IEJ
Iranian Endodontic Journal

Pulp, Root Canal and Peri-radicular Conditions: The Need for Re-classification

Paul V. Abbott [a*]

[a] IUWA Dental School, The University of Western Australia

Article Type: **Review Article**

Received: 14 Jan 2024
Revised: 05 Mar 2024
Accepted: 16 Apr 2024
Doi: *10.22037/iej.v19i3.44394*

Corresponding author: Paul V. Abbott, UWA Dental School, The University of Western Australia, 17 Monash Avenue, Nedlands, WA, 6009, Australia.

E-mail: paul.v.abbott@uwa.edu.au

Currently no standard, universally accepted, and clinically useful classification of pulp, root canal and peri-radicular conditions is used within the dental profession. Most published classifications are either too simple and miss many of the conditions reported to occur within the pulp, root canal and peri-radicular tissues, or they are too complex for use in clinical settings. Furthermore, many classifications have used inappropriate terminology that has either not been defined or has been poorly defined. The lack of standardisation leads to confusion amongst practitioners and potential uncertainty regarding treatment. It also limits the ability to communicate effectively, to teach appropriately, and to compare data and research findings throughout the world. When developing a classification of tissue conditions or diseases, it is essential that the classification is developed appropriately and meets the recommended criteria for effective clinical, educational and research use. It is also extremely important that correct and well-defined terminology is used since "words do matter". Popular terminology based on symptoms should be avoided as these have been proven to lead to inappropriate treatment decisions. This review discusses how classifications should be developed for pulp, root canal and peri-radicular conditions. It also discusses the deficiencies of some popular classifications and outlines the classifications that truly address the recommended criteria and reflect the physiological and pathological changes in the pulp, root canal and peri-radicular tissues. Hence, it is recommended that these latter classifications be adopted as the internationally-accepted classifications for future clinical use, as well as for educational, research and communication purposes.

Keywords: Classification; Dental Pulp; Peri-radicular Conditions; Root Canal Conditions

Introduction

Currently no universal or internationally-accepted classification of pulp, root canal and peri-radicular conditions exists. This largely stems from authors of textbooks and journal articles using different terminology and not following a systematic approach to developing their classifications. This is not a new problem as demonstrated by Tronstad in 1991 [1] when he compared the classifications of pulp and periapical conditions used in 5 major endodontic textbooks. His comparison clearly showed the lack of consistency in the terminology used in these textbooks. In 2007, Abbott and Yu [2] compared the classifications of pulp and root canal conditions published in 11 textbooks and two others used by professional organisations. This comparison also clearly demonstrated the lack of consistency in terminology used for these conditions.

It was also apparent that the majority of these classifications are either too simple and miss many conditions or they are too complex to be readily used clinically. In addition, most of these classifications have not included all of the conditions that have been reported to occur within the pulp, root canal and peri-radicular tissues (see below). A few examples of some missing conditions are:

- Some missing *Pulp* conditions: Pulpless and infected root canal system, hyperplastic pulpitis, pulp canal calcification, *etc.*
- Some missing *Root canal* conditions: Root-filled and infected root canal system, incomplete root canal treatment (RCT), *etc.*
- Some missing *Peri-radicular* conditions: Extra-radicular infection, foreign body reaction, external inflammatory resorption, *etc.*

The above are examples and many more conditions have also been missed. If a classification does not include all possible conditions, then it is deficient and should not be considered as suitable for clinical use.

Furthermore, most of the classifications use words or terms that are undefined or poorly defined, uncommon, from a different language (*e.g.* Latin) or not representative of the condition being described. When the published requirements for clinical classifications of any conditions and/or diseases of the body [2-7] are considered (see below), it is clear that most classifications of pulp, root canal and peri-radicular conditions in textbooks and those used in the dental literature are inadequate as they do not meet these criteria. The result is no current standard terminology exists which ultimately causes confusion amongst clinicians, researchers and students [8].

Over the past decade and a half, many practitioners have adopted the classifications proposed by the American Association of Endodontists (AAE) in 2009 [9]. However, there has been regular and considerable debate since then about the suitability of these classifications. The AAE classifications resulted from a series of papers prepared by committees of invited experts appointed by the AAE [10-12]. These papers were then discussed at what was designated as a "consensus conference" in Chicago in October 2008 [8]. There were 67 attendees at the conference and they had all had been invited by the AAE because of their research and publications in journals and/or textbooks regarding pulp, root canal and peri-radicular conditions [13]. Unfortunately, the level of consensus achieved at that conference was far from ideal and much debate has continued since then regarding the final recommendations made by the AAE. Several concerns were raised at the conference and these are discussed below.

Another confounding factor regarding pulp diagnoses is the long-held belief that histological findings and the clinical presentations of pulp conditions do not correlate. This was based on studies from the 1960's and 1970's [2, 14-18] which led to confusion amongst clinicians, and subsequently, the use of many poor, inappropriate diagnostic terms. However, in 2014, Ricucci *et al.* [19] reported very good correlations between histological findings and clinical diagnoses with 96.6% of cases clinically diagnosed with either a normal pulp or reversible pulpitis having the same histologic diagnosis, and 84.4% of teeth clinically diagnosed with irreversible pulpitis having the same histologic diagnosis. Hence, those old concepts and beliefs regarding pulp diagnosis must be re-considered.

Some other specialty disciplines within dentistry have developed classifications of the conditions specific to their disciplines; for example, the specialty of periodontics has revised its classification several times [20-27] and these have been widely accepted by the profession. However, the discipline of endodontics is lacking such an approach, which is to the detriment of the endodontic specialty and the dental profession in general, as well as students, educators, researchers and patients [8].

The aim of this review is to discuss the need for the dental profession, and specifically the specialty of endodontics to reconsider the classification of pulp, root canal and peri-radicular conditions. Prior to discussing and proposing a classification, it is important to discuss some of the terminology used and the background behind the various classifications and the controversies associated with them.

Terminology

In considering any classification, it is essential to define the terminology to be used since the actual words used do matter and they will help clinicians, researchers, educators and students to understand the various conditions and to communicate effectively [28, 29].

Another important aspect regarding terminology is the effect that words have on the patients. A systematic review of the medical literature [30] reported that different terminology affects the patient's decisions regarding the management options and, in particular, when "more medical" or "more precise" terms were used, patients had strongly preferred more invasive medical procedures [30]. A study by Bestall *et al.* [31] has also reported that patients may be more vulnerable to the words their clinicians use. Hence, terminology used by clinicians for diagnosis is important from both the patient and practitioner perspectives because it may influence management of the presenting diseases or conditions. Language is a powerful tool as it can shape both the understanding and management of diseases and other conditions [32].

The following discussion outlines some controversial and unclear terminology associated with many of the classifications for pulp, root canal and peri-radicular conditions. Recommendations for clear, well-defined terminology are provided.

"Conditions" versus "Diseases"?

Distinguishing between the terms *"conditions"* and *"diseases"* is important because not all conditions are true diseases [3, 6]. A *"disease"* is defined as *"a disordered or incorrectly functioning organ or system of the body resulting from (amongst other causes) the effect of infection, toxicity, or unfavourable environmental factors. It is considered as an illness or ailment"* [33]. Since many pulp conditions do not fit this definition, it is inappropriate to group them as *"diseases"*. Some examples of pulp conditions that are not "diseases" are: clinically normal pulps, pulp atrophy and pulp canal calcification. Likewise, not all conditions occurring within the peri-radicular tissues fit this definition of diseases-an example is apical periodontitis which is an inflammatory reaction that is part of the body's defense against the presence of bacteria in the root canal system, and as such, the tissues are functioning normally rather than being a *"disordered or incorrectly functioning organ or system"*.

In the medical sense, the term *"condition"* is defined as the *"state of health"* [34] so it can be applied to both healthy and diseased tissues. Hence, the term "condition" is more appropriate and will be used in this review unless the process being discussed is a true *"disease"* that fits the above definition.

"Pulp and root canal conditions" versus "Pulp"?

In the literature, the general heading of *"pulp disease"* is often used. However, this term is not appropriate since it does not include all conditions that occur within the pulp space (*i.e.* within the root canal system) and because not all teeth will have pulps [6]. There are several situations where teeth do not have pulps:

1. As a result of infection where the bacteria initially cause the pulp to necrose and then they remove the necrotic tissue so the root canal system becomes *"pulpless and infected"*. This occurs within a relatively short period of time ranging from 1-2 months [35]. Many classifications, and hence clinicians, describe these teeth as having a *"necrotic pulp"* but this is not accurate since there is no necrotic pulp tissue remaining within the root canal system. In addition, a necrotic pulp, in itself (that is, when not infected) does not cause apical periodontitis. Therefore, the tooth does not require any intervention such as RCT. Apical periodontitis only develops when the necrotic pulp becomes infected, and RCT will then be indicated [36-40].

It is also worth noting that many clinicians use the term *"necrotic tooth"* to describe a tooth that has a pulpless and infected root canal system which is even more inappropriate since the *tooth* is not *"necrotic";* it still has a viable periodontal ligament which is an integral part of the tooth organ, and such teeth can function normally. The only teeth that can truly be called *"necrotic teeth"* will be those that have been extracted!!

2. When the pulp has been removed by a dentist as part of RCT. If the treatment is not yet completed, then the tooth should be diagnosed as having had "*incomplete root canal treatment*". In such a situation, the root canal system may still be infected or it may not be infected, depending on the effectiveness of the initial treatment.

3. When the tooth has had a root canal filling placed at some earlier time. The root canal system may or may not be infected. If it is infected, then the infection may be either a continuing infection or a new infection.

