Title: Identification and Definition of Diagnostic Terminology for Periapical/Periradicular Health and Disease States

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Abstract

The purpose of this in-depth investigation was to identify, clarify and substantiate clinical terminology relative to periradicular/periapical 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, treatment and prognosis wherever possible along with establishing the basis for the metrics used in this diagnostic process. Moreover, diagnostic terminologies and their relevance to clinical situations were procured from an extensive historical search and correlated with contemporary concepts in disease processes, clinical assessments, histological findings (if appropriate) and standardized definitions that have been promulgated and promoted for use in the last 25 years in educational programs, test constructions, and for third party concerns.

Multiple search strategies based on Medical Subject Headings (MeSH terms) for endodontics were developed to examine MEDLINE & SCOPUS. The search was not limited to the English language and focused on diagnosis, dental journals only, human subjects and etiology with a particular emphasis on periapical periodontitis and periapical disease, along with pulpal necrosis and osteitis. Four search strategies were run that also included terms in a specificity or sensitivity category in an attempt to narrow the focus to the search. Furthermore, the searches attempted to identify evidence based and systematic reviews within the MeSH headings.

A total of 31 articles were obtained from these searches, 6 of which were in other languages. These six were sent to dental colleagues for translations and summaries relative to the specific focus of this study. At present an additional three articles are being processed that are not included in Tables 1-3.

Another dimension of this study required an investigation into the historical evolution of diagnostic states, their definitions, specific terminologies and their furtherance from enhanced and refined biological investigations and clinical applications. Classic and meaningful dental texts that influenced the evolution of the specialty of endodontics were reviewed.

Detailed definitions from the American Association of Endodontists, both in the Glossary in the American Board of Endodontics were considered. Furthermore, definitions and classifications from Periodontics, Oral Maxillofacial Pathology and the World Health Organizations were investigated and considered.

In general clinical terminology that is used routinely in the practice of endodontics is not based on scientific data. The diagnostic terms are generated on assumptions, by correlating certain signs symptoms and radiographic findings with what is presumed (not proven) to be the histopathology of a given clinical state. This view is also apparent from the historical literature that defined the specialty of endodontics.

There were no studies that specifically tried to assess the accuracy of the metrics used contemporarily for the classification of clinical periradicular disease states. 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 and other such markers of microbial impact, pathogenicity and virulence.
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 studies would include a range of signs and symptoms. Each study would not have to be all-inclusive, but could include a portion of these, or a single diagnostic entity.

Introduction

One of the hallmarks of any profession is its distinct lexicon and it 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 a personalized, empirical diction that is used to aggrandize 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 historical framework upon 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 periradicular diagnostic states, the instruments and tests used to determine these conditions, the clinician’s understanding of such and 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, the 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, while another might label it as an 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. Hence, in these circumstances treatment may be radical, altered, delayed or inappropriate, while the understanding of the exact clinical diagnostic status of the patient is vague and undefined.

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

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

Literature search

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

Harris CA. The Dental Art, Practical Treatise on Dental Surgery. Baltimore, Armstrong & Berry, 1839.

Only described disease entity that is pertinent is the alveolar abscess.

Alveolar abscess – deep seated, throbbing and painful disease, which at time is excruciating and continues with only occasional slight intermissions until matter is formed, when it, in a great degree, subsides, and is succeeded by slight paroxysm of heat and cold. He recognized that the abscess could open to the oral cavity following bone resorption, but did not provide a specific designation (e.g., fistula or sinus).


Focused on the inflammation of the alveolar periosteum (lining membrane of the sockets of the teeth); addresses general inflammation, identifying as being from “…a bad state of the system…” and local inflammation “involving the periosteal investment of the roots of one or two teeth.” Dealt with this inflammation as acute or chronic and refers to acute and chronic periostitis. These classifications could be septic or nonseptic.

Acute alveolar abscess – caused by the escape from the end of the root of putrid contents of the pulp chamber ando 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


While primarily a text that deals with the practical application of drugs and remedies in the treatment of disease, Buckley does identify four primary disease states for the periradicular tissues.

