Accessed October 9, 2018.

39. Girish G, Jacobson JA. Ultrasound. In: Weissman BN, editor. Imaging of Arthritis and Metabolic Bone Disease. Philadelphia: Saunders; 2009. p. 89–103.

Youngjun Kwak, DDS, MSD, PhD,* Jungkyu Choi, PhD,† Kiyeol Kim, PhD,‡ Su-Jung Shin, DDS, MSD, PhD,§ Sunil Kim, DDS, MSD, PhD,‖ and Euiseong Kim, DDS, MSD, PhD‖¶

The 5-Year Survival Rate of Nonsurgical Endodontic Treatment: A Population-based Cohort Study in Korea

SIGNIFICANCE

It was confirmed that teeth treated with nonsurgical endodontic treatment showed high survival rate after 5 years (initial RCT, 90.85%; re-RCT, 88.42%). The big data study confirmed that re-RCT showed a statistically significant higher rate of extraction in the 0–1-year (within 12 months) interval than in other time intervals.

From the *Department of Conservative Dentistry, Yonsei University College of Dentistry, Seoul, Korea; †Institute of Health Insurance and Clinical Research, National Health Insurance Service Ilsan Hospital, Goyang, Korea; ‡Dental Education Research Center, Yonsei University College of Dentistry, Seoul, Korea; §Department of Conservative Dentistry, Gangnam Severance Dental Hospital, Yonsei University College of Dentistry, Seoul, Korea; ‖Microscope Center, Department of Conservative Dentistry and Oral Science Research Center, Yonsei University College of Dentistry, Seoul, Korea; and ¶Department of Electrical & Electronic Engineering, Yonsei University College of Engineering, Seoul, Korea

Address requests for reprints to Dr Euiseong Kim or Sunil Kim, Microscope Center, Department of Conservative Dentistry and Oral Science Research Center, Yonsei University College of Dentistry, 50-1 Yonsei-Ro, Seodaemun-Gu, Seoul 03722, Korea.
E-mail addresses: seone1@yuhs.ac or endyendo@yuhs.ac
0099-2399/$ - see front matter

https://doi.org/10.1016/j.joen.2019.07.004

ABSTRACT

Introduction: The purpose of the current population-based study was to analyze the 5-year survival rate of teeth that received nonsurgical endodontic treatment. Specifically, the variables affecting the 5-year survival rates of endodontically treated teeth were analyzed. **Methods:** The present study included all endodontically treated teeth reported in 2010 in the National Health Insurance Cohort database of the Republic of Korea. By tracing the dental records of the sample to the end of 2015, the 5-year survival rates of the initial root canal treatment (RCT) and re-RCT were analyzed. Gender, age, institution type, diagnosis, arch type, tooth type, number of visits, and rubber dam usage were included in the analyses as confounding variables. **Results:** In total, 2,866,749 teeth received initial RCT, and 173,429 teeth received re-RCT. Five-year survival rates were 90.85% and 88.42%, respectively. The survival rate of teeth that received initial RCT was significantly higher than that of those that received re-RCT. Specifically, re-RCT showed a significantly higher rate of extraction within 12 months than during other intervals. Furthermore, the following characteristics significantly positively affected the 5-year tooth survival rate: being female, <20 years of age, hospital as the institution type, mandibular teeth, anterior teeth, initial RCT, and use of rubber dam. **Conclusions:** Because of its high 5-year survival rate, endodontic treatment is an effective method for maintaining natural teeth. *(J Endod 2019;45:1192–1199.)*

KEY WORDS

Endodontic treatment; initial root canal treatment; population-based study; re-RCT; survival rate

The purpose of endodontic treatment is to either maintain or restore natural teeth by preventing and eliminating apical periodontitis[1]. Recent studies have shown that endodontic treatment is effective for maintaining natural teeth with high success rates[1–3]. Although initial root canal treatment (RCT) is a promising treatment because of its high success and survival rates, failures may eventually occur. Recent studies have reported failure rates of 7%–16% after initial RCT[1,4,5]. After initial RCT failure, re-RCT is often performed because of its minimally invasive approach and high 5-year tooth survival rate of 89%[6]. However, Riis et al[7] reported re-RCT survival rate to be 76% in a randomized clinical trial, suggesting a lower survival rate compared with that of initial RCT.

Studies examining the success or survival rates of teeth that received endodontic treatment during a period of time may report varying results depending on the study design (prospective or retrospective) and whether the success or survival rate is investigated. In a prospective study, the survival rate of 759 teeth that received initial RCT was 95.4%, and that of 858 teeth that received re-RCT was 95.3%[8]. However, a retrospective study demonstrated success rates of 94.0% and 85.9% for initial and re-RCT in 1376 teeth and 624 teeth, respectively[9]. These results indicate that it is necessary to investigate 5-year outcomes by using multiple methods to accurately interpret success rates.

Recently, medical data pools have increased exponentially because of breakthroughs in data collection and storage technology. In endodontics, a population-based analysis for examining the survival rate of endodontically treated teeth can be efficient, complementing conventional methodologies[10]. The advantage of a population-based study analysis is the abundant information about the patients,

providers, clinical settings, and techniques that is quickly gathered[10,11]. This allows for new insights and scientific discoveries in many fields, including dental research, that may inform treatment outcomes.

The purpose of this population-based study was to investigate the 5-year survival rates of endodontically treated teeth. Data from the National Health Insurance Cohort Database (NHICD) provided by the Health Insurance Review and Assessment Service were analyzed to determine the variables affecting the 5-year survival rates of endodontically treated teeth. These results will help inform clinicians about the factors they need to consider when performing nonsurgical endodontic treatment.

MATERIALS AND METHODS

Study Population and Exclusion Criteria

The current study was approved by the Institutional Review Board of Yonsei University Dental Hospital (approval number: 2-2016-0039). Except for the cases that qualified for exclusion, all recorded data related to teeth that received nonsurgical endodontic treatment (ie, initial RCT, re-RCT) throughout the Republic of Korea in 2010 were included. By tracing the dental records of the sample to the end of 2015, the 5-year survival rates of initial RCT and re-RCT were analyzed. All of the extraction codes are included in the reimbursable items in the Korean National Health Insurance data set. Therefore, teeth that were not coded as extracted were assumed to be surviving at the end of 2015.