Therefore, when considering all of the conditions that can affect the pulp and the root canal system, it is more appropriate to use the general heading of *"pulp and root canal conditions"* [6] and this term will be used in this review.

"Peri-radicular" versus "Periapical"?

In the literature, the general heading of *"periapical disease"* is often used. However, the periapical region is only one small part of the tissues that surround the entire tooth root, all of which can respond or react to pulp inflammation and infection of the root canal system–that is, such responses are not restricted to the periapical region [6]. An infected root canal system can cause inflammation anywhere along the root surface due to the effects of lateral canals, perforations, internal resorption, trauma (such as luxations, avulsion), *etc.* In addition, various forms of external tooth resorption are unrelated to the pulp or root canal condition and they can manifest anywhere along the root surface [7, 41]. Hence, in order to have a comprehensive classification, it is more appropriate to use the term *"peri-radicular conditions"* as a heading for the group of conditions that can affect these tissues [6]. The term *"periapical conditions"* should only be used when specifically when referring to conditions that only affect the periapical tissues.

"Acute" and "Chronic" versus "Symptomatic" and "Asymptomatic"?

The use of the terms *"acute"* and *"chronic"* in a clinical sense is standard practice in medicine and dentistry and they are used without implying the histological responses that are occurring in the tissues [2, 3, 6]. For example, the terms "acute appendicitis" or "chronic appendicitis" are used by medical practitioners, depending on the severity of the presenting condition and the time that the symptoms and signs have been present. Likewise in dentistry, terms such as chronic periodontal disease, acute ulcerative gingivitis, acute pericoronitis, *etc.* are used. In endodontics, *"acute"* and *"chronic"* have been used for many years for pulp and peri-radicular conditions but in more recent times, a tendency towards not using them has emerged. Instead of these

terms, the words *"symptomatic"* and *"asymptomatic"* have become popular despite them being inappropriate. As stated by Gutmann *et al.* [12], there is *"little, if any, support in the historic or contemporary peer-reviewed literature for the use of these terms"*, and they *"have slowly crept into usage with little scientific basis for their applications or meanings"*. Unfortunately, the slow creep has developed into a rapid and almost universal acceptance of these terms, but still without any scientific justification.

The often-stated reason for not using *"acute"* and *"chronic"* is that these are *"histological terms"* and therefore they should not be used clinically. However, as stated above, these terms are standard in medicine and all other specialties of dentistry and therefore it is not an acceptable argument to say that they should not be used clinically.

Whenever words are being used to describe something, they need to be defined so their meaning and their intent of use is clear. Medical dictionaries define *"acute"* and *"chronic"* for clinical use as follows:

Acute

- "Of or relating to a disease or a condition with a rapid onset and a short, severe course" [6, 42].
- "Relating to an illness that has a rapid onset and follows a short but severe course" [43]].
- "Sudden onset of sharpness or severity" [44].

Chronic

- "Of long duration, used for a disease of slow progress and long continuance" [6, 45].
- "Continuing a long time or recurring frequently" and "relating to an illness or medical condition that is characterized by long duration or frequent recurrence" [46].
- "A condition that is continuing or occurring repeatedly or for a prolonged period of time" [47].

The pain associated with acute conditions is usually moderate to severe and the patient has typically presented to the practitioner seeking relief of the symptoms. In contrast, the pain associated with chronic conditions is usually mild and only occasionally present (*i.e.* it "*comes and goes*"). The patient may only mention the occasional pain or discomfort during a routine examination.

In contrast, medical dictionaries distinguish between *"symptomatic"* and *"asymptomatic"* purely on the presence or absence of the symptoms of a disease [48, 49] with no indication of the severity of the pain, the time of onset, how long the pain has been present and whether it is occasional, recurrent or constant. Hence, the information provided by these terms is quite basic and should be considered as inadequate to be able to formulate an accurate and useful diagnosis.

Hence, it is possible, definitely acceptable, and common practice in medicine and dentistry to use *"acute"* and *"chronic"* as clinical terms providing the appropriate definitions are understood and applied. These terms are also very appropriate for conditions of the pulp, root canal and peri-radicular tissues. Hence, they will be used in this review and clinicians are encouraged to use them in order to be consistent with other fields of dentistry and medicine.

A study by Bestall *et al.* [28] reported that dentists were statistically significantly less likely to treat pulp and periapical conditions labelled as "*asymptomatic"* compared to the same condition when designated as *"chronic"* (for example *"asymptomatic apical periodontitis"* compared to *"chronic apical periodontitis"*). There were also differences when conditions were labeled as *"acute"* rather than *"symptomatic"*. The study has extremely important implications because many teeth with potentially serious pulp, root canal and peri-radicular conditions would be left untreated by many dentists because of a lack of symptoms rather than treating the tooth to resolve the condition, or to remove the disease, and ultimately to improve the patient's health. The authors concluded that "diagnostic terminology is important in directing appropriate management of diseases rather than just managing symptoms" [28]. Similar findings were reported in a study of medical practitioners who were surveyed about their treatment recommendations for chronic malaria compared to asymptomatic malaria [50]. A statistically significantly higher number of practitioners would treat cases labeled as chronic malaria compared to those who would treat cases labelled as asymptomatic.

These studies clearly indicate that "words do matter" and the appropriate diagnostic terminology should always be used [28-32].

Furthermore, it is interesting and significant to note that the authors of the position paper regarding pulp and root canal conditions [11] for the AAE Consensus conference did not recommend the use of the terms *"symptomatic"* and *"asymptomatic"*, yet the AAE's recommended terminology from this conference did recommend these terms [9]. The latter recommendation appears to have been based on an online survey of the conference participants who were asked to answer a series of questions in an attempt to gain consensus for various situations [51]. *"Consensus"* for each question or scenario was considered to have been reached when 51% or more of respondents agreed or disagreed. However, *"consensus"* can have several meanings according to various dictionaries some examples are:

- The Cambridge Dictionary: "*a generally accepted opinion or decision among a group of people*" [52] and "*a situation in which all the people in a group agree about something*" [53].
- The Miriam-Webster Dictionary: "*general agreement, unanimity*" [54].
- The Collins Dictionary: "*general agreement among a group of people*" [55].
- The Oxford Dictionary: "*an opinion that all members of a group agree with*" [56].

All of the above definitions clearly state that either complete or general agreement is required in order to have true consensus. The 51% used by the AAE [51] is barely a majority, and hence it is not indicative of true consensus. Discussions during the conference also clearly indicated that there was no scenario that had complete or even general agreement. Therefore, the results of the survey should not have been used to claim "*consensus*" plus they should not have been used to develop the AAE's recommended classification [9], particularly when the final terms were not consistent with those proposed in the position papers that had been written by invited panels of experts who researched the literature to make their recommendations [10-12].

In addition to the above, some of the questions in the survey [51] were not well constructed and did not provide sufficient, or appropriate, choices of answers. Only a single response was possible and the response could only be "*strongly agree*", "*agree*", "*disagree*" or "*strongly disagree*". In addition, there was no opportunity for respondents to provide comments about each question and its possible answers. As an example of a poor question with restricted possible answers, participants were asked whether "*irreversible pulpitis should be further described as symptomatic or asymptomatic*". Consensus, according to the AAE criteria, was reached but with only 57% agreement which implies 43% disagreement. However, there was no option for participants to provide their preference for further describing irreversible pulpitis as chronic or acute-hence, the restricted answer options likely influenced the responses, and consensus was barely achieved. There were also inconsistent answers to questions involving the use of the terms "*symptomatic*", "*asymptomatic*", "*acute*" and "*chronic*"; for example, 63% disagreed that "*acute*" and "*chronic*" were preferred over "*symptomatic*" and "*asymptomatic*" for apical periodontitis yet 83% agreed that "*acute*" and "*chronic*" should be used for apical abscesses. Some questions about specific conditions had high consensus; for example, did two conditions (hyperplastic pulpitis/pulp polyp and internal resorption) represent pulp disease with both recording 84% agreement. However, these conditions were not included in the AAE's final recommended classification despite having been included in the recommended classification in the position paper about pulp and root canal conditions [11]. Interestingly, 91% of participants agreed that diagnostic terms for pulp and periapical diseases should be biologically based, yet the final recommendations [9] did not follow this approach. Further analysis of the survey results show other inconsistencies which are not supported by clinical or histological findings, but are based solely on opinions. Overall, the AAE classification [9] appears to have been based more on the results of the survey of personal opinions rather than on the position papers that had been written by invited panels of experts who researched the literature to make their recommendations [10-12].

Purposes and requirements of a classification

Establishing a diagnosis should always be the first stage of managing any condition or disease. In addition, in order to effectively manage the condition or disease, an accurate diagnosis is imperative as this will determine the various options that may be available for managing the particular condition or disease [2, 3, 6, 7]. In medicine and dentistry, there are many various conditions that can affect a single tissue or organ. Hence, it is essential that a diagnostic classification should include all of the different conditions that can possibly occur.

A diagnosis can be considered as a few words summarising a particular combination of the patient's symptoms, the clinical signs, radiographic observations and the results of various diagnostic tests [2, 3, 6, 7]. Since only a few words are used to summarise all this information and to diagnose a condition, it is essential to have a clear, simple and comprehensive classification of all related or similar conditions that can occur in each tissue or organ. Examples of this in dentistry include the classifications of pulp, root canal and peri-radicular conditions, the classification of periodontal conditions, and the classification of the various types of tooth resorption.

