

Identify and Define All Diagnostic Terms for Periapical/Periradicular Health and Disease States

James L. Gutmann, DDS,* J. Craig Baumgartner, DDS, MS, PhD,[†] Alan H. Gluskin, DDS,[‡] Gary R. Hartwell, DDS, MS,[§] and Richard E. Walton, DMD, MS^{||}

Abstract

Introduction: The purpose of this in-depth investigation was to identify, clarify, and substantiate clinical terminology relative to apical/periapical/periradicular diagnostic states, which is used routinely in the provision of endodontic care. Furthermore, the information gleaned from this investigation was used to link diagnostic categories to symptoms, pathogenesis, treatment, and prognosis wherever possible, along with establishing the basis for the metrics used in this diagnostic process. **Materials and Methods:** Diagnostic terminologies and their relevance to clinical situations were procured from extensive historic and electronic searches and correlated with contemporary concepts in disease processes, clinical assessments, histologic findings (if appropriate), and standardized definitions that have been promulgated and promoted for use in the last 25 years in educational programs and test constructions and for third-party concerns. **Results:** In general, clinical terminology that is used routinely in the practice of endodontics is not based on the findings of scientific investigations. The diagnostic terms are based on assumptions by correlating certain signs, symptoms, and radiographic findings with what is presumed (not proven) to be the underlying disease process of a given clinical state. There were no studies that specifically tried to assess the accuracy of the metrics used contemporarily for the classification of clinical disease states. **Conclusion:** A succinct diagnostic scheme that could be described thoroughly, agreed on unanimously, coded succinctly for easy electronic input, and ultimately used for follow-up analysis would not only drive treatment modalities more accurately, but would also allow for future outcomes assessment and validation. (*J Endod* 2009;35:1658–1674)

Key Words

Apical, periapical and periradicular disease, diagnostic categories, diagnostic terms

One of the hallmarks of any profession is its distinct lexicon; this is no different with the dental specialties. Within this framework of the lexicon, there should exist clarity, succinctness, and specificity that are based on sound biological principles and understandings, clinical realities, and daily usages. Even in a global society in which differences may exist, there must be a commonality of thought, a distinct explicitness of meaning, and a rational basis for the choice of terminology and its routine application, as opposed to personalized, empiric bias that is used to exaggerate an individual's thought process or perceived and unsupported interpretation. Oftentimes, the latter is identified as colloquial and bears little resemblance to the actual issue, event, or procedure at hand. Within the discipline of endodontics, this latter type of lexicon has been proffered in contemporary times regardless of the historic framework on which it has evolved.

On the other hand, the clinical discipline of endodontics and its scientific counterpart, endodontology, have been using a terminology in which biological concepts are commingled with that of the clinical, often leaving the reader or clinician confused, with the real issue being obfuscated by the dual meaning of commonly used and unclear definitions. Hence, the communication between and among colleagues can suffer as well. For example, the specific determination of pulpal and apical (also known as periapical/periradicular) diagnostic states, the instruments and tests used to determine these conditions, the clinician's understanding of such an ability to make the appropriate determination, and the patient's understanding of the issues at hand are often times confusing, with multiple clinicians arriving at vastly different interpretations of the same data. Furthermore, this can lead to treatment that may or may not be warranted or at least may not have a sound basis for application. For example, one practitioner may describe a clinical set of diagnostic data as being an abscess, whereas another might label it as an acute apical periodontitis, another an apical infection, and another a tooth that has had a "blow up." Moreover, neither definition nor interpretation identifies a treatment appropriate to the clinical reality that is present or how the information gleaned during the examination of the patient, if thorough and defining, was used to make the final determination of the patient's status. However, there is a perception by many clinicians that the treatment is actually not different with the different diagnostic terms stated, which raises the question as to whether the diagnosis should be a separate activity from the treatment. The diagnosis may affect the treatment plan, prognosis, and the need for supportive therapy in addition to the treatment itself.

The purpose of this investigation was to examine, through an extensive search of the literature, the scientific and clinical bases for periapical diagnostic states and their diagnostic terms in an attempt to answer the following questions.

1. How should the degree of periapical pain be quantified clinically?
2. What are the endodontically related conditions involving root-supporting tissues?
3. Based on the highest level of available evidence, what diagnostic terms best represent apical/periapical/periradicular health and the various forms of periapical/periradicular disease?
4. Which combination(s) of metrics provide the maximal accuracy for establishing periapical diagnoses?
5. What gaps in knowledge remain for developing and validating metrics and the resulting periapical diagnosis?

From *Baylor College of Dentistry, Dallas, TX; [†]Oregon Health & Science University, Portland, Oregon; [‡]Department of Endodontics, University of the Pacific, San Francisco, California; [§]Naval Postgraduate Dental School, Bethesda, Fairfax, Virginia; and ^{||}Department of Endodontics, University of Iowa College of Dentistry, Iowa City, Iowa.

Address correspondence to James L. Gutmann, DDS, 1416 Spenwick Terrace, Dallas, TX 75204-5529. E-mail address: jlgutmann@earthlink.net.

0099-2399/\$0 - see front matter

Copyright © 2009 American Association of Endodontists. doi:10.1016/j.joen.2009.09.028

Material and Methods

Literature Search

The historic basis for the classification of diseases of the root-supporting tissues and the terminology used to describe each diagnostic state were investigated by using information obtained from textbooks that have been identified as classic and meaningful in the evolution of the discipline of endodontics. Each text consulted is detailed later with the data gleaned from within. The initial purpose of providing this information in detail is for the contemporary reader to appreciate the historic evolutionary process of the diagnostic issues that have served as the basis for current thinking and applied terminology.

Historic Review

Harris (1) wrote that the only disease entity that was pertinent was the alveolar abscess. Alveolar abscess is defined as deep-seated, throbbing, and painful disease that at times is excruciating and continues with only occasional slight intermissions after matter is formed, when it, in a great degree, subsides, and is succeeded by slight paroxysms of heat and cold. He recognized that the abscess could open to the oral cavity after bone resorption but did not provide a specific designation (eg, fistula or sinus).

Tomes and Nowell (2) focused on the inflammation of the alveolar periosteum (lining membrane of the sockets of the teeth) and addressed general inflammation, identifying it as being from "...a bad state of the system..." and local inflammation "involving the periosteal investment of the roots of one or two teeth." They described this inflammation as acute or chronic and referred to acute and chronic periostitis. These two classifications could then be categorized further into septic or nonseptic.

- Acute alveolar abscess: caused by the escape from the end of the root of putrid contents of the pulp chamber and the root canals, thus inoculating with septic material the tissues external to the apex of the root.
- Chronic alveolar abscess: authors do identify the possibility of a "fistulous opening" developing.

Buckley (3) published a text that dealt with the practical application of drugs and remedies in the treatment of disease. He identified four primary disease states for the periapical tissues.

1. Nonseptic pericementitis
2. Septic pericementitis
3. Acute alveolar abscess—with and without the presence of a sinus tract
4. Pericemental abscess (inflammation of the pericemental tissues without the presence of an infection)

Blair (4) in his treatise on surgery and diseases of the mouth and jaws described "peridental" pathosis—pericementitis, alveolar abscess and alveolar fistula—as the only meaningful diagnostic states. This was probably true at the time as the treatment for these entities was the same—extraction.

Marshall (5) followed Buckley's schema with some additional classifications, such as a tooth may develop an acute or chronic, septic or nonseptic, and apical or lateral pericementitis. Furthermore, he provided a distinction between the acute and chronic apical abscess as follows:

- *Acute apical abscess*—develops from an infection of the pulp. Difference between the acute and chronic is based on the virulence of the organisms involved, the resistance of the patient and the relative extent of the tissue proliferation.

- *Chronic apical abscess*—long-continued infection maintained by organisms of relatively low virulence. Four general types:

1. Abscess is partially opened and there is drainage via the root canal
2. "Fistulous" opening is present—known as chronic abscess with sinus
3. Granuloma (chronic dentoalveolar abscess)
4. Variation of #2 with the "fistulous discharge" coming at the free margin of the gingival between the root and the alveolar wall

These four classifications were described from more of a histopathological standpoint by Gilmer (6) in 1914. Marshall appears to have characterized the chronic lesion radiographically in a manner that is still used today: "The roentgenograph is one of the best means at our disposal for determining the existence of a chronic condition. It is characterized by a rather well-defined radiolucent area, or dark shadow, which may or may not be exactly at the apex of the tooth."

Prinz (7, 8) in his text publications of 1928 and 1937, identified specific diseases of the "pericementum" with reference to inflammation of the "peridental" membrane.

Clinical observations for the inflammation of the peridental membrane were:

- The diseased tooth is readily located—the pain is steady in degree and in its position. No reflex symptoms are observed.
- The tooth is very sore to touch; occlusion in mastication or ordinary shutting of the teeth produces pain irrespective of thermal changes.
- Percussion induces pain.
- The tooth is raised in its socket and strikes before any of the others occlude.
- Pressure at first usually relieves the pain; later it is intensified. In the later stages, swelling is common.
- There is little reaction to temperature changes; cold may give relief, while heat does not materially affect it.
- The pain is localized, dull, steady, boring or throbbing in character; it is not paroxysmal, but greatly increased on assuming a recumbent position. Pain remains more or less constant without much reference to external conditions.
- Submaxillary lymph nodes may be swollen, tender and painful to pressure.

Classification—author refers to an excellent classification of disease of the "pericementum" from a pathological view point proposed by Arkövy in 1885 (9) as follows:

- *Periodontitis acuta*
 1. Periodontitis acute marginalis
 2. Periodontitis acuta apicalis
 3. Periodontitis acuta circumscripta (a—consecutive; and b—idiopathica)
 4. Periodontitis acute diffusa
 5. Periodontitis acuta purulenta (a—circumscripta; and b—diffusa)
 6. Abscessus apicalis
 7. Phlegmone acuta septic osteo-peridentalis (Periodontitis toxica)
- *Periodontitis chronic*
 1. Periodontitis chronic apicalis
 2. Periodontitis chronic diffusa
 3. Periodontitis chronic purulenta
 4. Periodontitis chronic granulomatosa
 5. Necrosis apicalis
 6. Necrosis totalis
 7. Caries alveolaris, seu osteoperiostitis alveolodentalis; or according to Magitot (seu Pyorrhoea alveolaris)

Prinz's Classification and Definitions

• Acute pericementitis

1. *Acute apical pericementitis* (acute dentoalveolar abscess)—an acute, exudative, destructive inflammation of the pericementum at the apical region of the tooth.
2. *Acute marginal pericementitis*—an acute, exudative, destructive inflammation of the pericementum restricted to its margin and clinically usually associated with marginal gingivitis.
3. *Acute diffuse suppurative pericementitis*—an acute circumscribed or diffuse suppurative destructive inflammation of the pericementum.
4. *Acute intraradicular pericementitis* (acute pericemental-paradental abscess)—an acute, circumscribed, destructive inflammation (abscess) within the pericementum arising from an infection, which enters at the gingival margin of a tooth and not from its apex. Usually, it occurs near the middle or lower-third portion of the tooth root or between the roots of teeth with living pulps.

• Chronic pericementitis

1. *Chronic apical suppurative pericementitis* (chronic dentoalveolar abscess)—a chronic circumscribed destructive suppuration of the pericementum accompanied by necrosis of the alveolar bone within the region of the affected tooth and the spontaneous discharge of pus. It arises as a sequence of a latent infection derived from a gangrenous pulp or from pulp remnants left in the apical region of a filled-root canal.
2. *Chronic proliferating pericementitis* (granuloma and radicular cyst)—a circumscribed chronic productive inflammation of the pericementum about the apex of a tooth resulting in the formation of granulation tissue known as a dental granuloma that, at times, develops into a radicular cyst.
(Note: the term granuloma as applied to the granulation tissue observed about the apex of the tooth appears to have been introduced into the dental literature by Arkövy. *Diagnostik der Zahnkrankheiten*, University of Budapest, Stuttgart 1885:203. [The term is probably a misnomer as the nature of the tissue is primarily granulomatous.])