Nonseptic pericementitis

Septic pericementitis

Acute alveolar abscess – with and without the presence of a sinus tract

Pericemental abscess (inflammation of the pericemental tissues without the presence of an infection)

Blair V. Surgery and Diseases of the Outh and Jaws. CV Mosby 1913
“Peridental” pathosis – pericementitis, alveolar abscess and alveolar fistula characterize the listing of the
diagnostic states in this text.

Marshall JA. Diseases of the Teeth, Their Diagnosis and Treatment. Lea & Febiger, Philadelphia 1926.

Followed Buckley’s schema with some additional classifications, such as a tooth may develop an acute or
chronic, septic or nonseptic, 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 or relatively low virulence.

Four general types:
Abscess is partially opened and there is drainage via the root canal
“Fistulous” opening is present – known as chronic abscess with sinus
Granuloma (chronic dento-alveolar abscess)
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 are described from more of histopathological standpoint by Gilmer TL.

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 H. Soft Structures of the Teeth and Their Treatment. Lea & Febiger, Philadelphia, 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:
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.
Pressue 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, whil 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 Arkøevy (1885) as follows:
Periodontitis acuta
Periodontitis acute marginalis
Periodontitis acuta apicalis
Periodontitis acuta circumscripta (a – consecutive; and b – idiopathica)
Periodontitis acute diffusa
Periodontitis acuta purulenta (a – circumscripta; and b – diffusa)
Abscessus apicalis
Phlegmone acuta septic osteo-peridentalis (Periodontitis toxica)
Periodontitis chronic
Periodontitis chronic apicalis
Periodontitis chronic diffusa
Periodontitis chronic purulenta
Periodontitis chronic granulomatosus
Necrosis apicalis
Necrosis totalis
Caries alveolaris, seu osteo-periostitis alveolodentalis; or according to Magitot (Seu Pyorrhœa alveolaris)

Prinz's classification and definitions
Acute pericementitis
Acute apical pericementitis (acute dento-alveolar abscess) – an acute, exudative, destructive inflammation of the pericementum at the apical region of the tooth.
Acute marginal pericementitis – an acute, exudative, destructive inflammation of the pericementum restricted to its margin and clinically usually associated with marginal gingivitis.
Acute diffuse suppurative pericementitis – an acute circumscribed or diffuse suppurative destructive inflammation of the pericementum.
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
Chronic apical suppurative pericementitis (Chronic dento-alveolar 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.
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 know as a dental granuloma which, 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 Arkævĭn 1885 – Diagnostik der Zahnkrankheiten, Stuttgart 1885, p. 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.” Furthermore, his 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:
Acute traumatic periodontitis – reaction of the periodontal tissues to injury caused by trauma of any kind.
Chemical periodontitis – a reaction of the materials that have been introduced into the root canal during root canal procedures – ranging from acute initially to chronic after longer exposures.
Acute infective periodontitis – requires the presence of pathogenic microorganisms; pain, swelling, heat, redness and occasionally fever
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 membrane and alveolar bone into granulation tissue (here also the concept of granulation tissue vs granulomatous tissue appears to be confused)
Acute dento-alveolar abscess – purulence, collateral edema and swelling of the soft tissues of the face (uses the term cellulitis at this stage of advanced abscess formation)
Acute exacerbation of chronic periapical inflammation (a concept that we have seen with various labels such as phoenix abscess, recrudescence, etc.
Condensing osteitis (bone sclerosis) due to chronic periapical inflammation


Classification 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:
Acute inflammation
Traumatic and chemical periodontitis – an acute inflammation of the apical periodontal tissues with no infection present.
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.
Acute dento-alveolar abscess – an acute inflammation of the periodontal tissue around the apical foramen attended by an increasing amount of inflammatory exudates and suppuration.
Chronic inflammation
Suppurative periodontitis (chronic abscess) – a destructive suppuration of the periodontal tissues about the apex of a diseased tooth, with an intermittent discharge of pus.
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
Also uses the same characteristics for the clinical signs and symptoms for inflammation of the periodontal membrane as detailed by Prinz above.
Provided a slight variation as to the disease of the periapical tissues as follows:

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.

Acute alveolar abscess – a localized collection of pus in the alveolar bone at the root apex of the tooth following death of the pulp, with extension of the infection through the apical foramen into the periapical tissues; serve local and sometimes general adverse reaction; advanced state of acute apical periodontitis.

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.