When developing a classification, the physiological and/or pathological changes that have occurred in the tissues and how these develop and progress over time must be understood. Furthermore, all classifications of dental and medical conditions should also satisfy the following criteria [2, 3, 6, 7] they should be:

1. **Possible to use in a clinical setting:** that is, the diagnosis can be determined based on the information gained from the patient's symptoms, clinical signs, tests, radiographs, *etc.*
2. **Meaningful**: that is, the various conditions reflect what is happening, or has already happened, in the involved tissues.
3. **Useful**: that is, the diagnosis directs the clinician to the management options for the particular condition.
4. **Clear**: that is, the diagnosis and terminology used can be understood by all relevant clinicians.

5. **Universal**: that is, the classification should be used throughout the world to standardise terminology and allow effective communication amongst clinicians, educators, students and researchers.

When considering the "Universal" criterion above, the World Health Organisation [4] has recommended that diagnostic classifications should enable easy storage, retrieval and analysis of health information for evidenced-based decision-making, to facilitate sharing and comparing of health information between hospitals, regions, settings and countries, and to allow data comparisons in the same location across different time periods.

The various classifications of pulp, root canal and peri-radicular tissues that have been published in the literature and those used by many clinicians have, unfortunately, not been developed according to the above basic principles. As stated above, there is very little consistency between the published classifications and many different terms have been used for the same conditions [1, 2, 8, 11]. Hence, it is timely and essential to reconsider the classifications of pulp, root canal and peri-radicular conditions to ensure they follow the above principles.

Developing a classification

Scarpelli *et al.* [5] have outlined that a classification of diseases and/or conditions should be based on one or more of the overarching approaches listed below. Any single disease or condition may fall within one or more of these types of classifications. The suggested approaches are to classify the diseases/conditions according to the relevant:

(1) Topography – by bodily region or system;
(2) Anatomy – by organ or tissue;
(3) Physiology – by function or effect;
(4) Pathology – by the nature of the disease processes;
(5) Aetiology – by the cause(s) of the diseases/conditions;
(6) Juristic – according to the legal circumstances of death, especially when it is sudden;
(7) Epidemiology – by incidence, distribution and control of the disorders; or
(8) Statistics – by analysis of the incidence and prevalence rate of the diseases/conditions.

Since pulp, root canal and peri-radicular conditions must involve teeth, the first two approaches (topography and anatomy) are unnecessary when developing a classification for these conditions. The juristic, epidemiology and statistics approaches are not relevant to clinical dentistry and can be ignored when developing classifications for pulp, root canal and peri-radicular conditions.

The fifth approach (aetiology) is not relevant for a classification of pulp, root canal and peri-radicular conditions since the causes of pathological changes within these tissues are all similar in that bacteria are usually involved [35-40, 57]. The entry pathway by which bacteria reach the pulp and root canal system may vary with the most common pathways being caries, cracks, fractures, restorations breaking down [58, 59]. Other pathways may be related to periodontal diseases and traumatic dental injuries. However, the pathological changes within the pulp and the root canal system follow the same course and therefore attempting to classify these conditions according to aetiology is not relevant, nor is it feasible. Most peri-radicular (and especially periapical) conditions develop because of bacteria being present within the root canal system [35-40, 60-63] and hence, again, classifying them according to aetiology is not relevant. Some peri-radicular conditions, such as several types of external resorption, have different aetiologies but attempts to classify them according to aetiology has led to inadequate classifications [7, 41]. The important aspect about aetiology is to ensure that it is assessed during a clinical examination so the cause of the disease (for example, the pathway of entry of the bacteria) can be removed as the initial part of treating the tooth [58, 59].

The third and fourth approaches to developing classifications listed above (physiology and pathology, respectively), can, and should, be used to develop classifications for pulp, root canal and peri-radicular conditions since these conditions are either a physiological response (for example, pulp canal calcification following trauma to a tooth) or a result of a pathological process (for example, pulpitis which progresses to pulp necrosis with infection and then the root canal system becomes pulpless and infected, which in turn can lead to various forms of apical periodontitis).

Using the physiological and pathological approaches to develop a classification is consistent with the second requirement for a classification [2, 3, 6, 7], as listed above; that is, it should be "meaningful" by reflecting the various conditions that are present or have already occurred, within the pulp, root canal and peri-radicular tissues. Hence, it is essential that clinicians understand the functions of the pulp, root canal and/or peri-radicular tissues and how the various conditions that affect them develop and progress over time if untreated.

Diagnosis is not only about assessing disease and abnormal conditions as it also includes the assessment of the health of the involved tissues [2, 3, 6]. Hence, a comprehensive classification should also include a category for tissues that are free of any disease or other changes. For the dental pulp, the term *"clinically normal pulp"* is recommended rather than *"normal"* or *"healthy"* pulp [2, 3, 6] since it is not possible to clinically assess a pulp as being entirely normal or healthy, particularly in a tooth that has had previous caries, a restoration, trauma, *etc.* as such a pulp may

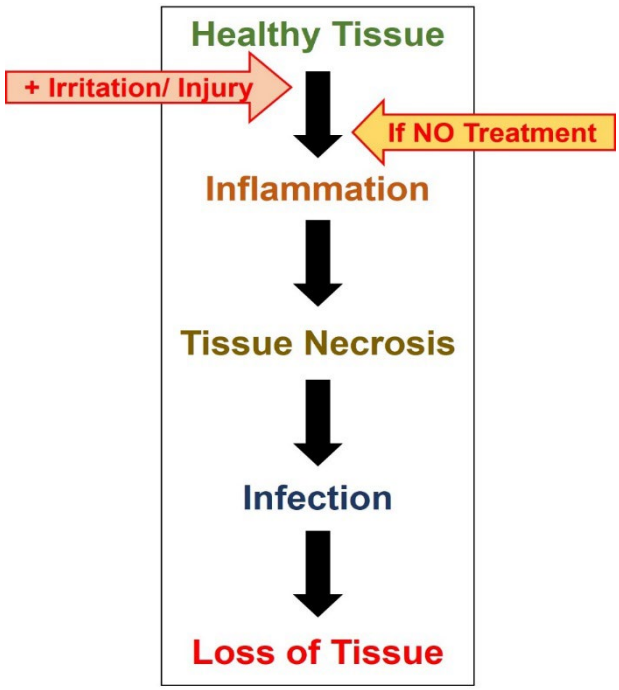

Figure 1. The stages of disease progression when any tissue of the body is irritated or injured and no treatment is provided (adapted from Abbott [6, 61-63] [6, 61-63])

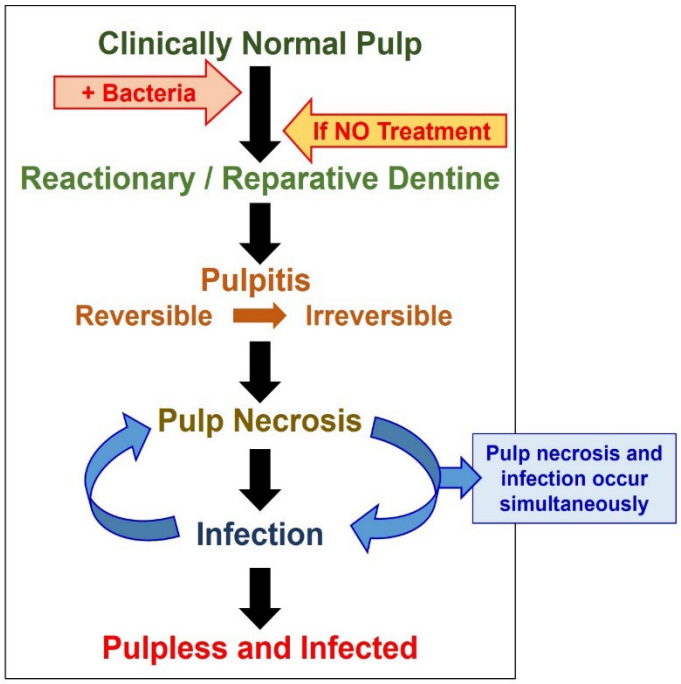

Figure 2. The stages of disease progression within the pulp and root canal system when a tooth is subject to bacterial invasion and no treatment is provided (adapted from Abbott [60-63] [6, 61-63])

be somewhat compromised histologically (for example, there may be some fibrosis) but it does not show any clinical signs of such changes. Similarly, the terms *"clinically normal periapical tissues"* and/or *"clinically normal peri-radicular tissues"* should be used where the clinical and radiographic examinations do not reveal any abnormalities within these tissues [3, 6, 61-63] since it is well-known that clinical and radiographic signs may be absent in the early stages of apical periodontitis, apical abscesses [60] and various types of resorption [7]. Using the term *"clinically normal"* recognises the limitations of the clinical examination and tests as well as the limitations of radiographs in being able to definitively determine the absence or presence of changes in the pulp, root canal and peri-radicular tissues [2, 3, 6, 61-63].

Changes, responses and reactions within the pulp, root canal and peri-radicular tissues

As discussed above, one of the criteria for a classification of conditions is that it should reflect what is happening in the tissues [2, 3, 6, 7]. In addition, a classification should be based on the physiological and/or pathological changes that occur [5]. Hence, it is important to understand all of the changes that may occur within the respective tissues. The changes in the pulp and root canal system followed by the changes in the peri-radicular tissues are outlined below. These changes in tissue conditions are the basis for the classifications published by Abbott [3, 6, 61-63] and Abbott and Yu [2].