The exclusion criteria for teeth that received initial RCT and re-RCT were as follows:

1. involvement of deciduous teeth;
2. involvement of third molars;
3. pulpotomy; and
4. data errors.

Each treatment method was identified by a specific treatment code (initial RCT: U0060, U0121, and U0126; re-RCT: U2245). Cases were considered to have data errors when extraction codes were applied before the release of endodontic treatment codes.

Confounding Variables

The confounding variables that were extracted from the insurance cohort database included the treatment type, gender, age, institution type, diagnosis, arch type, tooth type, canal filling method, number of visits, and use of rubber dam. Age was divided into 4 groups (ie, <20, 20–39, 40–64, ≥65). The institution type variable indicated where the patients underwent the treatment and was divided into 2 groups (ie, dental hospital [code number 41] and local dental clinic [code number 51]). The top 10 diagnoses codes (ie, asymptomatic irreversible pulpitis [NHICD code: caries of dentin and caries of cementum], symptomatic irreversible pulpitis [NHICD code: pulpitis], pulp necrosis [NHICD code: pulp necrosis and pulp degeneration], symptomatic apical periodontitis [NHICD code: acute apical periodontitis], asymptomatic apical periodontitis [NHICD code: chronic apical periodontitis and chronic periodontitis], chronic apical abscess [NHICD code: periapical abscess with sinus], and acute apical abscess [NHICD code: periapical abscess without sinus]) for endodontic treatment were selected within the scope of the NHICD. The arch-type variable indicated maxillary or mandibular teeth. The tooth-type variable indicated anterior, premolar, or molar teeth. The number of visits was categorized as single or multiple. Finally, use of a rubber dam by the medical professional was also investigated in the present study.

Statistical Analyses

The χ^2 test was performed to compare the cumulative survival rate for a fixed period (0–1 year, 1–2 years, 2–3 years, 3–4 years, 4–5 years, and 5 years) after nonsurgical endodontic treatment in 2010. For the primary analysis, the Cochran–Mantel–Haenszel χ^2 test, with an adjustment for the treatment type (ie, initial RCT, re-RCT), was conducted to test whether the extraction rate was different between groups. Specifically, the Cochran–Armitage trend test was performed to analyze the association between the extraction rate and the follow-up period after either initial RCT or re-RCT. Simple and multiple Cox regression analyses were used to evaluate the significance of the treatment method. Covariates included gender, age, institution type, arch type, tooth type, treatment type, number of visits, and use of rubber dam. Whereas the survival probability was calculated by using Kaplan–Meier analysis with a 95% confidence level, a log-rank test was conducted to determine whether significant differences existed. The SAS statistical package (version 9.4; SAS Institute, Cary, NC) was used to perform all the analyses in the present study. A P value $<.05$ was considered significant.

RESULTS

A flowchart describing the enrollment process for the current study can be found in Figure 1. The total number of teeth included in the database was 3,703,450. The following cases were excluded: 455,347 deciduous teeth, 47,896 third molars, 152,868 pulpotomy cases, and 7161 cases with data errors. After exclusion, 3,040,178 teeth were included in the initial RCT and re-RCT study. The number of teeth that received initial RCT was 2,866,749, with a 5-year survival rate of 90.85% (2,604,456/2,866,749). The number of teeth that underwent re-RCT was 173,429, with a 5-year survival rate of 88.42% (153,339/173,429) (Tables 1 and 2). The extraction rates after initial RCT were 1.58% (0–1 year), 1.46% (1–2 years), 1.58% (2–3 years), 1.72% (3–4 years), 1.85% (4–5 years), and 0.96% (5 years) The extraction rates after re-RCT were 3.24% (0–1 year), 1.81% (1–2 years), 1.83% (2–3 years), 1.79% (3–4 years), 1.94 (4–5 years), and 0.97% (5 years) (Table 1).

Figure 2 shows the cumulative survival probabilities of those teeth that received initial RCT and re-RCT, using the Kaplan–Meier survival analysis. The 5-year survival rate was higher for those teeth that underwent initial RCT than those that received re-RCT. All variables except the number of visits (P = .4298) were statistically significant (Table 2).

Table 3 illustrates the results of the Cochran–Armitage trend test for the extraction rate (100% survival rate) and the follow-up period after either initial RCT or re-RCT. Although the extraction rate was found to increase with time in the initial RCT cases, the re-RCT cases had a significantly higher extraction rate at the 12-month time point (1.58% for initial RCT vs 3.24% for re-RCT) (Tables 1 and 2). The difference between RCT and re-RCT extraction rates decreased during the 5-year period but remained higher in the re-RCT group. Those teeth that received re-RCT presented a statistically higher extraction rate in the 0–1-year interval than in all the other intervals.

Table 4 shows the results of the simple and multiple Cox proportional hazard analyses of contributing risk factors. These include the patient characteristics, institution type, arch type, tooth type, treatment type, number of visits, and use of rubber dam by the medical professional for tooth extraction after endodontic treatment. The tooth extraction rate for female patients was found to be significantly less than that for male patients (hazard ratio [HR] = 0.68; 95% confidence interval [CI], 0.67–0.68; $P < .0001$). Furthermore, the extraction rate was significantly lower in the group younger than 20 years than in the other age groups, with extraction rate increasing with age ($P < .0001$). The tooth extraction rate was statistically lower when extraction was performed at the hospital,

