Identification and Definition of Diagnostic Terms of Pulpal and Health Disease States

Consensus Conference on Diagnostic Terminology
American Association of Endodontists
Question 2 Subcommittee

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Executive Summary

Question 2: Identify and Define all Diagnostic Terms for Pulpal Health and Disease States

Consensus conference subcommittee 2 was charged with the identification and definition of all diagnostic terms for pulp health and disease states. Specifically this was accomplished by a systematic review of the literature in the areas of pain quantification, pulp pathophysiology, pulpal diagnostic terms and pulp testing metrics. The objective of this analysis was the establishment of evidence-based diagnostic nomenclature for clinically encountered pulpal conditions.

The following databases were searched for literature pertaining to our charge: Medline-Ovid, Pubmed, Web of knowledge, Cochrane Oral Health Group, EMBASE, SCOPUS, Google Scholar and Medstory. Non-English citations and non-human studies were excluded in searches for pain and pulpal metrics. Texts reviewed included Endodontics 6th Ed. Ingle JL, Bakland, LK, BC Decker, Hamilton Ontario, Canada 2008; Pathways of the Pulp 9th Ed. Cohen, S, Hargreaves, KM, Mosby Elsevier, St Louis, Mo. USA 2006; Principles and Practice of Endodontics 4th Ed. Torabinejad M, Walton RE, Saunders, Philadelphia, Pa., 2008; Encyclopedia of Pain Schmidt, RF, Willis, Springer, Berlin, Germany 2006; Essential Endodontology: Prevention and Treatment of Apical Periodontitis: Orstavik and Pitt Ford, 2007; Problems were encountered in consistency of terminology, a lack of high levels of evidence and inherent subjectivity in subject matter (diagnostic terminology). A lack of studies with high levels of evidence posed the most significant concern.

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

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

Based on pulp pathophysiology the diagnostic terms that best represent pulpal health and disease are
the following: Clinically Normal Pulp, Reversible and Irreversible Pulpitis, Pulp Necrosis, Root-filled
tooth without signs of infection, Root-filled tooth with signs of infection. Incomplete endodontic
treatment without signs of infection, Incomplete endodontic treatment with signs of infection, Pulp
Canal Calcification, Hyperplastic Pulpitis, Internal inflammatory resorption (active or inactive), and
Internal Surface Resorption. The subcommittee recognizes that there are other qualifiers such as the
perceived presence or absence of infection (i.e. necrotic pulp with infection). This is not always easily
determined clinically. It is recommended as a point of discussion in terms of adopting it as part of
terminology. It should be emphasized that levels of evidence (LOE) in the literature supporting the
use of specific clinical diagnostic terminology is generally very low in that the classification schemes
appear to be mainly the opinions of the various authors who provide logical arguments for their
choices in developing nomenclature based on studies with levels of evidence rarely exceeding lowest
level. They are usually related to clinical examination findings, however there is much uncertainty as
to the specific correlations between diagnostic information and the actual treatment needs of the
patient. More clinical study is needed in this area.

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

The pervasive impediment to our work was the observed lack of controlled clinical studies with high
levels of evidence in the area of pulpal diagnosis, diagnostic modalities and metrics for pain and
diagnostic measurements. Future work in these areas is imperative for the advancement of
Endodontics.

Identify and Define all Diagnostic Terms for Pulpal Health Disease States
1- Clinical Quantification of Pulpal Pain
The absolute measurement of pain on a scale common to all patients is not possible due to the
individual subjectivity of the pain response (11, 12). As a result, initial evaluations as well as the
effectiveness of interventions must be assessed using vague descriptors relative to the individual pain
experience such as ‘severe’, ‘spontaneous’ and ‘continuous’ or a subjective determination of the
increase or decrease in intensity. More precise forms of pain measurement are available but their
value in endodontic diagnosis and treatment has not been determined.

Several techniques for pain measurement in human subjects have been described. They include
Verbal rating scales(13-25), Numerical rating scales(18, 26), Visual analog scales(22, 27-37), Color
analog scales(38-42), Finger span expression(19, 43, 44), Calibrated questionnaires(9, 18, 45-48), and
Cortical evoked potentials(16, 17, 26, 31, 49-52). A brief description of each is presented.
Verbal rating scales are a list of verbal pain descriptors such as no pain, mild pain, moderate pain and
severe pain. The patient chooses the word which best describes their pain and a number is assigned to
this depending on its ranking in terms of intensity.
Numerical rating scales are a list of numbers, for example one to 100) with zero being no pain and 100
the most intense pain imaginable. The patient selects a number that corresponds to their pain
intensity.
Visual analog scales consist of a line with two end points of ‘no pain’ and ‘worst pain ever’. The
patient marks a point on the line that relates to the intensity of their pain. The distance of that point
from ‘no pain’ is the measure of pain intensity.

Color analog scales are used with children. A series of graded intensity colors are anchored at each
end by the terms ‘no pain’ and ‘worst pain.’
Calibrated questionnaires should really be ‘calibrated questionnaire’ as there is only one that has
gained widespread acceptance ‘The McGill Pain Questionnaire’. This consists of twenty groups of
descendants selected from the medical literature which describe either the sensory qualities of the pain,
the affective qualities of the pain or are evaluative describing the overall intensity of the experience.
These are displayed on a form which includes diagrams used for localization. A pain rating index is
determined on the rank values of the words. The McGill Pain Questionnaire has been translated into
at least sixteen languages and is very widely used. Its advantage is that it allows measurement of the
different components of the pain experience individually providing a three dimensional measure of
the experience whereas the other scales are uni-dimensional recording predominantly intensity.