- **Changes within the pulp and root canal system**

The first reaction whenever any tissue in the body is injured, irritated or stimulated will be inflammation (Figure 1). If no treatment is provided, or if the stimulus or irritating factor is not removed, the inflammation will persist and the tissue will typically necrose and become infected. Over time the necrotic tissue will be lost as the bacteria digest and remove it [6, 35, 64] (Figure 1). A typical example of this progressive reaction to injury and bacterial invasion is gangrene in the extremities of the body, such as the toes and fingers.

Pulp irritation commences once bacteria and/or their endotoxins reach the dentine. The dentinal tubules provide pathways for the irritant to stimulate the pulp. The pulp is a unique tissue in that can form hard tissue (*i.e.* reactionary, reparative and secondary dentine), which will be its first reaction to any irritation (Figure 2). The formation of reactionary or reparative dentine is the pulp's attempt to wall itself off from the irritant bacteria and/or their by-products. However, the pulp cannot maintain this function indefinitely and, if the bacteria are not removed and the pathway for their entry remains (*i.e.* no dental treatment is done), the pulp reacts in a similar manner to other soft tissues and becomes inflamed; that is, pulpitis develops [6, 57, 64, 65] (Figure 2).

Once inflammation develops, the pulp reacts in the same way as other soft tissues in the body react to the constant presence of bacteria; that is, the inflammation progresses and spreads

throughout the pulp. It then subsequently dies (necroses) and becomes infected. In teeth where the bacteria enter *via* a coronal pathway (such as caries, cracks or restoration breakdown), the processes of pulp necrosis and infection occur simultaneously as "bacterial front" progressively moves from the coronal pulp chamber through the root canal system towards the apical foramen/foramina. The pulp tissue apical to the "bacterial front" will be irreversibly inflamed whilst coronal to this "front", the tissue will be necrotic and infected. Once the entire pulp becomes necrotic and infected, and within a very short time, the necrotic pulp tissue will be digested and removed by the bacteria so the root canal system becomes pulpless and infected. This was reported by Jansson *et al.* [35] who exposed monkey premolar pulps, severed them at the apical foramen, left the tissue within the canals and then placed some plaque from the animal's teeth to infect the canals with oral bacteria. The cavities were left unrestored for 10-14 days to ensure the root canal systems were infected. One group of teeth had zinc oxide-eugenol (ZO-E) temporary restorations placed and teeth in the other group were left open until the animals were sacrificed at various time intervals of up to ten months. Histological examination revealed that the root canals in the "closed" teeth (*i.e.* those with ZO-E restorations) became pulpless within one month whilst root canals in the "open" teeth (*i.e.* no temporary restoration) became pulpless within two months [35]. This process by which an infected root canal system becomes pulpless is similar to the process of gangrene in the body's extremities [6].

From a clinical diagnostic perspective, the first stage of pulp disease is inflammation which is known as pulpitis since reparative or reactionary dentine formation cannot be assessed clinically. There are two major stages of pulpitis they are known as reversible and irreversible pulpitis and each stage can be chronic or acute [2].

Reversible pulpitis is considered as an early or less extensive form of inflammation, which usually only involves a small area of the pulp where the dentinal tubules are communicating with the source of the bacteria (*e.g.* the carious defect, the crack, the region where the restoration is breaking down, *etc.*). Typically, the symptoms are mild and the clinician's judgement is that conservative treatment (such as caries removal, pulp capping, partial pulpotomy, *etc.*), rather than RCT is likely to resolve the inflammation [2, 3].

Many patients with reversible pulpitis do not present to a dentist for treatment especially if it is chronic reversible pulpitis because the symptoms are very mild and/or only occasionally present. Hence, the pulp inflammation continues developing and spreads throughout the pulp to become irreversible pulpitis. This is a more advanced and extensive inflammatory process, typically involving a greater amount of the pulp. Initially, it will usually be chronic irreversible pulpitis which may be present for some time, depending on the patient's perception of pain and their willingness or otherwise to seek treatment. Over time, it will become acute irreversible pulpitis when the irritation increases or reaches a specific threshold [2, 3].

Some patients with irreversible pulpitis may not seek treatment as the pain may not be severe enough for them, or it may subside [66] when the pulp starts to necrose. The pulp necrosis will initially just involve part of the pulp that communicates with the irritant such as within a pulp horn where the dentine tubules communicate with the caries lesion. However, the necrosis will progressively spread apically to involve the entire pulp as the bacterial front moves throughout the pulp [2, 3]. During the period when necrotic and infected pulp tissue exists coronal to the bacterial front (as mentioned above) and irreversible pulpitis is present apical to the bacterial front, the tooth should be classified as having "pulp necrobiosis". Grossman proposed this term [67, 68] which is a very good and descriptive term since it implies a mixture of necrotic (necro-) and live (bio-) tissue. This necrobiosis stage of the pulp's disease process is usually relatively; short typically only for several hours or a few days. This is because the bacterial front usually moves relatively quickly towards the apical foramen/foramina. Once the front reaches the apical foramen/foramina, the entire pulp will be necrotic [2, 3]. Based on the study by Jansson *et al.* [35] and the conditions within the particular tooth (such as the presence or absence of a restoration, caries, *etc.*) plus the conditions within the root canal system (such as the anaerobic/aerobic environment), the tooth will very likely become pulpless and infected within 1-2 months [35]. Figure 3 demonstrates the progression of the pulp and root canal system through these various stages of their disease processes.

Teeth that have had previous endodontic treatment can either remain infected or they can become infected again over time, and both situations can lead to a range of peri-radicular conditions [69]. Continuation of an infection within the root canal system is often related to inadequate endodontic treatment whilst a new infection will be a result of further caries, a crack, a fracture or breakdown of the restoration in the tooth. These are the exact same pathways by which bacteria enter teeth to cause pulp disease, as discussed above [58]. Previous endodontic treatment can include a pulp cap (indirect or direct), a partial pulpotomy, a pulpotomy, a partial pulpectomy or pulpectomy followed by a root canal filling. The primary, and most important, aspect to assess when examining teeth with previous endodontic treatment is whether there are any signs of infection of the root canal system [2, 3]. These signs usually manifest

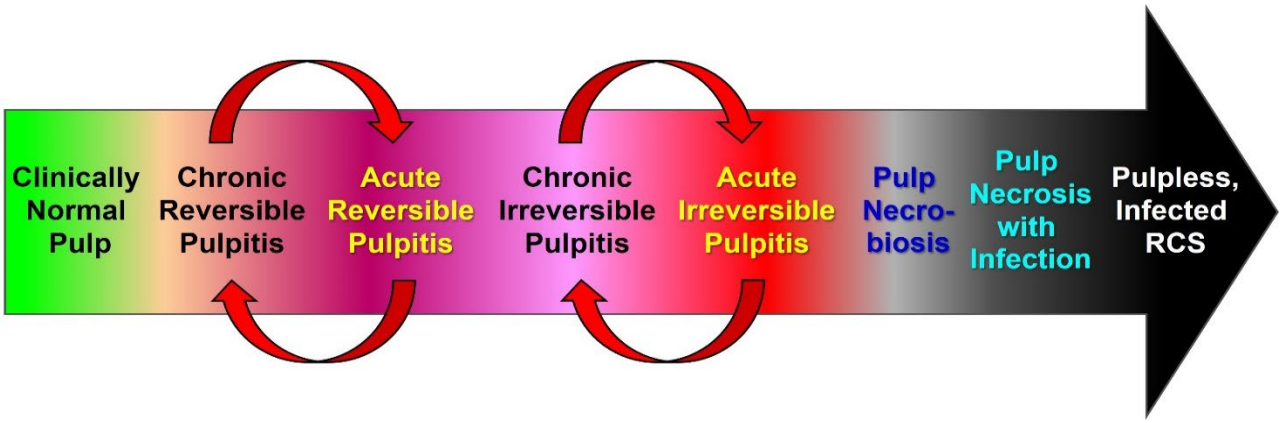

Figure 3. Schematic representation of the progression of pulp and root canal conditions through the various stages of the disease process; root canal system (RCS), root canal treatment (RCT) (reproduced from Abbott [3] with permission)

as peri-radicular conditions and the peri-radicular responses to infections of teeth with previous endodontic treatment are the same as those that occur with teeth infected for the first time, as outlined below; that is, apical periodontitis which may be followed by an apical abscess, extra-radicular infection, periapical cyst, *etc.* [60-63].

Over time, pulps may develop physiological changes such as pulp atrophy, or pulp canal calcification. The pulp can also have degenerative changes such as pulp hyperplasia (sometimes called pulp polyp), and various forms of internal resorption [2, 3, 6, 7]. These conditions may be a result of various factors such as ageing (*i.e.* atrophy, pulp canal calcification), irritation from caries, cracks, fractures or restorations breaking down (*i.e.* pulp canal calcification, pulp hyperplasia, internal surface resorption, internal inflammatory resorption), or specific events such as trauma to the tooth (*i.e.* pulp canal calcification, internal inflammatory resorption). In some cases, there may be no obvious cause (such as internal replacement resorption). These physiological and degenerative conditions are usually only noted through radiographic examination of the teeth and therefore some clinicians consider them to just be radiographic observations rather than true diagnoses of specific conditions or changes of the pulp. However, they do represent changes that have been reported in the literature to occur within the pulp and/or root canal system and therefore they should be considered as separate conditions. It is also essential to assess the state of the pulp or the root canal system when any of these physiological or degenerative conditions are noted [2, 3, 6]. As an example, the pulp or root canal system of a tooth with pulp canal calcification may have any of the stages of the pulp/root canal disease process that are outlined above; it may have reversible pulpitis (chronic or acute), irreversible pulpitis (chronic or acute), or it may be pulpless and infected, *etc.*

Clinicians must also recognise and understand that the pulp and root canal status are dynamic. The various conditions, and especially diseases, are progressive and they move through the various stages of the overall pulp/root canal disease processes. None of these conditions occur instantaneously or spontaneously and they will always progress from one condition to the next over time if not treated (Figures 2, 3). Hence, the symptoms that a patient describes on presentation to a dentist will depend on the current condition or stage of the disease process at the time of assessment.