Subacute alveolar abscess – arbitrary classification of case which 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 case of chronic abscess, which flare up and present mildly acute symptoms. 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.

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 Malassez.

This review of the historical roots of the endodontic diagnostic schemes and advocated terminology sets the stage for considering the contemporary assessment of these parameters. While the historical 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 exists to validate the metrics or provide quantifiable parameters of assessment. Furthermore, historical classifications did not necessarily delve into the infectious nature of the apical or periapical lesion that was present. Moreover, the clinical diagnostic states did not identify a repair-predictive value for each category or description.

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/periradicular health and disease states. As a basis for the search, the following published article was used as a framework for investigation, data procurement and assessment.


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

The first search strategy was run using only the the first four MeSH headings and limiting the search to dental journals.
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<th>Searches</th>
<th>Results</th>
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<tbody>
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<td>1</td>
<td>Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis/di, et</td>
<td>1477</td>
</tr>
<tr>
<td>2</td>
<td>Limit to dentistry journals</td>
<td>675</td>
</tr>
</tbody>
</table>

References and abstracts of the 675 articles were reviewed for relevance to the question asked with 22 chosen for further in-depth review (Table 1).

A second search strategy was run that limited the search to diagnosis, evidence based and systematic reviews.

<table>
<thead>
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<th>Searches</th>
<th>Results</th>
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<tbody>
<tr>
<td>1</td>
<td>Periapical Periodontitis/di, et or Periapical diseases/di, et or dental pulp necrosis/di et or osteitis/di, et</td>
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<td>2</td>
<td>Limit to dentistry journals</td>
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<tr>
<td>3</td>
<td>Limit 2 to (dentistry journals and “diagnosis (sensitivity)”</td>
<td>284</td>
</tr>
<tr>
<td>4</td>
<td>Limit 3 to (evidence based medicine reviews and systematic reviews)</td>
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</table>

References and abstracts of the 284 articles were reviewed for relevance to the question asked with 9 chosen for further in-depth review (Table 2).

A third search strategy was run that limited the search to the English language and human language along with a diagnostic sensitivity/specificity.

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<td>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]</td>
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</tr>
<tr>
<td>2</td>
<td>Limit 1 to (English language and humans)</td>
<td>4827</td>
</tr>
<tr>
<td>3</td>
<td>Limit 2 to (dentistry journals and “diagnosis (sensitivity)”</td>
<td>284</td>
</tr>
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<td>4</td>
<td>From 3 keep 1-284</td>
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<tr>
<td>5</td>
<td>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]</td>
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</tr>
<tr>
<td>6</td>
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<tr>
<td>7</td>
<td>Limit 6 to (english language and humans)</td>
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<tr>
<td>8</td>
<td>Limit 7 to “diagnosis (specificity)”</td>
<td>42</td>
</tr>
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</table>

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

The fourth search strategy attempted to narrow the field of investigation.

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<th>Searches</th>
<th>Results</th>
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<tbody>
<tr>
<td>1</td>
<td>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]</td>
<td>8739</td>
</tr>
<tr>
<td>2</td>
<td>Limit 1 to (english language and humans)</td>
<td>4827</td>
</tr>
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</table>
There were no articles within the 136 identified that were appropriate for our question at hand. Additional articles not identified in the search, but identified in manual searching are listed in Table 3 and were reviewed for relevance along with the other 31 article identified in Tables 1 & 2.

A search strategy using SCOPUS and the previously identified MeSH terms was also performed. Ninety-five (95) articles were identified with 2 specific articles being assessed further for relevance (See Table 3**).

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

1. Current and contemporary textbooks including specific issues of journals dedicated entirely to the topical issues (Table 4)
2. Glossary of Endodontic Terms published by the AAE and the American Board of Endodontics (Table 5)
5. Glossary of Periodontal Terms - The American Academy of Periodontology 2001, 4th edition (Table 7)
6. Terms used in Oral & Maxillofacial Pathology (Table 8)

The information obtained from the above sources was used to address the purposes of this study that were identified in the introduction of the paper. This work was accomplished by a team of appointed clinicians/academicians/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.

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 psychologic components. It is those psychologic 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 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, periradicular pain is often of a persistent quality accompanied by high levels of apprehension and anxiety. Objective measurement is often difficult when the data is so truly subjective and multidimensional.