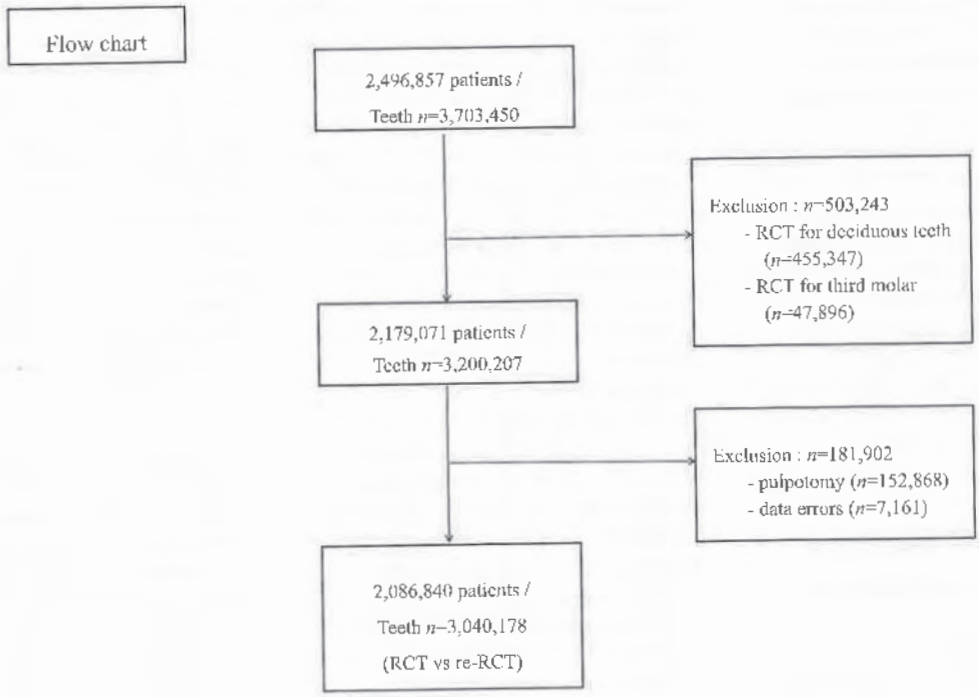


FIGURE 1 – Flowchart describing the enrollment process for the current study.

as well as when it was performed on mandibular teeth and anterior teeth ($P < .0001$). Moreover, the extraction rate of teeth that received re-RCT was 1.44 times higher than that of teeth that underwent initial RCT (HR = 1.44; 95% CI, 1.42–1.46; $P < .0001$). Finally, the extraction rate was significantly lower when rubber dam was used (HR = 0.84; 95% CI, 0.83–0.85; $P < .0001$) than when it was not used. In contrast, a statistical difference in the extraction rate based on the number of visits was not observed ($P = .5745$).

DISCUSSION

This retrospective, population-based analysis included the majority of RCT and re-RCT teeth extractions among the entire Korean population in 2010. Because Koreans are obliged to join the health insurance, all nonsurgical endodontic treatments conducted in the Republic of Korea would be included in the present study except for those in a few overseas Koreans. All teeth reported to receive endodontic treatment in any clinical field in the Republic of Korea in 2010 were included in the current research. Therefore, a large sample size (>3 million teeth) and the long follow-up periods (>5 years) after endodontic treatment rendered results more reliable and eliminated the various biases in previous studies. Furthermore, because of the advantages provided by population-based analysis, a vast amount of data obtained between 2010 and 2015 was quickly available to observe the role of multiple variables on teeth extraction. Importantly, considering that few investigations have used population-based analysis in the endodontic field, the current study could have strong significance and implications.

Ng et al[8] reported the 4-year cumulative survival rates of teeth that received initial RCT and re-RCT as statistically similar (95.4% and 95.3%, respectively). However, in the present study, the 5-year survival rate of teeth that received initial RCT was found to be significantly higher than that of those teeth that received re-RCT (Tables 1–3). Loss of tooth structure may contribute to a lower survival rate of teeth undergoing re-RCT than those undergoing initial RCT. Shemesh et al[12] reported that retreatment procedures could significantly damage the root and result in cracks and fractures. Another factor affecting the difference in survival rate between initial RCT and re-RCT is efficiency of disinfection. Preexisting root canal filling materials, post and core, and separated files can limit the

TABLE 1 - The 5-Year Survival Rates of Teeth That Received Initial RCT and re-RCT in 2010

	Initial RCT (n = 2,866,749)				Re-RCT (n = 173,429)				P value
	Survival teeth (n, %)		Extracted teeth (n, %)		Survival teeth (n, %)		Extracted teeth (n, %)		
Time interval									
0–1 year	2,821,414	98.42	45,335	1.58	167,808	96.76	5621	3.24	<.0001
1–2 years	2,779,675	96.96	41,739	1.46	164,666	94.95	3142	1.81	
2–3 years	2,734,380	95.38	45,295	1.58	161,489	93.12	3177	1.83	
3–4 years	2,684,908	93.66	49,472	1.72	158,396	91.33	3093	1.79	
4–5 years	2,631,972	91.81	52,936	1.85	155,033	89.39	3363	1.94	
5 years	2,604,456	90.85	27,516	0.96	153,339	88.42	1694	0.97	

P values were calculated by χ^2 tests.

TABLE 2 - The 5-Year Survival Rates of Teeth That Received Initial RCT and re-RCT in 2010 Based on the Confounding Variables (n = 3,040,178)

Variables	Initial RCT (n = 2,866,749)		Re-RCT (n = 173,429)		P value
	Survival teeth (n, %)		Survival teeth (n, %)		
Gender					
Male	1,265,870	89.19	64,059	86.11	<.0001
Female	1,338,586	92.47	89,280	90.15	
Age (y)					
<20	217,169	96.21	9321	94.23	<.0001
20–39	718,699	95.84	55,131	93.44	
40–64	1,279,866	89.34	71,544	86.42	
≥65	388,722	84.77	17,343	79.73	
Institution type					
Hospital	98,408	92.45	12,112	90.89	<.0001
Local clinic	2,506,048	90.79	141,227	88.21	
Top 10 diagnoses codes					
Asymptomatic irreversible pulpitis					
Caries of dentin	56,829	93.77	2393	90.00	<.0001
Caries of cementum	29,742	93.45	811	89.32	
Symptomatic irreversible pulpitis					
Pulpitis	2,009,701	91.23	74,635	88.43	
Pulp necrosis					
Pulp necrosis	48,536	90.66	4002	90.09	
Pulp degeneration	26,468	92.03	2237	90.02	
Symptomatic apical periodontitis					
Acute apical periodontitis	50,079	90.32	2413	88.36	
Asymptomatic apical periodontitis					
Chronic apical periodontitis	41,472	87.97	6672	89.38	
Chronic periodontitis	29,001	86.77	2170	86.59	
Chronic apical abscess					
Periapical abscess with sinus	42,591	86.75	7992	86.86	
Acute apical abscess					
Periapical abscess without sinus	168,175	87.63	41,747	88.31	
Arch type					
Maxillary teeth	1,474,345	90.74	84,924	89.23	<.0001
Mandibular teeth	1,130,111	90.99	68,415	87.42	
Tooth type					
Anterior	588,646	91.48	36,959	92.16	<.0001
Premolar	734,889	91.50	40,142	88.72	
Molar	1,280,921	90.19	76,238	86.56	
No. of visits					
Single	1634	90.88	592	90.11	.4298
Multiple	2,602,822	90.85	152,747	88.41	
Use of rubber dam					
Without rubber dam	2,328,964	90.62	129,906	88.14	<.0001
With rubber dam	275,492	92.82	23,433	89.98	