- **Changes within the peri-radicular tissues**

The first response of the peri-radicular tissues to irritation will be inflammation which is similar to other tissues in the body (Figure 4). If no treatment is provided, these tissues will progress through various conditions. The tissues within the periapical region are the most commonly affected by changes within the pulp and/or root canal system, and therefore the periapical region should be considered as a dynamic interface between the pulp/root canal system and the body's immune or defense system [60-63].

Most peri-radicular (and especially periapical) conditions are a direct result of changes within the pulp and the root canal system [60-63]. The most common irritants leading to peri-radicular changes will be the presence of bacteria and their endotoxins within an infected root canal system [60]. The relationship between an infected root canal system and the periapical tissue responses has been widely investigated [35-40, 60-63, 70-78] and is demonstrated in Figure 5. Clinicians should understand that the bacteria are not usually present within the periapical or peri-radicular tissues themselves unless there is an abscess, cellulitis, or an extra-radicular infection. In some patients, when there is no treatment of an infected root canal system, some bacteria from the root canal system may enter the

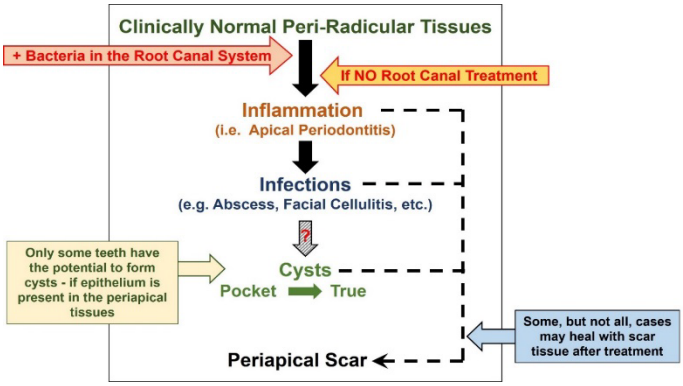

Figure 4. The progression of the peri-radicular tissues through different conditions when the tooth has an infected root canal system and no treatment is provided except where indicated for periapical scars which sometimes form after root canal treatment or periapical surgery (reproduced from Abbott [6] with permission)

periapical and/or peri-radicular tissues *via* the apical foramen or other pathways (such as lateral canals, dentinal tubules with cementum defects caused by external inflammatory resorption, *etc.*). In these cases, the periapical and/or peri-radicular tissues can become infected which leads to the formation of an abscess, facial cellulitis, or an extra-radicular infection [60-63, 70-78].

If no treatment is provided to the tooth, the peri-radicular inflammation will continue and progress so that, over time, a larger periapical or peri-radicular radiolucency will be evident radiographically. A periapical cyst may form in a very small number of cases but this can only occur if there is epithelium present within the periapical tissues. In a study by Nair *et al.* [77] where extracted teeth and their periapical tissues were examined histologically and with transmission electron microscopy, about 50% of teeth had such epithelium and therefore these teeth were the only ones that had the potential to form periapical cysts. However, only 15% did actually form cysts. Nair *et al.* [77] also described two forms of cysts with the periapical pocket cyst likely forming first and then progressing over time to become a periapical true cyst. Nair *et al.* [77] defined a pocket cyst as an epithelial-lined cavity that has an opening which communicates with the infected root canal system whereas a true cyst has no such opening and the cyst is completely lined by epithelium. True cysts are also self-propagating and will not respond to RCT. True cysts will need to be surgically removed whereas it is believed that a pocket cyst may heal following disinfection of the root canal system *via* standard RCT [60-63, 77].

Many years ago, several studies [35-40] reported that periapical inflammation (that is, apical periodontitis) only occurred when the root canal system was infected with bacteria.

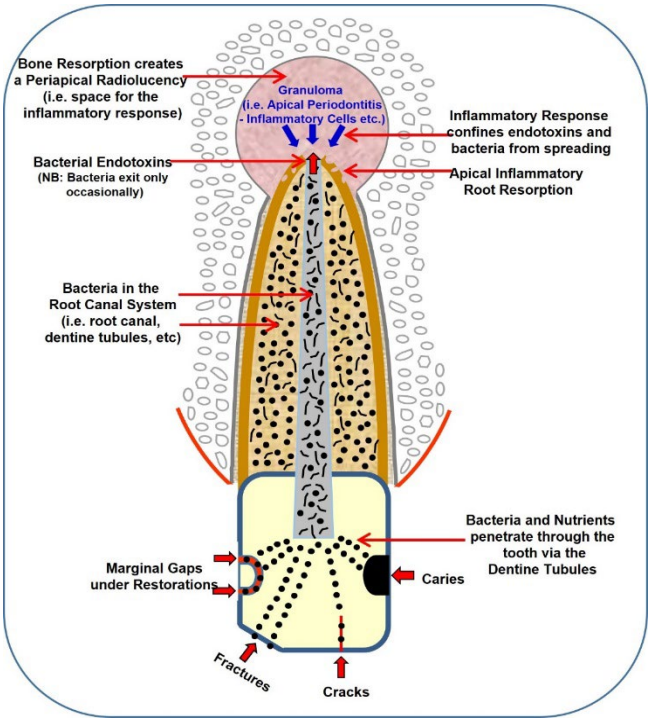

Figure 5. Schematic diagram showing the common pathways of entry of bacteria into teeth to cause an infected root canal system and the resultant periapical response (reproduced from Dashper *et al.* [64] with permission)

However; more recent studies have shown that the inflammatory response can develop earlier when the pulp is inflamed. In these cases, the periapical inflammation is considered to be an extension of the pulp's inflammatory response since there is only one direction in which the inflammation can spread - that is, apically and out into the periapical tissues [79]. Hence, when teeth have any form of reversible or irreversible pulpitis, they may also have clinical and/or radiographic signs of apical periodontitis – such as widened periodontal ligament spaces, tenderness to percussion, or condensing osteitis [2, 3, 6, 79-81].

Accidental trauma can also damage the periapical tissues. Concussion, subluxation, lateral luxation, extrusion, intrusion and avulsion all damage the periodontal ligament and/or cementum [82, 83]. If the injury is mild (*e.g.* concussion, subluxation) and appropriate treatment is provided, then the tissues will usually heal in a short period of time [83]. These injuries are "short-term insults" to the peri-radicular tissues and healing is expected once the injury has been appropriately managed (Figure 6). However, injuries that cause more severe damage (such as avulsion, intrusion, lateral luxation and extrusion) often result in various forms of tooth resorption particularly external inflammatory resorption and external replacement resorption [83]. Other forms of tooth resorption can

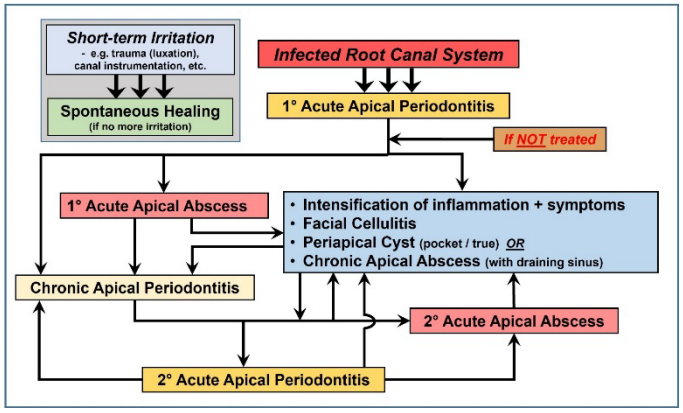

Figure 6. The interactions between the different periapical conditions that are a result of an infected root canal system. This illustrates the dynamic nature of these periapical conditions (adapted from Abbott [6, 61-63]) (1^0: Primary; 2^0: Secondary)

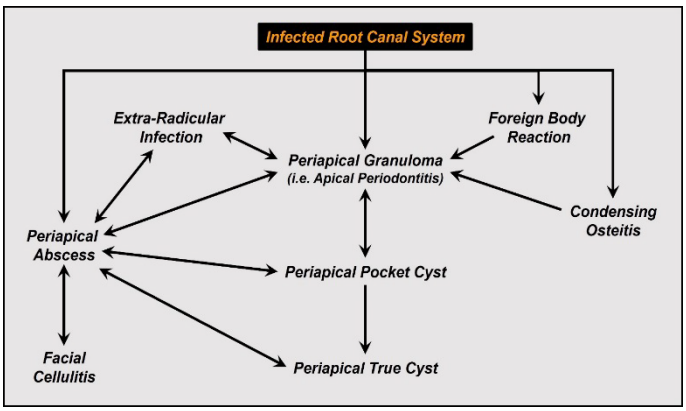

Figure 7. Schematic representation of the interactions between the various forms of periapical conditions arising from an infected root canal system (adapted from Abbott [6, 61-63])

also occur with various other causes and they will have their unique clinical and radiographic features (see Abbott and Lin [7] for a comprehensive review and classification of tooth resorption).

Traumatic occlusion is another cause of peri-radicular inflammation without infection of the root canal system [84, 85]. A premature contact or an occlusal interference associated with traumatic occlusion are continuous, constant irritations to the periodontal ligament which will result in peri-radicular inflammation.

