

# Identify and Define All Diagnostic Terms for Pulpal Health and Disease States

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## Abstract

**Introduction:** Consensus Conference Subcommittee 2 was charged with the identification and definition of all diagnostic terms for pulpal health and disease states by using a systematic review of the literature. **Methods:** Eight databases were searched, and numerous widely recognized endodontic texts were consulted. For each reference the level of evidence was determined, and the findings were summarized by members of the subcommittee. Highest levels of evidence were always included when available. Areas of inquiry included quantification of pulpal pain, the designation of conditions that can be identified in the dental pulp, diagnostic terms that can best represent pulpal health and disease, and metrics used to arrive at such designations. **Results and Conclusions:** On the basis of the findings of this inquiry, specific diagnostic terms for pulpal health and disease are suggested. In addition, numerous areas for further study were identified. (*J Endod* 2009;35:1645–1657)

## Key Words

Dental pulp, diagnosis, metrics

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An evidence-based practice centers on the integration of the best clinically relevant scientific information with the patient's desires and the individual practitioner's clinical practice abilities. Its ultimate goal is to enhance patient care through the development of appropriate treatment modalities for specific clinical presentations. The development of standard terminology is at the center of patient care in that it facilitates communication between the patient and doctor as well as between dental health professionals. Presently in endodontics there is no standard diagnostic nomenclature consensus for pulpal status in health or disease. The objective of this analysis was the establishment of evidence-based diagnostic nomenclature for clinically encountered pulpal conditions.

## Materials and Methods

The following databases were searched for literature pertaining to our charge: MEDLINE-Ovid, PubMed, Web of Knowledge, Cochrane Oral Health Group, EMBASE, SCOPUS, Google Scholar, and Medstory. Non-English language citations and nonhuman studies were excluded in searches for pain and pulpal metrics. Texts reviewed included the following: *Endodontics*, 6th ed, Ingle JI, Bakland LK, BC Decker, Hamilton, Ontario, Canada, 2008; *Pathways of the Pulp*, 9th ed, Cohen S, Hargreaves KM, Mosby-Elsevier, St Louis, MO, 2006; *Principles and Practice of Endodontics*, 4th ed, Torabinejad M, Walton RE, Saunders, Philadelphia, PA, 2008; *Encyclopedia of Pain*, Schmidt RF, Willis WD, Springer, Berlin, Germany, 2006; *Essential Endodontology: Prevention and Treatment of Apical Periodontitis*, Ørstavik D, Pitt Ford TR, Blackwell Publishing, Oxford, United Kingdom, 2007. Problems were encountered in consistency of terminology, a lack of high levels of evidence, and inherent subjectivity in subject matter (diagnostic terminology). A lack of studies with high levels of evidence posed the most significant concern.

## Results

### Subquestion #1: How Should the Degree of Pulpal Pain Be Quantified Clinically?

The absolute measurement of pain on a scale common to all patients is not possible as a result of the individual subjectivity of the pain response (1, 2). As a result, initial evaluations as well as the effectiveness of interventions must be assessed by using vague descriptors relative to the individual pain experience such as "severe," "spontaneous," and "continuous" or a subjective determination of the increase or decrease in intensity. More precise forms of pain measurement are available, but their value in endodontic diagnosis and treatment has not been determined.

Several techniques for pain measurement in human subjects have been described. They include verbal rating scales (3–15), numeric rating scales (8, 16), visual analog scales (12, 17–27), color analog scales (28–32), finger span expression (9, 33, 34), calibrated questionnaires (8, 35–39), and cortical evoked potentials (6, 7, 16, 21, 40–43). A brief description of each is presented.

Verbal rating scales are a list of verbal pain descriptors such as no pain, mild pain, moderate pain, and severe pain. The patient chooses the word that best describes their pain, and a number is assigned to this, depending on its ranking in terms of intensity.

Numeric rating scales are a list of numbers, for example, 0–100, with 0 being no pain and 100 the most intense pain imaginable. The patient selects a number that corresponds to their pain intensity.

**TABLE 1.** Use of Dolorimetry Technique in Observations of Pulpal Pain

Verbal rating scales	Numeric rating scales	Visual analog scales	Color analog scales	Calibrated questionnaires	Finger Span Scale	Cortical evoked potentials
16	3	16	2	2	3	5

Visual analog scales consist of a line with 2 end points of “no pain” and “worst pain ever.” The patient marks a point on the line that relates to the intensity of their pain. The distance of that point from “no pain” is the measure of pain intensity.

Color analog scales are used with children. A series of graded in intensity colors are anchored at each end by the terms “no pain” and “worst pain.”

Calibrated questionnaires should really be calibrated questionnaire because there is only one that has gained widespread acceptance, the McGill Pain Questionnaire. This consists of 20 groups of descriptors selected from the medical literature that describe the sensory qualities of the pain, the affective qualities of the pain, or are evaluative describing the overall intensity of the experience. These are displayed on a form that includes diagrams used for localization. A pain rating index is determined on the rank values of the words. The McGill Pain Questionnaire has been translated into at least 16 languages and is very widely used. Its advantage is that it allows measurement of the different components of the pain experience individually, providing a 3-dimensional measure of the experience, whereas the other scales are predominantly 1-dimensional.

Finger span scaling has largely been used in children because it overcomes the complexities of other scales that children might have difficulty understanding. The finger span concept is first demonstrated by holding the thumb and forefinger of one hand together. The patient is told that the fingers in this position represent “no hurt” (or “no pain”). Then a spread of a small distance between the fingers is shown to represent a “tiny” hurt, and a somewhat wider distance is “medium” hurt. When the forefinger and thumb are moved as far apart as possible, this is “most possible hurt.” The span in each instance is measured.

Cortical evoked potentials are components of an electroencephalogram taken while applying a noxious stimulus and can be used with an unconscious subject.

Table 1 shows the number of times a dolorimetry technique has been used in observations of pulpal pain. The numbers are numbers of reports and thus biased by investigators who have used the same approach in multiple studies. In some studies more than 1 approach to pain measurement was used. All techniques are included in the count individually, resulting in some reports being counted more than once in the table.

## Measurement of Pulpal Pain

A systematic review of the literature revealed no published reports of quantifying pulpal pain in a truly clinical situation. All available reports resulted from experimental settings in which the effect of some variable such as analgesic, local anesthetic, exercises, or orthodontic tooth movement on the perception of pain was determined by measuring the pain after pulpal stimulation. There are many reports of efficacy testing of local anesthetics that use the failure to respond to an electrical pulp tester as an indicator of effective anesthesia. This is not quantification but the reporting of an “all or none response.” These reports were not included in this survey. Some studies of local anesthetic solutions do use pain scales, and they have been included (8–16, 18, 20, 22–29, 33, 36, 38, 39, 44, 45).

Although some of the studies reviewed for this article are of high level in that they were randomized, clinical trials, none of them exam-

ined the efficacy of the various scales in describing pulpal pain. This represents a significant deficit of knowledge in the area of pulpal pain assessment. The most prevalent approach endodontists use to assess pulpal pain is an informal verbal descriptor scale, with terms such as severe, intermittent, or spontaneous being widely used. The visual analog scale has achieved wide acceptance in the experimental field, having the important attributes of simplicity and a facile conversion to numbers. The scale is clinically useful, particularly with long-term pain, and serves as a valuable tool for the monitoring and assessment of clinical interventions. Calibrated questionnaires (essentially the McGill Pain Questionnaire) have very broad acceptance in many areas, but they would be less appropriate and more time-consuming in the setting of the dental office than either verbal descriptor or visual analog scales. The use of finger span and color analog scales is generally confined to very young subjects and would be of limited application in the dental office. Although electroencephalography would be an exciting extension to endodontic practice, its acceptance is unlikely, rendering the use of cortical evoked potentials a distant possibility.

## Subquestion #2: What Are the Conditions That Can Be Identified and Described with Respect to the Dental Pulp?

Various states of pulpal health and disease exist, and historically, many classification systems have been used to designate them. The diagnostic systems that have been advocated can be combined into 2 main types, histopathologic classification systems and clinical classification systems, yet most have used a combination of the 2 types of terminology (46–53). Because pulpal inflammatory disease is a progressive temporal continuum, a disease state that changes through time, there exist a large number of potential histopathologic descriptors of pulpal disease states. Clinically, however, only a limited number of pulpal conditions can be described on the basis of examination findings for a patient. Several studies have shown that there is little or no correlation between clinical diagnostic findings and the histopathologic state of the pulp (54–63). Because histopathologic diagnosis is not truly available to the endodontic clinician and because diagnosis is needed to perform clinical endodontic treatment, then the various disease states of the pulp must be described by using a clinical classification scheme.

Clinical classification is based on the use of a diagnostic methodology to produce data that can be interpreted to develop a pulpal diagnosis. The information collected is the patient's chief complaint, their medical and dental history, and the results of objective testing. The information is used to develop a diagnosis and a plan of treatment. It is usually helpful to format the process to increase efficiency and consistency. One such systematic format is given the name S.O.A.P., which is an acronym for Subjective findings, Objective tests, Assessment (or Appraisal), and Plan of treatment (49).

One of the earlier attempts to describe clinical pulpal states of health and disease was by Morse et al (51), and it is a variation of this system that we use today (50, 64). New systems of classification continue to arise as attempts are made to enhance the accuracy and clinical relevance of diagnostic terminology (65). By eliminating terminology that relates to the clinically inaccessible histopathologic state of the pulp, the list of conditions that can be identified and described with respect to the dental pulp becomes manageable.

Levels of evidence in the literature supporting the use of specific clinical diagnostic terminology are generally very low in that the classification schemes appear to be mainly the opinions of the various authors, who provide logical arguments for their choices in developing nomenclature on the basis of studies with levels of evidence rarely exceeding Level 4. They are usually related to clinical examination findings; however, there is much uncertainty as to the specific correlations between diagnostic information and the actual treatment needs of the patient (53). More clinical study is needed in this area.