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

Cortical evoked potentials are components of and electroencephalograph taken while applying a
noxious stimulus and can be used with an unconscious subject.
Measurement of pulpal pain (8, 13, 14, 18-26, 28, 30, 32-39, 43, 45-48, 51-63) A systematic review of the
literature revealed no published reports of quantifying pulpal pain in a truly clinical situation. All
available reports resulted from experimental settings in which the effect of some variable such an
analgesic, local anesthetic, exercises, orthodontic tooth movement etc. on the perception of pain was
determined by measuring the pain after pulpal stimulation. There are many reports of efficacy testing
of local anesthetics that use the failure to respond to an electrical pulp tester as an indicator of
effective anesthesia. This is not quantification but the reporting of an ‘all or none response’. These
reports were not included in this survey. Some studies of local anesthetic solutions do use pain scales
and they have been included.
While some of the studies reviewed for this submission are of high level in that they were randomized clinical trials, none of them examined the efficacy of the various scales in describing pulpal pain. This represents a significant deficit of knowledge in the area of pulpal pain assessment. The most prevalent approach endodontists use to assess pulpal pain is an informal Verbal Descriptor Scale with the terms such as severe, intermittent, or spontaneous being widely used. The Visual Analog Scale has achieved wide acceptance in the experimental field having the important attributes of simplicity and a facile conversion to numbers. The scale is clinically useful particularly with long term pain and serves as a valuable tool for the monitoring and assessment of clinical interventions. Calibrated questionnaires (essentially the McGill Pain Questionnaire) have very broad acceptance in many areas but would be less appropriate and more time consuming in the setting of the dental office than either Verbal Descriptor or Visual Analog scales. The use of Finger Span and Color Analog scales is generally confined to very young subjects and would be of limited application in the dental office. While electroencephalography would be an exciting extension to endodontic practice its acceptance is unlikely rendering the use of Cortical Evoked Potentials a distant possibility.

2- Identify and Describe Pulpal Conditions

Various states of pulpal health and disease exist and, historically, many classification systems have been used to designate them. The diagnostic systems that have been advocated can be combined into two main types: histopathologic classification systems and clinical classification systems yet most have used a combination of the two types of terminology (1-8). Since pulpal inflammatory disease is a progressive temporal continuum, a disease state that changes through time, there exist a large number of potential histopathologic descriptors of pulpal disease states. Clinically, however, only a limited number of pulpal conditions can be described based upon examination findings for a patient. Several studies have shown that there is little or no correlation between clinical diagnostic findings and the histopathologic state of the pulp (9-18). Since histopathologic diagnosis is not truly available to the endodontic clinician, and because diagnosis is needed to perform clinical endodontic treatment, then the various disease states of the pulp must be described using a clinical classification scheme.

Clinical classification is based upon the use of a diagnostic methodology to produce data that can be interpreted to develop a pulpal diagnosis. The information collected is the patient’s chief complaint, their medical and dental history, and the results of objective testing. The information is used to develop a diagnosis and a plan of treatment. It is usually helpful to format the process to increase efficiency and consistency. One such systematic format is given the name S.O.A.P. which is an acronym for Subjective findings, Objective tests, Assessment (or Appraisal) and Plan of treatment (4).

One of the earlier attempts to describe clinical pulpal states of health and disease was by Morse et al (6) and it is a variation of this system that we use today (5, 19). New systems of classification continue to arise as attempts are made to enhance the accuracy and clinical relevance of diagnostic terminology (20). By eliminating terminology that relates to the clinically inaccessible, histopathologic state of the pulp, the list of conditions that can be identified and described with respect to the dental pulp becomes manageable.

Levels of evidence (LOE) in the literature supporting the use of specific clinical diagnostic terminology is generally very low in that the classification schemes appear to be mainly the opinions of the various authors who provide logical arguments for their choices in developing nomenclature based on studies with levels of evidence rarely exceeding level 4. They are usually related to clinical examination findings, however there is much uncertainty as to the specific correlations between diagnostic information and the actual treatment needs of the patient (8). More clinical study is needed in this area.
The conditions of the pulp that can be identified and described will be listed in the following section.
The clinical manifestations of these conditions and the objective findings relating to them will accompany each descriptor.

Clinically Normal Pulp - This descriptor is mentioned in several classifications (8, 20) and is equivalent in meaning to vital asymptomatic (6) or healthy pulp (8). The term normal pulp appears to be more relevant to the clinical situation since it relates to the clinical presentation of the pulp. The words “vital” and “healthy” are inappropriate since vitality cannot be determined through clinical examination or “vitality” testing and pulps may be decidedly unhealthy and yet respond in a clinically normal manner. This descriptor indicates that all clinical signs are within normal limits (14) and that the tooth is asymptomatic. Depending on the age of the tooth, there may or may not be evidence of calcification of the pulp and there may be pulpal fibrosis. The pulp will generally respond to cold or electrical stimuli and the response will not linger for more than few seconds but will usually not respond to heat (20). Percussion, palpation, and bite tests will elicit no pain and the radiographic appearance will be normal.