Behavioral Signs of Pain

Assessment requires the clinician to be aware of non-verbal signs of pain. Behavioral signs include crying, facial grimacing, clenched jaw, restless legs, 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 patient’s 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 (1).

On the numerical rating scale, the patient is asked to identify how much pain they are 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.

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 where they think their pain is.

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 or she has no pain or feels sad because he or she has some or a lot of pain. The person is asked to choose the face that best describes how he or she is feeling. This rating scale can be used by individuals’ age 3 years and older.

Numerical 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 their 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 (2). It is also understood that verbal descriptors provide misleading information in terms of both accuracy and sensitivity (3). 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 (4).

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 and spontaneous pain, tenderness to palpation and percussion, swelling and purulent exudates (5). Increased endotoxin in carious dentin was also directly correlated to irreversible pulpitis by assay that employed the Quantitative Chromogenic test using Limulus Lysate (6). In a study investigating symptomatic periapical pathosis, results provided compelling evidence that herpesviruses participate in the pathogenesis of symptomatic periapical pathosis. Slots, et.al, showed 100% of 25 symptomatic periapical specimens harvested during apicoectomy contained the presence of human cytomegalovirus (HCMV). cDNA methodology was used to identify transcription of herpesviral genes (7).

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 allodynia in a tooth are not quantitative. Most involve percussion applications which can vary greatly. A study by Khan et. al 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 to control teeth in the same patient (8). Pressure algometers have also shown high reliability for assessing pressure pain thresholds in the temporomandibular joint and masticatory muscles of patients compared to controls (9).

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. in a Danish study reported a combination of signs and symptoms yielding a correct diagnosis of pulpal degeneration in 82% of cases: 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 (10). Others have found a relationship exists between radiolucency size and the presence of amalgam restorations in patients who develop clinical signs of infection. They recommend early intervention based on these predictors (11).

The PennEndo database has also 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 pathology, whereas dull pain was more likely associated with periapical pathology. Percussion and palpation tests were powerful in differentially diagnosing between pulpal and periapical conditions (12).

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 and other such markers of pathogenicity and
virulence.

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

Those texts that provide a complete, comprehensive, descriptive and generally similar list of diagnostic
terms are 1, 2, 4, 5, 7, 8, 12, 18, 19, 22, 23, 25, 27 and 28, (Table 4) as well as the AAE documents (Table 5).

Those that do not supply a logical, complete or descriptive list of terms are 9, 10, 11, 13, 14, 15, 16, 17, 20
and 24 (Table 4).

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 3, 6, 21 and 26 (Table 4).

Evaluation of the data from the first 14 textbooks (Table 4), added manuscripts (Tables 1-3) and the AAE
documents (Table 5) 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 a periapical/periradicular classification. #1, 18, 22 (Table 4), AAE
defines this descriptively (Table 5).

2) Periapical pain with no, or minimal (“widening” or “thickening”) of the apical 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 descriptions as well
as descriptions in 4, 7 and 19.

3) Slight or no periapical pain with slight to marked apical and/or lateral (a few textbooks) radiographic
changes.

4) Periapical pain of varying degrees of swelling (none to extensive) with evidence of purulence.
Radiographic changes from none to marked; little mention of systemic manifestations.

5) Slight to no periapical pain. Draining sinus tract. Radiographic changes from none to marked.

“Other” findings and matching terms are included in many of the 14 Category 1 texts and documents
(Table 3). There is little consistency with the lists of “other” terms.

Terminology definitions that are generally consistent are as follows:
1) Acute refers to pain or other significant signs (example: swelling) and only a few texts relate it to duration and/or histologic findings.

2) Chronic refers to no or slight pain and only a few texts relate it to duration and/or histological findings.

3) Abscess refers to evidence of purulence (or 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.

Osteitis is inflammation of bone peripheral to the apical periodontium.