The conditions of the pulp that can be identified and described are listed in the following section. The clinical manifestations of these conditions and the objective findings relating to them accompany each descriptor.

### Clinically Normal Pulp

This descriptor is mentioned in several classifications (8, 20) and is equivalent in meaning to vital asymptomatic (51) or healthy pulp (53). The term *normal pulp* appears to be more relevant to the clinical situation because it relates to the clinical presentation of the pulp. The words *vital* and *healthy* are inaccurate because vitality cannot be determined through clinical examination or vitality testing, and pulps might be decidedly unhealthy and yet respond in a clinically normal manner. This descriptor indicates that all clinical signs are within normal limits (59), and that the tooth is asymptomatic. Depending on the age of the tooth, there might or might not be evidence of calcification of the pulp, and there might be pulpal fibrosis. The pulp will generally respond to cold or electrical stimuli, and the response will not linger for more than a few seconds, but it will usually not respond to heat (65). Percussion, palpation, and bite tests elicit no pain, and the radiographic appearance is normal.

### Reversible Pulpitis

This descriptor refers to a pulpal state that implies the presence of mild pulpal inflammation and that the pulp is capable of healing (46, 47, 49, 50–53, 64, 65) if appropriate therapy (ie, removal of the irritant) is performed. Reversible pulpitis is a result of caries, trauma, defective or new restorations and is characterized by a mild to severe pain response to stimuli (usually thermal but possibly to biting pressure in a cracked tooth) (65–67). The pain resolves within seconds of removal of the stimulus. There is no response to percussion or palpation of the alveolus, and the radiographic appearance is generally normal. Reversible pulpitis should be distinguished clinically from dentin hypersensitivity, which is a phenomenon of fluid movement in the dentinal tubules and is not necessarily related to pulpal inflammation. The presentation of these 2 entities is very similar except that dentinal hypersensitivity can occur in the absence of the typical etiologic agents of pulpitis such as caries or faulty/new restoration. The etiology for this is exposed root dentin (46, 49, 65).

### Irreversible Pulpitis

This descriptor refers to a pulpal state that implies the presence of a more severe degenerative process that will not heal and that, if left untreated, will result in pulpal necrosis followed by apical periodontitis. Pulpectomy or extraction is required to alleviate the symptoms and prevent apical periodontitis (46, 47, 49–53, 64, 65). Several classifications have broken this entity down into 2 types. The common factor in both of these is the requirement for endodontic therapy to treat the tooth. The first type is asymptomatic irreversible pulpitis, and the second is symptomatic irreversible pulpitis (49, 64, 65).

Asymptomatic irreversible pulpitis is a pulpal state characterized by evidence of the need for endodontic therapy in the absence of clinical

symptoms or pain. Irreversible inflammation of the pulp is produced by carious exposure (47, 68, 69), caries excavation, or trauma (46, 49, 64) necessitating root canal therapy. Despite being “painless,” this form of pulpitis is expected to progress to pulp necrosis without treatment (63, 70).

Symptomatic irreversible pulpitis is a pulpal state characterized by mild to severe pain that lingers after removal of a stimulus (53) or that might be spontaneous (49). It implies a more severe degenerative inflammatory pulpal process that, if left untreated, will result in pulpal necrosis. The tooth will exhibit pain when exposed to thermal irritants (heat and/or cold) (53) that are prolonged well beyond the removal of the stimulus. The pain might be sharp or dull, depending on the type of pulpal nerve fibers responding to the inflammatory mediators (71) and peptides (72). A-delta fibers mediate sharp pain, with C fibers mediating dull throbbing pain (53, 73), and it might be localized or referred (1, 49, 74). The etiology of irreversible pulpitis might be deep caries (69) or restorations, pulp exposure, cracks, or any other pulpal irritants. The tooth might or might not be percussion or bite sensitive, and the radiographic appearance might be unremarkable except for the presence of the etiologic agent (65). Occasionally, if the inflammatory process has extended into the periapical area, thickening of the periodontal ligament space (49, 65) or condensing osteitis (chronic focal sclerosing osteomyelitis) (75) might be visible. The treatment for irreversible pulpitis is root canal therapy or extraction of the tooth.

### Pulp Necrosis

The end result of irreversible pulpitis (asymptomatic or symptomatic) (49) and, in many cases, dental trauma (46, 53) is necrosis of the pulp tissue (47, 48, 50–52). Because this event rarely occurs suddenly (except for cases of dental trauma), there occurs a variable period of time when the pulp will be partially necrotic. The area of cell death expands until the entire pulp necroses. Subsequent bacterial invasion will ultimately result in an infected root canal system (46, 47, 53, 76) and, without treatment, apical periodontitis. Teeth with necrosis of the pulp will present with variable symptoms ranging from none to severe pain, bite sensitivity, and hyperocclusion (77) of periradicular origin. Occasionally, the tooth containing a necrotic pulp can become discolored (46, 78) as a result of altered translucency of the tooth structure or hemolysis of red blood cells during pulp decomposition. Radiographically, the appearance can vary from apparently normal to exhibiting a large periradicular radiolucency. The one thing that usually distinguishes pulp necrosis from the other pulpal states is the absence of sensitivity to thermal or electrical pulp tests. Occasionally, the necrotic pulp might respond to heat application (49). Of all the histopathologic pulpal states, necrosis is the one that is most reliably predicted from clinical testing (54, 56), with high correlations between negative pulp tests and necrosis of the pulp, although this finding is not universally supported (58). Partial pulp necrosis (necrobiosis) (65) is very difficult to diagnose especially in multi-rooted teeth, which might have different pulp states in different roots within the same tooth. This can occasionally give rise to positive responses to thermal and electric pulp tests, combined with signs and symptoms of infected necrotic pulp (46, 65). The distinction between partial and full necrosis becomes important when dealing with immature teeth that have an open apex. To decide whether to perform apexogenesis or apexification on these teeth, one must decide whether the entire pulp is necrotic. The definitive test for this is to enter the pulp chamber and remove necrotic tissue until a vital pulp stump is reached (53).

### Hyperplastic Pulpitis (Pulp Polyp)

This rarely found entity occurs when caries invades the pulp in an immature tooth with open apices (46, 47, 50, 68, 79–82). The



enhanced blood supply created by the open apices allows the immature pulp to better resist bacterial invasion than a more mature pulp (81, 83), and the opening through the carious lesion into the oral cavity establishes a pathway for drainage of pulpal inflammatory exudates. Acute inflammation then subsides, and chronic inflammatory tissue proliferates through the opening (68). Clinically, this appears as a fleshy mass of tissue connected to the pulp space that appears to be growing out of the tooth, and the tissue is frequently epithelialized. Free-floating cells of the oral mucosa are “seeded” onto the proliferating granulomatous tissue, resulting in a stratified squamous epithelium (47), and the resultant lesion is rarely painful except when masticatory forces cause irritation and bleeding (68). Radiographically, there appears to be a deep carious lesion connecting to the pulp space, and the root ends are immature. Treatment for this entity is either endodontic therapy or extraction because this condition is considered to be irreversible (47).

### Internal Resorption

Internal resorption of the tooth structure is a pathologic state of the pulp in which multinucleated clastic cells within the pulp tissue begin to remove the dentinal walls of the pulp space. It is generally idiopathic in that the trigger for the metaplastic transformation of normal pulp cells into clastic ones is unknown. Several hypotheses have been proposed (84, 85), and it is possible that it might be a combination of these that starts the resorptive process. The resorption sometimes moves swiftly and then might be followed by a time of slower or no growth in the size of the lesion (47). Internal resorption is generally painless and is usually found clinically through routine radiographic screening, when it appears generally as an ovoid enlargement of the pulp space (86) in which the original borders of the pulp space become distorted or disappear altogether (84, 85, 87). The lesion stays associated with the root canal on angled radiographs (84, 85). The tooth might respond to pulp sensibility tests, but occasionally the tests might be negative if there is partial necrosis with the advancing resorptive lesion within the living portion of the pulp tissue subjacent to the necrotic tissue (65, 84, 85). If perforation of the tooth structure has occurred and the tissue in the pulp space is exposed to oral fluids, pain might occur (84, 85). The crown of the tooth might appear pink in color (47, 87) as a result of thinning of the tooth structure, allowing the color of the underlying granulomatous tissue to be visible; however, this might also be due to undermining, subepithelial external root resorption (84, 85). Internal root resorption is considered a form of irreversible pulpitis and requires root canal therapy to halt the process (47).

### Pulp Calcification

Degenerative changes to the pulp such as pulp calcification or pulp atrophy/fibrosis are related to aging or sublethal injury resulting in chronic irritation to the pulp. The pulp responds by fibrosing or calcifying (88, 89). Generally, pulp fibrosis or atrophy is a histologic change that is not clinically discernible unless the pulp space is entered during the initial phases of root canal therapy, so its value as a diagnostic term is questionable. Pulp calcification, however, is usually clinically detectable before treatment and can directly affect the prognosis of treatment, in that severely calcified teeth are predisposed to tooth perforation during the search for canals (90). This entity is also sometimes referred to as pulp canal obliteration (65) or calcific metamorphosis (91, 92), but both terms appear to be inaccurate because the canal is rarely completely obliterated (93), and there is actually no “metamorphosis” of the tooth, just a progressive deposition of dentin (secondary or tertiary) resulting in radiographically apparent shrinkage of the pulp

canal space (46). Calcification, per se, does not necessarily imply that progressive inflammation of the pulp or pulp necrosis will occur. In fact, pulp necrosis is found in less than 7% of traumatically induced calcified pulps (94). Lastly, the mineral content of the tertiary dentin represents more than just calcium hence the term pulp canal mineralization would be a more accurate term.