It was in Kronfeld's opinion "...that every inflammatory reaction occurring in the soft tissue and bone surrounding the root surface should be given the general name periodontitis, and that this term should be employed in the general description of any inflammatory conditions in this area regardless of the etiology or type of inflammation" (10). Furthermore, he felt that older terms such as pericementitis and periapical abscess should be abandoned because not every inflammation of the periapical tissues cause abscess formation. He listed the periodontitis categories as follows:

- Traumatic periodontitis
- Chemical periodontitis
- Infective periodontitis

This approach therefore led to the following classification:

1. *Acute traumatic periodontitis*—reaction of the periodontal tissues to injury caused by trauma of any kind.
2. *Chemical periodontitis*—a reaction of the materials that have been introduced into the root canal during root canal procedures ranging from initially acute to chronic after longer exposures.
3. *Acute infective periodontitis*—requires the presence of pathogenic microorganisms; pain, swelling, heat, redness and occasionally fever.

4. *Chronic infective periodontitis*—requires the long-term persistence of microorganisms; "no clinical symptoms at all or only a temporary feeling of slight uneasiness or discomfort." Chronic osteitis with transformation of the periodontal membranes and alveolar bone into granulation tissue (here also the concept of granulation tissue vs. granulomatous tissue appears to be confused).
5. *Acute dentoalveolar abscess*—purulence, collateral edema and swelling of the soft tissues of the face (uses the term cellulitis at this stage of advanced abscess formation).
6. *Acute exacerbation of chronic periapical inflammation*—a concept that we have seen with various labels such as phoenix abscess, recrudescence, and so on.
7. *Condensing osteitis* (bone sclerosis)—due to chronic periapical inflammation.

Coolidge's classification was very much in line with Kronfeld but note carefully the use of the term "inflammation" to primarily describe the disturbances of the apical periodontal tissues (11):

• Acute inflammation

1. *Traumatic and chemical periodontitis*—an acute inflammation of the apical periodontal tissues with no infection present.
2. *Apical periodontitis from infection*—an acute inflammation of the apical periodontal tissue due to the invasion of these tissues by pathogenic microorganisms through the apical foramen.
3. *Acute dentoalveolar abscess*—an acute inflammation of the periodontal tissue around the apical foramen attended by an increasing amount of inflammatory exudates and suppuration.

• Chronic inflammation

1. *Suppurative periodontitis* (chronic abscess)—a destructive suppuration of the periodontal tissues about the apex of a diseased tooth with an intermittent discharge of pus.
2. *Proliferative periodontitis*
 - *Granuloma*—circumscribed area of chronic inflammation in the periodontal tissues surrounding the apical foramen, which is filled with a mass of granulation tissue.
 - *Radicular cyst*—if granulation tissue is surrounded by epithelium.
 - *Condensing osteitis*—sclerotic bone with little or no granulation tissue.

Coolidge also uses the same characteristics for the clinical signs and symptoms for inflammation of the periodontal membrane as detailed by Prinz above.

Grossman provided a slight variation as to the disease of the periapical tissues as follows (12, 13):

1. *Acute apical periodontitis*—an acute inflammation of the apical periodontal membrane as a result of irritation via the root canal or from trauma. Tenderness or pain on the tooth when tapped or pushed in a certain direction; pain can be severe making closure of the teeth difficult.
2. *Acute alveolar abscess*—a localized collection of pus in the alveolar bone at the root apex of the tooth after death of the pulp, with extension of the infection through the apical foramen into the periapical tissues; severe local and sometimes general adverse reaction; advanced state of acute apical periodontitis.
3. *Chronic alveolar abscess*—a long-standing, low-grade infection of the periapical alveolar bone; generally symptomless and may be detected during routine x-ray examination or because of the presence of a fistula; swelling is seldom present.



Figure 1. Examples of what could be referred to as an “apical” lesion based on clinician interpretation and past educational and clinical experiences. (A) A radiograph of maxillary left first molar that exhibits, based on location, an “apical” lesion or radiolucency at the apex of the mesial buccal root. The palatal root may not be considered as having a specific apical lesion but could be interpreted as having a “thickened periodontal ligament space.” (B) A radiograph of mandibular left second molar that exhibits based on location, an “apical” lesion or radiolucency at the apex of the mesial and distal roots. (C) A radiograph of maxillary left first molar that exhibits, based on location, an “apical” lesion or radiolucency at the apex of the palatal root.

4. *Subacute alveolar abscess*—arbitrary classification of cases that do not run a short, severe course as in the acute alveolar abscess, nor yet a symptomless, long-standing course as in chronic alveolar abscess, but present symptoms having some of the characteristics of each. It is applied particularly to cases of chronic abscess, which flare up and present mildly acute symptoms.
5. *Granuloma*—a growth of granulation tissue continuous with the periodontal membrane resulting from death of the pulp with extension of infection, or diffusion of bacterial toxins, periapically.
6. *Cyst*—slowly growing epithelial-lined sac at the apex of a tooth; presupposes physical, chemical or bacterial injury resulting in death of the pulp, followed by stimulation of the epithelial debris of a/k/a rest of Malassez.

This review of the historic roots of the endodontic diagnostic schemes and advocated terminology set the stage for considering the contemporary assessment of these parameters. Although the historic literature focused on the use of percussion, palpation, and mobility testing, along with radiographic findings to support diagnoses

of the periapical tissues, little if any data exist to validate the metrics or provide quantifiable parameters of assessment. Furthermore, historic classifications did not necessarily delve into the infectious nature of the periapical lesion that was present. Moreover, the clinical diagnostic states did not identify a repair-predictive value for each category or description.

Also noted in the historic review was the use of specific terminology that described the location and nature of the disease process, namely the terms apical, periapical, periradicular, acute, chronic, abscess, and periodontitis. In this respect, the terms “apical” and “periapical” were predominate with the term “periradicular” not being advocated until the mid-1980s and defined as “enclosing or surrounding the root portion of the tooth” (14). Examples of what could be defined as “apical,” “periapical” and “periradicular” are shown in Figures 1 through 3 and may be open to clinician interpretation. Although the terms can be used interchangeably and apical may be the preferred term, a review of the radiographic characteristics depicted in Figures 1 through 3 shows how easily these terms can and will be used based on clinician assessment because there are no set criteria parameters of measurement in the literature to distinguish usage of these terms. The

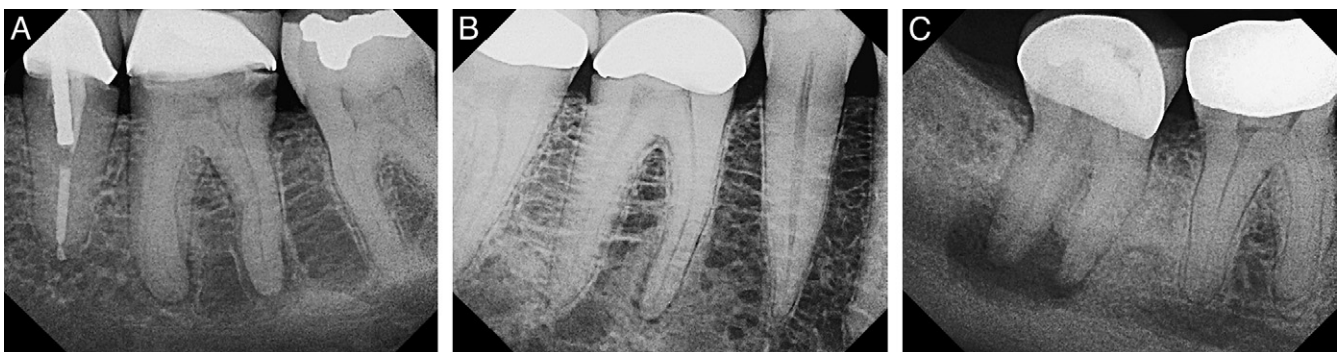


Figure 2. Examples of what could be referred to as a “periapical” lesion based on clinician interpretation and past educational and clinical experiences. (A) A radiograph of mandibular left first molar that exhibits, based on location, a “periapical” lesion or radiolucency at the apex of the mesial and distal roots. The distinction in the use of the term “periapical” versus “apical” would be based on the position of the radiolucencies that are located both apically and 3 to 4 mm coronally or “periapically” around the root end. (B) A radiograph of mandibular right first molar that exhibits, based on location, a “periapical” lesion or radiolucency at the apex of the mesial and distal roots. Although some clinicians may describe this as a thickened periodontal ligament space on the mesial, the width of the ligament space far exceeds that of 2 mm, which is considered as an acceptable criteria. (C) A radiograph of mandibular right second molar that exhibits, based on location, a “periapical” lesion or radiolucency at the apex of the mesial and distal roots. Some clinicians may describe this as a “periradicular” lesion because of its extensive encompassing of the root structure.

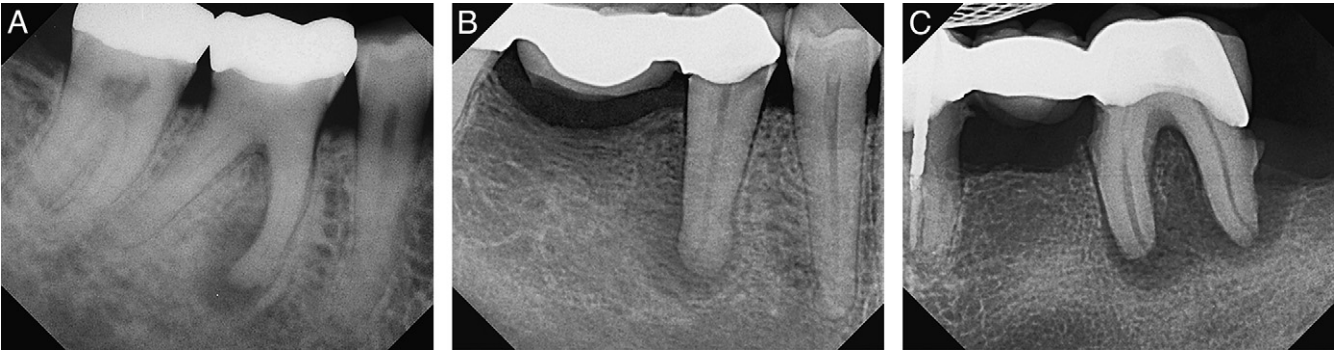


Figure 3. Examples of what could be referred to as a “periradicular” lesion based on clinician interpretation and past educational and clinical experiences. (A) A radiograph of mandibular left second premolar that exhibits, based on location, a “periradicular” lesion or radiolucency that extends significantly from the apex along the length of the distal root wall. (B) A radiograph of mandibular right first molar that exhibits, based on location, a “periradicular” lesion or radiolucency at the apex of the mesial root that extends coronally along the mesial root wall to the furcation. (C) A radiograph of mandibular right second premolar that exhibits, based on location, a “periradicular” lesion or radiolucency at the apex that extends coronally along the distal root wall. (D) A radiograph of mandibular left second molar that exhibits, based on location, a “periradicular” lesion or radiolucency at the apex of the mesial and distal roots that extends coronally into the furcation and up the distal root wall.

terms “acute” and “chronic” appeared to have suffered misinterpretation over the years, that being the interchangeable usage of these terms to describe both clinical situations and histologic findings. Historically, there is no evidence or suggestion to support the concepts of the use of the terms symptomatic or asymptomatic when referring to a diseased state at the end of a root.