Reversible Pulpitis - A pulpal state that implies the presence of mild pulpal inflammation and that the pulp is capable of healing (1, 2, 4-8, 19, 20) if appropriate therapy (i.e. removal of the irritant) is performed. Reversible pulpitis is secondary to caries, trauma, defective, or new restorations and is characterized by a mild to severe pain response to stimuli (usually thermal but possibly to biting pressure in a cracked tooth) (20-22). The pain resolves within seconds of removal of the stimulus. There is no response to percussion or palpation of the alveolus and the radiographic appearance is generally normal. Reversible pulpitis should be distinguished clinically from dentin hypersensitivity that is a phenomenon of fluid movement in the dentinal tubules and is not necessarily related to pulpal inflammation. The presentation of these two entities is very similar except that dentinal hypersensitivity can occur in the absence of the typical etiologic agents of pulpitis such as caries or faulty / new restoration. The etiology for this is exposed root dentin (1, 4, 20).

Irreversible Pulpitis – A pulpal state that implies the presence of a more severe degenerative process that will not heal and that, if left untreated, will result in pulpal necrosis followed by apical periodontitis. Pulpectomy or extraction is required to alleviate the symptoms and prevent apical periodontitis (1, 2, 4-8, 19, 20). Several classifications have broken this entity down into two types. The common factor in both of these is the requirement for endodontic therapy to treat the tooth. The first type is asymptomatic irreversible pulpitis and the second is symptomatic irreversible pulpitis(4, 19, 20).

Asymptomatic irreversible pulpitis is a pulpal state characterized by evidence of the need for endodontic therapy in the absence of clinical symptoms or pain. Irreversible inflammation of the pulp is produced by carious exposure (2, 23, 24), caries excavation, or trauma (1, 4, 19) necessitating root canal therapy. Despite being “painless” this form of pulpitis is expected to progress to pulp necrosis without treatment (18, 25).

Symptomatic irreversible pulpitis is a pulpal state characterized by mild to severe pain that lingers after removal of a stimulus (8) or that may be spontaneous (4). It implies a more severe degenerative inflammatory pulpal process that, if left untreated, will result in pulpal necrosis. The tooth will exhibit pain when exposed to thermal irritants (heat and/or cold) (8) that is prolonged well beyond the removal of the stimulus. The pain may be sharp or dull, depending on the type of pulpal nerve fibers responding to the inflammatory mediators (26) and peptides (27) (A delta fibers mediate sharp pain with c fibers mediating dull throbbing pain) (8, 28) and it may be localized or referred (4, 29). The etiology of irreversible pulpitis may be deep caries (24) or restorations, pulp exposure, cracks, or any other pulpal irritants. The tooth may or may not be percussion or bite sensitive and the
radiographic appearance may be unremarkable except for the presence of the etiologic agent (20). Occasionally, if the inflammatory process has extended into the periapical area, thickening of the periodontal ligament space (4, 20) or condensing osteitis (chronic focal sclerosing osteomyelitis) (30) may be visible. The treatment for irreversible pulpitis is root canal therapy or extraction of the tooth.

Pulp Necrosis – The end result of irreversible pulpitis (asymptomatic or symptomatic) (4) and, in many cases, dental trauma (1, 8) is necrosis of the pulp tissue (2, 3, 5-7). Because this event rarely occurs suddenly (except for cases of dental trauma), there occurs a variable period of time when the pulp will be partially necrotic. The area of cell death expands until the entire pulp necroses. Subsequent bacterial invasion will ultimately result in an infected root canal system (1, 2, 8, 31) and, without treatment, apical periodontitis. Teeth with necrosis of the pulp will present with variable symptoms ranging from none, to severe pain, bite sensitivity, and hyperocclusion (32) of periradicular origin. Occasionally, the tooth containing a necrotic pulp can become discolored (1, 33) due to altered translucency of the tooth structure or hemolysis of red blood cells during pulp decomposition. Radiographically, the appearance can vary from apparently normal to exhibiting a large periapical radiolucency. The one thing that usually distinguishes pulp necrosis from the other pulpal states is the absence of sensitivity to thermal or electrical pulp tests. Occasionally, the necrotic pulp may respond to heat application (4). Of all of the histopathologic pulpal states, necrosis is the one that is most reliably predicted from clinical testing (9, 11) with high correlations between negative pulp tests and necrosis of the pulp although this finding is not universally supported (13). Partial pulp necrosis (necrobiosis) (20) is very difficult to diagnose especially in multi-rooted teeth which may have different pulp states in different roots within the same tooth. This can occasionally give rise to positive responses to thermal and electric pulp tests combined with signs and symptoms of infected necrotic pulp (1, 20). The distinction between partial and full necrosis becomes important when dealing with immature teeth that have an open apex. To decide whether to perform apexogenesis or apexitification on these teeth, one must decide on whether the entire pulp is necrotic or not. The definitive test for this is to enter the pulp chamber and remove necrotic tissue until a vital pulp stump is reached (8).

Hyperplastic Pulpitis (Pulp Polyp) – This rarely found entity occurs when caries invades the pulp in an immature tooth with open apices (1, 2, 5, 23, 34-37). The enhanced blood supply created by the open apices allows the immature pulp to better resist bacterial invasion than a more mature pulp (36, 38) and the opening through the carious lesion into the oral cavity establishes a pathway for drainage of pulpal inflammatory exudates. Acute inflammation subsides and chronic inflammatory tissue proliferates through the opening (23). Clinically this appears as a fleshy mass of tissue connected to the pulp space that appears to be growing out of the tooth and the tissue is frequently epithelialized. Free-floating cells of the oral mucosa are “seeded” onto the proliferating granulomatous tissue resulting in a stratified squamous epithelium (2) and the resultant lesion is rarely painful except when masticatory forces cause irritation and bleeding (23). Radiographically, there appears a deep carious lesion apparently connecting to the pulp space and the root ends are immature. Treatment for this entity is either endodontic therapy or extraction since this condition is considered to be irreversible (2).