### Previously Initiated Treatment

Occasionally, a tooth that has had endodontic therapy previously started but not completed will present for diagnosis (64). These teeth would have undergone previous pulpotomy or pulpectomy, and the history and clinical examination should reveal this. These teeth might or might not present with signs and symptoms of pulpal or periradicular disease (65), and radiographic evidence of access into the pulp space and the possible presence of radiopaque interappointment medicaments such as calcium hydroxide paste would be found. In these cases as with any necrotic pulp or pulpless tooth, given time, the pulp space will become infected, and apical periodontitis would be expected to ensue (65), and so completion of endodontic therapy would be necessary.

### Previous Endodontic Therapy

Many times teeth that have had previous endodontic therapy are examined by dentists (65). This entity is also referred to as previously treated (64); however, this terminology is perhaps not specific enough to endodontics to make it an appropriate term for this condition. Various treatment modalities would fall under this diagnostic category including teeth that have undergone nonsurgical root canal therapy, surgical root canal therapy, and therapeutic pulpotomy with calcium hydroxide (the Cvek pulpotomy) (85, 95, 96) or with mineral trioxide aggregate (97) to induce apexogenesis. The history and both the clinical and radiographic examinations should indicate the existence of previous endodontic therapy. For treatment designed to preserve the pulp (pulpotomy), the important question to be answered is whether the treated pulp remains healthy, and for teeth with completed full endodontic therapy, it is whether the pulp spaces are infected (85, 98). This will usually be determined by the response of the periradicular tissues (99) and the clinical determination as to whether bacterial ingress from the coronal aspect is likely to have occurred (100, 101). The technical quality of the root canal filling will also need to be assessed, but this cannot be completely addressed by inspecting the radiograph because this will only show a 2-dimensional representation of the obturation and perhaps the presence of an iatrogenic complication such as perforation of the root or a separated instrument. The assessed technical quality of the root filling alone cannot give any indication as to whether the root canal space is infected (65). However, the decision as to whether to treat the tooth with this diagnosis (nonsurgical/surgical retreatment or extraction) will be determined by diagnosing the presence or absence of apical periodontitis, by a thorough knowledge of outcomes assessments (102), and by whether other considerations (such as restorative needs) require that treatment be instituted (103).

The classification presented in Table 2 has been proposed previously by Abbott and Yu (65) and Abbott (104–106). It is a simple yet comprehensive clinical diagnostic system that uses terminology outlined above and relating to the clinical findings. It is based on the progression of pulp diseases through the various stages discussed above. It also includes normal pulp tissue, which is an entity that should be diagnosed and given recognition when there are no signs of disease.

The previously described clinical pulpal diagnoses are those that can be described and differentiated by using the diagnostic methods routinely available today. The author realizes that universal agreement on the terminology presented here will not be easily obtained, and much legitimate debate will now ensue.

**TABLE 2.** Comprehensive Clinical Diagnostic System

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Clinically normal pulp: based on clinical examination and test results
Reversible pulpitis
Acute
Chronic
Irreversible pulpitis
Acute
Chronic
Necrobiosis (part of pulp necrotic and infected; the rest is irreversibly inflamed)
Pulp necrosis
No sign of infection
Infected
Pulpless, infected root canal system
Degenerative changes
Atrophy
Pulpal canal mineralization
Partial
Total
Hyperplasia
Internal resorption
Surface
Inflammatory
Replacement
Previous root canal treatment
No sign of infection
Infected
Technical standard (based on the radiographic appearance)
Adequate
Inadequate
Other problems: eg, perforation, missed canals, fractured instrument

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There might come a time when diagnostic methods will arise that will have greater specificity and sensitivity and that are so inexpensive and efficient that future clinicians will be able to discriminate other pulpal conditions more accurately than we can today. Perhaps advances in areas such as measuring pulpal blood flow or high-resolution, 3-dimensional imaging will allow practitioners to correlate better between pulpal histopathologic states and clinically detectable phenomena. This could lead to an expansion of this terminology and to greater accuracy in patient treatment, and efforts must continue in that direction.

However, the more pressing need at this time is to develop a more reliable body of scientific evidence to validate or correct the current diagnostic process and thus help us to enhance clinical care. Our patients deserve at least that much.

### **Subquestion #3: On the Basis of the Highest Level of Available Evidence, What Diagnostic Terms Best Represent Pulpal Health and the Various Forms of Pulpal Disease?**

Many different classification systems have been advocated for pulp diseases over the years, although most of them are based on histologic findings. Table 3 (65) has been reproduced as a summary of many of these classifications. Abbott (104–106) and Abbott and Yu (65) have also proposed a classification system of their own that varies from those in Table 2. Typically, these classifications mix clinical and histologic terms, resulting in many misleading terms and diagnoses for the same clinical condition. This creates confusion and uncertainty in clinical practice when a rational treatment plan needs to be established to target a specific pathologic entity.

## **Clinically Normal Pulp**

All classifications of tissue conditions should include tissue that has not been harmed in any way, that is, normal or healthy tissue (65). The clinical tests available to dentists to assess the state of the dental pulp are relatively crude. These tests are not entirely reliable because they are usually only testing the ability of the pulp to respond to a stimulus (ie, pulp sensibility), and this does not provide much information about whether the pulp is healthy. Hence, it is more appropriate to classify the pulp as being a clinically normal pulp when there is an absence of any signs or symptoms of pulp disease being present (65).

## **Pulpitis**

The first response of a dental pulp to a stimulus is inflammation. Hence, the most appropriate term to use is pulpitis, because the suffix *-itis* is defined in dictionaries as indicating inflammation of the tissue whose name it is attached to, ie, the pulp (107, 108).

Some teeth with pulpitis can be clinically managed via conservative means (such as a simple restoration or a sedative dressing followed by a restoration), whereas others require more radical treatment, which implies removal of the pulp either as part of endodontic treatment or via extraction of the tooth. Because these clinical treatments vary so greatly, it is essential that clinicians differentially diagnose which pulps can be managed conservatively and which ones require removal. This implies that subcategories of classification are required for teeth with pulpitis. The generally accepted terms are reversible pulpitis and irreversible pulpitis, although some dispute exists as to the applicability of these terms. At this time, there is no undisputed evidence to support or refute the use of these 2 terms.

Reversible pulpitis implies that the inflammation within the pulp can be reversed, that is, the pulp will heal after treatment with either normal or fibrous tissue. (Note that both forms of responses result in clinically normal pulp tissue, although the exact nature of the healing response cannot be predicted). From a clinical perspective, it is recognized that it is not possible to accurately determine this state of pulpitis in all cases. However, it is generally accepted that teeth with relatively mild symptoms will have reversible pulpitis.

Teeth with more severe symptoms are usually diagnosed as having irreversible pulpitis, and therefore the pulp or tooth will be removed. Currently, differentiating between reversible and irreversible pulpitis is largely done on an empirical basis. It is also not known whether pulps are ever truly irreversibly inflamed, that is, could all pulps with inflammation recover if conservative treatment strategies were used? This question requires further research to establish an answer.

## **Necrosis**

If an inflamed dental pulp is not treated and continues to be subject to the irritant or injurious factor, then it will die at some stage. The term *necrosis* is defined as “death of cells or tissues through injury or disease, especially in a localized area of the body” (109). Hence, its use in a classification of pulp diseases is entirely appropriate. It is recognized that in the disease continuum, partial necrosis can exist. This is usually confirmed clinically during treatment and is significant in terms of the extent of possible canal infection. It is for the most part a histologic finding with either partial or full necrosis that endodontic therapy is still indicated.

## **Teeth with Previous Root Fillings**

Teeth with existing root canal fillings need to be assessed as part of the routine clinical and radiographic examination of a patient. The most important aspect of this assessment is to determine whether the root canal system is infected because an infected canal will cause apical

**TABLE 3.** Comparative Terminology and Classifications of Pulp Diseases Used by Various Authors and Organizations (2–14)

World Health Organization <sup>2</sup>	Weine <sup>3</sup>	Ingle <sup>4</sup>	Seltzer and Bender <sup>5</sup>	Cohen and Burns <sup>6</sup>	Tronstad <sup>7</sup>	
(Note: Normal pulp not mentioned)	(Note: Normal pulp not mentioned)	Healthy pulp	(Note: Normal pulp not mentioned)	Within normal limits, normal pulp, calcific metamorphosis	Healthy pulp	
Pulpitis: initial (hyperemia), acute, suppurative (pulpal abscess), chronic, chronic ulcerative, chronic hyperplastic (pulpal polyp), other unspecified pulpitis, pulpitis unspecified	Pulpitis: hyperalgesia (reversible pulpitis), hypersensitive dentin, hyperemia, painful pulpitis, acute pulpalgia (acute pulpitis), chronic pulpalgia (subacute pulpitis), nonpainful pulpitis, chronic ulcerative pulpitis, chronic pulpitis (no caries), chronic hyperplastic pulpitis (pulp polyp)	Pulpitis: hyper-reactive pulpalgia, hypersensitivity, hyperemia, acute pulpalgia, incipient, moderate, advanced, chronic pulpalgia, hyperplastic pulpitis	Pulpitis: incipient form of chronic pulpitis, acute pulpitis, chronic partial pulpitis with partial necrosis, chronic total pulpitis with partial liquefaction necrosis, chronic partial pulpitis (hyperplastic form)	Pulpitis: reversible, irreversible, asymptomatic, irreversible pulpitis, hyperplastic pulpitis, internal resorption, canal calcification, symptomatic irreversible pulpitis	Pulpitis: asymptomatic pulpitis, symptomatic pulpitis	
Necrosis of the pulp	Pulp necrosis	Pulp necrosis, liquefaction, sicca	Pulp necrosis	Necrosis: partial, complete	Necrotic pulp	
Pulp degenerations, denticles, pulpal calcification, pulpal stones	Pulp degeneration, atrophy, dystrophic calcification	Pulp degeneration, atrophic pulpositis, calcific pulpositis	Pulp degeneration, atrophic pulp, dystrophic mineralization			
Abnormal hard tissue formation in pulp, secondary or irregular dentin	Internal resorption	Internal resorption				
American Association of EndodontistsGlossary <sup>8</sup>	Harty <sup>9</sup>	Walton and Torabinejad <sup>10</sup>	Grossman <sup>11</sup>	Castellucci <sup>12</sup>	Stock <sup>13</sup>	Bergenholtz <sup>14</sup>
Normal pulp	Normal pulp	(Note: Normal pulp not mentioned)	(Note: Normal pulp not mentioned)	Healthy pulp	Normal pulp	Pulpa sana
Pulpitis: reversible pulpitis, irreversible pulpitis	Pulpitis: reversible pulpitis, irreversible pulpitis	Pulpitis: reversible pulpitis, irreversible pulpitis, hyperplastic pulpitis	Hyperemia, pulpitis, acute pulpitis, chronic ulcerative pulpitis, chronic hyperplastic pulpitis	Pulpitis: Hyperemia, pulpitis irreversible	Concussed pulp, reversible pulpitis, irreversible pulpitis	Pulpitis
Pulp necrosis	Necrosis	Pulpal necrosis Pulp calcification, internal (intracanal) resorption	Necrosis Pulp degeneration, calcific, fibrous, atrophic, internal resorption	Necrosis	Pulpal necrosis Internal resorption	Necrosis pulpa