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

The following are the four categories described above that matched with the nomenclature (Tables 4 & 5): 

Periapical pain with no to minimal radiographic changes:
Acute apical periodontitis is most common (5, 7, 8, 12, 13, 18, 23, 25, 26, 27, 28, AAE).
Acute periradicular periodontitis (2, 4)
Acute periradicular inflammation (19,20)
Symptomatic apical periodontitis (1, 15, 22, 25)

Slight or no periapical pain with radiographic changes:
Chronic apical periodontitis (5, 8, 12, 18, 20, 25, 26, 27, 28, AAE)
Chronic periradicular periodontitis (2, 4)
Asymptomatic apical periodontitis (1, 22, 25)
Other variations (7, 14, 19, 23)

Pain and/or swelling. Varying (none to marked) radiographic changes:
Acute apical abscess (1, 12, 20, 22, 25)
Acute periradicular abscess (2,19,18, AAE)
Other variations (4, 7, 8, 23)

No to slight pain. Draining sinus tract:
Chronic apical abscess (1, 12, 22, 25)
Chronic periradicular abscess (2, 18, AAE)
Other variations (4, 8, 19, 20, 23, 27, 28)

Additional categories include:

Condensing osteitis (or a variation): referring to an increase in bony trabecular patterns, is included in 12, 15, 18,19, 22, 23, and AAE.
“Non-endodontic” or “other pathosis”: is included in 5, 12, 14, 15, 22, and 23.

The literature from the MEDLINE search was reviewed to determine if there are correlations with periapical clinical signs and symptoms with histopathology (Tables 1-3). The objective was to define categories of diagnostic pathologies 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”.

In summary there is very little evidence at any level. Three articles that do correlate clinical findings with histopathological responses are studies are detailed and summarized below.

1. Maixner D, Green T, Walton R. Histologic examination of condensing osteitis. J Endod 18:196, 1992. 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 that there was an increase in trabeculation, consistent with the radiographic appearance. “Condensing osteitis” is an accurate term.

2. Harrison J, Larson W. The Epithelized Oral Sinus Tract. Oral Surg Oral Med Oral Pathol 1976;42:511-17. 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 granulation 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. “Chronic”, as indicating longer duration, would be appropriate.

3. Baumgartner JC, Picket A, Muller J. Microscopic Examination of Oral sinus Tracts and Their Associated Periapical Lesions. J Endod 1984;10:146-152. 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, 20/30 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. The diagnostic terms are generated on assumptions, by correlating certain signs 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 historical literature that defined the specialty of endodontics.

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

Symptomatic Apical Periodontitis (SAP)
The most common metric associated with SAP is pain upon biting, eating, teeth coming into contact and percussion testing. (13-19) The only study that attempted to relate pain to percussion to histopathologic findings was reported by Seltzer, Bender and Ziontz in 1963 (13). They looked at histologic sections of the apical tissue associated with the apices of 166 extracted teeth and found that percussion pain was present in all cases of partial of 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 which accompanies the presence of inflammatory cells could be responsible for the painful reaction of the tooth to percussion. Since 1963 several publications (14-16, 18,19) have reported that in addition to the edema present, inflammatory mediators and immunologic reactions occurring in the stimulated PDL are also factors associated with the painful response of the tooth to percussion. The response of teeth with SAP 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 SAP cases. The radiographic findings will vary from no observable change to only a widening of the PDL space at the apex of the root.(15-19)

Asymptomatic Apical Periodontitis (AAP)

The most common metrics associated with AAP are the presence of an asymptomatic tooth with a necrotic pulp and the presence of a periradicular radiolucency. (14-19) Fish (20) in 1939 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 asymptomatic, the results of the pulp tests reflect the presence of a necrotic pulp and there is the presence of a periradicular radiolucency on the radiograph. There is no metric associated with the size of the radiolucency as 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.(14-19)

Most of the references (14-16, 18,19,21) 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. None of the sources noted here actually conducted studies to attempt to correlate the histologic findings they report with the clinical diagnosis of AAP. There are a multitude of 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, et.) but none have attempted to relate their findings to the metrics noted for this clinical classification of periradicular lesion.

Chronic Apical Abscess (CAA)

The metrics for the teeth in the CAA classification mimic those for the AAP group with one additional feature. In addition to the tooth being asymptomatic, pulp tests reflecting the presence of a necrotic pulp and the presence of a periradicular radiolucency, there is the presence of a sinus tract.(14-19) Exudate may or may not be expressed from the sinus tract depending on how active the periradicular lesion is at the time of the intraoral examination.