Internal Resorption – Internal resorption of the tooth structure is a pathological state of the pulp in which multinucleated stastic cells within the pulp tissue begin to remove the dentin of the walls of the pulp space. It is generally idiopathic in that the trigger for the metaeplastic transformation of normal pulp cells into stastic ones is unknown. Several hypotheses have been proposed (39, 40) and it is possible that it may be a combination of these that starts the resorptive process. The resorption sometimes moves swiftly and then may be followed by a time of slower or no growth in the size of the lesion (2). Internal resorption is generally painless and is usually found clinically through routine radiographic screening when it appears generally as an ovoid enlargement of the pulp space (41) in
which the original borders of the pulp space become distorted or disappear altogether (39, 40, 42). The lesion stays associated with the root canal on angled radiographs (39, 40). The tooth may respond to pulp sensibility tests, but occasionally the tests may be negative if there is partial necrosis with the advancing resorptive lesion within the living portion of the pulp tissue subjacent to the necrotic tissue (20, 39, 40). If perforation of the tooth structure has occurred and the tissue in the pulp space is exposed to oral fluids, pain may occur (39, 40). The crown of the tooth may appear pink in color (2, 42) due to thinning of the tooth structure allowing the color of the underlying granulomatous tissue to be visible, however, this may also be due to undermining, subepithelial external root resorption (39, 40). Internal root resorption is considered a form of irreversible pulpitis and requires root canal therapy to halt the process (2).

Pulp Calcification – Degenerative changes to the pulp such as pulp calcification or pulp atrophy / fibrosis are related to aging or sub lethal injury resulting in chronic irritation to the pulp. The pulp responds by fibrosing or calcifying (43, 44). Generally, pulp fibrosis or atrophy is a histological change that is not clinically discernible unless the pulp space is entered during the initial phases of root canal therapy, so its value as a diagnostic term is questionable. Pulp calcification, however, is usually clinically detectible prior to treatment and can directly affect the prognosis of treatment in that severely calcified teeth are predisposed to tooth perforation during the search for canals (45). This entity is also sometimes referred to as pulp canal obliteration (20) or calcific metamorphosis (46, 47) but both terms appear to be inaccurate since the canal is rarely completely obliterated (48) and there is actually no “metamorphosis” of the tooth, just a progressive deposition of dentin (secondary or tertiary) resulting in radiographically apparent shrinkage of the pulp canal space (1). Calcification, per se, does not necessarily imply that progressive inflammation of the pulp or pulp necrosis will occur. In fact, pulp necrosis is found in less than 7% of traumatically induced calcified pulps (49).

Previously Initiated Treatment – Occasionally a tooth that has had endodontic therapy previously started but not completed will present for diagnosis (19). These teeth would have undergone previous pulpotomy or pulpectomy and the history and clinical examination should reveal this. These teeth may or may not present with signs and symptoms of pulpal or periradicular disease (20) and radiographic evidence of access into the pulp space and the possible presence of radiopaque interappointment medicaments such as calcium hydroxide paste would be found. In these cases as with any necrotic pulp or pulpless tooth, given time, the pulp space will become infected and apical periodontitis would be expected to ensue (20) and so completion of endodontic therapy would be necessary.

Previous Endodontic Therapy – Many times, teeth that have had previous endodontic therapy will be examined by dentists (20). This entity is also referred to a “Previously Treated” (19) however this terminology is perhaps not specific enough to Endodontics to make it an appropriate term for this condition. Various treatment modalities would fall under this diagnostic category including teeth that have undergone non-surgical root canal therapy, surgical root canal therapy, and therapeutic pulpotomy with calcium hydroxide (the Cvek pulpotomy (40, 50, 51)) or with mineral trioxide aggregate (52) to induce apexogenesis. The history and both the clinical and radiographic examinations should indicate the existence of previous endodontic therapy. For treatment designed to preserve the pulp (pulpotomy) the important question to be answered is whether the treated pulp remains healthy and for teeth with completed full endodontic therapy, it is whether the pulp spaces are infected or not (20, 53). This will usually be determined by the response of the periradicular tissues (54), and the clinical determination as to whether bacterial ingress from the coronal aspect is likely to have occurred (55, 56). The technical quality of the root canal filling will also need to be assessed, but this cannot be completely addressed by inspecting the radiograph since this will only show a two dimensional representation of the obturation and perhaps the presence of an iatrogenic complication, such as perforation of the root or a separated instrument. The assessed technical quality
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of the root filling alone cannot give any indication as to whether the root canal space is infected or not
however (20). The decision as to whether or not to treat the tooth with this diagnosis (non-surgical /
surgical retreatment or extraction) will be determined by diagnosing the presence or absence of apical
periodontitis, by a thorough knowledge of outcomes assessments (57) and by whether other
considerations (such as restorative needs) require that treatment be instituted (58).

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

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

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

3- Diagnostic Terms that Best Represent Pulpal Health and Disease

Many different classification systems have been advocated for pulp diseasover the years although
most of them are based on histological findings – Table 2 in the appendix from Abbott & Yu (2007)1
has been reproduced as a summary of many of these classifications. Abbott15-17 and Abbott & Yu1
have also proposed a classification system of their own which varies from those in the Table.
Typically, these classifications mix clinical and histological terms resulting in many misleading terms
and diagnoses for the same clinical condition. This creates confusion and uncertainty in clinical
practice when a rational treatment plan needs to be established in order to target a specific
pathological entity.