From Abbott PV, Yu C. A clinical classification of the status of the pulp and the root canal system. Aust Dent J 2007;52:(1 Suppl):S17–S31. Reproduced with permission from the Australian Dental Journal.

periodontitis. It is also important to assess the technical standard of the root canal filling because this might determine whether further treatment is required and/or feasible. Such determination is usually based on the radiographic appearance of the root canal filling.

If there are no signs or symptoms to suggest that a root-filled tooth is infected, then the management of such a tooth might be simply one of observation and reassessment. In other cases, the root filling might be judged as being technically unsatisfactory and requiring replacement before further restoration of the tooth. Hence, specific diagnostic terms are required for these situations. Because the tooth is not infected, it would be appropriate to say it is “a root-filled tooth with no signs of infection” (65). The phrase *no signs of infection* does not necessarily imply that the root canal system is not infected, but merely that there is no clinical or radiographic evidence of it being infected at the time of examination.

Teeth that have root canal fillings might become infected at any time once a pathway of entry for microorganisms becomes available. The management of such a tooth requires specific considerations and treatment techniques. Hence, a specific diagnostic category or term is required. The proposed term is “infected root canal system in a root-filled tooth” (65).

### Teeth with Incomplete Endodontic Treatment

Patients might present to dentists and/or endodontists with a tooth that has had endodontic treatment commenced at some time in the past, but the treatment was not completed. There are a wide variety of possible reasons why the treatment might not have been completed (eg, patient did not return for treatment, patient was referred to a specialist for further treatment); these might or might not be relevant to the diagnosis in all cases. It is important to distinguish these cases from other conditions outlined above and below because their clinical management might be different.

If a tooth has had endodontic treatment commenced but not completed and it has no signs of the root canal system being infected, then the tooth could be classified as having “incomplete endodontic treatment with no signs of infection” (65). The phrase *no signs of infection* does not necessarily imply that the root canal system is not infected, but merely that there is no clinical or radiographic evidence of it being infected at the time of examination (65).

If a tooth has had endodontic treatment commenced but not completed and there are signs of the root canal system being infected, then the tooth could be classified as having “an infected root canal system and incomplete endodontic treatment.” Any other findings that would complicate further management of the tooth (eg, perforation, untreated canal) should be listed as part of the diagnosis (65).

### Teeth with Degenerative and/or Physiologic Changes to the Pulp

Dental pulps undergo physiologic changes just like all other tissues in the body. Such changes are not pathologic in nature, and they might be difficult to diagnose clinically. Likewise, some pulps might undergo degenerative changes over time. If there are clinical or radiographic manifestations of the degeneration, it is important to consider these conditions as part of the diagnostic process and therefore to include them in a classification of the “Status of the Pulp and the Root Canal System.”

Typical conditions are pulp canal calcification, either part of the normal aging process or it can be an indication of long-standing irritation to the pulp. Calcification is defined as “abnormal deposition of calcium salts within tissue” (110). Hyperplasia is defined as “an abnormal increase in cells in a tissue or organ, excluding tumor forma-

tion, whereby the bulk of the tissue or organ is increased” (111). This term can be used when there has been an overgrowth of granulation tissue originating from the pulp, and it might result in the development of a pulp polyp. It has been suggested that the inflammation might be limited to the pulp chamber and that the apical pulp tissues might be normal, except for some vasodilatation and minimal chronic inflammation. Because this condition is associated with inflammation, the term should be hyperplastic pulpitis.

### Teeth with Internal Resorption

Three forms of internal root resorption have been reported, although varying terminology has been used to describe them. The different forms of internal resorption require different clinical management, and therefore it is essential that they be differentially diagnosed from one another. The proposed terminology is internal surface resorption, when just minor areas of the root canal wall have been resorbed (112). This resorption might be self-limiting and might repair if the pulp is relatively healthy and if the irritating stimulus has been removed from the tooth.

Internal inflammatory resorption occurs when an inflammatory response within the pulp (ie, pulpitis) leads to activation of dentinoclastic cells, which resorb the dentin walls of the root canal and then progress through the dentin toward the cementum (113). This resorption is believed to be a result of the presence of microorganisms within the coronal part of the root canal that cause pulpitis in the pulp apical to the resorptive area (113). Hence, a tooth with active internal inflammatory resorption will have some necrotic and infected pulp tissue as well as some pulp tissue with irreversible pulpitis. If the condition is defined as such, then there is no need to mention each of these conditions in the diagnosis. The dentinoclasts present in internal inflammatory resorption will only remain alive and active as long as there is a viable blood supply to the apical part of the pulp. If this blood supply is lost, then the apical part of the pulp will necrose, and the dentinoclasts will also die. Thus, the internal inflammatory resorption will no longer be active. Typically, the necrotic apical pulp tissue is then digested and removed by the microorganisms, and the entire canal will become pulpless (as described above), resulting in apical periodontitis. Once apical periodontitis is evident, it is highly likely that the resorption is no longer active, which will make clinical management somewhat easier and less involved. Hence, it is important to distinguish between active and nonactive states of internal inflammatory resorption.

Internal replacement resorption is a metaplastic type of change to the dental pulp in which the pulp first is replaced by bone, and then subsequently the dentin is replaced by bone (113). This condition must be distinguished from the other 2 types of internal resorption mentioned above because its clinical management is quite different, ie, the tooth can be extracted, or it can be left untreated and simply reviewed until extraction is required.

### Subquestion #4: Which Combination(s) of Metrics Provides the Maximal Accuracy for Establishing Pulpal Diagnoses?

Inconsistent definitions of pulpal disease have led many researchers to dichotomize pulpal status into general categories that are defined as vital or nonvital (114). Others have elected to further categorize vital pulp status according to the severity of inflammation and, in particular, whether the inflammation is reversible or irreversible (115). In an effort to interpret research findings in a meaningful way, this review attempts to address the evidence for metrics for establishing diagnoses of (1) vital versus nonvital pulp and (2) normal pulp versus reversible pulpitis versus irreversible pulpitis. The best method for arriving at the



**TABLE 4.** Accuracy of Cold Testing

Reference	Gold standard	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Seltzer et al (128)	Histology	0.78	0.81	0.47	0.94
Dummer et al (132)	Histology	0.68	0.70	0.33	0.91
Petersson et al (133)	Clinical <sup>a</sup>	0.83	0.93	0.89	0.90
Evans et al (121)	Clinical <sup>b</sup>	0.92	0.89	—	—
Gopikrishna et al (120)	Clinical <sup>c</sup>	0.81	0.92	0.92	0.81

<sup>a</sup>In Petersson et al (1999), gold standard was determined by “direct pulp inspection.”

<sup>b</sup>In Evans et al (1999), pulpal status was “confirmed by pulpectomy.”

<sup>c</sup>In Gopikrishna et al (2007), pulpal status was evaluated by direct visual inspection.

agreed on definition for pulpal disease, which might or might not be impractical or desirable to use within clinical practice, is termed the gold standard test or reference test. The results from such a gold standard test for pulp diagnosis is used to compare with the diagnostic test being evaluated for the determination of testing accuracy. Studies assessing diagnostic accuracy for pulpal disease testing have used 2 different gold standard tests: a clinically derived measure (eg, presence of necrotic tissue on accessing a tooth would indicate that the tooth was nonvital) and a histologically derived measure (eg, on extracted teeth for which the history of symptoms has been established and/or on which pulp tests have been performed) (114, 115). It must be recognized that because the progression of pulpal disease might result in periradicular changes, metrics used to establish a periradicular diagnosis might aid in the determination of a pulpal diagnosis. For example, if one arrives at an endodontic diagnosis of apical periodontitis, the implication is that there is an inflammation of the periodontal ligament caused by infection of the pulp or necrotic pulp space.

### Metrics for Diagnosis of Vital versus Nonvital Pulp

A diagnosis of vital versus nonvital pulp is relatively straightforward when compared with determining a diagnosis of normal pulp versus reversible pulpitis versus irreversible pulpitis. This is because the interpretation of the findings from the pulp tests can be dichotomized (ie, response versus no response) (114). Furthermore, the gold standards for studies on metrics for determining vital versus nonvital pulp are more readily discernible (ie, determination of necrotic pulp tissue on endodontic access or on histologic examination after extraction). Thus, there is relatively more evidence related to determination of vital versus nonvital pulp. The tests for which some level of accuracy for determining pulp status has been determined are cold, heat, electric, laser Doppler flowmetry, and pulse oximetry (53).