The periapical region should be considered as the interface between the root canal system (or the pulp, if it is present) and the body's defense (or immune) system [2, 60-63]. Periapical and peri-radicular conditions will remain as long as the root canal system stays infected [60]. The response of the peri-radicular tissues is dynamic and it can change over time. There are several possibilities that may be present, but not all at the same time [60-63]. That is, the response can change, and this depends on the interactions that occur between the bacteria in the root canal system and the host's defense system [60-63]. The former is largely dependent on the type of bacteria present, their virulence, the local conditions (for example, anaerobic *versus* aerobic), and the availability of nutrients. The host's response is also dependent on many factors such as the patient's general state of health and how effective their immune system is. All of these tooth and patient factors can change over time, and thus the nature of the peri-radicular response can also change over time. Figure 7 demonstrates the potential interactions between the different peri-radicular conditions that can occur [60-63].

Figure 6 demonstrates the progression of the various peri-radicular conditions when no treatment has been provided [3, 60-63]. Patients may present to a dentist for treatment at any stage of this disease process but the most common time will be when there is an acute condition present since the patient will usually be experiencing pain, which is likely to be severe. However, some patients may not present immediately upon noting pain, and the pain may subside as the acute inflammation becomes chronic. This then allows time for further progression of the disease process. A common scenario would be for a patient to have a short episode of pain with primary acute apical periodontitis which then progresses to chronic apical periodontitis, during which time there is little or only occasional mild pain and a periapical radiolucency develops [6]. The patient may then have occasional episodes of pain when bacteria exit the canal and enter the periapical tissues to cause secondary acute apical periodontitis but the body's defense system may control this and the periapical tissues return to a state of chronic apical periodontitis. This cycle may occur several times before the pain eventually increases to the point where the patient seeks dental treatment [6]. Other scenarios are also possible such as when an acute apical abscess occurs and the patient notices the swelling and pain, or when a chronic apical abscess develops and the patient notices a draining sinus and seeks treatment [6].

Recommended classifications

The classifications recommended below meet all of the criteria discussed above [2, 3, 6, 7] for classifications of diseases and conditions of the human body that is, they are: *1*) possible to use clinically; *2*) meaningful; *3*) useful; *4*) clear; and *5*) universal. They also follow the Scarpelli *et al.* [5] approach of using the physiological and pathological changes within the tissues to develop the classifications, plus they meet the WHO criteria [4] for classifications of conditions as the data obtained when using these classifications can be easily stored, retrieved, analyzed for evidence-based decision-making, shared, and compared between health service providers across different locations.

- **Classification of pulp and root canal conditions**

Table 1 outlines the classification of pulp and root canal conditions that has been adapted from Abbott and Yu [2] and Abbott [3, 6, 61-63]. The concept behind this classification is to include all of the conditions that have been reported to occur in the pulp and root canal system. It also follows the general stages of disease progression that occur (Figure 1) with the terminology adapted to suit pulp and root canal conditions (Figures 2, 3). These conditions are grouped according to their general nature namely, healthy tissue (no signs of disease), inflammation (pulpitis), tissue necrosis and infection (pulp necrosis), tissue destruction (pulpless teeth), previously-treated teeth, and the physiological and degenerative changes that occur over time.

- **Classification of peri-radicular conditions**

Table 2 outlines the classification of peri-radicular conditions that has been adapted from Abbott [3, 6, 60-63]. The concept behind this classification is to recognise and include all of the conditions that have been reported to occur in the peri-radicular tissues. It also follows the general stages of progression for peri-radicular conditions (Figures 4, 6, 7). These conditions are grouped according to their general nature namely, healthy tissues (no signs of disease), inflammatory conditions, infections, cysts, periapical scars and external tooth resorption.

Comparison of classifications

Specific comparisons could be made between the many different classifications that have been published in various textbooks and journal articles, plus those recommended by various professional organisations. There is no doubt that there are numerous differences and deficiencies in the various classifications and such a discussion would be time consuming and unlikely to be helpful. Hence, the following discussion will be limited to comparisons of the above recommended classifications that have been developed according to strict criteria that recognise the tissue changes that occur with the AAE classifications which have become popular but were largely based on opinions rather than the tissue changes that occur and they did not follow the recommended criteria for the development of a classification.

- **Pulp and root canal conditions: The *"Abbott & Yu* classification" versus the *"AAE classification"***

The AAE classification of pulp and root canal conditions [9] has only seven conditions whereas the Abbott & Yu classification [2] has 17 conditions. The latter is based on changes that have been reported to occur within the pulp and the root canal system. Hence, the AAE classification must be considered to be quite

Table 1. Classification of the status of the pulp and root canal conditions adapted from Abbott and Yu [2] and Abbott [61-63]

No signs of disease		
Clinically Normal Pulp (Based on history, clinical examination, tests, radiographs, *etc.*)		
Pulpitis		*** Notes**
Reversible pulpitis	Acute *	Acute. Rapid onset and a short, severe course; moderate to severe pain; patient is seeking urgent relief of the symptoms. Chronic. Long-standing; no pain or only mild and occasional pain; patient not seeking urgent relief.
	Chronic *	
Irreversible pulpitis	Acute *	
	Chronic *	
Pulp necrosis		
Pulp necrobiosis (Part of pulp is necrotic & infected; the rest is irreversibly inflamed)		
Pulp necrosis with no signs of infection		
Necrotic and infected pulp		
Pulpless teeth		
• Pulpless and infected root canal system		
• Teeth with previous root canal treatment		
Root-filled with NO signs of infection		
Root-filled and infected		
Incomplete endodontic treatment* (*e.g.* treatment started elsewhere) *Note: Also specify whether the root canal system is infected or not		
Technical standard (based on the radiographic appearance)		Adequate Inadequate
Other problems: specify the problem: *e.g.* perforation, untreated canal(s), fractured instrument, silver point, *etc.*)		
Degenerative changes Note: MUST also specify the diagnosis/condition of the pulp or the root canal system**		
• **Pulp atrophy ****		
• **Pulp canal calcification ****		Partial Complete" (based on radiographic appearance)
• **Pulp hyperplasia (A form of pulpitis; hence MUST specify the pulp status also)**		
• **Internal resorption**		Internal surface resorption Internal inflammatory resorption Internal replacement resorption

Table 2. Classification of the status of the peri-radicular tissues -adapted from Abbott [61-63]

No signs of disease		
• **Clinically normal periapical / peri-radicular tissues** *(Based on the history, clinical examination, tests, radiographs, etc.)*		
Inflammatory conditions		
Apical periodontitis-	**Acute**	**Primary** *(i.e. no periapical radiolucency)*
		Secondary *(i.e. has a periapical radiolucency)*
		Chronic *(i.e. has a radiolucency)*
		Condensing osteitis *(i.e. radiopacity) (A form of chronic apical periodontitis)*
Foreign body reaction		
Infections		
• **Apical abscess**	**Acute**	**Primary** *(i.e. no periapical radiolucency)*
		Secondary *(i.e. has a peiapical radiolucency)*
		Chronic *(i.e. has a draining sinus)*
• **Facial cellulitis**		
• **Extra-radicular infection**		
Cysts		
• **Periapical pocket cyst**		
• **Periapical true cyst**		
Scar		
• **Periapical scar**		
External resorption:		
• **External surface resorption**		
• **External inflammatory resorption**		Lateral
		Apical
• **External replacement resorption**		
• **External invasive resorption**		
• **External pressure resorption**		
• **External orthodontic resorption**		
• **External physiological resorption**		
• **External idiopathic resorption**		

Table 3. A comparison of the Abbott & Yu [2] classification of pulp and root canal conditions with that of the AAE [9]. Conditions listed on the same horizontal line denote pulp or root canal conditions that present with the same symptoms, clinical signs, tests results and radiographic findings

Abbott & Yu (2007) [2]	**AAE (2009)** [9]	**Notes and Comparisons**
Clinically normal pulp	**Normal pulp**	The AAE term does not clearly indicate that this diagnosis is based on a clinical assessment and the pulp may not be completely normal.
Acute reversible pulpitis	**Reversible pulpitis**	The AAE classification does not account for acute (painful) or chronic (occasionally has symptoms) cases of reversible pulpitis.
Chronic reversible pulpitis		
Acute irreversible pulpitis	**Symptomatic irreversible pulpitis**	The AAE classification uses inappropriate terminology and focusses presentation on symptoms rather than the tissue changes in the pulp.
Chronic irreversible pulpitis	**Asymptomatic irreversible pulpitis**	
Pulp necrobiosis	-	***Not accounted for in the AAE classification.***
Pulp necrosis with infection	**Pulp necrosis**	The AAE classification does not account for the presence or absence of infection associated with pulp necrosis.
Pulp necrosis with no signs of infection		
Pulpless and infected root canal system	-	***Not accounted for in the AAE classification.***
Previous endodontic treatment with an infected root canal system	-	In the Abbott & Yu classification, the term *"Previous Endodontic Treatment"* includes all forms of pulp and/or /root canal treatment (*e.g.* pulp cap, partial pulpotomy, pulpotomy, partial pulpectomy, previous root canal filling, root canal treatment that has been commenced but not yet completed, *etc.*). The nature of the previous treatment should be clearly stated in the clinical notes as per the history, radiographic report, and clinical findings.
Previous endodontic treatment with no signs of infection		
	Previously treated	The AAE classification separates these two scenarios but neither category in the AAE classification accounts for the presence or absence of infection of the root canal system.
	Previously initiated treatment	
Pulp atrophy	-	Not accounted for in the AAE classification.
Pulp canal calcification	-	Not accounted for in the AAE classification.
Pulp hyperplasia	-	Not accounted for in the AAE classification.
Internal surface resorption	-	Not accounted for in the AAE classification.
Internal inflammatory resorption	-	Not accounted for in the AAE classification.
Internal replacement resorption	-	Not accounted for in the AAE classification.
17	7	Total Number of Conditions in Each Classification

Table 4. A comparison of the Abbott [63] classification of periapical/peri-radicular conditions with that of the AAE [9]. Conditions listed on the same horizontal line denote periapical/peri-radicular conditions that present with the same symptoms, clinical signs, tests results and radiographic findings.