Clinically Normal Pulp: All classifications of tissue conditions should include tissue that has not been
harmed in any way – that is, normal or healthy tissue.1 The clinical tests available to dentists to assess
the state of the dental pulp are relatively crude. These tests are not entirely reliable since they are
usually only testing the ability of the pulp to respond to a stimulus (i.e. pulp sensibility) and this does
not provide much information about whether the pulp is healthy or not. Hence, it is more appropriate
to classify the pulp as being a “clinically normal pulp” when there is an absence of any signs or
symptoms of pulp disease being present.1

Pulpitis: The first response of a dental pulp to a stimulus is inflammation. Hence, the most
appropriate term to use is “pulpitis” since the suffix “-itis” is defined in dictionaries as indicating
inflammation of the tissue whose name it is attached to – i.e. the pulp.19,20

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

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

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

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

Teeth with Previous Root Fillings: Teeth with existing root canal fillings need to be assessed as part of the routine clinical and radiographic examination of a patient. The most important aspect of this assessment is to determine whether the root canal system is infected or not since an infected canal will cause apical periodontitis. It is also important to assess the technical standard of the root canal filling as this may determine whether further treatment is required and/or feasible. Such determination is usually based on the radiographic appearance of the root canal filling.

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

Teeth that have root canal fillings may become infected at any time once a pathway of entry for micro-organisms becomes available. The management of such a tooth requires specific considerations and treatment techniques. Hence, a specific diagnostic category or term is required. The proposed term is “Infected root canal system in a root-filled tooth”.1

Teeth with Incomplete Endodontic Treatment: Patients may present to Dentists and/or Endodontists with a tooth that has had endodontic treatment commenced at some time in the past but the treatment was not completed. There are a wide variety of possible reasons why the treatment may not have been completed (e.g. patient did not return for treatment, patient was referred to a specialist for
further treatment, etc) – these may or may not be relevant to the diagnosis in all cases. It is important
to distinguish these cases from other conditions outlined above and below because their clinical
management may be different.

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

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

Teeth with Degenerative and/or Physiological Changes to the Pulp

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

Typical conditions are “Pulp Canal Calcification” – either part of the normal ageing process or it can
be an indication of long-standing irritation to the pulp. Calcification is defined as “abnormal
deposition of calcium salts within tissue” “Hyperplasia” - is defined as “an abnormal increase in
cells in a tissue or organ, excluding tumor formation, whereby the bulk of the tissue or organ is
increased”. This term can be used when there has been an overgrowth of granulation tissue
originating from the pulp and it may result in the development of a pulp polyp. It has been suggested
that the inflammation may be limited to the pulp chamber and that the apical pulp tissues may be
normal, except for some vasodilatation and minimal chronic inflammation. As this condition is
associated with inflammation, the term should be hyperplastic pulpitis.

Teeth with Internal Resorption

Three forms of internal root resorption have been reported although varying terminology has been
used to describe them. The different forms of internal resorption require different clinical
management and therefore it is essential that they be differentially diagnosed from one another. The
proposed terminology is: “Internal Surface Resorption” - when just minor areas of the root canal wall
have been resorbed. This resorption may be self-limiting and may repair if the pulp is relatively
healthy and if the irritating stimulus has been removed from the tooth. “Internal Inflammatory
Resorption” - occurs when an inflammatory response within the pulp (i.e. pulpitis) leads to
activation of dentinoclastic cells which resorb the dentine walls of the root canal and then progresses
through the dentine towards the cementum. This resorption is believed to be a result of the
presence of micro-organisms within the coronal part of the root canal which causes pulpitis in the
pulp apical to the resorptive area. Hence, a tooth with active internal inflammatory resorption will
have some necrotic and infected pulp tissue as well as some pulp tissue with irreversible pulpitis. If
the condition is defined as such, then there is no need to mention each of these conditions in the
diagnosis. The dentinoclasts present in internal inflammatory resorption will only remain alive and
active as long as there is a viable blood supply to the apical part of the pulp. If this blood supply is
lost then the apical part of the pulp will necrose and the dentinoclasts will also die. Thus, the internal
inflammatory resorption will no longer be active. Typically, the necrotic apical pulp tissue is then
digested and removed by the micro-organisms and the entire canal will become pulpless (as
described above), resulting in apical periodontitis. Once apical periodontitis is evident, it is highly likely that the resorption is no longer active which will make clinical management somewhat easier and less involved. Hence, it is important to distinguish between active and non-active states of internal inflammatory resorption. “Internal Replacement Resorption” - is a metaplastic-type of change to the dental pulp where first the pulp is replaced by bone and then subsequently the dentine is replaced by bone. 30 This condition must be distinguished from the other two types of internal resorption mentioned above since its clinical management is quite different – i.e. the tooth can be extracted or it can be left untreated and simply reviewed until extracted is required.

4- Which Combination of Metrics Provides the Maximal Accuracy or Establishing Pulpal Diagnoses?