Comparison of studies that address pulp testing methods is challenging, given the variations in factors such as testing methodology (eg, stimulus type, method of application, definition of response, location of stimulus), tooth variables (eg, restorations, caries, past trauma, recession, tooth type), and patient variables (eg, age, gender, anxiety, oral habits, systemic diseases). This challenge is especially apparent when one attempts to interpret the findings of studies that address the use of the cold test. Materials used as a coolant include CO<sub>2</sub> snow, ice stick, 1,1,1,2 tetrafluoroethane, ethyl chloride, and dichlorodifluoromethane. Application methods include direct application, cotton swab, cotton pellet, and cotton roll. Given the inherent challenges, it

is not surprising to find considerable variability between studies. The findings from the selected studies related to the accuracy of these tests are summarized in Tables 4–8. One can conclude from the information presented in these tables that there is considerable variability in the sensitivity and specificity of cold and heat tests and in the sensitivity of electric pulp tests. Thus, the studies suggest that there is no agreement as to whether cold and heat tests, when used in the absence of other tests, can reliably determine the presence of diseased (ie, nonvital) pulp or for cold, heat, and electric tests to identify teeth without disease (ie, vital pulp) (116). There is less variability in findings for specificity of electric pulp tests, suggesting that this test is more consistent at identifying teeth without disease (ie, vital pulp) (117, 118). In addition, it appears that heat tests have lower positive predictive values than cold or electric tests. Thus, a lack of response to a heat test appears to be less likely to be predictive of a vital pulp (119).

Cold, heat, and electric tests assess the responsiveness of the pulpal innervation, as opposed to the vitality of the pulp tissue. They are, therefore, of less value in conditions in which the innervation of the pulp tissue is compromised (eg, after trauma) (120). As a result, a pulp with vascularity and vital cells, but with severed or compromised nerves, might be misdiagnosed as being nonvital by these tests. An alternative to assessing the responsiveness of pulpal innervations is assessing blood circulation of the tissue. Two such tests, laser Doppler flowmetry and pulse oximetry, have been included in this article because the results of these tests have been referenced to a gold standard (121). The findings summarized in Tables 7 and 8 show that both laser Doppler flowmetry and pulse oximetry have higher sensitivity and specificity than cold, heat, and electric tests. Thus they appear to be more likely to identify nonvital pulp and vital pulp. This is most likely because laser Doppler flowmetry and pulse oximetry provide a measure of vitality that does not rely on intact and functioning innervations, but rather they are a measure of intrapulpal blood flow. However, limitations of these tests include any condition that limits the ability of the test to distinguish the vascular blood flow. The limitations would include teeth undergoing calcific changes, such as in teeth with a history of trauma, full coverage or deep restorations, or physiologic conditions associated with aging. In addition, care must be taken to avoid false-positive findings that might occur if the adjacent gingiva is not masked.

### Other Clinical Measures of Pulp Disease

In addition to tests for pulp responsiveness and pulpal blood flow, other factors have been used in an attempt to determine pulp status.

**TABLE 5.** Accuracy of Heat Testing

Reference	Gold standard	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Seltzer et al (128)	Histology	0.78	0.81	0.47	0.94
Dummer et al (132)	Histology	0.68	0.70	0.33	0.91
Petersson et al (133)	Clinical <sup>a</sup>	0.86	0.41	0.48	0.83

<sup>a</sup>In Petersson et al (1999), gold standard was determined by “direct pulp inspection.”



**TABLE 6.** Accuracy of Electric Pulp Testing

Reference	Gold standard	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Seltzer et al (128)	Histology	0.98	—	—	—
Petersson et al (133)	Clinical <sup>a</sup>	0.72	0.93	0.88	0.84
Evans et al (121)	Clinical <sup>b</sup>	0.87	0.96	—	—
Gopikrishna et al (120)	Clinical <sup>c</sup>	0.71	0.92	0.91	0.74

<sup>a</sup>In Petersson et al (1999), gold standard was determined by “direct pulp inspection.”

<sup>b</sup>In Evans et al (1999), pulpal status was “confirmed by pulpectomy.”

<sup>c</sup>In Gopikrishna et al (2007), pulpal status was evaluated by direct visual inspection.

Evans et al (121) reported that the presence of external root resorption, periapical radiolucency, crown discoloration, tenderness to percussion, and history of pain were all found to have a high specificity (0.97 or better) but low sensitivity (0.49 or lower) for nonvitality. However, the authors failed to disclose the clinical criteria that were used for assessment of these characteristics, making it impossible to validate their findings. A clinical finding of carious pulp exposure has been reported in endodontic textbooks as indicating an irreversible pulpitis (53, 122–124). This has been based, in large part, on histologic evaluation of extracted teeth with deep carious lesions (125). No articles were found that used (1) a standardized method for determining when pulp was exposed during caries removal, along with (2) a gold standard for determination of accuracy of caries excavation as a metric for determining reversible versus irreversible pulpitis.

### Identification of Reversible versus Irreversible Pulpitis

Studies that have attempted to determine accuracy (or have enough information in the report to establish accuracy) of metrics for determining diagnoses of reversible versus irreversible pulpitis are less common than studies that determine accuracy of metrics for determining vital versus nonvital pulp. Some researchers have attempted to correlate the results of diagnostic tests with categories of pulpal inflammation (119). Hyman and Cohen (116) summarized the results of 4 articles that histologically evaluated teeth after pulp tests. The metric that was evaluated in this table was from teeth that had an “abnormal reaction to cold test,” and the gold standard was histologic evidence of pulpal inflammation (Table 9). When compared with the determination of vital versus nonvital pulp tissue, the determination of reversible versus irreversible pulpitis by using cold has relatively lower sensitivity, specificity, and positive predictive values. Studies have not been conducted in which pulse oximetry and laser Doppler flowmetry have been used to differentiate between reversible and irreversible pulpitis.

### History of the Presenting Symptoms

In addition to using pulp tests to determine the severity of pulpal inflammation, some researchers have attempted to evaluate whether the history of presenting symptoms could be used as a metric for determining pulp status. Grushka and Sessle (126) have used the McGill Pain Questionnaire to differentiate types of toothache pain, and they determined that self-reports of toothache pain seem to be valid predictors of whether pulp inflammation is reversible. The methodology used by Grushkas and Sessle for determining reversible versus irreversible pulpitis was only defined as the use of “standard dental diagnostic

procedures.” Thus, a gold standard, such as pulp status upon endodontic access or extraction and histology, was not used. In addition, the statistical analysis does not allow for determination of the accuracy of metrics used for diagnosis. Other authors have addressed the history of presenting symptoms as a metric for determining a pulp diagnosis. For example, Bender (127) has reported that the more severe pulpal pain is and the longer it had been present, the more likely it is that irreversible inflammation has been present. Another predictive factor for determining whether pulpal inflammation is irreversible is a history of being spontaneous. In some cases the spontaneous pain was so severe as to wake the patient from sleep (128).

### Limitations of Using History of Presenting Symptoms

Although the history of presenting symptoms might be useful as an aid in determining a pulpal diagnosis, it is worth noting that none of the studies that have addressed the history of presenting symptoms have resulted in sensitivity, specificity, positive predictive value, or negative predictive value of the symptoms. In addition, studies that have assessed the history of symptoms for teeth with necrotic pulps have shown that 26%–60% of the cases had no history of pain (129, 130). Thus, although a history of presenting symptoms would, for some patients, aid in determining the pulpal diagnosis, for many patients the history would not yield predictive value. Table 10 illustrates the challenges of developing metrics for pulpal diagnosis (and specifically reversible versus irreversible pulpitis) on the basis of history of the presenting symptoms.

### Identified Deficiencies in Available Evidence

There are several areas in which there is a lack of knowledge concerning the accuracy of metrics for determining pulp diagnoses. An ideal metric, or combination of metrics, would result in a definitive diagnosis that would lead to known outcome, thereby suggesting treatment options if the predicted outcome is undesirable. In general, pulp tests are more sensitive and specific when used to determine vitality of pulp tissue, as compared with determining the severity of pulpal inflammation. Given that an extensive review of the highest levels of evidence has shown that “the preoperative presence of apical periodontitis has a dominant, negative effect on the outcome of nonsurgical endodontic treatment,” the goal of pulp testing should be to prevent apical periodontitis and thereby optimize the outcomes of endodontic treatment (131). Alternately stated, the goal of pulp testing should be not only to determine when the pulp has become nonvital (and most likely infected, resulting in the likelihood of apical periodontitis) but also to

**TABLE 7.** Accuracy of Pulse Oximetry and Pulpal Vitality

Reference	Gold standard	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Gopikrishna et al (120)	Clinical <sup>a</sup>	1.00	0.95	0.95	1.00

<sup>a</sup>In Gopikrishna et al (2007), pulpal status was evaluated by direct visual inspection.

TABLE 8. Accuracy of Laser Doppler Flowmetry

Reference	Gold standard	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Evans et al (121)	Clinical <sup>a</sup>	1.0	1.0	—	—

<sup>a</sup>In Evans et al (1999), pulpal status was “confirmed by pulpectomy.”

determine when the pulpal inflammation is irreversible. The ability to determine when inflammation of the pulp has become irreversible would, therefore, guide the practitioner and patient in treatment choices (ie, nonsurgical root canal treatment vs extraction) and preempt subsequent necrosis, infection, and apical periodontitis.

Discussion

Subquestion #5: What Gaps in Knowledge Remain for Developing and Validating Metrics and the Resulting Pulpal Diagnoses?