P values were calculated by Cochran–Mantel-Haenszel χ^2 tests.

penetration of disinfectants and decrease the effectiveness of re-RCT[13,14]. Removing contaminated root canal material is important in re-RCT; however, this process is very challenging[15]. The bacteria detected in persistent infection have more diverse bacterial communities than those detected in primary infection and may have a negative effect on survival rate[16]. Finally, microorganisms detected in persistent infection, such as *Enterococcus faecalis*, tend to be more resistant to intracanal medicaments[17].

Moreover, the extraction rate of teeth that received initial RCT (1.58%) was less than half that of those teeth that received re-RCT (3.24%) in the 0–1-year period. Similar patterns were observed in the other periods, ie, 1.46% vs 1.81%, 1.58% vs 1.83%, 1.72% vs 1.79%, 1.85% vs 1.94%, and 0.96% vs 0.97%, respectively. Those teeth that received re-RCT presented the highest extraction rate in the 0–1-year period; this gradually decreased during the following years. Therefore, teeth that receive re-RCT require both an active recall check within 1 year from the treatment and an effort to maintain the natural teeth through either additional re-RCT or apical surgery when needed rather than extraction at an early point. These findings validate previous findings indicating the occurrence of chronic apical periodontitis or healing within the first year after endodontic treatment[17].

In the current study, it was noted that the survival rate of teeth after nonsurgical endodontic treatment was significantly higher in female patients than in male patients. Several reasons for this difference are possible. These include prosthetic problems, vertical root fracture (VRF), periodontal problems, endodontic problems, and dental caries[18,19]. With regard to VRF, a previous study indicated that male patients are at higher risk of tooth extraction than female patients[20], considering that they present stronger occlusal bite forces, increased attrition, a habit of chewing harder and tougher food, and a less flexible supporting bone structure than that presented by female patients[21]. In addition, such a result

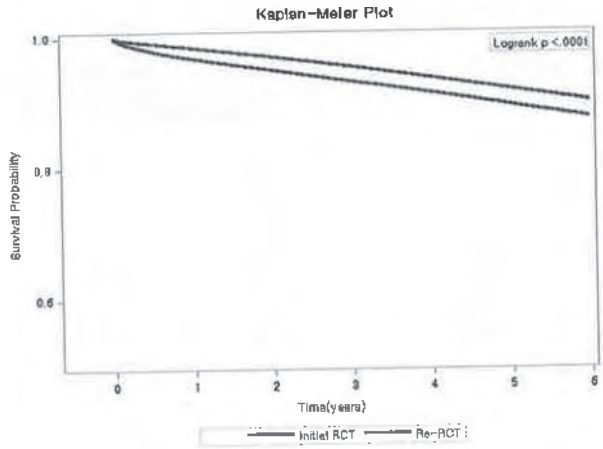


FIGURE 2 – Cumulative survival probabilities for teeth that received initial RCT and re-RCT.

can be interpreted as a loss of the protective pulpal reflexes because of the reduction of the crucial protective feedback and pulp proprioception in male patients[22]. However, several previous studies have reported the difference in incidence of extraction between male and female patients to be statistically insignificant[19,23,24]. This may result from the differences in eating and oral habits among Koreans as well as the increased statistical power of population-based studies because of their large sample sizes. The incidence of teeth extraction because of periodontal problems after endodontic treatment varied between 5.5% and 40.3%[19,25]. In addition, a previous report described that periodontitis occurred more frequently in male patients because female patients had better oral hygiene than male patients, and access to dental care is easier for female patients[26,27]. Therefore, there are many potential contributing factors to the statistically significant difference between male and female treatment outcomes.

In this study, the survival rate decreased with age, which is in concordance with previous investigations[23,28]. There are several reasons for considering young age as a positive prognostic factor for tooth survival rate. For instance, VRF is less common in the younger age groups because their teeth are more resilient to the masticatory force[20]. Furthermore, young people have more residual tooth structure compared with older people because they have undertaken fewer restorative interventions[4].

In addition, a previous population-based cohort study showed that larger dental institutions are positively correlated with improved outcome[28], which is in concordance with the results from our investigation. This may be a result of the advanced devices used, such as the microscope and cone-beam computed tomography, or the improved skillful techniques used, including re-RCT and apical surgery. The survival rate appears to be even better in difficult cases when treated in dental hospitals[23]. In part, this may be due to the use of rubber dams by students and residents in educational institutions, which positively affects the survival rates[29].

Similar to previous studies, the difference in survival rates between one-visit and multiple-visit RCTs was not significant in the current investigation. According to a recent study, a trial comparing one-visit and multiple-visit RCTs described them to have similar success rates[30]. In addition, a systematic review and meta-analysis of cases with apical periodontitis identified an absence of difference in the healing rates between the 2 treatment methods (P = .3809)[31], confirming the results obtained in the present study.

Although the rate of endodontic treatment with rubber dam was 10.62% in the current study, it was 16.5% in the population-based study of the National Health Insurance Research Database in Taiwan[32]. According to previous research, the ratio of the use of rubber dam varied from 10% to 89%[29]. Use of rubber dams has been associated with reportedly higher tooth survival rates in several studies[24,26,29,32,33]. Considering that it is possible to reduce 90%–98% of the microorganisms in dental treatment by using a rubber dam[33], using it during initial RCT results in a statistically higher survival rate[24].