Abbott (2004) [63]	AAE (2009) [9]	Notes and Comparisons
Clinically normal periapical tissues	**Normal apical tissues**	• The AAE term does not clearly indicate that this diagnosis is based on clinical and radiographic (with or without CBCT) assessments and the periapical/peri-radicular tissues may not be completely normal.
Primary acute apical periodontitis	**Symptomatic apical periodontitis**	• The AAE classification does not account for primary or secondary apical periodontitis, with primary conditions having no periapical radiolucency (indicating the first stage of the disease process, and usually very painful) whilst secondary conditions do have a periapical radiolucency indicating that a chronic condition (chronic apical periodontitis or a chronic apical abscess) has been present for some time prior to the acute exacerbation occurring. • The AAE classification also uses inappropriate terminology and focusses presentation on symptoms rather than focussing on the history and the tissue changes that are occurring in the periapical/peri-radicular tissues. • The AAE classification is inconsistent with its use of "acute" and "symptomatic" when comparing the terminology used for apical periodontitis and apical abscesses.
Secondary acute apical periodontitis		
Chronic apical periodontitis (Radiolucency)	**Asymptomatic apical periodontitis**	• The AAE classification uses inappropriate terminology and focusses on the patient presenting with or without symptoms rather than focussing on the history and the tissue changes that are occurring in the periapical/peri-radicular tissues. • The AAE classification is inconsistent with its use of the terms "chronic" and "asymptomatic" when comparing the terminology used for apical periodontitis and apical abscesses.
Condensing osteitis	**Condensing osteitis**	No difference between the two classifications for this condition.
Primary acute apical abscess	**Acute apical abscess**	• The AAE classification does not account for primary or secondary acute apical abscesses, with primary conditions having no periapical radiolucency (indicating the first stage of the infection process, and usually very painful) whilst secondary conditions do have a periapical radiolucency indicating that a chronic condition (chronic apical periodontitis or a chronic apical abscess) has been present for some time prior to the acute exacerbation occurring. • ***ALSO***: The AAE classification is inconsistent with its use of *"acute"* and *"symptomatic"* when comparing the terminology used for apical periodontitis and apical abscesses (and it is also inconsistent with terms used for pulp conditions).
Secondary acute apical abscess		
Chronic apical abscess	**Chronic apical abscess**	• ***No difference between the two classifications for this condition.*** • ***BUT: The AAE classification is inconsistent with its use of the terms "chronic" and "asymptomatic" when comparing the terminology*** used for apical periodontitis and apical abscesses (and it is also inconsistent with terms used for pulp conditions).
Facial cellulitis	-	Not accounted for in the AAE classification.
Extra-radicular infection	-	Not accounted for in the AAE classification.
Foreign body reaction	-	Not accounted for in the AAE classification.
Periapical pocket cyst	-	Not accounted for in the AAE classification.
Periapical true cyst	-	Not accounted for in the AAE classification.
Periapical Scar	-	Not accounted for in the AAE classification.
External Surface Resorption	-	Not accounted for in the AAE classification.
External inflammatory resorption	-	Not accounted for in the AAE classification.
External replacement resorption	-	Not accounted for in the AAE classification.
External invasive resorption	-	Not accounted for in the AAE classification.
External pressure resorption	-	Not accounted for in the AAE classification.
External orthodontic resorption	-	Not accounted for in the AAE classification.
Physiological resorption	-	Not accounted for in the AAE classification
Idiopathic resorption	-	Not accounted for in the AAE classification
22	**6**	Total number of conditions in each classification

deficient as it has a shortfall of 10 conditions that have all been reported to occur in teeth in many published research projects and case reports. Therefore, since these conditions can, and do, occur in teeth, they cannot, and must not, be ignored. Hence, they should all be included in a classification of pulp and root canal conditions. In addition, the AAE classification uses inappropriate terminology (as discussed above *i.e.* symptomatic, asymptomatic) which is also inconsistent with the accompanying AAE classification of periapical conditions. The missing conditions and other differences between these two classifications are demonstrated in Table 3.

- **Peri-radicular conditions: The *"Abbott classification"* versus the *"AAE classification"***

The AAE classification of peri-radicular conditions [9] has only ***six*** conditions which are notably limited to the periapical tissues only that is, the AAE classification does not include any conditions that affect other parts of the peri-radicular tissues. In contrast, the Abbott classification [63] has ***22*** conditions. The latter classification is based on changes that occur within the peri-radicular tissues, all of which have been reported in the literature, as discussed above. Therefore, they cannot, and must not, be ignored. Hence, they should be included in a classification of peri-radicular conditions. Hence, the AAE classification must be considered to be quite deficient as it has a shortfall of ***16*** conditions, in addition to using inappropriate and inconsistent terminology, as discussed above (*i.e.* symptomatic, asymptomatic, acute, chronic). The missing conditions and other differences between these two classifications are demonstrated in Table 4.

Conclusions

There is a lack of standard, and clinically useful, classifications of pulp, root canal and peri-radicular conditions within the dental profession, and especially within the published literature. This leads to confusion amongst clinicians and potential uncertainty regarding treatment. It also limits the ability to communicate effectively, to teach appropriately, and to compare data and research findings throughout the world. When developing a classification of tissue conditions or diseases, it is essential that the classification meets the recommended criteria so it can be used effectively in clinical, educational and research settings. It is also extremely important that correct and well-defined terminology is used since "words do matter". Popular terminology based on symptoms should be avoided as these have been proven to lead to many inappropriate treatment decisions. The only published classifications that truly address the criteria outlined above, and in particular, reflect the physiological and pathological changes that occur in the pulp, root canal and peri-radicular tissues are those developed by Abbott & Yu [2] (for pulp and root canal conditions see Table 1) and Abbott [63] (for peri-radicular conditions see Table 2). Hence, it is recommended that these classifications be adopted as the internationally-accepted classifications for future clinical use, as well as for educational, research and communication purposes.

Acknowledgment

The author would like to thank the many colleagues and students who have worked on the various research projects and assisted with the publications mentioned in this paper, as well as all those who have contributed through many discussions and correspondence over the years.

Conflict of interest

None.

Funding support

None.

References

1. Tronstad L. Clinical endodontics. A textbook. 2 ed. Stuttgart: Thieme; 1991. p. 76-8.
2. Abbott PV, Yu C. A clinical classification of the status of the pulp and the root canal system. Aust Dent J. 2007;52(1 Suppl):S17-31.
3. Abbott P. Examination and diagnosis of pulp, root canal, and periapical/peri-radicular conditions. Ingle's Endodontics 7: PMPH-USA Limited; 2019. p. 215-66.
4. Organization WH. International Statistical Classification of Diseases and related health problems: Alphabetical index: World Health Organization; 2004.
5. Scarpelli D, Robbins J, Robbins S, Burrows WJEB. Human disease. 2020.
6. Abbott PVJEa, guidelines ebc. Pulp, root canal, and periradicular conditions. 2022:85-116.
7. Abbott PV, Lin S. Tooth resorption-Part 2: A clinical classification. Dent Traumatol. 2022;38(4):267-85.
8. Glickman GN. AAE Consensus Conference on Diagnostic Terminology: background and perspectives. J Endod. 2009;35(12):1619-20.
9. AAE Consensus conference recommended diagnostic terminology. J Endod. 2009;35(12):1634.
10. Glickman GNJJoe. AAE Consensus Conference on Diagnostic Terminology: background and perspectives. 2009;35(12):1619-20.
11. Levin LG, Law AS, Holland GR, Abbott PV, Roda RS. Identify and define all diagnostic terms for pulpal health and disease states. J Endod. 2009;35(12):1645-57.
12. Gutmann JL, Baumgartner JC, Gluskin AH, Hartwell GR, Walton RE. Identify and define all diagnostic terms for