In the area of clinical quantification of pulpal pain, it was observed that the majority of studies were performed in experimental settings in which the effects of a variable on pain perception were measured. The applicability therefore to endodontic patient populations is limited because the predictive value for pulpal pathology was not tested in a clinical setting. Verbal rating scales, numeric rating scales, visual analog scales, color analog scales, calibrated questionnaires, and finger span scaling were reviewed in the context of pulpal pain assessment. Of these, an informal verbal descriptor scale was found to be the most commonly used by endodontists in patient assessment. Both the visual analog scale and the calibrated questionnaire have been used in experimental settings; however, their utility in practice is limited because of time and resource constraints.

Conditions that can be identified and described with regard to the dental pulp are divided into histologic and clinical classifications. For the purposes of the development of an evidence-based diagnostic terminology, clinical classifications are the most appropriate. The clinically normal pulp is that pulp that is free from symptoms and vital. Inflammation of the pulp or pulpitis is a broad category that can be further divided into reversible or irreversible, depending on the degree and character of presenting symptoms. The demarcation is significant because endodontic intervention is recommended for the latter. These 2 categories can be further divided on the basis of symptoms or the lack thereof. Asymptomatic irreversible pulpitis and symptomatic irreversible pulpitis have different presentations but the same therapeutic outcome. Presumably every tooth with decay, minor trauma, or periodontal disease has asymptomatic reversible pulpitis. Minor symptoms of sweet or thermal sensitivity represent symptomatic reversible pulpitis. Pulp necrosis is characterized by necrosis of the pulp tissue. Total necrosis is the most easily diagnosed entity, whereas partial necrosis can be the most difficult. Hyperplastic pulpitis is a rare condition usually described in immature teeth with gross pulpal exposures. Internal resorption is the result of clastic cells that are stimulated by inflammatory mediators to resorb dentin. Although painless, it can threaten tooth retention if left unchecked. Pulp calcification is the result of degenerative changes in the dental pulp, with exuberant dentinogenesis as a result

of chronic irritation of the pulp. The categories of previously initiated treatment (incomplete) and previously treated pertain to those teeth that have had endodontic treatment either initiated or completed.

On the basis of pulp pathophysiology, the diagnostic terms that best represent pulpal health and disease are the following:

- Clinically normal pulp
- Reversible and irreversible pulpitis
- Pulp necrosis
- Root-filled tooth without signs of infection
- Root-filled tooth with signs of infection
- Incomplete endodontic treatment without signs of infection
- Incomplete endodontic treatment with signs of infection
- Pulp canal mineralization
- Hyperplastic pulpitis
- Internal inflammatory resorption (active or inactive)
- Internal surface resorption

The subcommittee recognizes that there are other qualifiers such as the perceived presence or absence of infection (ie, necrotic pulp with infection). This is not always easily determined clinically. It is recommended as a point of discussion in terms of adopting it as part of terminology. It should be emphasized that levels of evidence in the literature supporting the use of specific clinical diagnostic terminology are generally very low, in that the classification schemes appear to be mainly the opinions of the various authors who provide logical arguments for their choices in developing nomenclature on the basis of studies with levels of evidence rarely exceeding the lowest level. They are usually related to clinical examination findings; however, there is much uncertainty as to the specific correlations between diagnostic information and the actual treatment needs of the patient. More clinical study is needed in this area.

Metrics for establishing pulpal diagnoses were reviewed by our committee. As a result of the lack of evidence that supports the metrics for pulpal diagnosis, it is not possible at this time to determine which metric, or combination with other metrics or history responses, provides the best accuracy for determining pulpal diagnoses. This is particularly important when discriminating between reversible and irreversible pulpitis. Future studies should focus on standardized methods for obtaining a history of presenting symptoms, developing algorithms for pulp diagnoses that incorporate the history of presenting symptoms, results of pulpal tests, and clinical findings. This will facilitate the development of sensitivity, specificity, positive predictive values, and negative predictive values by establishing a gold standard. The identification of biologic markers for reversible and irreversible pulpal inflammation will be of immense value in determining the need for endodontic intervention and the prevention of apical periodontitis.

TABLE 9. Abnormal Response to Cold Testing and Irreversible Pulpitis

Reference	Gold standard <sup>a</sup>	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Seltzer et al (128)	Histology	0.41	0.76	0.34	0.81
Dummer et al (132)	Histology	0.63	0.80	0.48	0.88
Garfunkle et al (119)	Histology	0.57	—	—	—

From Hyman JJ, Cohen M. The predictive value of endodontic diagnostic tests. *Oral Surg Oral Med Oral Pathol* 1984;58:343–6.

**TABLE 10.** Metrics for pulpal diagnosis (and specifically reversible versus irreversible pulpitis) on the basis of history of the presenting symptoms

Reversible pulpitis	Irreversible pulpitis
Sensitivity to mild discomfort	Pain might be absent or present
Short duration or shooting sensation	History of pain is usually given
Not severe	Pain is often moderate to severe
Infrequent episodes of discomfort	Pain is often spontaneous
Seldom hurts to bite unless tooth also fractured or restoration is loose and occlusion is affected	Pain is increasing in frequency, often to the point of being continuous
Could result in irreversible pulpitis if source not removed	Pain usually lingers, especially with increasing episodes
Symptoms usually subside immediately after removal if cause	Patient often requires some type of analgesic
	Might be able to identify specific or multiple stimuli
	Pain radiates or is diffuse or might be localized

Modified from Clinical characteristics of pulpitis. In: Dumsha TC, Gutmann JL. Problems in managing endodontic emergencies. In: Gutmann JL, Dumsha TC, Lovdahl PE, Hovland EJ, eds. Problem solving in endodontics. 3rd ed. St Louis: Mosby-Year Book, Inc., 1997:229–52.