This study has some limitations. All patients who received nonsurgical endodontic treatment throughout the Republic of Korea in 2010 were followed up to determine whether endodontically treated teeth were extracted by the end of 2015. Because the amount of data collected during this procedure exceeded the amount of data that the Health Insurance Review and Assessment Service could provide, only the first disease names for endodontic treatment stored in the NHICD were included in the present study, whereas the second and other disease names were not considered in the analyses. Furthermore, we assumed that the teeth that were not reported as extracted were surviving. However, some teeth may have been extracted outside of the national health insurance system. Such cases would reduce the reported survival rate.

In the present study, tooth survival rates after endodontic treatment were evaluated by the presence of the extraction code in the NHICD. In addition to the extraction, the evaluation of patient symptoms, the function of the teeth, and the assessment of the periapical radiographs may influence the results. Furthermore, although the main factor affecting the survival rate of teeth over time after endodontic treatment is the remaining coronal

TABLE 3 - Results of the Cochran–Armitage Trend Test Between the Rate of Extraction and the Follow-up Period after Either Initial RCT or re-RCT (n = 3,040,178)

Initial RCT			P value	Re-RCT			P value
Total initial RCT		44.99*	<.0001	Total Re-RCT		19.77	<.0001
0–1 y vs	1–2 y	9.91	<.0001	0–1 y vs	1–2 y	25.27	<.0001
	2–3 y	4.55*	<.0001		2–3 y	23.95	<.0001
	3–4 y	20.90*	<.0001		3–4 y	24.08	<.0001
	4–5 y	34.84*	<.0001		4–5 y	19.82	<.0001
1–2 y vs	2–3 y	14.36*	<.0001	1–2 y vs	2–3 y	1.20*	.229
	3–4 y	30.59*	<.0001		3–4 y	0.90*	.3663
	4–5 y	44.42*	<.0001		4–5 y	5.12*	<.0001
2–3 y vs	3–4 y	16.24*	<.0001	2–3 y vs	3–4 y	0.29	.77
	4–5 y	30.09*	<.0001		4–5 y	3.91*	<.0001
3–4 y vs	4–5 y	13.88*	<.0001	3–4 y vs	4–5 y	4.18*	<.0001

P values were calculated by Cochran–Armitage trend tests.
*Indicates increase in the extraction rate with time.

TABLE 4 - Total Simple and Multiple Cox Proportional Hazard Analyses of the Contributing Risk Factors, Which Include the Patients' Characteristics, Institution Type, Arch Type, Tooth Type, Treatment Type, Number of Visits, and Use of the Rubber Dam for Tooth Extraction after Either Initial RCT or re-RCT (n = 3,040,178)

Variables	Simple Cox regression		Multiple Cox regression	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Gender				
Female vs male	0.69 (0.68–0.69)	<.0001	0.68 (0.67–0.68)	<.0001
Age (y)				
20–39 vs <20	1.13 (1.10–1.15)	<.0001	1.09 (1.07–1.12)	<.0001
40–64 vs <20	2.91 (2.85–2.97)	<.0001	2.93 (2.87–2.99)	<.0001
≥65 vs <20	4.28 (4.18–4.37)	<.0001	4.56 (4.46–4.66)	<.0001
Institution type				
Local clinic vs hospital	1.22 (1.20–1.25)	<.0001	1.07 (1.05–1.09)	<.0001
Arch type				
Mandibular teeth vs maxillary teeth	0.99 (0.98–0.99)	.0004	0.93 (0.92–0.94)	<.0001
Tooth type				
Premolar vs anterior	1.02 (1.01–1.03)	.0001	1.11 (1.10–1.12)	<.0001
Molar vs anterior	1.19 (1.18–1.20)	<.0001	1.47 (1.45–1.48)	<.0001
Treatment type				
Re-RCT vs initial RCT	1.30 (1.28–1.32)	<.0001	1.44 (1.42–1.46)	<.0001
No. of visits				
Multiple visits vs single visit	1.00 (0.88–1.14)	1.0000	0.96 (0.85–1.10)	.5745
Use of rubber dam				
With R/D vs without R/D	0.77 (0.76–0.78)	<.0001	0.84 (0.83–0.85)	<.0001

CI, confidence interval; HR, hazard ratio; R/D, rubber dam.
P values were calculated by simple and multiple Cox regressions.

tooth structure, we were not able to investigate this variable in the current study[34]. In addition, it was not reported whether prosthetic treatment was performed after endodontic treatment, because prosthetic treatment in the Republic of Korea is not included in the insurance data. Moreover, it is not certain whether the extraction was caused by an endodontic problem.

ACKNOWLEDGMENTS

Youngjun Kwak and Jungkyu Choi contributed equally to this work as first author.

This study was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education (grant no. 2018R1D1A1A09081906).

The authors deny any conflicts of interest related to this study.

REFERENCES

1. Ng LYE, Mann V, Rahbaran S, et al. Outcome of primary root canal treatment: systematic review of the literature—part 2: influence of clinical factors. Int Endod J 2008;41:6–31.
2. Burry JC, Stover S, Eichmiller F, Bhagavatula P. Outcomes of primary endodontic therapy provided by endodontic specialists compared with other providers. J Endod 2016;42:702–5.
3. Chen SC, Chueh LH, Hsiao CK, et al. An epidemiologic study of tooth retention after nonsurgical endodontic treatment in a large population in Taiwan. J Endod 2007;33:226–9.
4. Ng YL, Mann V, Gulabivala K. Tooth survival following non-surgical root canal treatment: a systematic review of the literature. Int Endod J 2010;43:171–89.
5. Torabinejad M, Anderson P, Bader J, et al. Outcomes of root canal treatment and restoration, implant-supported single crowns, fixed partial dentures, and extraction without replacement: a systematic review. J Prosthet Dent 2007;98:285–311.
6. Salehrabi R, Rotstein I. Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment. J Endod 2010;36:790–2.
7. Riis A, Taschieri S, Del Fabbro M, Kvist T. Tooth survival after surgical or nonsurgical endodontic retreatment: long-term follow-up of a randomized clinical trial. J Endod 2018;44:1480–6.