Contemporary Literature Search

A more contemporary perspective on the question at hand was pursued through a library search of MEDLINE that focused on the following challenge: identify and define all diagnostic terms for periapical health and disease states. As a basis for the search, an evidence-based study was used as a framework for investigation, data procurement, and assessment (15).

A MEDLINE search was conducted using the following specific MeSH terms: periapical periodontitis, periapical diseases, dental pulp necrosis, osteitis, diagnosis, and etiology. An attempt was made to use the term classification, but this was much too broad for this focused search.

The first search strategy (Table 1) was run using only the first four MeSH headings and limiting the search to dental journals.

References and abstracts of the 675 articles were reviewed for relevance that was highly specific to the question asked with 22 chosen for further in-depth review (Table 2).

A second search strategy (Table 3) was run that limited the search to diagnosis, evidence-based and systematic reviews.

References and abstracts of the 284 articles were reviewed for relevance to the question asked with 9 chosen for further in-depth review (Table 4).

A third search strategy (Table 5) was run that limited the search to the English language and human studies, along with a diagnostic sensitivity/specificity.

TABLE 1. Search of First 4 MeSH Headings in Dental Journals Only

#	Searches	Results
1	Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis/di, et	1,477
2	Limit to dentistry journals	675

There were no articles within the 42 identified that were appropriate for the question at hand.

The fourth search strategy (Table 6) attempted to narrow the field of investigation.

There were no articles within the 136 identified that were appropriate for the question at hand.

Additional articles not identified in the search, but identified in manual searching are listed in Table 7 and were reviewed for relevance along with the other 31 articles identified in Tables 2 and 4.

A search strategy using SCOPUS and the previously identified MeSH terms was also performed. Ninety-five articles were identified with 2 specific articles being assessed further for relevance (See Table 7**).

Further information on the questions at hand was obtained from the following sources.

- Current and contemporary textbooks including specific issues of journals dedicated entirely to the topical issues (Table 8)
- *Glossary of Endodontic Terms* published by the AAE and terms from the American Board of Endodontics (Table 9)
- National Board Examination’s Endodontics Test Construction Committee—source uses the *Glossary of Endodontic Terms* published by the American Association of Endodontists.
- *Application of the International Classification of Diseases to Dentistry and Stomatology* – 3rd Edition, World Health Organization, Geneva, 1995 (Table 10)
- *Glossary of Periodontal Terms*—The American Academy of Periodontology 2001, 4th edition (Table 11)
- Terms used in oral and maxillofacial pathology (Table 12)

The information obtained from the previously described sources was used to address the purposes of this study that were identified in the introduction of the article. This work was accomplished by a team of appointed clinicians/academics/scientists who each had the responsibility of addressing one of the specific questions posed in the purpose of this study. Each content expert had access to a 61-page working document that was of compilation of all searched and documented data. Data and its interpretation were compiled with the results being presented below on an individual basis relative to the specific questions proffered.

TABLE 2. Initial MEDLINE Search: Remaining Articles for Review

- Abbott PV, Yu C. A clinical classification of the status of the pulp and the root canal system. *Aus Dent J* 2007;52(1 Suppl):S17–31.
- Nair PNR. Pathogenesis of apical periodontitis and the causes of endodontic failures. *Crit Rev Oral Biol Med* 2004;15:348–81.
- Lopez-Marcos JF. Aetiology, classification and pathogenesis of pulp and periapical disease. *Medicina Oral, Patologia Oral y Cirugia Bucal* 2004;9:Suppl:58–62, 52–7.
- West JD. Endodontic diagnosis. Mystery or mastery? *Dent Today* 2004;23:80–7.
- Abbott PV. The periapical space—a dynamic interface. *Aust Endod J* 2002;28:96–107.
- McCaul LK, McHugh S, Saunders WP. The influence of specialty training and experience on decision making in endodontic diagnosis and treatment planning. *Int Endod J* 2001;34:594–606.
- Chugal NM, Clive JM, Spångberg LS. A prognostic model for the assessment of the outcome of endodontic treatment: effect of biologic and diagnostic variables. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001;91:342–52.
- Abbott PV. The periapical space—a dynamic interface. *Ann Royal Austral Coll Dent Surg* 2000;15:223–34.
- Carrotte PV. Current practice in endodontics: 2. Diagnosis and treatment planning. *Dent Update* 2000;27:388–91.
- Kuc I, Peters E, Pan J. Comparison of clinical and histologic diagnoses in periapical lesions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000;89:333–7.
- Pierce A. Pulpal injury: pathology, diagnosis and periodontal reactions. *Aust Endod J* 1998;24:60–5.
- Nair PNR. Apical periodontitis: a dynamic encounter between root canal infection and host response. *Periodontol* 2000;1997;13:121–48.
- Mandel E, Machtou P, Torabinejad M. Clinical diagnosis and treatment of endodontic and periodontal lesions. *Quintessence Int* 1993;24:135–9.
- Navarrete P, Martinez B. [Comparative study of clinico-radiographic diagnosis and histopathological examination of pulp necrosis]. [Spanish] *Odontol Chilena* 1988;36:3–8.
- Jimenez Planas A, Llamas Cadaval R, Castro Fernandez A, et al. [Clinical and radiographic study of 255 cases of apical periodontitis]. [Spanish] *Revista de Actualidad Estomatol Espanola* 1988;48:73–4, 77–8, 81–2.
- 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.
- 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.
- Smulson MH. Classification and diagnosis of pulpal pathoses. *Dent Clin North Am* 1984;28:699–723.
- Moorer WR, Thoden van Velzen SK, Wesselink PR, Genet JM, Kersten HW. [Chronic apical periodontitis]. [Dutch] *Neder Tijds Tandheelkunde*. 1980;87:318–26.
- Fischer CH. [Sclerotic osteitis and cementoma]. [German] *Deutsche Stomatol (Berlin)* 1968;18:495–502.
- Brose D, Schilder R. [Apical periodontitis]. [German] *Zahntechnik* 1982;23:110–6.
- Apt H, Busch I, Arnold A. [The diagnosis and therapy of periapical periodontitis]. [German] *Stomatol der DDR* 1982;32:392–9.
- Fischer CH. [Ossifying osteitis and its significance for the diagnosis of pulpitis]. [German] *Deutsche Zahnärztliche Zeitschrift* 1966;21:1451–8.

Results

How should the degree of periapical pain be quantified clinically?

Dental pain is undeniably an unpleasant sensory and emotional experience associated with actual tissue damage. Current models of pain view it as a complex experience, consisting of both physiologic and psychological components. It is those psychological components that have made the quantification of dental pain so fraught with interpretive narration and personal overlays, resulting in subjective judgments about how much pain actually exists for the patient. Most patients are fairly uniform in their recognition of thresholds of pain, but they vary greatly in their reactions to the experience. Emotional factors such as anxiety and stress can lower the pain threshold and heighten patient response negating a real understanding of “how much it actually hurts.”

A major problem in pain studies is the quantification or measurement of the pain. Often, attempts at measurement treat tooth pain as if it

were specifically a sensory component (nociceptive pain) only varying in intensity. Clinically, periapical pain is often of a persistent quality accompanied by high levels of apprehension and anxiety. Objective measurement is often difficult when the data are so truly subjective and multidimensional.

Behavioral Signs of Pain

Assessment requires the clinician to be aware of nonverbal signs of pain. Behavioral signs include crying, facial grimacing, clenched jaw, restless legs, and holding or protecting the face as well as palliative aids such as an ice pack or a description of analgesics taken for the pain.

Pain Scales

There has been an ongoing body of literature in the discipline of pain that has been concerned with the development of assessment tools to help patients describe their pain. The pain scale is one tool commonly used to describe the intensity of the pain or how much pain the patient is feeling. The most common pain scales include the Numerical Rating Scale, the Visual Analog Scale, the Categorical Scale, and the Pain Faces Scale (16).

On the Numerical Rating Scale, the patient is asked to identify how much pain he/she is having by choosing a number from 0 (no pain) to 10 (the worst pain imaginable). As the numbers get higher, they stand for pain that is getting worse. The rating scale can be used to describe:

- how the pain feels at its worst
- how the pain feels most of the time
- how the pain feels at its least
- how the pain changes with endodontic therapy

TABLE 3. Search Limited to Diagnosis, Evidence-based, and Systemic Reviews

#	Searches	Results
1	Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis/di, et	1,477
2	Limit to dentistry journals	675
3	Limit #2 to (dentistry journals and “diagnosis (sensitivity)”)	284
4	Limit #3 to (evidence-based medicine and systematic reviews)	0

TABLE 4. Second Medline Search: Remaining Articles for Review

Fleury A, Regan JD. Endodontic diagnosis: clinical aspects. *J Irish Dent Assoc* 2006;52:28–38.
Carrotte P. Endodontics: part 2. Diagnosis and treatment planning. *Br Dent J* 2004;197:231–8.
Halse A, Molven O, Fristad I. Diagnosing periapical lesions—disagreement and borderline cases. *Int Endod J* 2002;35:703–9.
Stheeman SE, Mileman PA, van 't Hof MA, et al. Diagnostic confidence and the accuracy of treatment decisions for radiopaque periapical lesions. *Int Endod J* 1995;28:121–8.
Steiman HR. Endodontic diagnostic techniques. *Curr Opinion Dent* 1991;1:723–8.
Halse A, Molven O. A strategy for the diagnosis of periapical pathosis. *J Endod* 1986;12:534–8.
Hayes RL, Corcoran JF, Zillich RM. Diagnosis and treatment of diseases of the dental pulp and periapical tissues. *J Mich Dent Assoc* 1981;63:313–7.
Morse DR, Seltzer S, Sinai I, et al. Endodontic classification. *J Am Dent Assoc* 1977;94:685–9.
Jones EH. Roentgenology in endodontics. 2. Integration of roentgenology and vitality tests in the assessment of periapical pathoses. *Aust Dent J* 1966;11:310–5.

The visual analog scale is a straight line with the left end of the line representing no pain, and the right end of the line representing the worst pain. Patients are asked to mark on the line the location of their pain.

The Categorical Pain Scale has four categories: none, mild, moderate, and severe. Patients are asked to select the category that best describes their pain. They can also quantify their pain with a numeric descriptor.

None (0) Mild (1-3) Moderate (4-6) Severe (7-10)

The Pain Faces Scale uses six faces with different expressions on each face. Each face is a person who feels happy because he/she has no pain or feels sad because he/she has some or a lot of pain. The person is asked to choose the face that best describes how he/she is feeling. This rating scale can be used by individuals' age 3 years and older.

Numeric scales communicate limited data. On a scale of 0 to 10, one person's 8 is not the same as another's. If nothing else, the literature recognizes that pain scales seem to communicate the patient's urgency in wanting his/her pain addressed. Pain at 7 to 10 is a serious problem. Pain at level 10 is perceived as an emergency by the patient and should usually be treated as an emergency by the clinician.

The literature shows that reliability and validity of verbal descriptors can vary when analgesics are used (17). It is also understood that verbal descriptors provide misleading information in terms of both accuracy and sensitivity (18). However, verbal descriptors may provide

a more sensitive tool over visual scales for separating intensity and unpleasantness as a distinction about the quality of the pain (19).