## References

- Turk DC, Dworkin RH, Burke LB, et al. Developing patient-reported outcome measures for pain clinical trials: IMMPACT recommendations. *Pain* 2006;125:208–15.
- Dionne RA, Bartoshuk L, Mogil J, Witter J. Individual responder analyses for pain: does one pain scale fit all? *Trends Pharmacol Sc* 2005;26:125–30.
- Akpata ES, Behbehani J. Effect of bonding systems on post-operative sensitivity from posterior composites. *Am J Dent* 2006;19:151–4.
- Akpata ES, Sadiq W. Post-operative sensitivity in glass-ionomer versus adhesive resin-lined posterior composites. *Am J Dent* 2001;14:34–8.
- Al-Negrish ARS, Hababbeh R. Flare up rate related to root canal treatment of asymptomatic pulpally necrotic central incisor teeth in patients attending a military hospital. *J Dent* 2006;34:635–40.
- Coda B, Tanaka A, Jacobson RC, Donaldson G, Chapman CR. Hydromorphone analgesia after intravenous bolus administration. *Pain* 1997;71:41–8.
- Coda BA, Hill HF, Schaffer RL, Luger TJ, Jacobson RC, Chapman CR. Enhancement of morphine analgesia by fenfluramine in subjects receiving tailored opioid infusions. *Pain* 1993;52:85–91.
- Palace DA, Reid K, Rayens MK. The influence of deep (odontogenic) pain intensity, quality, and duration on the incidence and characteristics of referred orofacial pain. *J Orofac Pain* 1996;10:232–9.
- Franzen OG, Ahlquist ML. The intensive aspect of information processing in the intradental A-delta system in man: a psychophysiological analysis of sharp dental pain. *Behav Brain Res* 1989;33:1–11.
- Gangarosa Sr LP, Ciarlone AE, Neaverth EJ, Johnston CA, Snowden JD, Thompson WO. Use of verbal descriptors, thermal scores and electrical pulp testing as predictors of tooth pain before and after application of benzocaine gels into cavities of teeth with pulpitis. *Anesth Prog* 1989;36:272–5.
- Klages U, Ulusoy O, Kianifard S, Wehrbein H. Dental trait anxiety and pain sensitivity as predictors of expected and experienced pain in stressful dental procedures. *Eur J Oral Sci* 2004;112:477–83.
- McGrath PA, Gracely RH, Dubner R, Heft MW. Non-pain and pain sensations evoked by tooth pulp stimulation. *Pain* 1983;15:377–88.
- Mengel MK, Stiefenhofer AE, Jyvasjarvi E, Kniffki KD. Pain sensation during cold stimulation of the teeth: differential reflection of A delta and C fiber activity? *Pain* 1993;55:159–69.
- Nusstein J, Kennedy S, Reader A, Beck M, Weaver J. Anesthetic efficacy of the supplemental X-tip intraosseous injection in patients with irreversible pulpitis. *J Endod* 2003;29:724–8.
- Owatz CB, Khan AA, Schindler WG, Schwartz SA, Keiser K, Hargreaves KM. The incidence of mechanical allodynia in patients with irreversible pulpitis. *J Endod* 2007;33:552–6.
- Klement W, Medert HA, Arndt JO. Nalbuphine does not act analgetically in electrical painful tooth pulp stimulation in man. *Pain* 1992;48:269–74.
- de Paz Villanueva LEC. *Fusobacterium nucleatum* in endodontic flare-ups. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002;93:179–83.
- Doroschak AM, Bowles WR, Hargreaves KM. Evaluation of the combination of flurbiprofen and tramadol for management of endodontic pain. *J Endod* 1999;25:660–3.
- Ehrmann EH, Messer HH, Clark RM. Flare-ups in endodontics and their relationship to various medicaments. *Aus Endod J* 2007;33:119–30.
- Hsiao-Wu GW, Susarla SM, White RR. Use of the cold test as a measure of pulpal anesthesia during endodontic therapy: a randomized, blinded, placebo-controlled clinical trial. *J Endod* 2007;33:406–10.
- Kemppainen P, Waltimo A, Waltimo T, Kononen M, Pertovaara A. Differential effects of noxious conditioning stimulation of the cheek by capsaicin on human sensory and inhibitory masseter reflex responses evoked by tooth pulp stimulation. *J Dent Res* 1997;76:1561–8.
- Khan AA, McCreary B, Owatz CB, et al. The development of a diagnostic instrument for the measurement of mechanical allodynia. *J Endod* 2007;33:663–6.
- Leavitt AH, King GJ, Ramsay DS, Jackson DL. A longitudinal evaluation of pulpal pain during orthodontic tooth movement. *Ortho Craniofac Res* 2002;5:29–37.
- Lier BB, Rosing CK, Aass AM, Gjermo P. Treatment of dentin hypersensitivity by Nd:YAG laser. *J Clin Period* 2002;29:501–6.
- Oliveira PC, Volpato MC, Ramacciato JC, Ranali J. Articaine and lignocaine efficiency in infiltration anesthesia: a pilot study. *Br Dent J* 2004;197:45–6. discussion 33.
- Rosenberg PA, Amin KG, Zibari Y, Lin LM. Comparison of 4% articaine with 1:100,000 epinephrine and 2% lidocaine with 1:100,000 epinephrine when used as a supplemental anesthetic. *J Endod* 2007;33:403–5.
- Whitworth JM, Kanaa MD, Corbett IP, Meechan JG. Influence of injection speed on the effectiveness of incisive/mental nerve block: a randomized, controlled, double-blind study in adult volunteers. *J Endod* 2007;33:1149–54.
- Oztas N, Ulus T, Bodur H, Dogan C. The wand in pulp therapy: an alternative to inferior alveolar nerve block. *Quint Int* 2005;36:559–64.
- Naidu S, Loughlin P, Coldwell SE, Noonan CJ, Milgrom P. A randomized controlled trial comparing mandibular local anesthesia techniques in children receiving nitrous oxide-oxygen sedation. *Anesth Prog* 2004;51:19–23.
- Munshi AK, Hegde AM, Latha R. Use of EMLA: is it an injection free alternative? *J Clin Ped Dent* 2001;25:215–9.
- McConahay T, Bryson M, Bulloch B. Clinically significant changes in acute pain in a pediatric ED using the Color Analog Scale. *Am J Emerg Med* 2007;25:739–42.
- Bulloch B, Tenenbein M. Validation of 2 pain scales for use in the pediatric emergency department. *Pediatrics* 2002;110:e33.
- Ahlquist M, Franzen O, Coffey J, Pashley D. Dental pain evoked by hydrostatic pressures applied to exposed dentin in man: a test of the hydrodynamic theory of dentin sensitivity. *J Endod* 1994;20:130–4.
- Merkel S. Pain assessment in infants and young children: the Finger Span Scale. *Am J Nurs* 2002;102:55–6.
- Sessle BJ, Hu JW, Amamo N, Zhong G. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurons in trigeminal subnucleus caudalis (medullary dorsal horn) and its implications for referred pain. *Pain* 1986;27:219–35.
- Mellor AC, Dorman ML, Girdler NM. The use of an intra-oral injection of ketorolac in the treatment of irreversible pulpitis. *Intl Endod J* 2005;38:789–92.
- Ahlquist ML, Franzen OG. Encoding of the subjective intensity of sharp dental pain. *Endod Dent Trauma* 1994;10:153–66.
- Newton JT, Buck DJ. Anxiety and pain measures in dentistry: a guide to their quality and application. *J Am Dent Assoc* 2000;131:1449–57.
- Harazaki M, Takahashi H, Ito A, Isshiki Y. Soft laser irradiation induced pain reduction in orthodontic treatment. *Bulletin of Tokyo Dental College* 1998;39:95–101.
- Motohashi K, Umino M, Fujii Y. An experimental system for a heterotopic pain stimulation study in humans. *Brain Res* 2002;10:31–40.
- Suri A, Kaltenbach ML, Grundy BL, Derendorf H. Pharmacodynamic evaluation of codeine using tooth pulp evoked potentials. *J Clin Pharmacol* 1996;36:1126–31.
- Ahlquist ML, Franzen OG. Inflammation and dental pain in man. *Endod Dent Trauma* 1994;10:201–9.
- Lekic D, Cenic D. Pain and tooth pulp evoked potentials. *Clin Electroencephalogr* 1992;23:37–46.
- Ngassapa D. Correlation of clinical pain symptoms with histopathological changes of the dental pulp: a review. *E African Med J* 1996;73:779–81.
- Akpata ES, Behbehani J. Effect of bonding systems on post-operative sensitivity from posterior composites. *Am J Dent* 2006;19:151–4.
- Smulson MH, Sieraski SM. Histophysiology and diseases of the dental pulp. In: Weine FS, ed. *Endodontic therapy*. 5th ed. St Louis: Mosby; 1996:84–165.
- Simon JHS, Walton RE, Pashley DH, Dowden WE, Bakland LK. *Pulpal pathology*. In: Ingle JI, Bakland LK, eds. *Endodontics*. 4th ed. Baltimore: Williams & Wilkins; 1994:419–38.