Inconsistent definitions of pulpal disease have led many researchers to dichotomize pulpal status into general categories that are defined as vital or non-vital. Others have elected to further categorize vital pulp status according to the severity of inflammation and, in particular, whether the inflammation is reversible or irreversible. In an effort to interpret research findings in a meaningful way, this paper will address the evidence for metrics for establishing diagnoses of 1) vital vs. non-vital pulp, and 2) normal pulp vs. reversible pulpitis vs. irreversible pulpitis. The best method for arriving at the agreed upon definition for pulpal disease, which may or may not be impractical or desirable to use within clinical practice, is termed the “gold standard” test or reference test. The results from such a “gold standard” test for pulp diagnosis is used to compare with the diagnostic test being evaluated for the determination of testing accuracy. Studies assessing diagnostic accuracy for pulpal disease testing have used two different “gold standard” tests; a clinically derived measure (e.g., presence of necrotic tissue upon accessing a tooth would indicate that the tooth was non-vital) and a histologically derived measure (e.g., on extracted teeth for which the history of symptoms have been established and/or on which pulp tests have been performed). It must be recognized that because the progression of pulpal disease may result in periapical changes, and therefore metrics used to establish a periapical diagnosis may aid in the determination of a pulpal diagnosis. For example, if one arrives at an endodontic diagnosis of apical periodontitis, the implication is that there is inflammation of the periodontal ligament caused by infection of the pulp or necrotic pulp space.

Metrics for Diagnosis of Vital vs. Non-vital Pulp

A diagnosis of vital vs. non-vital pulp is relatively straightforward when compared to determining a diagnosis of normal pulp vs. reversible pulpitis vs. irreversible pulpitis. This is because the interpretation of the findings from the pulp tests can be dichotomized (i.e., response vs. no response). Furthermore, the “Gold Standards” for studies on metrics for determining vital vs. non-vital pulp are more readily discernable (i.e., determination of necrotic pulp tissue upon endodontic access or upon histological examination after extraction). Thus, there is relatively more evidence related to determination of vital vs. non-vital pulp. The tests for which some level of accuracy for determining pulp status has been determined are cold, heat, electric, laser Doppler flowmetry, and pulse oximetry.

Comparison of studies that address pulp testing methods is challenging, given the variations in factors such as testing methodology (e.g., stimulus type, method of application, definition of response, location of stimulus) tooth variables (e.g., restorations, caries, past trauma, recession, tooth type) patient variables (e.g., age, gender, anxiety, oral habits, systemic diseases). This challenge is exemplified when one attempts to interpret the findings of studies that address the use of the cold test. Materials used as a coolant include CO2 snow, ice stick, 1,1,1,2 Tetrafluoroethane, ethyl chloride, and dichlorodifluoromethane. Application methods include direct application, cotton swab, and cotton pellet, and cotton roll. Given the inherent challenges, it is not surprising to find considerable variability between studies. The findings from the selected studies related to the accuracy of these tests are summarized in Tables 4-8 in the Appendix. One can conclude from the information presented in these tables that there is considerable variability in the sensitivity and specificity of cold and heat tests, and in the sensitivity of electric test pulp tests. Thus, the studies suggest that there is no agreement as to whether cold and heat tests, when used in the absence of other tests, can reliably
determine the presence of diseased (i.e., non-vital) pulp, or for cold, heat and electric tests to identify
teeth without disease (i.e., vital pulp). There is less variability in findings for specificity of electric
pulp tests, suggesting that this test is more consistent at identifying teeth without disease (i.e., vital
pulp). In addition, it appears that heat tests have lower positive predictive values than cold or electric
tests. Thus, a lack of response to a heat test appears to be less likely to be predictive of a vital pulp.

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

Other Clinical Measures of Pulp Disease
In addition to tests for pulp responsiveness and pulpal blood flow, other factors have been used in an
attempt to determine pulp status. Evans et al (1999) reported that the presence of external root
resorption, periapical radioluency, crown discoloration, tenderness to percussion, and history of
pain were all found to have a high specificity (0.97 or better), but low sensitivity (0.49 or lower), for
non-vitality. However, the authors failed to disclose the clinical criteria that were used for assessment
of these characteristics, making it impossible to validate their findings. A clinical finding of carious
pulp exposure has been reported in endodontic text books (e.g., Berman et al, 2005; Ingle et al 2002;
Torabinejad and Walton, 2002; Siggurdson, 2007) as indicating an irreversible pulpitis. This has been
based, in large part, on histological evaluation of extracted teeth with deep carious lesions (e.g.,
Torneck, 1974). No articles were found that used 1) a standardized method for determining when
pulp was exposed during caries removal, along with 2) a “gold standard” for determination of
accuracy of caries excavation as a metric for determining reversible vs. irreversible pulpitis.