Assays and Genetic Markers

There is a body of evolving research that links bacterial markers and genetic assays with symptomatic teeth. The quantification of endotoxin concentration by assay in necrotic root canals and dentin has a positive relationship to symptomatic teeth. A positive association has been found between endotoxin (lipopolysaccharide) and spontaneous pain, tenderness to palpation and percussion, swelling, and purulent exudates (20). Increased endotoxin in carious dentin was also directly correlated to irreversible pulpitis by assay that used the Quantitative Chromogenic Test using *Limulus* lysate (21). In a study investigating symptomatic periapical pathosis, results provided compelling evidence that herpesviruses participate in the pathogenesis of symptomatic periapical pathosis. Slots et al (22) showed 76% of 25 symptomatic periapical specimens harvested during root-end resection contained the presence of human cytomegalovirus and/or Epstein-Barr virus. Complementary DNA methodology was used to identify transcription of herpesviral genes (22).

Metrics. Mechanical allodynia, defined as a reduction in mechanical pain threshold, is an essential diagnostic feature of inflammation of the periodontal ligament. Traditional methods for measuring mechanical

TABLE 5. Search Limited to English Language and Human Studies, Diagnostic Sensitivity/Specificity

#	Searches	Results
1	Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis.mp. [mp=title, original title, abstract, name of substance word, subject heading word]	8,739
2	Limit #1 to (English language and human studies)	4,827
3	Limit #2 to (dentistry journals and "diagnosis (sensitivity)")	284
4	From #3 keep 1-284	0
5	Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis.mp. [mp=title, original title, abstract, name of substance word, subject heading word]	121,467
6	Limit #5 to dentistry journals	3,434
7	Limit #6 to (English language and human studies)	2,201
8	Limit #7 to "diagnosis (sensitivity)"	42

TABLE 6. Narrowing the Field of Investigation

#	Searches	Results
1	Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis.mp. [mp=title, original title, abstract, name of substance word, subject heading word]	8,739
2	Limit #1 to (English language and human studies)	4,827
3	Limit #2 to (dentistry journals and "diagnosis (sensitivity)")	284
4	From #3 keep 1-284	0
5	Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis.mp. [mp=title, original title, abstract, name of substance word, subject heading word]	121,467
6	Limit #5 to dentistry journals	3,434
7	Limit #6 to (English language and human studies)	2,201
8	Limit #7 to "diagnosis (sensitivity)"	823
9	Limit #8 to (dentistry journals and "diagnosis (sensitivity)")	823
10	Limit #9 to "etiology (sensitivity)"	136

TABLE 7. Articles Identified Through Manual Searching

- Schulz J, Gutterman JR. The diagnostic correlator: an endodontic teaching device. *Oral Surg* 1975;40:537-47.
- Taatz H, Stiefel A. [The pathomorphology of chronic apical periodontitis] [German] *Zahn Mund Kieferheilkd Zentralbl* 1977;65:611-25.
- Barańska-Gachowska M, Luciak M, Biernawska K. [Correlations between the clinical histological diagnosis in chronic inflammatory processes of periapical tissues] [Polish] *Czas Stomatol* 1979;32:19-35.
- Antrim DD, Bakland LK, Parker MW. Treatment of endodontic urgent care cases. *Dent Clin North Am* 1986;30:549-72.
- Montgomery S, Ferguson CD. Endodontics: diagnostic, treatment planning and prognostic considerations. *Dent Clin North Am* 1986;33:48.
- Abu Asma NE, Abd el-Rassak MY, es-Saaïd HY. Correlation between histological and radiological appearance of periapical radiolucencies. *Egypt Dent J* 1990;36:245-59.
- Grossman LI. Comment on endodontic classification. Letter to the editor. *J Am Dent Assoc* 1997;95:183-4.
- **Zehnder M, Gold, SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. *J Clin Periodontol* 2002;29:663-761.
- **Molven O, Halse A, Fristad I. Long-term reliability and observer comparisons in the radiographic diagnosis of periapical disease. *Int Endod J* 2002;35:142-7.

allodynia in a tooth are not quantitative. Most involve percussion applications, which can vary greatly. A study by Khan et al (23) evaluated the reliability of a bite force transducer to measure mechanical pain thresholds, which might have application as a quantitative diagnostic aid for measuring mechanical allodynia in patients with apical periodontitis. The results of the Khan study confirmed that the digital force transducer was a reliable method to measure mechanical pain thresholds when compared with control teeth in the same patient. Pressure algometers have also shown high reliability for assessing pressure pain thresholds in the temporomandibular joint and masticatory muscles of patients compared with controls (24).

Integrated Signs and Predictors. There is evidence in the literature based on moderate to large numbers of patients in clinical trials that portrays a differential diagnostic approach to signs of periapical disease that may predict the quality of symptoms. Klausen et al (25) reported a combination of signs and symptoms yielding a correct diagnosis of pulpal degeneration in 82% of cases including constant pain, sensitivity to temperature changes, an extruded feeling in the tooth, impaired mouth opening, mobility, and tenderness to palpation in

the apical area. These signs and symptoms in various combinations were highly accurate predictors of disease (25). Others have found that a relationship exists between radiolucency size and the presence of amalgam restorations in patients who develop clinical signs of infection. They recommended early intervention based on these predictors (26).

The PennEndo database also has yielded correlations that have meaning for the differential diagnosis of periapical pain based on advanced statistical analyses. Using a logistic regression model, the study has elucidated signs and symptoms that correlate to improve differential diagnoses. Sharp pain was more likely associated with pulpal pathosis, whereas dull pain was more likely associated with periapical pathosis. Percussion and palpation tests were powerful in differentially diagnosing between pulpal and periapical conditions (27).

Based on available evidence, it appears the most fruitful avenues of research in areas that may lead to better and more accurate quantification modalities of periradicular pain may lie in devices that allow direct measurements of pain thresholds as well as assays of markers of pathogenicity and virulence.

TABLE 8. Contemporary Texts and Adjunctive Publications

1. Ingle J, Bakland L. Ingle's endodontics. 6th ed. Hamilton: Ontario BC Decker; 2009.
2. Cohen S, Hargreaves K. Pathways of the pulp. 9th ed. St. Louis: Mosby; 2006.
3. Gutmann JL, Dumsha TC, Lovdahl P, et al. Problem solving in endodontics. 4th ed. St. Louis: Mosby; 2004.
4. Dumsha TC, Gutmann JL. Clinician's endodontic handbook. 3rd ed. Hudson, OH: Lexi-Comp; 2005.
5. Castellucci A. Endodontics. Vol. I. Florence: Edizioni Odontoiatriche Il Tridente S.r.l.; 2004.
6. Seltzer S. Endodontology, biologic considerations. 2nd ed. Philadelphia: Lea & Febiger; 1988.
7. Nicholls E. Endodontics. 3rd ed. Bristol: John Wright & Sons; 1984.
8. Ørstavik D, Pitt Ford TR. Essential endodontology. London: Blackwell; 1998.
9. Bergenholtz G, Horsted-Bandslev P, Reit C. Textbook of endodontology. London: Blackwell Munksgaard; 2003.
10. Weine F. Endodontic therapy. 6th ed. St. Louis: Mosby; 2004.
11. Beer R, Baumann M, Kim S. Endodontology. Stuttgart, NY: Thieme; 2000.
12. Daniel J. Advanced endodontics for clinicians. Bangalore: J & J Publishers; 1998.
13. Pitt Ford TR. Harty's endodontics in clinical practice. London: Elsevier Science Ltd; 2004.
14. Mumford JM, Jedyakiewicz NM. Principles of endodontics. London: Quintessence Publishing; 1988.
15. Tronstad L. Clinical endodontics. 2nd ed. Stuttgart, NY: Thieme; 2003.
16. Al Kandari A, Daniel JG. Current concepts in endodontics. Kuwait: Al Alfain Publishing; 1997.
17. Whitworth JM. Rational root canal treatment in practice-quintessentials series. 2nd ed. London: Quintessence Publishing Co Ltd; 2002.
18. Johnson W. Color atlas of endodontics. Philadelphia: Saunders; 2002.
19. Stock CJR, Gulabivala K, Walker RT, et al. Endodontics. 2nd ed. London: Mosby-Wolfe; 1995.
20. Pitt Ford TR, Rhodes JS, Pitt Ford HE. Endodontics. Problem solving in clinical practice. London: Martin Dunitz Ltd; 2002.
21. Trope M, Debelian G. Endodontics manual for the general dentist. London: Quintessence Publishing Co Ltd; 2005.
22. Torabinejad M, Walton R. Endodontic principles and practice. 4th ed. St. Louis: Saunders/Elsevier; 2009.
23. Grossman L, Oliet S, DelRio C. Endodontic practice. 11th ed. Philadelphia: Lea & Febiger; 1988.
24. Morse D. Clinical endodontology. Springfield, IL: C Thomas; 1974.
25. Ingle JI. PDQ endodontics. Hamilton, Ontario: BC Decker; 2005.
26. Huuonen S, Ørstavik D. Radiological aspects of apical periodontitis. *Endod Topics* 2002;1:3-25.
27. Sigurdsson A. Pulpal diagnosis. *Endod Topics* 2003;5:12-25.
28. Abbott PV. Classification, diagnosis and clinical manifestations of apical periodontitis. *Endod Topics* 2004;8:36-54.

TABLE 9. Glossary of Endodontic Terms Published by the AAE and Terms From the American Board of Endodontics

Glossary of Endodontic Terms: American Association of Endodontists, 2003

Acute periradicular abscess—Acute apical abscess—"An inflammatory reaction to pulpal infection and necrosis characterized by rapid onset, spontaneous pain, tenderness of the tooth to pressure, pus formation and eventual swelling of associated tissues." (Synonyms: acute periapical abscess, acute alveolar abscess, dentoalveolar abscess, phoenix abscess, recrudescence abscess, secondary apical abscess)

Chronic periradicular abscess—Suppurative periradicular periodontitis (Chronic apical abscess, Chronic periradicular abscess, Chronic periapical abscess)—"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." (Synonyms: chronic alveolar abscess, chronic dentoalveolar abscess, suppurative apical periodontitis)

Acute periradicular (apical) periodontitis—"Inflammation usually of the apical periodontium producing clinical symptoms including painful response to biting and percussion."

Chronic periradicular (apical) periodontitis—"Inflammation and destruction of apical periodontium that is of pulpal origin, appears as a periradicular radiolucent area and does not produce clinical symptoms."

Subacute periradicular periodontitis—"Inflammation usually of the apical periodontium producing mild clinical symptoms; not as severe as acute periradicular periodontitis."

Focal Sclerosing Osteomyelitis (Condensing osteitis, periradicular osteosclerosis, sclerosing osteitis, sclerotic bone)—"A diffuse radiopaque lesion believed to represent a localized bony reaction to a low-grade inflammatory stimulus, usually seen at the apex of a tooth (or its extraction site) in which there has been a long-standing pulp pathosis."

Pulpal and Periapical Diagnostic Terminology: American Board of Endodontics, 2007

Normal apical tissues—"Teeth with normal periradicular tissues that will not be abnormally sensitive to percussion or palpation testing. The lamina dura surrounding the root is intact and the periodontal ligament space is uniform."

Symptomatic apical periodontitis—"Inflammation, usually of the apical periodontium, producing clinical symptoms including painful response to biting and percussion. It may or may not be associated with an apical radiolucent area."

Asymptomatic apical periodontitis—"Inflammation and destruction of apical periodontium that is of pulpal origin, appears as an apical radiolucent area and does not produce clinical symptoms."

Acute apical abscess—"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."

Chronic apical abscess—"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."

What are the endodontically related conditions involving root-supporting tissues? Based on the highest level of available evidence, what diagnostic terms best represent periapical health and the various forms of periapical disease?

Textbook sources were separated into two categories according to the completeness and logic of the descriptions of diagnostic terminology as well as including the clinical criteria for each term. A third category comprised texts that do not include a listing of diagnostic categories because of an unsuitable theme.