8. Ng YL, Mann V, Gulabivala K. A prospective study of the factors affecting outcomes of non-surgical root canal treatment: part 2—tooth survival. Int Endod J 2011;44:610–25.
9. Imura N, Pinheiro ET, Gomes BP, et al. The outcome of endodontic treatment: a retrospective study of 2000 cases performed by a specialist. J Endod 2007;33:1278–82.
10. Dhawan A, Brand JC, Rossi MJ, Lubowitz JH. Big data: progress or a big headache? Arthroscopy 2018;34:649–51.
11. Brennan PF, Bakken S. Nursing needs big data and big data needs nursing. J Nurs Scholarsh 2015;47:477–84.
12. Shemesh H, Roeleveld AC, Wesselink PR, Wu MK. Damage to root dentin during retreatment procedures. J Endod 2011;37:63–6.
13. Bergenholtz G, Lekholm U, Milthon R, et al. Retreatment of endodontic fillings. Scand J Dent Res 1979;87:217–24.
14. Mollo A, Botti G, Prinicipi Goldoni N, et al. Efficacy of two Ni-Ti systems and hand files for removing gutta-percha from root canals. Int Endod J 2012;45:1–6.
15. Tzanetakis GN, Azcarate-Peril MA, Zachaki S, et al. Comparison of bacterial community composition of primary and persistent endodontic infections using pyrosequencing. J Endod 2015;41:1226–33.
16. Tennert C, Fuhrmann M, Wittmer A, et al. New bacterial composition in primary and persistent/secondary endodontic infections with respect to clinical and radiographic findings. J Endod 2014;40:670–7.
17. Orstavik D. Time-course and risk analyses of the development and healing of chronic apical periodontitis in man. Int Endod J 1996;29:150–5.
18. Olcay K, Ataoglu H, Belli S. Evaluation of related factors in the failure of endodontically treated teeth: a cross-sectional study. J Endod 2018;44:38–45.
19. Toure B, Faye B, Kane AW, et al. Analysis of reasons for extraction of endodontically treated teeth: a prospective study. J Endod 2011;37:1512–5.
20. Yoshino K, Ito K, Kuroda M, Sugihara N. Prevalence of vertical root fracture as the reason for tooth extraction in dental clinics. J Investig Clin Dent 2015;19:1405–9.
21. Koc D, Dogan A, Bek B. Bite force and influential factors on bite force measurements: a literature review. Eur J Dent 2010;4:223–32.
22. Awawdeh L, Hemaidat K, Al-Omari W. Higher maximal occlusal bite force in endodontically treated teeth versus vital contralateral counterparts. J Endod 2017;43:871–5.
23. Lazarski MP, Walker 3rd WA, Flores CM, et al. Epidemiological evaluation of the outcomes of nonsurgical root canal treatment in a large cohort of insured dental patients. J Endod 2001;27:791–6.
24. Lin PY, Huang SH, Chang HJ, Chi LY. The effect of rubber dam usage on the survival rate of teeth receiving initial root canal treatment: a nationwide population-based study. J Endod 2014;40:1733–7.
25. Fuss Z, Lustig J, Tamse A. Prevalence of vertical root fractures in extracted endodontically treated teeth. Int Endod J 1999;32:283–6.
26. Eke PI, Dye BA, Wei L, et al. Update on prevalence of periodontitis in adults in the United States: NHANES 2009 to 2012. J Periodontol 2015;86:611–22.
27. Costa FO, Lages EJ, Cota LO, et al. Tooth loss in individuals under periodontal maintenance therapy: 5-year prospective study. J Periodontal Res 2014;49:121–8.
28. Lee CB, Chang YH, Wen PC, Li CY. Association of failed root canal treatment with dentist and institutional volumes: a population-based cohort study in Taiwan. J Endod 2017;43:1628–34.
29. Ahmed HM, Cohen S, Levy G, et al. Rubber dam application in endodontic practice: an update on critical educational and ethical dilemmas. Aust Dent J 2014;59:457–63.
30. Dennis D. Similar success rates were achieved in single- and multiple-visit endodontic treatment. J Evid Based Dent Pract 2018;18:343–5.
31. Sathorn C, Parashos P, Messer HH. Effectiveness of single- versus multiple-visit endodontic treatment of teeth with apical periodontitis: a systematic review and meta-analysis. Int Endod J 2005;38:347–55.
32. Lin HC, Pai SF, Hsu YY, et al. Use of rubber dams during root canal treatment in Taiwan. J Formos Med Assoc 2011;110:397–400.

33. Cochran MA, Miller CH, Sheldrake MA. The efficacy of the rubber dam as a barrier to the spread of microorganisms during dental treatment. J Am Dent Assoc 1989;119:141–4.

34. Al-Nuaimi N, Patel S, Austin RS, Mannocci F. A prospective study assessing the effect of coronal tooth structure loss on the outcome of root canal retreatment. Int Endod J 2017;50:1143–57.

CLINICAL RESEARCH

Differences in Inflammation and Bone Resorption between Apical Granulomas, Radicular Cysts, and Dentigerous Cysts

Manuel Weber, MD, DMD, Jutta Ries, PhD,* Maike Büttner-Herold, MD,† Carol-Immanuel Geppert, MD,‡ Marco Kesting, MD, DMD,* and Falk Wehrhan, MD, DMD**

SIGNIFICANCE

Apical granulomas, radicular cysts, and dentigerous cysts show immunologic differences. High infiltration of HLA-DR– and CD83-positive proinflammatory cells is associated with radicular cyst formation. Additionally, bone resorption markers MCSF and Gal3 are significantly increased in radicular cysts compared with apical granulomas. The development of apical periodontists toward granulomas or radicular cysts could be immunologically controlled. Therefore, the use of root filling materials with anti-inflammatory properties might be beneficial.