48. Seltzer S, Bender IB. The dental pulp: biologic considerations in dental procedures. 3rd ed. Philadelphia: Lippincott; 1984.
49. Berman LH, Hartwell GR. Diagnosis. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 9th ed. St Louis: Mosby-Elsevier; 2006:2–39.
50. Glickman GN, Mickel AK, Levin LG, Fouad AF, Johnson WT. Glossary of endodontic terms. 7th ed. Chicago: American Association of Endodontists; 2003.
51. Morse DR, Seltzer S, Sinai I, Biron G. Endodontic classification. *J Am Dent Assoc* 1977;94:685–9.
52. Torabinejad M. Pulp and periradicular pathosis. In: Walton RE, Torabinejad M, eds. Principles and practice of endodontics. 3rd ed. Philadelphia: WB Saunders; 2002:34–7.
53. Sigurdsson A. Clinical manifestations and diagnosis. In: Ørstavik D, Pitt-Ford TR, eds. Essential endodontology. 2nd ed. Oxford: Blackwell Munksgaard; 2008:235–61.
54. 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* 1963;16:846–71.
55. 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* 1963;16:969–77.
56. Johnson RH, Dachi SF, Haley JV. Pulpal hyperemia: a correlation of clinical and histologic data from 706 teeth. *J Am Dent Assoc* 1970;81:108–17.
57. Baume LJ. Diagnosis of diseases of the pulp. *Oral Surg Oral Med Oral Pathol* 1970;29:102–16.
58. Dummer PM, Hicks R, Huws D. Clinical signs and symptoms in pulp disease. *Int Endod J* 1980;13:27–35.
59. Hyman JJ, Cohen ME. The predictive value of endodontic diagnostic tests. *Oral Surg Oral Med Oral Pathol* 1984;58:343–6.
60. Garfunkel A, Sela J, Ulmanský M. Dental pulp pathosis: clinicopathologic correlations based on 109 cases. *Oral Surg Oral Med Oral Pathol* 1973;35:110–7.
61. Langeland K. Management of the inflamed pulp associated with deep carious lesion. *J Endod* 1981;7:169–81.
62. Lundy T, Stanley HR. Correlation of pulpal histopathology and clinical symptoms in human teeth subjected to experimental irritation. *Oral Surg Oral Med Oral Pathol* 1969;27:187–201.
63. Hasler JE, Mitchell DF. Painless pulpitis. *J Am Dent Assoc* 1970;81:671–7.
64. American Board of Endodontics. Pulpal & periapical diagnostic terminology. Chicago: American Board of Endodontics; 2007.
65. Abbott PV, Yu C. A clinical classification of the status of the pulp and the root canal system. *Austral Dent J* 2007;52(Suppl):S17–31.
66. Lynch CD, McConnell RJ. The cracked tooth syndrome. *J Can Dent Assoc* 2002;68:470–5.
67. Krell KV, Rivera EM. A six year evaluation of cracked teeth diagnosed with reversible pulpitis: treatment and prognosis. *J Endod* 2007;33:1405–7.
68. Trowbridge HO. Histology of pulpal inflammation. In: Hargreaves KM, Goodis HE, eds. Seltzer and Bender's dental pulp. Chicago: Quintessence; 2002:227–45.
69. Reeves R, Stanley HR. The relationship of bacterial penetration and pulpal pathosis in carious teeth. *Oral Surg Oral Med Oral Pathol* 1966;22:59–65.
70. Michaelson PL, Holland GR. Is pulpitis painful? *Int Endod J* 2002;35:829–32.
71. Goodis HE, Bowles WR, Hargreaves KM. Prostaglandin E2 enhances bradykinin-evoked iCGRP release in bovine dental pulp. *J Dent Res* 2000;79:1604–7.
72. Bowles WR, Withrow JC, Lepinski AM, Hargreaves KM. Tissue levels of immunoreactive substance P are increased in patients with irreversible pulpitis. *J Endod* 2003;29:265–7.
73. Hargreaves KM. Pain mechanisms of the pulpodentin complex. In: Hargreaves KM, Goodis HE, eds. Seltzer and Bender's dental pulp. Chicago: Quintessence; 2002:181–203.
74. Glick DH. Locating referred pulpal pains. *Oral Surg Oral Med Oral Pathol* 1962;15:613–23.
75. Torabinejad M, Walton RE. Periradicular lesions. In: Ingle JI, Bakland LK, eds. Endodontics. 4th ed. Baltimore: Williams & Wilkins; 1994:439–64.
76. Baumgartner JC. Pulpal infections including caries. In: Hargreaves KM, Goodis HE, eds. Seltzer and Bender's dental pulp. Chicago: Quintessence; 2002:281–307.
77. Klausen B, Helbo M, Dabelsteen E. A differential diagnostic approach to the symptomatology of acute dental pain. *Oral Surg Oral Med Oral Pathol* 1985;59:297–301.
78. Messer HH. Permanent restorations and the dental pulp. In: Hargreaves KM, Goodis HE, eds. Seltzer and Bender's dental pulp. Chicago: Quintessence Books; 2002:345–69.
79. Dixon AD, Peach R. Fine structure of epithelial and connective tissue elements in human dental polyps. *Arch Oral Biol* 1965;10:71–81.
80. Caliskan MK, Oztup F, Caliskan G. Histological evaluation of teeth with hyperplastic pulpitis caused by trauma or caries: case reports. *Int Endod J* 2003;36:64–70.
81. Kawashima N, Suda H. Immunopathological aspects of pulpal and periapical inflammations. In: Ørstavik D, Pitt-Ford TR, eds. Essential endodontology. 2nd ed. Oxford: Blackwell Munksgaard; 2008:44–80.
82. Fouad AF, Levin LG. Pulpal reactions to caries and dental procedures. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 9th ed. St Louis: Mosby-Elsevier; 2006:514–40.
83. Avery J. Repair potential of the pulp. *J Endod* 1981;7:205–12.
84. Levin LG, Trope M. Root resorption. In: Hargreaves KM, Goodis HE, eds. Seltzer and Bender's dental pulp. Chicago: Quintessence; 2002:425–47.
85. Trope M, Blanco L, Chivian N, Sigurdsson A. The role of endodontics after dental traumatic injuries. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 9th ed. St Louis: Mosby-Elsevier; 2006:610–49.
86. Andreasen JO, Andreasen FM. Textbook and color atlas of traumatic injuries to the teeth. 3rd ed. Copenhagen and St Louis: Munksgaard, Mosby; 1994.
87. Weine FS. Diagnosis and treatment planning. In: Weine FS, ed. Endodontic therapy. 5th ed. St Louis: Mosby; 1996:28–83.
88. Stanley HR, Pereira JC, Spiegel E, Broom C, Schultz M. The detection and prevalence of reactive and physiologic sclerotic dentin, reparative dentin and dead tracts beneath various types of dental lesions according to tooth surface and age. *J Oral Pathol* 1983;12:257–89.
89. Pashley DH, Liewehr FR. Structure and functions of the dentin-pulp complex. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 9th ed. St Louis: Mosby-Elsevier; 2006:460–513.
90. Newton CW, Coil JM. Geriatric endodontics. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 9th ed. St Louis: Mosby-Elsevier; 2006:883–917.
91. Schindler WG, Gullickson DC. Rationale for the management of calcific metamorphosis secondary to traumatic injuries. *J Endod* 1988;14:408–12.
92. Smith JW. Calcific metamorphosis: a treatment dilemma. *Oral Surg Oral Med Oral Pathol* 1982;54:441–4.
93. Kuyk JK, Walton RE. Comparison of the radiographic appearance of root canal size to its actual diameter. *J Endod* 1990;16:528–33.
94. Holcomb JB, Gregory WB Jr. Calcific metamorphosis of the pulp: its incidence and treatment. *Oral Surg Oral Med Oral Pathol* 1967;24:825–30.
95. Cvek M. Treatment of non-vital permanent incisors with calcium hydroxide: I—follow-up of periapical repair and apical closure of immature roots. *Odontologisk Revy* 1972;23:27–44.
96. Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture. *J Endod* 1978;4:232–7.
97. El-Meligy OA, Avery DR. Comparison of mineral trioxide aggregate and calcium hydroxide as pulpotomy agents in young permanent teeth (apexogenesis). *Pediatr Dent* 2006;28:399–404.
98. Sjogren U, Figdor D, Persson S, Sundqvist G. Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. *Int Endod J* 1997;30:297–306.
99. Nair PN, Sjogren U, Figdor D, Sundqvist G. Persistent periapical radiolucencies of root-filled human teeth, failed endodontic treatments, and periapical scars. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;87:617–27.
100. Ray HA, Trope M. Periapical status of endodontically treated teeth in relation to the technical quality of the root filling and the coronal restoration. *Int Endod J* 1995;28:12–8.
101. Aquilino SA, Caplan DJ. Relationship between crown placement and the survival of endodontically treated teeth. *J Prosthet Dent* 2002;87:256–63.
102. Friedman S, Abitbol S, Lawrence HP. Treatment outcome in endodontics: the Toronto Study—phase 1: initial treatment. *J Endod* 2003;29:787–93.
103. Roda RS, Gittleman BH. Non-surgical retreatment. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 9th ed. St Louis: Mosby Elsevier; 2006:944–1010.
104. Abbott PV. The periapical space: a dynamic interface. *Ann R Australas Coll Dent Surg* 2000;15:223–34.
105. Abbott PV. Classification, diagnosis and clinical manifestations of apical periodontitis. *Endod Topics* 2004;8:36–54.
106. Abbott P. Endodontics and dental traumatology: an overview of modern endodontics—teaching manual. Perth: The University of Western Australia; 1999. 11–5.
107. -itis. Dictionary.com. Merriam-Webster's medical dictionary. Merriam-Webster, Inc. Available at: <http://dictionary.reference.com/browse/itis>. Accessed May 04, 2008.
108. Pulpitis. Dictionary.com. Merriam-Webster's medical dictionary. Merriam-Webster, Inc. Available at: <http://dictionary.reference.com/browse/pulpitis>. Accessed May 04, 2008.
109. Necrosis. Dictionary.com. Merriam-Webster's medical dictionary. Merriam-Webster, Inc. Available at: <http://dictionary.reference.com/browse/necrosis>. Accessed May 04, 2008.
110. Calcification. Dictionary.com. The American Heritage Stedman's medical dictionary. Houghton Mifflin Company. Available at: <http://dictionary.reference.com/browse/calcification>. Accessed May 04, 2008.



111. Hyperplasia. Dictionary.com. The American Heritage Stedman's medical dictionary. Houghton Mifflin Company. Available at: <http://dictionary.reference.com/browse/hyperplasia>. Accessed May 04, 2008.
112. Andreasen FM, Andreasen JO, Cvek M. Root fractures. In: Andreasen JO, Andreasen FM, Andersson L, eds. Textbook and color atlas of traumatic injuries to the teeth. 4th ed. Oxford, UK: Wiley-Blackwell Publishing; 2007:344–8.
113. Andreasen FM, Andreasen JO, Cvek M. Root fractures. In: Andreasen JO, Andreasen FM, Andersson L, eds. Textbook and color atlas of traumatic injuries to the teeth. 4th ed. Oxford, UK: Wiley-Blackwell Publishing; 2007:393.
114. Peters DD, Baumgartner JC, Lorton L. Adult pulp diagnosis: I—evaluation of the positive and negative responses to cold and electrical pulp tests. *J Endod* 1994; 20:506–11.
115. Bhaskar SN, Rappaport HN. Dental vitality tests and pulp status. *J Am Dent Assoc* 1973;86:409–11.
116. Hyman JJ, Cohen M. The predictive value of endodontic diagnostic tests. *Oral Surg Oral Med Oral Pathol* 1984;58:343–6.
117. Fuss Z, Trowbridge H, Bender IB, Rickoff B, Sorin S. Assessment of reliability of electrical and thermal pulp testing agents. *J Endod* 1986;12:301–5.
118. Seltzer S, Bender IB, Nazimov H. Differential diagnosis of pulp conditions. *Oral Surg* 1965;19:383–91.
119. Garfunkle A, Sela J, Ulmansky M. Dental pulp pathosis; clinicopathological correlations based on 109 cases. *Oral Surg* 1973;35:110–4.
120. Gopikrishna V, Tinagupta K, Kandaswamy D. Comparison of electrical, thermal, and pulse oximetry methods for assessing pulp vitality in recently traumatized teeth. *J Endod* 2007;33:531–5.
121. 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. *Endo Dent Traumatol* 1999;15:284–90.
122. Berman LH, Hartwell GR. Diagnosis. In: Cohen S, Hargreaves K, eds. Pathways of the pulp. St Louis: Mosby-Elsevier; 2005:2–39.
123. Ingle JI, Heithersay GS, Hartwell GR, et al. Endodontic diagnostic procedures. In: Ingle JI, Bakland LK, eds. Endodontics. Baltimore: Williams & Wilkins; 2002: 203–58.
124. Torabinejad M, Walton R. In: Walton RE, Torabinejad M, eds. Principles and practice of endodontics. 3rd ed. Philadelphia: WB Saunders Co; 2002: 49–71.
125. Torneck CD. A report of studies into changes in the fine structure of the dental pulp in human caries pulpitis. *J Endod* 1981;7:8–16.
126. Grushka M, Sessle BJ. Application of the McGill pain questionnaire to the differentiation of toothache pain. *Pain* 1984;19:49–50.
127. Bender IB. Reversible and irreversible pulpitis: diagnosis and treatment. *Aus Endo J* 2000;26:10–4.
128. Seltzer S, Bender IB, Ziontz M. The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. *Oral Surg* 1963;16:846–71.
129. Barbakow FH, Cleaton-Jones P, Friedman D. An evaluation of 566 cases of root canal therapy in general dental practice: 2—postoperative observations. *J Endod* 1980;6:485–9.
130. Beveridge Brown. The measurement of human dental intrapulpal pressure and its response to clinical variables. *Oral Surg Oral Med Oral Pathol* 1965;19: 655–8.
131. Friedman S. Expected outcomes in the prevention and treatment of apical periodontitis. In: Ørstavik D, Pitt Ford TR, eds. Essential endodontology: prevention and treatment of apical pathosis. Oxford: Blackwell; 2007: 408–69.
132. Dummer PMH, Hicks R, Huws D. Clinical signs and symptoms in pulp disease. *Int Endod J* 1980;13:27–35.
133. Petersson K, Soderstrom C, Kiani-Anaraki M, Levy G. Evaluation of the ability of thermal and electrical tests to register pulp vitality. *Endod Dent Traumatol* 1999;15:127–31.