1. Those texts that provide a complete, comprehensive, descriptive and generally similar list of diagnostic terms are listed as 1, 2, 4, 5, 7, 8, 12, 18, 19, 22, 23, 25, 27, and 28 (Table 8) as well as the AAE/ABE documents (Table 9).
2. Those that do not supply a logical, complete, or descriptive list of terms are listed as 9, 10, 11, 13, 14, 15, 16, 17, 20, and 24 (Table 8).
3. Texts that do not deal with subjects that require overall diagnosis and treatment planning and therefore do not describe a complete list of diagnostic terms are listed as 3, 6, 21, and 26 (Table 8).

Evaluation of the data from the first 14 textbooks (Table 8), added manuscripts (Tables 2, 4, and 7), and the AAE/ABE documents (Table 9) found that there is general consistency with these 14 texts and documents of category 1. Most include four general diagnostic categories with variations in the nomenclature. The following are general comments:

1. Few of the textbooks list normal as an periapical classification (Fig. 4) (1, 18, 22 [Table 8]). The AAE defines this descriptively (Table 9).
2. Periapical pain with no or minimal ("widening" or "thickening") of the apical periodontal ligament (PDL) space is a repeated description. Text #1 generally belongs in this group. Radiographic changes

are also described as "may or may not be associated with an apical radiolucent area." This is identical to the AAE/ABE descriptions as well as descriptions in references listed as 4, 7, and 19.

3. Slight or no periapical pain with slight to marked apical and/or lateral radiographic changes is indicated diagnostically by a few textbooks.
4. Periapical pain with varying degrees of swelling (none to extensive) with evidence of purulence is used in a few texts, whereas radiographic changes from none to marked, with little mention of systemic manifestations is also indicated in some texts.
5. Slight to no periapical pain, along with a draining sinus tract has been indicated, with radiographic changes from none to marked.

"Other" findings and matching terms are included in many of the 14 category 1 texts and documents (Table 7). There is little consistency with the lists of "other" terms.

Terminology definitions that are generally consistent are as follows (See also Table 13 for organization use of terms):

1. Acute refers to pain or other significant signs (example: swelling) and only a few texts relate it to duration and/or histologic findings. This latter reference to histologic findings appears to be of only historic interest and, therefore, should not negate or dismiss the use of the term acute.
2. Chronic refers to no or slight pain and only a few texts relate it to duration and/or histological findings. This latter reference to histologic findings appears to be of only historic interest and, therefore, should not negate or dismiss the use of the term chronic.
3. Abscess refers to evidence of purulence (pus or exudates).
4. Apical, periapical and periradicular seem interchangeable.
5. Periodontitis is used in most descriptions and refers to inflammation of the periodontium with a pulpal etiology and is not to be misconstrued as being periodontal disease.
6. Osteitis is inflammation of bone peripheral to the apical periodontium.

TABLE 10. Application of the International Classification of Diseases to Dentistry and Stomatology (3rd ed), World Health Organization (WHO), Geneva, 1995

K04 DISEASES OF PULP AND PERIAPICAL TISSUES	
K04.0 Pulpitis	
K04.00 Initial (hyperaemia)	
K04.01 Acute	
K04.02 Suppurative [pulpal abscess]	
K04.03 Chronic	
K04.04 Chronic, ulcerative	
K04.05 Chronic, hyperplastic [pulpal polyp]	
K04.08 Other specified pulpitis	
K04.09 Pulpitis, unspecified	
K04.1 Necrosis of pulp	
Pulpal gangrene	
K04.2 Pulp degeneration	
Denticles	
Pulpal calcification	
Pulpal stones	
K04.3 Abnormal hard tissue formation in pulp	
K04.3X Secondary or irregular dentine	
Excludes: pulpal calcifications (K04.2)	pulpal stones (K04.2)
K04.4 Acute apical periodontitis of pulpal origin	
Acute apical periodontitis	
K04.5 Chronic apical periodontitis	
Apical granuloma	
K04.6 Periapical abscess with sinus	
Includes: dental abscess with sinus	
dentoalveolar abscess with sinus	
periodontal abscess of pulpal origin	
K04.60 Sinus to maxillary antrum	
K04.61 Sinus to nasal cavity	
K04.62 Sinus to oral cavity	
K04.63 Sinus to skin	
K04.69 Periapical abscess with sinus, unspecified	
K04.7 Periapical abscess without sinus	
Dental abscess }	
Dentoalveolar abscess }	without sinus
Periodontal abscess of pulpal origin }	
K04.8 Radicular cyst	
Includes: cyst	
• apical periodontal	
• periapical	
K04.80 Apical and lateral	
K04.81 Residual	
K04.82 Inflammatory paradental	
Excludes: developmental lateral periodontal cyst (K09.04)	
K04.89 Radicular cyst, unspecified	
K04.9 Other and unspecified diseases of pulp and periapical tissues	

Source: World Health Organization. Application of the International Classification of Diseases to Dentistry and Stomatology – 3rd Edition, Geneva, 1995:66-7.

7. There are some inconsistent variations of terms in individual textbooks when the periapical status is universally understood. Condensing osteitis (CO), periradicular osteosclerosis, condensing apical periodontitis, and focal sclerosing osteomyelitis are examples.

The following are the four categories described above that matched with the nomenclature (See Tables 8 and 9 for the numeric listing of the texts indicated below):

1. Periapical pain with no to minimal radiographic changes:
 - a. Acute apical periodontitis is most common (5, 7, 8, 12, 13, 18, 23, 25, 26, 27, 28, AAE/ABE)
 - b. Acute periradicular periodontitis (2, 4)

- c. Acute periradicular inflammation (19, 20)
 - d. Symptomatic apical periodontitis (1, 15, 22, 25)
2. Slight or no periapical pain with radiographic changes:
 - a. Chronic apical periodontitis (5, 8, 12, 18, 20, 25, 26, 27, 28, AAE/ABE)
 - b. Chronic periradicular periodontitis (2, 4)
 - c. Asymptomatic apical periodontitis (1, 22, 25)
 - d. Other variations (7, 14, 19, 23)
 3. Pain and/or swelling. Varying (none to marked) radiographic changes:
 - a. Acute apical abscess (1, 12, 20, 22, 25)
 - b. Acute periradicular abscess (2, 18, 19, AAE/ABE)
 - c. Other variations (4, 7, 8, 23)
 4. No to slight pain. Draining sinus tract:
 - a. Chronic apical abscess (1, 12, 22, 25)
 - b. Chronic periradicular abscess (2, 18, AAE/ABE)
 - c. Other variations (4, 8, 19, 20, 23, 27, 28)

Additional categories include (see texts numerically indicated in the designated Tables):

5. Condensing osteitis (or a variation), referring to an increase in bony trabecular patterns, is included in references 12, 15, 18, 19, 22, 23 (Table 8), and AAE (Table 9).
6. "Nonendodontic" or "other pathosis:" is included in references 5, 12, 14, 15, 22, and 23.

The literature from the MEDLINE search was reviewed to determine if there are correlations of periapical clinical signs and symptoms with the actual pathogenesis (Tables 2, 4, and 7). The objective was to define categories of diagnostic terminologies consistent with the periapical diseases. The highest level of evidence would be from clinical studies that include a large number of patients with varied signs and symptoms and differing periapical pathoses. The review also included other sources for journal titles. For example, the bibliographies from other pertinent or associated references were examined for relevance along with textbooks. Articles were carefully read to assess whether each would fit the category of "highest level of evidence" or even "moderate level of evidence."

High levels—randomized controlled trials and cohort studies

Moderate levels—case controlled studies

In summary, there is very little evidence at any level to correlate clinical signs and symptoms with histopathological processes. Three articles that do correlate clinical findings with histopathological responses are detailed and summarized and evaluated below.

- Marimen et al (28): Cadaver jaws were radiographed. Those periapical areas that radiologically were consistent with condensing osteitis were resected and examined histologically. There was inflammation in the medullary bone, and there was an increase in trabeculation, consistent with the radiographic appearance. "Condensing osteitis" is an accurate term.
- Harrison and Larson (29): Ten sinus tracts that extended to apical lesions were surgically removed and serially sectioned at right angles to the tract. The known duration of the sinus tracts was from 8 weeks to 3 years. One sinus tract was lined with stratified squamous epithelium. Nine of the sinus tracts were lined with granulomatous tissue consisting of new capillaries, collagen, lymphocytes, and plasma cells. One specimen had a moderate infiltrate of polymorphonuclear leukocytes. Thus, the term chronic as a histological term is not appropriate for the one specimen. "Chronic," as indicating longer duration (course of the disease), would be appropriate.

TABLE 11. Glossary of Periodontal Terms — The American Academy of Periodontology 2001