From the Departments of *Oral and Maxillofacial Surgery and †Nephropathology and ‡Institute of Pathology, Friedrich-Alexander University Erlangen-Nürnberg, Erlangen, Germany

Address requests for reprints to Dr Manuel Weber, Department of Oral and Maxillofacial Surgery, Friedrich-Alexander University Erlangen-Nürnberg, Glückstraße 11, 91054 Erlangen, Germany.
E-mail address: manuel.weber@uk-erlangen.de
0099-2399/$ - see front matter

https://doi.org/10.1016/j.joen.2019.06.014

ABSTRACT

Introduction: Dental cysts can be of inflammatory (radicular cysts) or noninflammatory (dentigerous cysts) origin. Apical periodontitis is a necrosis of the pulp and infection of the root canal causing the development of apical granulomas or radicular cysts. The immunology of granuloma and cyst formation is important because modern root filling materials are immunologically active and can contribute to the resolution of apical granulomas. In contrast, radicular cysts often require apicectomy. A better understanding of the pathophysiology of inflammation and bone resorption in apical periodontitis could be the basis for developing new root filling materials with superior immunomodulatory properties. **Methods:** Forty-one apical granulomas, 23 radicular cysts, and 23 dentigerous cysts were analyzed in this study. A tissue microarray of the 87 consecutive specimens was created, and human leukocyte antigen–DR isotype (HLA-DR)-, CD83-, receptor activator of nuclear factor kappa B ligand–, macrophage colony-stimulating factor (MCSF)-, galectin-3 (Gal3)-, CD4-, and CD8-positive cells were detected by immunohistochemistry. Tissue microarrays were digitized, and the expression of markers was quantitatively assessed. **Results:** HLA-DR, CD83, MCSF, and Gal3 expression was significantly ($P < .05$) higher in radicular cysts compared with apical granulomas. HLA-DR, CD83, MCSF, receptor activator of nuclear factor kappa B ligand, and Gal3 expression in dentigerous cysts was significantly ($P < .05$) lower than in both periapical lesions (apical granulomas and radicular cysts). CD4 and CD8 infiltration was not statistically different between apical granulomas and radicular cysts. Dentigerous cysts showed a significantly ($P < .05$) lower T-cell infiltration than apical periodontitis. The CD4/CD8 ratio was not significantly different between the analyzed groups. **Conclusions:** The development of radicular cysts in apical periodontitis is associated with an increased expression of myeloid inflammatory markers and bone resorption parameters. Antigen-presenting cells and myeloid cells might be more relevant for the pathogenesis of apical periodontitis than T cells. Increased inflammation might promote the formation of radicular cysts and more pronounced bone resorption. *(J Endod 2019;45:1200–1208.)*

KEY WORDS

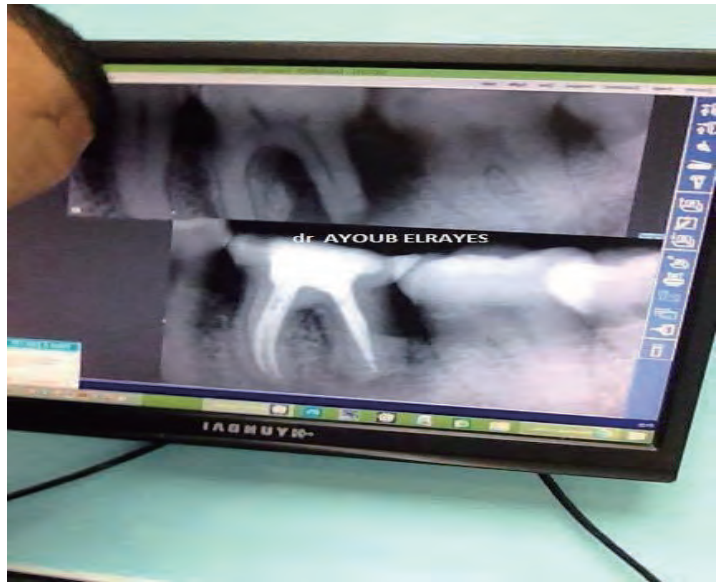
Apical granuloma; apical periodontitis; bone resorption; follicular cyst; periapical lesion; radicular cysts

Infection and necrosis of the dental pulp causes apical periodontitis, which manifests as apical granulomas or radicular cysts. Both periapical lesions have an inflammatory origin but show a different clinical course[1]. In contrast to radicular cysts, dentigerous cysts have no inflammatory cause[2] and are classified as developmental odontogenic cysts[3]. In most cases, apical periodontitis is initially treated by orthograde endodontic treatment[4]. If an apical lesion develops into a cyst, endodontic therapy alone is sometimes not sufficient, and apicectomy or even extraction of the affected teeth is required[4].

3. Case Record

My cas from June 2015

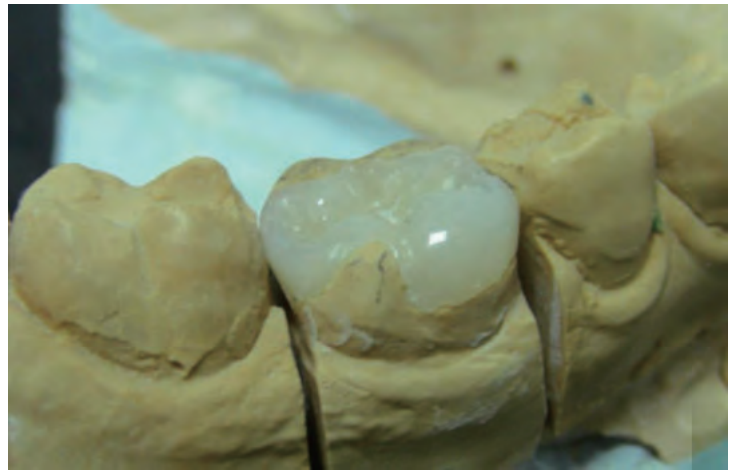
Apical inflammatory root resorption on lower molar tooth # 46
my colleague preferred the option of extraction or root resection coz of massive bone resorption shown in the radiography +age of the pt(65 years) and the resorption. The pt was motivated to try, and the absence of mobility and good oral hygiene motivated me for endo treatment preparation was with reciproc 25 for the mesial canals and i prefered hand files (apical 30) then 30 Hero 4% for the distal, copious irrigation 2.5 % sodium hypocrite alternatively with EDT.
As no apical constriction detected an intermediate filling the canals with calcium hydroxide for 2 weeks, i changed 2 times every tow weeks. (but this was only coz there was no time for this pt)
Then after one month and half when i detected the difference ,, and an apical constriction was found obturated with warm vertical condensation.
6 months later he came and the result was good ,,, with the glass ionomer temporary filling still in place .