Acute Apical Abscess (AAA)
The metrics for 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. Some cases may not demonstrate any evidence of periradicular changes on the radiograph while other cases may demonstrate changes that range from a widened PDL space to a frank radiolucent lesion. The patient may or may not exhibit systemic manifestations to include the presence of a fever and/or lymphadenopathy.(14-19, 21)

Condensing Osteitis (CO)

The one common metric for CO is the presence of a radiopacity at the apex of the root of a tooth.(14-19, 21) It 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 (22) published in 1992 gave a rare histologic picture of this type of lesion because in most instances these lesions do not require a biopsy. 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 periapical histologic, oral pathologic diagnosis. The Seltzer et al article (13) was the only on with a sample size worthy of referencing. The abstract 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 correlated included signs, symptoms, pulp test results, percussion, palpation and radiographic findings. Contemporarily, the most extensive sections related to apical periodontitis were found in Ørstavik & Pitt Ford (16).

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 evidenced-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/periradicular infectious process.

Studies, with valid and achievable protocols are necessary that include patients with a range of signs and symptoms, 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. Patient who are having their teeth extracted for periodontal, prosthetic or orthodontic purposes may serve at the best model 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:

Determine clinical signs and symptoms

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

Swelling: Presence or absence and degree and location

Sinus tract: Presence or absence

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

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.

Sections would be examined for:

Inflammatory cells: nature and distribution and relative numbers

Purulence: Presence or absence

Sinus tract: Presence or absence

Nature of surrounding bone: Increased or decreased

Possibly consider some sections using Brown & Brenn for bacterial identification

Advanced tests for microbial assessment and analysis

Correlate 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 repair-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.

Additionally, there appears to be little if any support peer-reviewed literature support for the use of the clinical terms “symptomatic and asymptomatic” in conjunction with presently used diagnostic terminology and disease states (This could refer to pulp or periradicular tissues). These terms have slowly crept into usage with little scientific basis for their applications or meanings. Diagnostic states based on symptomology were attempt in 1977 (21) with little validity other than empiricism and a failed attempt to divide histological diagnoses from clinical diagnoses. Their use was recently promulgated and perpetuated again without any rationale or scientific bases (12) (American Board of Endodontics). The use of these ambiguous terms may very lead to overtreatment, with little rationale other to say to a patient that you have a tooth with asymptomatic irreversible pulpitis or asymptomatic apical periodontitis that apparently must be made purely on a “subjective” assessment of radiographic findings; which could mean let’s treat anything that walks in the door. Protocols to assess the validity, accuracy and preciseness of these terms on a clinical basis with considerations for the patient’s level and perception of pain/sensitivity are 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 upon 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 also allow for future outcomes assessment and validation.
References – Not listed in any table or detailed in the text but indicated in the text with only a numerical designation.

Jaywant SS, Pai AV. A comparative study of pain measurement in acute burn patients. IJOT 2004; 35:13-17
Brennan MT, Runyon MS, Battis JJ, Fox PC, Kent ML, Cox TL, Norton HJ, Lockhart PB. Odontogenic signs and symptoms as predictors of odontogenic infection. JADA 2006; 137:62-66
Table 1 Initial Medline Search – Remaining Articles for Review


Table 2 Second Medline Search – Remaining Articles for Review


Table 3 – Articles Identified Through Manual Searching


Table 4 – Contemporary Texts and Adjunctive Publications

Seltzer S. Endodontology, Biologic Considerations. 2nd ed. Phila: Lea & Febiger, 1988
Nicholls E. Endodontics. 3rd ed. Bristol:John Wright & Sons, 1984
Weine F. Endodontic Therapy. 6th ed. St. Louis: Mosby, 2004
Chronic Grossman clinical grade

Table 5 – Glossary of Endodontic Terms Published by the AAE and the American Board of Endodontics


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.”


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


Table 7 - Glossary of Periodontal Terms - The American Academy of Periodontology 2001

ABSCESS: 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 a 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. (Cemento): 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.


GRANULOMATOUS TISSUE: A distinctive morphologic pattern of inflammation consisting of histiocytes that have been transformed into epitheloid 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 suppurring cavity.


Table 8 - Terms used in Oral & 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 pulpite (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 hat has separated from the adjacent vital bone.