ABSCCESS:	Localized collection of purulent exudates (pus) in a cavity formed by the disintegration of tissues.
ACUTE A.:	An abscess of relative short duration, typically producing pain and local inflammation.
APICAL A.:	Inflammatory condition characterized by formation of purulent exudates involving the dental pulp or pulpal remnants and the tissues surrounding the apex of a tooth.
CHRONIC A.:	1. Abscess of comparatively slow development with little evidence of inflammation. There may be an intermittent discharge of purulent matter. 2. Long-standing collection of purulent exudates. It may follow an acute abscess. See: Abscess, Residual.
GINGIVAL A.:	A localized purulent infection that involves the marginal gingival or interdental papilla.
PERICORONAL A.:	A localized purulent infection within the tissue surrounding the crown of a partially erupted tooth.
PERIODONTAL A.:	(Parietal A.): Localized purulent inflammation in the periodontal tissues; also called lateral periodontal abscess.
PULPAL A.:	Inflammation of the dental pulp characterized by the formation of purulent exudate.
RESIDUAL A.:	Abscess produced by the residues of a previous inflammatory process.
WANDERING A.:	Abscess in which purulent material flows along a course of decreased resistance and discharges at a distant point.
ACUTE:	1. Sharp, severe. 2. Denoting the swift onset and course of a disease.
CELLULITIS:	A diffuse inflammation; the term usually applies to purulent inflammation within loose subcutaneous tissue.
CHRONIC:	Continuing over a long period of time. Used to describe a disease state of long duration.
CYST:	A pathologic cavity lined by epithelium and usually containing fluid or semisolid material.
APICAL PERIODONTAL C.:	The most common odontogenic cyst; involving the apex of a root and resulting from the inflammatory reaction to a nonviral pulp.
DENTIGEROUS C.:	Forms around the crown of an unerupted tooth or odontoma.
DEVELOPMENTAL C.:	Results from a formative aberration.
GINGIVAL C.:	Found within the gingival, most commonly in the mandibular canine-premolar region. Believed to be derived from epithelial rests on the dental lamina.
INCISIVE CANAL C.:	(Nasopalatine Duct C. and Median Anterior Maxillary C.): A developmental, non-odontogenic cyst originating from embryonic remnants within the incisive canal.
KERATINIZING ODONTOGENIC C.:	(Calcifying and Keratinizing Odontogenic D.; Gorlin's C.): An odontogenic cyst found most often in the mandibular canine and premolar region; has distinct microscopic features including basal epithelial cells that resemble ameloblasts, large prematurely keratinized eosinophilic cells (ghost cells), and the production of an amorphous material referred to as "dentinoid;" may be totally cystic or predominantly solid.
KERATOCYST:	Developmental odontogenic cyst of the dental lamina in which the epithelial cells produce keratin; known for its aggressive nature and high recurrence rate.
LATERAL PERIODONTAL C.:	A small cyst of the periodontal ligament found most often in the mandibular canine and premolar areas; associated with a vital tooth and postulated to originate from the rests of Malassez, the rests of the dental lamina, or a supernumerary tooth bud.
ODONTOGENIC C.:	A class of cysts derived from odontogenic epithelium, such as Primordial, dentigerous, and lateral periodontal cysts.
PERIODONTAL C.:	See: Cyst, Lateral Periodontal C.
PRIMORDIAL C.:	An odontogenic cyst resulting from degeneration of the enamel organ of a developing tooth bud.
RADICULAR C.:	A cyst along the root of a tooth. Previously the term often was used synonymously with what is now more accurately referred to as an apical periodontal cyst.
RESIDUAL C.:	A cyst in the maxilla or mandible that remains after the associated tooth has been removed.
RETENTION C.:	Caused by retention of glandular secretion.
DYSPLASIA:	Abnormality of development; in pathology, alteration in size, shape, and organization of cells.
PERIAPICAL CEMENTAL D.:	(Cementoma): A process of unknown origin in which the periapical bone of vital teeth is replaced first by a fibrous type of connective tissue, and then by an osseocementoid tissue. During its early stages this abnormality appears radiolucent and with time the center becomes opaque. It is classified as an odontogenic tumor.
FILLING, RETROGRADE:	An amalgam or other restoration placed in the apical portion of a tooth to seal the root canal following surgical removal of a periapical lesion and/or the end of the root.
GRANULOMA:	A reactive nodule consisting of modified macrophages resembling epithelial cells surrounded by a rim of mononuclear cells, usually lymphocytes, and often containing giant cells.
APICAL G.:	Circumscribed granulomatous tissue adjacent to the apex of a tooth.
CENTRAL GIANT CELL G.:	Usually restricted to the jaw bones, this lytic lesion displays loose fibrillar connective tissue, numerous capillaries, and multinuclear giant cells; a histologic appearance similar to the bony lesions of hyperparathyroidism.
PERIPHERAL GIANT CELL G.:	Considered an unusual proliferative response of the tissues to injury, this lesion always occurs on the gingival or alveolar mucosa. Histologically, it is a non-encapsulated mass of delicate connective tissue cells, numerous capillaries, and multinucleated giant cells.
PYOGENIC G.:	Localized, painless protuberant, exophytic gingival mass that is attached by a sessile or pedunculated base from the gingival margin or more commonly from an interproximal space.
PREGNANCY-ASSOCIATED P.G.:	A pyogenic granuloma resulting from dental plaque and hormones during pregnancy.
GRANULOMATOUS TISSUE:	A distinctive morphologic pattern of inflammation consisting of histiocytes that have been transformed into epithelioid cells that are surrounded by mononuclear cells, usually lymphocytes. Seen in the granulomatous diseases, such as tuberculosis, syphilis, sarcoidosis, and leprosy.
PULPITIS:	Inflammation of the dental pulp.
PUS:	A product of inflammation consisting of leukocytes, degenerated tissue elements, tissue fluids. And microorganism.
RADICULAR:	Pertaining to the root of a tooth and its adjacent structures.
REFRACTORY:	Persistent; patients or sites that continue to demonstrate disease after appropriate therapy.
RESECTION:	Excision of some portion of a structure such as bone, gingiva, or a tooth root.
RETROFILLING:	A method of sealing the root canal of a tooth by an apical approach.
SINUS:	A cavity or hollow space in a bone or other tissue such as the dilated channels for venous blood in the cranium or liver.
S. TRACT:	A fistula or tract leading to a suppurating cavity.

Source: The American Academy of Periodontology. Glossary of Periodontal Terms. 4th ed. 2001:1-53.

TABLE 12. Terms Used in Oral and Maxillofacial Pathology

ACUTE APICAL PERIODONTITIS – Periapical periodontal ligament fibers exhibiting acute inflammation but no abscess formation in vital or nonvital teeth.
CELLULITIS – The acute and edematous spread of an acute inflammatory process. If an abscess is not able to establish drainage, it may spread diffusely through fascial planes of the soft tissue.
CONDENSING OSTEITIS (FOCAL SCLEROSING OSTEOMYELITIS) – Localized areas of bone sclerosis associated with the apices of teeth with pulpitis (from large carious lesions or deep coronal restorations) or pulpal necrosis.
CUTANEOUS SINUS – A draining dental abscess channelized through the overlying skin.
OSTEOMYELITIS – An acute or chronic inflammatory process in the medullary spaces or cortical surfaces of bone that extends away from the initial site of involvement.
ACUTE OSTEOMYELITIS – Exists when an acute inflammatory process spreads through the medullary spaces of the bone and there has been insufficient time for the body to react to the presence of the inflammatory infiltrate.
CHRONIC OSTEOMYELITIS – Exists when the defensive response leads to the production of granulation tissue which forms dense scar tissue and walls off the infected area. The encircled dead space acts as a reservoir for bacteria. There may be pain, swelling, sinus formation, purulent discharge, sequestrum, tooth loss, or traumatic fracture.
DIFFUSE SCLEROSING OSTEOMYELITIS – An increased radiodensity around sites of chronic infection such as apical inflammatory disease.
PARULIS – A mass of subacutely inflamed granulation tissue at the intraoral opening of a sinus tract.
PERIAPICAL ABSCESS – The accumulation of acute inflammatory cells at the apex of a nonvital tooth. May be symptomatic or asymptomatic.
PERIAPICAL CYST – Epithelium at the apex of a nonvital tooth can be stimulated to form a true epithelium lined cyst. The lumen will be filled with fluid and cellular debris.
PERIAPICAL GRANULOMA – Refers to a mass of chronically inflamed granulation tissue at the apex of a nonvital tooth. May arise after a periapical abscess or may transform into periapical cysts. Because the lesion does not show true granulomatous inflammation microscopically, the term Apical Periodontitis may be more appropriate.
PERIAPICAL SCAR – The defect created by periapical inflammatory lesions may fill with dense collagenous tissue rather than normal bone. This occurs most frequently when both facial and lingual cortical plates have been lost.
PHOENIX ABSCESS – An acute exacerbation of a chronic periapical inflammatory lesion.
SEQUESTRUM – A fragment of necrotic bone that has separated from the adjacent vital bone.

Source: Neville B, Damm D, Allen C, Bouquot J. Pulpal and Periapical Disease. In: Oral & Maxillofacial Pathology. 2nd ed. Philadelphia; W.B. Saunders Co, 2001;107-36.

- Baumgartner et al (30): Fifteen intraoral sinus tracts were biopsied along with their associated periapical lesion. An additional 15 sinus tracts were also biopsied for microscopic examination. All sinus tracts were serially sectioned. All 30 sinus tracts had epithelium extending to the level of the rete pegs. However, 20 of 30 sinus tracts did not have epithelium extending below the rete pegs. Ten of the 30 sinus tracts had epithelium extending down the sinus tract. In two cases, the cystic epithelium appeared to merge with the sinus tract epithelium. Of the 15 periapical biopsies submitted blind, 7 were periapical abscesses, 4 periapical granulomas, and 4 periapical cysts. Thus, even roots with a patent sinus tract suggesting a draining abscess were judged by the pathologist to be associated with lesions that microscopically were apical cysts or granulomas in addition to abscesses.

In general, clinical terminology that is used routinely in the practice of endodontics is not based on scientific data. Many of those terms

with contemporary advocacy fail to have historic support. The diagnostic terms are generated on assumptions, by correlating certain signs, symptoms, and radiographic findings with what was presumed (not proven) to be the histopathology of a given clinical state. Furthermore, the nature of the infectious process that led to these periapical/periradicular states is not addressed. This view is also apparent from the historic literature that defined the specialty of endodontics.

Which combination(s) of metrics provide the maximal accuracy for establishing periapical diagnoses?

Acute or Symptomatic Apical Periodontitis. The most common metric associated with this diagnosis is pain upon biting, eating, teeth coming into contact, and percussion testing (31–37). The only study that attempted to relate pain to percussion to histopathologic findings was reported by Seltzer et al (31) in 1963. They looked at histologic sections of the apical tissue associated with the apices of



Figure 4. Three mandibular molars with varying degrees of coronal challenges but with evidence of normal tissues around the root ends. (A) Heavily restored molar with significantly reduced pulp chamber and evidence of calcifications in the chamber. (B) Molar with occlusal wear, shallow restoration, and pulp chamber that is diminished in size compared with the second molar. (C) Molar with significant invasive or extraradicular resorption present.

TABLE 13. Usage of Descriptive Terms by Organization

Organization	Table #	Descriptive Terms
AAE	5	Uses acute and chronic to describe course of the disease; uses periradicular and apical interchangeably; uses both abscess and periodontitis to describe the nature of the disease process
ABE	5	Uses acute and chronic to describe course of the disease; uses apical <i>only</i> to describe location of the disease; uses symptomatic and asymptomatic to describe clinical symptomology
WHO	6	Uses apical and periapical; acute and chronic to describe the course of the disease; uses abscess periodontitis to describe the nature of the disease process
AAP	7	Uses apical and periapical; acute and chronic to describe the course of the disease
OMP	8	Uses periapical; acute and chronic to describe the course of the disease

166 extracted teeth and found that percussion pain was present in all cases of partial or total necrosis of the pulp. The presence of percussion pain was significantly greater in these cases than when no pulp necrosis was noted. Their conclusion was that a positive percussion test was important for detection of partial or total pulp necrosis. They also stated that the exact reason for the periradicular inflammation could not be determined in these cases, but they concluded that the edema that accompanies the presence of inflammatory cells could be responsible for the painful reaction of the tooth to percussion. Since 1963, several publications have reported that in addition to the edema present, inflammatory mediators and immunologic reactions occurring in the stimulated periodontal ligament are also factors associated with the painful response of the tooth to percussion (32–34, 36, 37).

The response of teeth with this diagnosis to palpation, electric, and thermal pulp tests are not reliable metrics as there are no consistent responses of the tested teeth to these tests. The results from these tests vary from case to case. The same is true for findings associated with the interpretation of periapical radiographs in these cases. The radiographic findings will vary from no observable change to only a widening of the PDL space at the apex of the root (Fig. 5) (33–37).

Chronic or Asymptomatic Apical Periodontitis. The most common metrics associated with chronic or asymptomatic apical periodontitis are the presence of an asymptomatic tooth with a necrotic pulp and the presence of a radiolucency at the end of the root (32–37). In 1939, Fish (38) was one of the first to attempt to measure the events occurring in the PDL and alveolar bone as pulpal disease advances into the periradicular tissues. The histologic findings were published as the “zones of Fish.”

As the name of the classification suggests, the teeth in this category are generally chronic or asymptomatic; the results of the pulp tests

reflect the presence of a necrotic pulp, and there is the presence of a radiolucency on the radiograph (Fig. 6). There is no metric associated with the size of the radiolucency because this will vary from case to case. There may or may not be an altered sensation or slight sensitivity to percussion or palpation in these cases, but there will be no extreme responses to either test (32–37).

Most of the references report that the histology associated with this clinical classification reflects the presence of lymphocytes, plasma cells and macrophages denoting the chronic nature of the lesion (32–34, 36, 37, 39). None of the sources noted here actually conducted studies to attempt to correlate the histologic findings they report with this clinical diagnosis. There are low-level of evidence reports in the dental literature where biopsy specimens from the apex of roots have been studied histologically to establish a diagnosis (e.g., periapical granuloma, periapical cyst, etc.), but none have attempted to relate their findings to the metrics noted for this clinical classification of the bony lesion present.

Chronic Apical Abscess. The metrics for the teeth in the chronic apical abscess classification mimic those from the previous group with one additional feature. In addition to the tooth being asymptomatic, pulp tests reflect the presence of a necrotic pulp and the presence of radiolucency at the end of the root; there is the presence of a sinus tract (Fig. 7) (32–37). Exudate may or may not be expressed from the sinus tract depending on how active the lesion is at the time of the intraoral examination. This may also include cases in which there is isolated probing that leads to the apex of the offending tooth.