Identification of Reversible vs. Irreversible Pulpitis
Studies that have attempted to determine accuracy (or have enough information in the report to
establish accuracy) of metrics for determining diagnoses of reversible vs. irreversible pulpitis are less
common than studies that determine accuracy of metrics for determining vital vs. non-vital pulp.
Some researchers (e.g., Garfunkle et al, 1973) have attempted to correlate the results of diagnostic tests
with categories of pulpal inflammation. Hyman and Cohen (1984) summarized the results of four
papers that histologically evaluated teeth following pulp tests. The metric that was evaluated in this
table was from teeth that had an “abnormal reaction to cold test” And the “Gold Standard was
histological evidence of pulpal inflammation. (Table 9, Appendix) When compared to the
determination of vital vs. non-vital pulp tissue, the determination of reversible vs. irreversible
pulpitis using cold has relatively lower sensitivity, specificity, and positive predictive values. Studies
have not been conducted in which pulse oximetry and laser Doppler flowmetry have been used to
differentiate between reversible and irreversible pulpitis.
History of the presenting symptoms: In addition to using pulp tests to determine the severity of pulp inflammation, some researchers have attempted to evaluate whether the history of presenting symptoms could be used as a metric for determining pulp status. Grushka and Sessle (1984) have used the McGill pain questionnaire to differentiate types of toothache pain, and determined that self-reports of toothache pain seem to be valid predictors of whether pulp inflammation is reversible or not. The methodology used by Grushka and Sessle for determining reversible vs. irreversible pulpitis was only defined as the use of “standard dental diagnostic procedures”. Thus, a “Gold Standard”, such as pulp status upon endodontic access, or extraction and histology, was not used. In addition, the statistical analysis does not allow for determination of the accuracy of metrics used for diagnosis. Other authors have addressed the history of presenting symptoms as a metric for determining a pulp diagnosis. For example, Bender (2000) has reported that the more severe pulpal pain is, and the longer it had been present, the more likely it is that irreversible inflammation has been present. Another predictive factor for determining whether pulpal inflammation is reversible is a history of being spontaneous. In some cases the spontaneous pain was so severe as to wake the patient from sleep Bender et al (1963).

Limitations of using History of Presenting Symptoms: While the history of presenting symptoms may be useful as an aid in determining a pulpal diagnosis, it is worth noting that none of the studies that have addressed the history of presenting symptoms have resulted in sensitivity, specificity, positive predictive value, or negative predictive value of the symptoms. In addition, studies that have assessed the history of symptoms for teeth with necrotic pulps have shown that 26-60% of the cases had no history of pain (Barbakow et al, 1981; Beveridge and Brown, 1965). Thus, while a history of presenting symptoms would, for some patients, aid in determining the pulpal diagnosis, for many patients, the history would not yield predictive value. Table 10 (Appendix) exemplifies the challenges of developing metrics for pulpal diagnosis (and specifically reversible vs. irreversible pulpitis) based upon history of the presenting symptoms.

Identified Deficiencies in Available Evidence: There are several areas where there is a lack of knowledge concerning the accuracy of metrics for determining pulp diagnoses. An ideal metric, or combination of metrics, would result in a definitive diagnosis that would lead to known outcome, thereby suggesting treatment options if the predicted outcome is undesirable. In general, pulp tests are more sensitive and specific when used to determine vitality of pulp tissue, as compared to determining the severity of pulpal inflammation. Given that an extensive review of the highest levels of evidence has shown that “the preoperative presence of apical periodontitis has a dominant, negative effect on the outcome of nonsurgical endodontic treatment” (Friedman, 2007), the goal of pulp testing should be to prevent apical periodontitis and thus, maximize outcomes of endodontic treatment. In other words, the goal of pulp testing should be not only to determine when the pulp has become non-vital (and most likely infected, resulting in the likelihood of apical periodontitis), but also to determine when the pulpal inflammation had become irreversible. The ability to determine when inflammation of the pulp has become irreversible would, therefore, guide the practitioner and patient in treatment choices (i.e., nonsurgical root canal treatment vs. extraction), and pre-empt the subsequent necrosis, infection, and apical periodontitis.
References


36. Rosenberg PA, Amin KG, Zibari Y, Lin LM. Comparison of 4% articaine with 1:100,000 epinephrine and 2% lidocaine with 1:100,000 epinephrine when used as a supplemental anesthetic. Journal of Endodontics 2007;33:403-405.


Identify and Describe Pulpal Conditions


Diagnostic Terms that Best Represent Pulpal Health and Disease


Abbott PV. Classification, diagnosis and clinical manifestations of apical periodontitis. Endod Topics 2004;8:36-54.


Which Combination of Metrics Provides the Maximal Accuracy or Establishing Pulpal Diagnoses? (Levels of Evidence are in bold.)


Fulling HJ, Andreasen JO. Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing. Scand J Dent Res 1976;84:286–90.3b


Grushka M, Sessle BJ. Application of the McGill pain questionnaire to the differentiation of toothache pain. Pain 1984:19:49-5. 4


Hargreaves KM. Pain mechanisms of the pulpodentin complex. Seltzer and Bender’s Dental Pulp, Hargreaves, Goodis and Seltzer, Chicago:Quintessence, 2002; 5


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

Table 2
The classification presented in the Table below has been proposed previously by Abbott.1,15-17 It is a simple yet comprehensive clinical diagnostic system that utilizes terminology outlined above and relating to the clinical findings. It is based on the progression of pulp diseases through the various stages discussed above. It also includes “normal” pulp tissue which is an entity that should be diagnosed and given recognition when there are no signs of disease.