Resource：https://www.facebook.com/DentalCaseEveryDay/

As u requested Dr.Mohamed Alkhatib:)... and thanxx for asking . Onlays cementation materials :

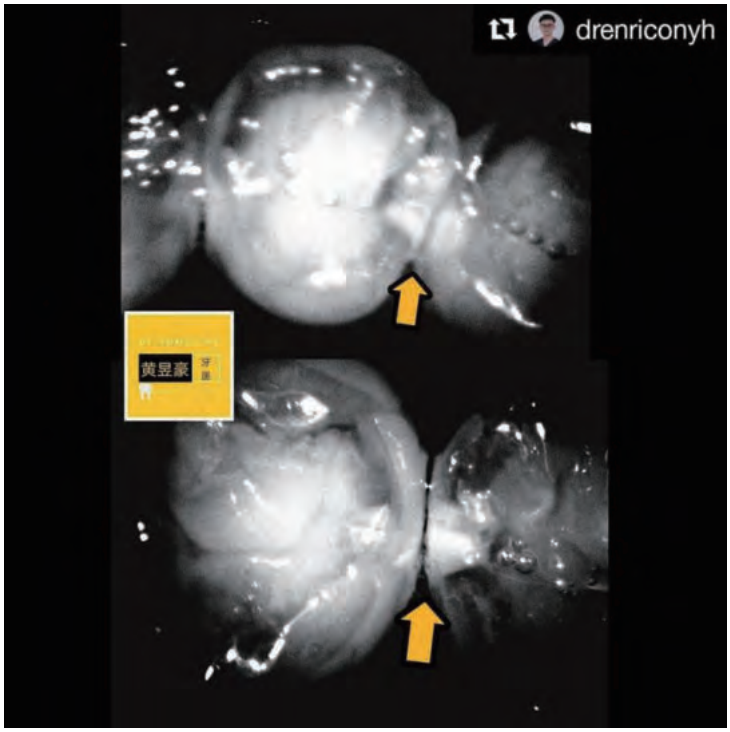
Dual Cure Resin Cement is available in five popular shades: White, White Opaque, Yellow, Brown and Clear. It is easy to use and is packaged in a convenient dual-chambered syringe with mixing tips.
Dual Cure Resin Cement is ideal for the cementation of implant prostheses, crowns, bridges, inlays, onlays, Maryland bridges and veneers. The cement is radio-opaque and provides high compressive and flexural strength and low solubility, which is required for these types of restorations.



Resource：https://www.facebook.com/AlphaDental.BI/?__tn__=%2Cd%2CP-R&eid=ARD2fMHZZSLdxjD0xlRPH5RdM36nSDz87Hyn03OZQe152jYP9QieAoon0ZMY63MLN7qbK1ay5q3BK5lQ

1.The use of a Hi-tech Intraoral camera to "scan" hidden caries in between teeth was used in this case. No x-ray was used for this case. The white spot shows a demineralized tooth structure. Available at Taman Bukit Indah.

2.Re-Crowning of front teeth after root canal treatment to protect, prolong and increase the chance of keeping your tooth in the mouth for a much longer time.
Restoring function and giving back a smile that is no longer awkward.

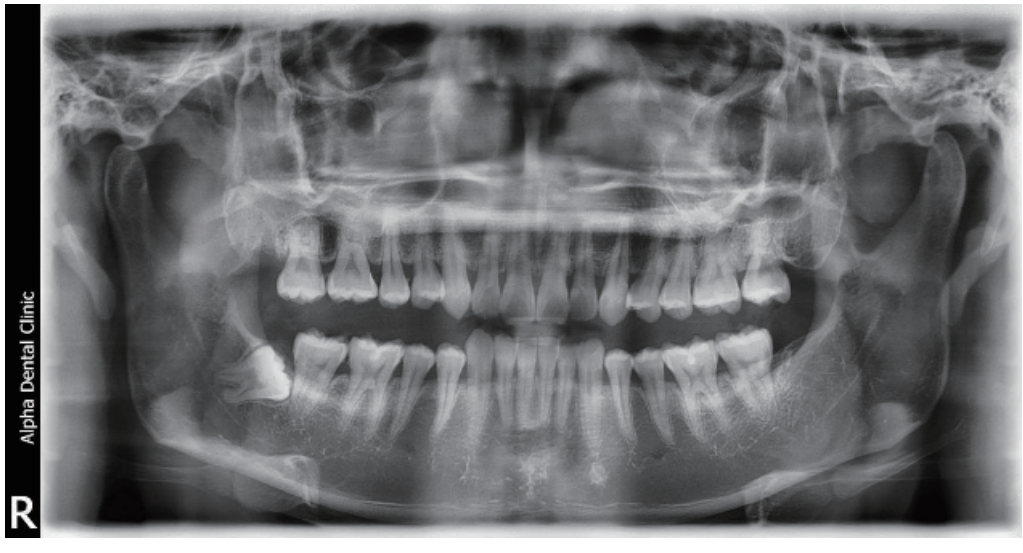


Do you want to see your own teeth with Superman's eyes?

Dental x-ray is one of the most effective tools to confirm a dentist's findings in your mouth. A small xray can be used to see the severity of a cavity (caries), the condition of the gum, and also if there is any infection in the jaw bone underneath the teeth. Whereas a big xray (OPG) can be used to check and monitor the condition of all your wisdom teeth (upper and lower jaw) and also an overview of your oral cavity (teeth, gums, bone, sinus, joints, etc).

Our clinic uses high tech digital x-rays with very little radiation, way lesser than what you would receive during an air-flight overseas.

Schedule an appointment with us today to have your condition checked!



From Orifice To Apex

Reading club

ART AND SCIENCE DIAGNOSIS

EXAMINATION AND TESTING

CLINICAL CLASSIFICATION OF PULPAL AND PERIAPICAL DISEASES

WWW.FANTA-DENTAL.COM