Acute Apical Abscess. The metrics for acute apical abscess (AAA) reflect a very painful tooth with pulp testing reflecting pulp necrosis. Swelling is generally present, and it may be localized to the mucogingival area or it may involve fascial planes and spaces. The tooth is very sensitive to percussion and palpation and may exhibit varying degrees of mobility. Often the tooth may be elevated in the alveolar socket because of pressure from the inflamed tissues around the root. Some cases may not show any evidence of anatomic changes on the radiograph, whereas other cases may show changes that range from a widened PDL space to a frank radiolucent lesion (Fig. 8). The patient may or may not exhibit systemic manifestations to include the presence of a fever and/or lymphadenopathy (32–37, 39).

CO. The one common metric for condensing osteitis (CO) is the presence of a radiopacity at the apex of the root of a tooth (Fig. 9) (32–37, 39). This finding is thought to be a proliferative bone response to a chronic irritant. This entity may manifest a variety of signs and symptoms. There may or may not be sensitivity to percussion and/or palpation. Also, there may or may not be a positive response to electric and thermal pulp tests. An abstract published in 1992 gave a rare histologic picture of this type of lesion because, in most instances, these lesions do not require a biopsy (28). The abstract reported that the lesion was made up of a dense mass of bone trabeculae with small marrow spaces, and the bone was lined with active osteoblasts.

General Comments

Most of the references used to characterize the metrics are from textbooks. No attempts were made to add all of the articles that were related to a particular histologic, oral, pathologic diagnosis. The Seltzer et al (38) article is the only one with a sample size worthy of referencing. The published abstract by Marimen et al (28) was a case report but was the only one that reflected the histologic picture of a CO lesion. In searching further, there were no studies that specifically tried to assess the accuracy of the metrics used contemporarily for these periradicular clinical classifications. In this assessment, the metrics used and

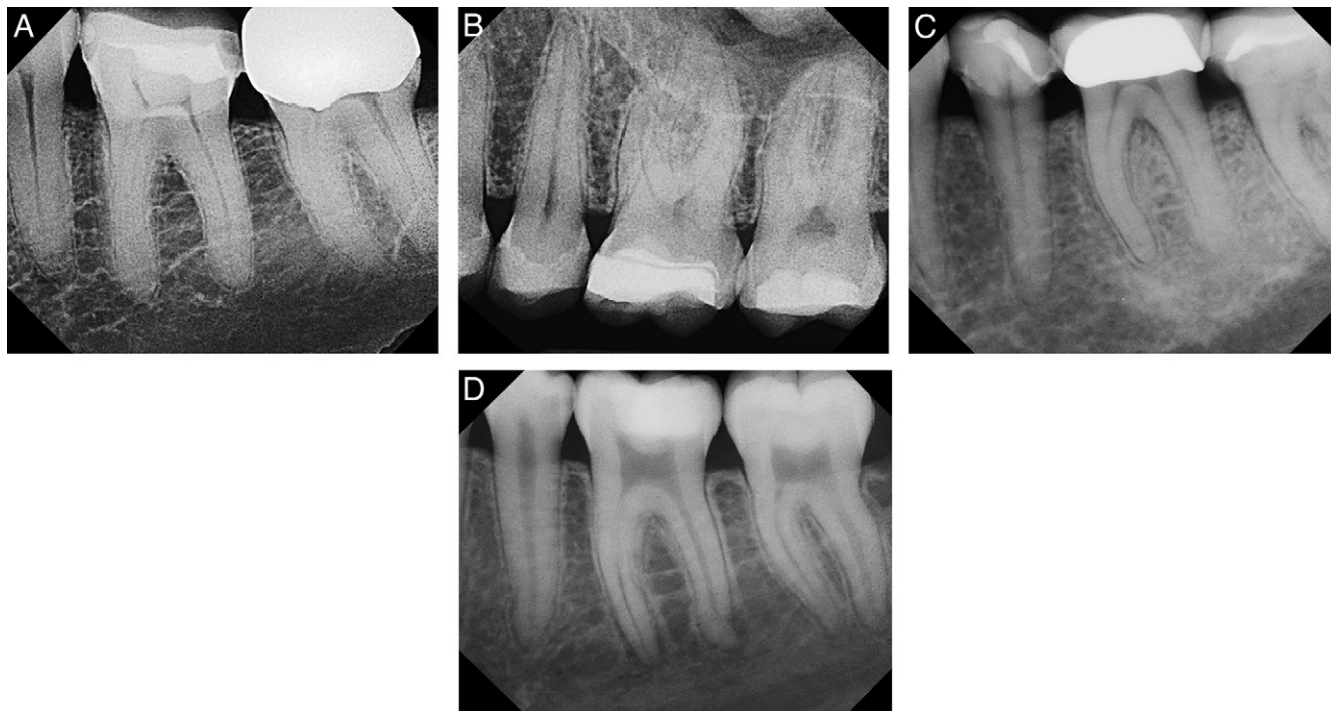


Figure 5. Examples of teeth with acute signs and symptoms and varying degrees of changes around the root ends. (A) Mandibular left first molar with a deep and extensive restoration. The patient experiences severe pain to cold, pain to percussion, and palpation, but the tooth has normal appearing tissues around the root apex. (B) Maxillary left first molar with extensive restoration, diminished pulp chamber. The patient has moderate pain to cold, spontaneous radiating pain and pain to biting, but the tooth has normal-appearing tissues around the root apices. (C) Mandibular left first molar with extensive restoration and diminished pulp chamber. The patient cannot bite on the tooth, and there is evidence of a slight thickening of the periodontal ligament space around the mesial root. (D) Mandibular left first molar with an extensive restoration and very large pulp chamber. The patient has intermittent pain to cold and moderate pain to biting and percussion tooth. There are significant apical changes around both the distal and the mesial roots, yet during pulp testing the tooth responded severely to cold. A-D represent a wide variety of apical changes found with a diagnosis of acute or symptomatic apical periodontitis, changes that may be interpreted differently by clinicians.

correlated included signs, symptoms, pulp test results, percussion, palpation, and radiographic findings. Contemporarily, the most extensive sections related to the classifications of periodontitis were found in Ørstavik and Pitt Ford (34).

What gaps in knowledge remain for developing and validating metrics and the resulting periapical diagnosis?

What is missing in response to both the main question and this question, and its pursuant challenges identified in this assessment, is the availability of evidence-based studies that include higher levels of data for the specific diagnostic terminology and metrics of determining the clinical diagnostic states. Moreover, the diagnostic classification schemes presently proffered do not address any relationship to the periapical infectious process.

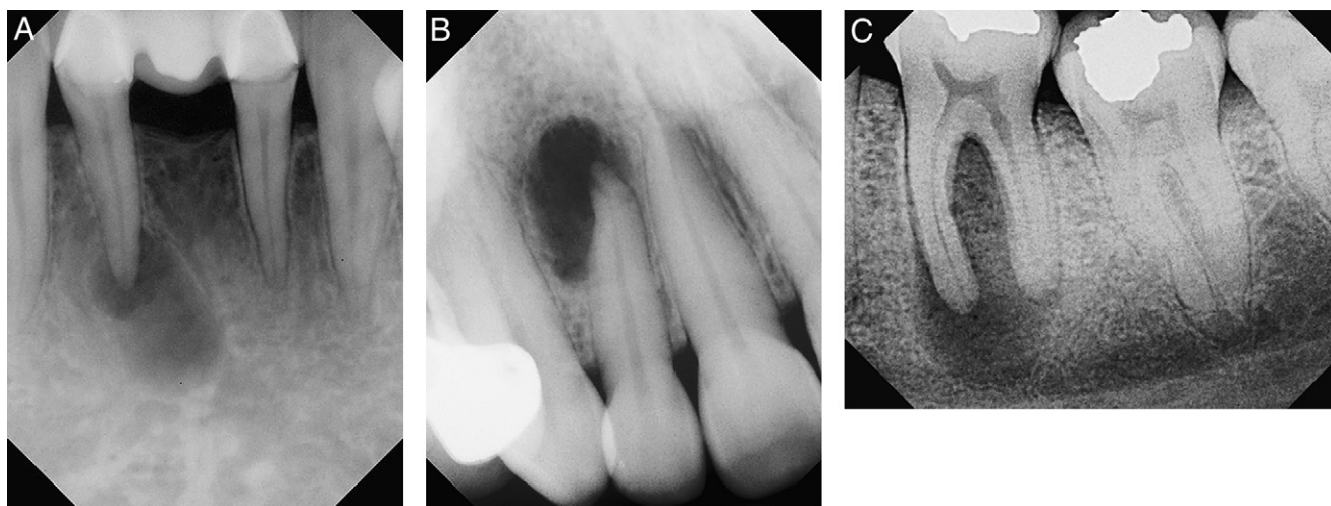


Figure 6. Examples of teeth with significant changes radiographically around the root ends. All teeth exhibit no symptoms or clinical signs of pathosis. (A-C) There is no pain to percussion or palpation and the teeth do not respond to pulp testing. These are typical representations of chronic or asymptomatic apical periodontitis.

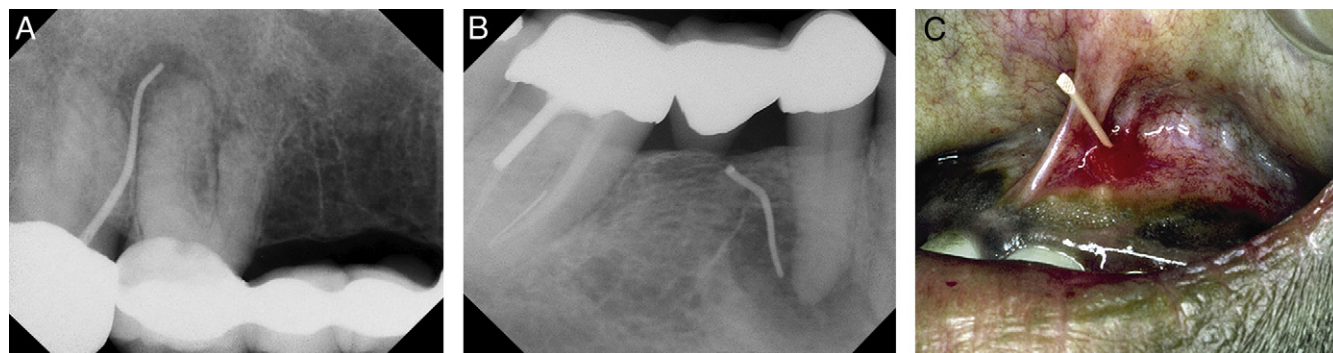


Figure 7. (A and B) Examples of teeth with chronic apical abscess exhibiting traceable sinus tracts as can be seen on the radiographs. Historically, this was known as suppurative periodontitis or chronic alveolar abscess. (C) Clinical photograph showing the presence of a double sinus tract tracing.