<table>
<thead>
<tr>
<th>Clinically Normal Pulp (based on clinical examination and test results)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reversible Pulpitis - Acute</td>
</tr>
<tr>
<td>- Chronic</td>
</tr>
<tr>
<td>Irreversible Pulpitis- Acute</td>
</tr>
<tr>
<td>- Chronic</td>
</tr>
<tr>
<td>Necrobiosis (Part of pulp necrotic &amp; infected; the rest is irreversibly inflamed)</td>
</tr>
<tr>
<td>Pulp Necrosis- No sign of infection</td>
</tr>
<tr>
<td>- Infected</td>
</tr>
<tr>
<td>Pulpless, Infected Root Canal System</td>
</tr>
<tr>
<td>Degenerative Changes</td>
</tr>
<tr>
<td>Atrophy</td>
</tr>
<tr>
<td>Pulpal canal calcification - Partial</td>
</tr>
<tr>
<td>- Total</td>
</tr>
<tr>
<td>Hyperplasia</td>
</tr>
<tr>
<td>Internal resorption - Surface</td>
</tr>
<tr>
<td>- Inflammatory</td>
</tr>
<tr>
<td>- Replacement</td>
</tr>
<tr>
<td>Previous Root Canal Treatment</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>No sign of infection</td>
</tr>
<tr>
<td>Infected</td>
</tr>
<tr>
<td>Technical standard (based on the radiographic appearance)</td>
</tr>
<tr>
<td>- Adequate</td>
</tr>
<tr>
<td>- Inadequate</td>
</tr>
<tr>
<td>Other problems - e.g. Perforation, Missed canals, Fractured instrument, etc.</td>
</tr>
</tbody>
</table>
## Comparative terminology and classifications of pulp diseases used by various authors and organisations

<table>
<thead>
<tr>
<th>World Health Organisation</th>
<th>Wein3</th>
<th>Ingle4</th>
<th>Seltzer &amp; Bender5</th>
<th>Cohen &amp; Burns6</th>
<th>Tronstad7</th>
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<tr>
<td>(NOTE: Normal pulp not mentioned)</td>
<td>(NOTE: Normal pulp not mentioned)</td>
<td>Healthy Pulp</td>
<td>(NOTE: Normal pulp not mentioned)</td>
<td>Within Normal Limits Normal pulp Calcific metamorphosis</td>
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<tr>
<td>Initial (hyperaemia)</td>
<td>Hyperalgesia (reversible pulpitis)</td>
<td>Hyper-reactive pulpalgia</td>
<td>Incipient form of chronic pulpitis</td>
<td>Reversible</td>
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<td>Hypersensitive dentine</td>
<td>Hypersensitivity</td>
<td>Acute pulpitis</td>
<td>Irreversible</td>
<td>Irreversible pulpitis</td>
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<td>Suppurative (pulpal abscess)</td>
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<td>Chronic partial pulpitis with partial necrosis</td>
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<td>Chronic</td>
<td>Painful pulpitis</td>
<td>Acute pulpalgia (acute pulpitis)</td>
<td>Chronic total pulpitis with partial liquefaction necrosis</td>
<td>Irreversible</td>
<td>Irreversible pulpitis</td>
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<td>Chronic ulcerative</td>
<td>Acute pulpalgia (subacute pulpitis)</td>
<td>Chronic ulcerative pulpitis</td>
<td>Chronic partial pulpitis (hyperplastic form)</td>
<td>Asymptomatic</td>
<td>Symptomatic pulpitis</td>
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<td>Chronic hyperplastic (pulp polyp)</td>
<td>Nonpainful pulpitis</td>
<td>Chronic pulpalgia</td>
<td>Hyperplastic pulposis</td>
<td>Internal resorption</td>
<td>Symptomatic pulpitis</td>
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<td>Other unspecified pulpitis</td>
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<td>Pulp degeneration Atrophic pulposis</td>
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<td>Pulpal stones</td>
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<td>Calcific pulposis</td>
<td>Dystrophic calcification</td>
<td>Dystrophic mineralization</td>
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<td>Abnormal hard tissue formation in pulp</td>
<td>Internal Resorption</td>
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<tr>
<td>Secondary or irregular dentine</td>
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<td>Pulp calcification Internal (intracanal) resorption</td>
<td>Pulp degeneration Calcific Fibrous Atrophic Internal resorption</td>
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Table 4:
Cold Testing

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<th>NPV</th>
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a In Petersson et al (1999), Gold Standard was determined by “direct pulp inspection”.
b In Evans et al (1999), Pulpal status was “confirmed by pulpectomy.”
c In Gopikrishna et al (2007a), Pulpal status was evaluated by direct visual inspection

Table 5
Heat Testing

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<tr>
<th>Reference</th>
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<th>NPV</th>
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a In Petersson et al (1999), Gold Standard was determined by “direct pulp inspection”.

Table 6
Electric Pulp Testing

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a In Petersson et al (1999), Gold Standard was determined by “direct pulp inspection”.
b In Evans et al (1999), Pulpal status was “confirmed by pulpectomy.”
c In Gopikrishna et al (2007a), Pulpal status was evaluated by direct visual inspection

Table 7
Pulse Oximetry

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c In Gopikrishna et al (2007a), Pulpal status was evaluated by direct visual inspection"
Table 8
Laser Doppler Flowmetry

<table>
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<td>Clinicalb</td>
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Table 9
Abnormal reaction to cold test detecting an irreversible pulpitis (Table taken from Hyman and Cohen (1984))

<table>
<thead>
<tr>
<th>Reference</th>
<th>“Gold Standard”</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
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</thead>
<tbody>
<tr>
<td>Seltzer et al</td>
<td>Histology</td>
<td>0.41</td>
<td>0.76</td>
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<td>0.81</td>
</tr>
<tr>
<td>Dummer et al</td>
<td>Histology</td>
<td>0.63</td>
<td>0.80</td>
<td>0.48</td>
<td>0.88</td>
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<tr>
<td>Garfunkle et al</td>
<td>Histology</td>
<td>0.57</td>
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</tbody>
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Table 6

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

Modified from Clinical Characteristics of Pulpitis, in Problems in Managing Endodontic Emergencies, Dumsha and Gutmann (1997)

Table 10

<table>
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