Studies with valid and achievable protocols that include patients with a range of signs and symptoms are necessary; however, the ability to have studies that are at the highest level of evidence may not be possible. Each study would not have to be all inclusive but could include a portion of these or a single diagnostic entity. Quantification, if possible, would be desirable. Patients who are having their teeth extracted for periodontal, prosthetic, or orthodontic purposes may serve as the best models to be able to not only do the clinical testing and quantification but also the histopathological and microbial assessment. The design(s) for consideration would be as follows:

A. Determine clinical signs and symptoms

1. Pain: differing degrees of subjective findings, from none to severe
2. Pain on percussion and palpation and mastication, from none to severe

3. Swelling: presence or absence and degree and location

4. Sinus tract: presence or absence

5. Radiographic findings: no changes to “widened” to visible resorptive lesion. Increased periapical, peripheral bone density; in this regard, the Periapical Index scoring method may be a starting point or may serve as a highly viable tool to implement studies in a rapid fashion

B. Obtain teeth and surrounding apical tissues. The teeth would be extracted (preferred) or at least the apical third would be resected with a block of adjacent bone and periodontium, all as a unit. Root and bone would be demineralized, serial or step-serial sectioned, and stained with hematoxylin and eosin or other appropriate stains. Sections would be examined for:

1. Inflammatory cells—nature and distribution and relative numbers
2. Purulence—presence or absence

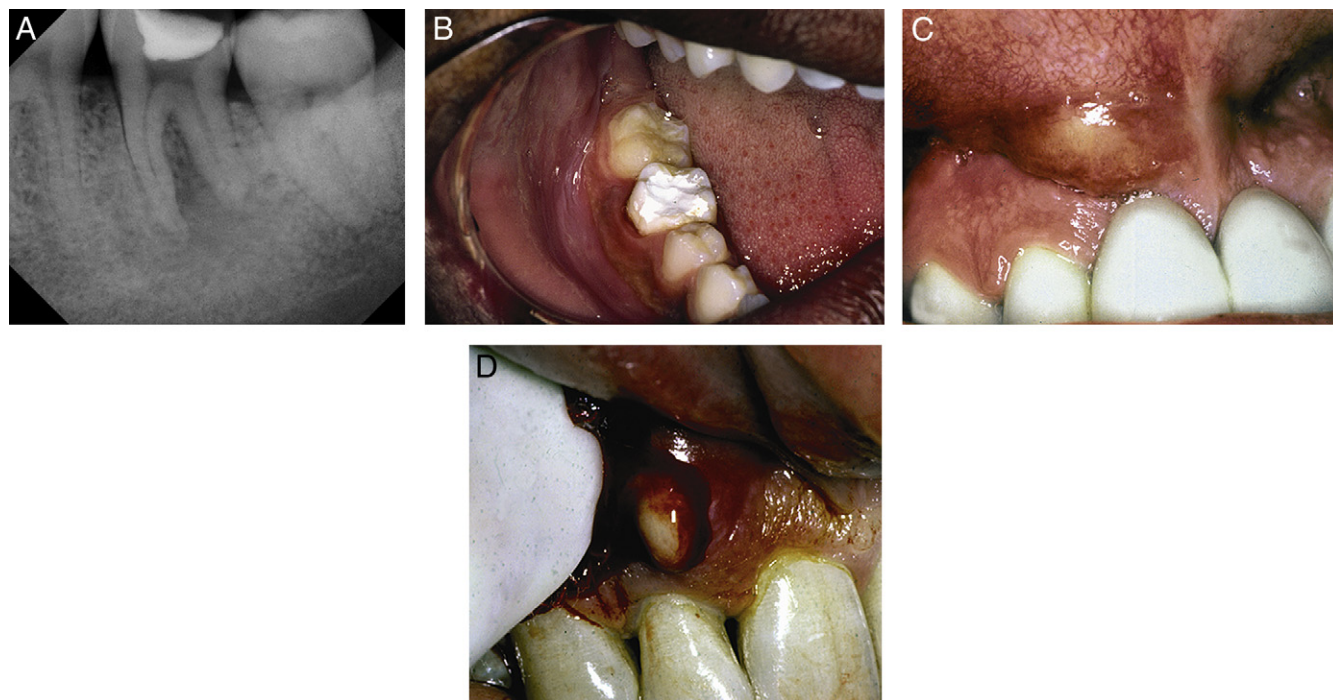


Figure 8. (A) Mandibular molar with significant bone loss, mobility, and symptoms indicative of an AAA. (B) Clinically, swelling is present adjacent to the molar in A. (C) Swelling over maxillary incisors that represent a cellulitis of pulpal origin. (D) Effective elimination of the purulence with incision and drainage of a swelling over the maxillary lateral incisor in another case of AAA.



Figure 9. Examples of three mandibular molars exhibiting dense bone around the root apices in conjunction with slight to moderate radiolucent changes (A-C).

3. Sinus tract—presence or absence
4. Nature of surrounding bone—increased or decreased
5. Possibly consider some sections using Brown and Brenn or in situ molecular techniques for bacterial identification
6. Advanced tests for microbial assessment and analysis
- C. Correlation of clinical findings with histopathological and microbial findings.

Concomitant with the lack of scientific support for the clinical diagnostic states is the lack of a diagnostic scheme that provides a healing-predictive, treatment-oriented diagnosis. This also should be considered in developing research protocols. This would provide the clinician with greater support in the transition from diagnosis to appropriate treatment. Moreover, it may tend to standardize treatment regimens globally based on sound diagnostic categories and determinations, thereby eliminating or minimizing empiricism and mistreatment.

There appears to be little if any support in the historic or contemporary peer-reviewed literature for the use of the clinical terms “symptomatic” and “asymptomatic” in conjunction with presently used diagnostic terminology and disease states (refer to pulp or periapical tissues). These terms have slowly crept into usage with little scientific basis for their applications or meanings. Diagnostic states based on symptomatology were proposed in 1977, with little validity other than empiricism and a failed attempt to distinguish histologic diagnoses from clinical diagnoses (39). Their use was recently promulgated and perpetuated again with minimal rationale or scientific bases (27) (Table 9, American Board of Endodontics). The arbitrary use of these ambiguous terms, without taking into account the historic basis for the endodontic diagnostic scheme, may very well lead to overtreatment. There is little rationale for their use other than to say to a patient that you have a tooth with asymptomatic irreversible pulpitis, a diagnosis that could easily be made on a single pulp test or assembly of inconclusive data, or asymptomatic apical periodontitis that apparently must be made purely on a “subjective” assessment of radiographic findings. Both of these assessments could mean that treatment would be recommended for any tooth at any time at the whim of the clinician. Protocols to assess the validity, accuracy, and precision of these terms on a clinical basis with considerations for the patient’s level and perception of pain/sensitivity are therefore essential.

Finally, in this digital/computer age of documentation, it would seem reasonable to have a succinct diagnostic scheme that could be described thoroughly, agreed on unanimously, coded succinctly for easy electronic input, and ultimately used for follow-up analysis. In essence, not only would this type of diagnostic scheme drive treatment modalities more accurately, but it would also allow for future outcomes assessment and validation.

References

1. Harris CA. The dental art, practical treatise on dental surgery. Baltimore: Armstrong & Berry; 1839.
2. Tomes CS, Nowell WS. A system of dental surgery (originally written by Sir John Tomes). 5th ed. London: J & A Churchill; 1906.
3. Buckley JP. Modern dental material medica, pharmacology and therapeutics. Philadelphia: P. Blakiston’s Son & C; 1910.
4. Blair V. Surgery and diseases of the mouth and jaws. St. Louis: The CV Mosby Company; 1913.
5. Marshall JA. Diseases of the teeth, their diagnosis and treatment. Philadelphia: Lea & Febiger; 1926.
6. Gilmer TL. Etiology, diagnosis and treatment of acute and chronic abscess. Dent Rev 1914;28:427–9.
7. Prinz H. Soft structures of the teeth and their treatment. Philadelphia: Lea & Febiger; 1928.
8. Prinz H. Soft structures of the teeth and their treatment. 2nd ed. Philadelphia: Lea & Febiger; 1937.
9. Arkövy J. Diagnostik der Zahnkrankheiten und der Durch Zahnleiden bedingten Kiefererkrankungen. Stuttgart: Universitat Budapest; 1885.
10. Kronfeld R. Histopathology of the teeth and their surrounding structures. Philadelphia: Lea & Febiger; 1933.
11. Coolidge EG. Clinical pathology and treatment of the dental pulp and periodontal tissues. Philadelphia: Lea & Febiger; 1939.
12. Grossman LI. Root canal therapy. Philadelphia: Lea & Febiger; 1940.
13. Grossman LI. Root canal therapy. 2nd ed. Philadelphia: Lea & Febiger; 1946.
14. An annotated glossary of terms used in endodontics. 4th ed. Chicago: American Association of Endodontists; 1984:11.
15. Kim MY, Lin J, White R, Niederman R. Benchmarking the endodontic literature on MEDLINE. J Endod 2001;27:470–3.
16. Jaywant SS, Pai AV. A comparative study of pain measurement in acute burn patients. International Journal of Thermophysics 2004;35:13–7.
17. Gracely RH, Dubner R. Reliability and validity of verbal descriptor scales of painfulness. Pain 1987;29:175–85.
18. Gillam DG, Bulman JS, Newman HN. A pilot assessment of alternative methods of quantifying dental pain with particular reference to dentine hypersensitivity. Comm Dent Health 1997;14:92–6.
19. Duncan GH, Bushnell MC, Lavigne GJ. Comparison of verbal and visual analogue scales for measuring the intensity and unpleasantness of experimental pain. Pain 1989;37:295–303.
20. Jacinto RC, Gomes BPFA, Shah HN, et al. Quantification of endotoxins in necrotic root canals from symptomatic and asymptomatic teeth. J Med Micro 2005;54:777–83.
21. Khabbaz MG, Anastasiadis PL, Sykaras SN. Determination of endotoxins in caries: association with pulpal pain. Int Endod J 2000;33:132–7.
22. Slots J, Nowzari H, Sabeti M. Cytomegalovirus infection in symptomatic periapical pathosis. Int Endod J 2004;37:519–24.
23. 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.
24. Bernhardt O, Schiffman EL, Look JO. Reliability and validity of a new fingertip shaped pressure algometer for assessing pressure pain thresholds in the temporomandibular joint and masticatory muscles. J Orofac Pain 2007;21:29–38.
25. 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.

26. Brennan MT, Runyon MS, Batts JJ, et al. Odontogenic signs and symptoms as predictors of odontogenic infection. *J Am Dent Assoc* 2006;137:62–6.
27. Iqbal M, Kim S, Yoon F. An investigation into differential diagnosis of pulp and periapical pain: a PennEndo database study. *J Endod* 2007;33:548–51.
28. Marimen D, Green TL, Walton RE, et al. Histologic examination of condensing osteitis. *J Endod* 1992;18:196.
29. Harrison J, Larson W. The epithelized oral sinus tract. *Oral Surg Oral Med Oral Pathol* 1976;42:511–7.
30. Baumgartner JC, Pickett A, Muller J. Microscopic examination of oral sinus tracts and their associated periapical lesions. *J Endod* 1984;10:146–52.
31. Seltzer S, Bender IB, Ziontz M. The dynamics of pulp inflammation: correlation between diagnostic data and acute histologic findings in the pulp. *Oral Surg* 1963;16:846–71.
32. Johnson WT. Color atlas of endodontics. Philadelphia: WB Saunders Co; 2002:10–1.
33. Tronstad L. Clinical endodontics. 2nd ed. New York: Thieme; 2003:46–50, 54–5.
34. Ørstavik D, Pitt Ford TR. Essential endodontology: prevention and treatment of apical periodontitis. 1st ed. London: Blackwell Science Ltd; 1998:68–70, 83–4, 94, 136–7, 166–174.
35. Cohen S, Hargreaves KM. Pathways of the pulp. 6th ed. St. Louis: Mosby-Elsevier; 2006:36–7.
36. Ingle JI, Bakland LK. Endodontics. 5th ed. Hamilton, Ontario: BC Decker; 2002. 179–86.
37. Torabinejad M, Walton RE. Endodontics: principles and practice. 4th ed. St. Louis: Saunders-Elsevier; 2008:58–63.
38. Fish EW. Bone infection. *J Am Dent Assoc* 1939;26:691–712.
39. Morse DR, Seltzer S, Sinai I, et al. Endodontic classification. *J Am Dent Assoc* 1977; 94:685–9.