periapical/periradicular health and disease states. J Endod. 2009;35(12):1658-74.
13. Consensus Conference on Diagnostic Terminology List of Invitees. J Endod. 2009;35(12):1622-4.
14. Baume LJ. Diagnosis of diseases of the pulp. Oral Surg Oral Med Oral Pathol. 1970;29(1):102-16.
15. Seltzer S, Bender IB, Ziontz M. The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. Oral Surg Oral Med Oral Pathol. 1963;16:846-71 contd.
16. Garfunkel A, Sela J, Ulmansky M. Dental pulp pathosis. Clinicopathologic correlations based on 109 cases. Oral Surg Oral Med Oral Pathol. 1973;35(1):110-7.
17. Johnson RH, Dachi SF, Haley JV. Pulpal hyperemia-a correlation of clinical and histologic data from 706 teeth. J Am Dent Assoc. 1970;81(1):108-17.
18. Mulsen M, Sieraski SJEtteSLM. Histophysiology and diseases of thedental pulp. 1989:128-50.
19. Ricucci D, Loghin S, Siqueira JF, Jr. Correlation between clinical and histologic pulp diagnoses. J Endod. 2014;40(12):1932-9.
20. Highfield J. Diagnosis and classification of periodontal disease. Aust Dent J. 2009;54 Suppl 1:S11-26.
21. Suzuki JB. Diagnosis and classification of the periodontal diseases. Dent Clin North Am. 1988;32(2):195-216.
22. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol. 1999;4(1):1-6.
23. Armitage GC. Classifying periodontal diseases--a long-standing dilemma. Periodontol 2000. 2002;30:9-23.
24. Periodontology AAo. Proceedings of the world workshop in clinical periodontics: American Academy of Periodontology; 1989.
25. Chapple ILC, Mealey BL, Van Dyke TE, Bartold PM, Dommisch H, Eickholz P, Geisinger ML, Genco RJ, Glogauer M, Goldstein M, Griffin TJ, Holmstrup P, Johnson GK, Kapila Y, Lang NP, Meyle J, Murakami S, Plemons J, Romito GA, Shapira L, Tatakis DN, Teughels W, Trombelli L, Walter C, Wimmer G, Xenoudi P, Yoshie H. Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018;89 Suppl 1:S74-s84.
26. Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH, Flemmig TF, Garcia R, Giannobile WV, Graziani F, Greenwell H, Herrera D, Kao RT, Kebschull M, Kinane DF, Kirkwood KL, Kocher T, Kornman KS, Kumar PS, Loos BG, Machtei E, Meng H, Mombelli A, Needleman I, Offenbacher S, Seymour GJ, Teles R, Tonetti MS. Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018;89 Suppl 1:S173-s82.
27. Jepsen S, Caton JG, Albandar JM, Bissada NF, Bouchard P, Cortellini P, Demirel K, de Sanctis M, Ercoli C, Fan J, Geurs NC, Hughes FJ, Jin L, Kantarci A, Lalla E, Madianos PN, Matthews D, McGuire MK, Mills MP, Preshaw PM, Reynolds MA, Sculean A, Susin C, West NX, Yamazaki K. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018;89 Suppl 1:S237-s48.
28. Bestall S, Flynn R, Charleson G, Abbott PV. Assessment of Australian Dentists' Treatment Planning Decisions Based on Diagnosis. J Endod. 2020;46(4):483-9.
29. Abbott PV. Words matter vitally. Br Dent J. 2021;231(6):315.
30. Nickel B, Barratt A, Copp T, Moynihan R, McCaffery K. Words do matter: a systematic review on how different terminology for the same condition influences management preferences. BMJ Open. 2017;7(7):e014129.
31. Bedell SE, Graboys TB, Bedell E, Lown B. Words that harm, words that heal. Arch Intern Med. 2004;164(13):1365-8.
32. McCaffery K, Nickel B, Moynihan R, Hersch J, Teixeira-Pinto A, Irwig L, Barratt A. How different terminology for ductal carcinoma in situ impacts women's concern and treatment preferences: a randomised comparison within a national community survey. BMJ Open. 2015;5(11):e008094.
33. Disease. Available from: Dictionary.com DISEASE Definition & Usage Examples|Dictionary.com Accessed 5th January 2024.
34. "Condition". Dictionary.com. Condition Definition & Usage Examples|Dictionary.com Accessed 5th January 2024.
35. Jansson L, Ehnevid H, Lindskog S, Blomlöf L. Development of periapical lesions. Swed Dent J. 1993;17(3):85-93.
36. Korzen BH, Krakow AA, Green DB. Pulpal and periapical tissue responses in conventional and monoinfected gnotobiotic rats. Oral Surg Oral Med Oral Pathol. 1974;37(5):783-802.
37. Sundqvist G. Bacteriological studies of necrotic dental pulps: Umeå University; 1976.
38. Möller AJ, Fabricius L, Dahlén G, Ohman AE, Heyden G. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. Scand J Dent Res. 1981;89(6):475-84.
39. Fabricius L, Dahlén G, Ohman AE, Möller AJ. Predominant indigenous oral bacteria isolated from infected root canals after varied times of closure. Scand J Dent Res. 1982;90(2):134-44.
40. Sundqvist G. Associations between microbial species in dental root canal infections. Oral Microbiol Immunol. 1992;7(5):257-62.
41. Lin S, Moreinos D, Kaufman AY, Abbott PV. Tooth Resorption - Part 1: The evolvement, rationales and controversies of tooth resorption. Dent Traumatol. 2022;38(4):253-66.
42. Acute. Available from: Dictionary.com - in The American Heritage Stedman's Medical Dictionary. Source location: Houghton Mifflin Company. http://dictionary.reference.com/browse/acute Accessed: 3rd April 2015.
43. Acute. Available from: Dictionary.com ACUTE Definition & Usage Examples|Dictionary.com Accessed 5th January 2024.
44. "Acute". Merriam-Webster. Available from: Acute Definition & Meaning-Merriam-Webster Accessed 5th January 2024.
45. Chronic. Available from: Dictionary.com - The American Heritage Stedman's Medical Dictionary. Source location: Houghton Mifflin Company. http://dictionary.reference.com/browse/chronic Accessed: 3rd April 2015.
46. "Chronic". Dictionary.com chronic Definition & Usage Examples| Dictionary.com. Accessed 5th January 2024.

47. Chronic". Merriam-Webster. Available from: Chronic Definition & Meaning-Merriam-Webster Accessed 5th January 2024.
48. "Symptomatic". Merriam-Webster. Available from: Symptomatic Definition & Meaning-Merriam-Webster. Accessed 5th January 2024.
49. Asymptomatic". Merriam-Webster [Available from: Asymptomatic Definition & Meaning-Merriam-Webster Accessed 5th January 2024.
50. Chen I, Clarke SE, Gosling R, Hamainza B, Killeen G, Magill A, O'Meara W, Price RN, Riley EM. "Asymptomatic" Malaria: A Chronic and Debilitating Infection That Should Be Treated. PLoS Med. 2016;13(1):e1001942.
51. Glickman GN, Bakland LK, Fouad AF, Hargreaves KM, Schwartz SA. Diagnostic terminology: report of an online survey. J Endod. 2009;35(12):1625-33.
52. Consensus. Available from: Consensus|English meaning-Cambridge Dictionary Accessed 5th January 2024.
53. Consensus. Available from: Consensus|meaning-Cambridge Learner's Dictionary Accessed 5th January 202.
54. Consensus. Available from: Consensus Definition & Meaning-Merriam-Webster. Accessed 5th January 2024.
55. Consensus. 2024.
56. Consensus. Available from: consensus noun-Definition, pictures, pronunciation and usage notes|Oxford Advanced Learner's Dictionary at OxfordLearnersDictionaries.com. 2024.
57. Kakehashi S, Stanley HR, Fitzgerald RJ. THE Effects Of Surgical Exposures Of Dental Pulps In Germ-Free And Conventional Laboratory Rats. Oral Surg Oral Med Oral Pathol. 1965;20:340-9.
58. Abbott PV. Assessing restored teeth with pulp and periapical diseases for the presence of cracks, caries and marginal breakdown. Aust Dent J. 2004;49(1):33-9; quiz 45.
59. Kwang S, Abbott PV. Bacterial contamination of the fitting surfaces of restorations in teeth with pulp and periapical disease: a scanning electron microscopy study. Aust Dent J. 2012;57(4):421-8.
60. Nair PN. Apical periodontitis: a dynamic encounter between root canal infection and host response. Periodontol 2000. 1997;13:121-48.
61. Abbott PV. The periapical space-a dynamic interface. Ann R Australas Coll Dent Surg. 2000;15:223-34.
62. Abbott PV. The periapical space-a dynamic interface. Aust Endod J. 2002;28(3):96-107.
63. Abbott PVJEt. Classification, diagnosis and clinical manifestations of apical periodontitis. 2004;8(1):36-54.
64. Dashper S, Nastri A, Abbott PV. Odontogenic Bacterial Infections. In: Farah C, Balasubramaniam R, McCullough M, eds. Contemporary Oral Medicine. Springer International Publishing, Cham. 2018;819-70.
65. Kakehashi S, Stanley HR, Fitzgerald R. The exposed germ-free pulp: effects of topical corticosteroid medication and restoration. Oral Surg Oral Med Oral Pathol. 1969;27(1):60-7.
66. Michaelson PL, Holland GR. Is pulpitis painful? Int Endod J. 2002;35(10):829-32.
67. LI G. Endodontic Practice. 9 ed. Philadelphia: Lea & Febiger; 1978.
68. Grossman LI, S O. Diagnosis and treatment of endodontic emergencies. Chicago: Quintessence Publishing Co; 1981.
69. Abbott PVJEt. Diagnosis and management planning for root-filled teeth with persisting or new apical pathosis. 2008;19(1):1-21.
70. Ramachandran Nair PN, Schroeder HE. Periapical actinomycosis. J Endod. 1984;10(12):567-70.
71. Ramachandran Nair PN. Light and electron microscopic studies of root canal flora and periapical lesions. J Endod. 1987;13(1):29-39.
72. Tronstad L, Barnett F, Riso K, Slots J. Extraradicular endodontic infections. Endod Dent Traumatol. 1987;3(2):86-90.
73. Yusuf H. The significance of the presence of foreign material periapically as a cause of failure of root treatment. Oral Surg Oral Med Oral Pathol. 1982;54(5):566-74.
74. Koppang HS, Koppang R, Solheim T, Aarnes H, Stølen SO. Cellulose fibers from endodontic paper points as an etiological factor in postendodontic periapical granulomas and cysts. J Endod. 1989;15(8):369-72.
75. Nair PN, Sjögren U, Krey G, Kahnberg KE, Sundqvist G. Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: a long-term light and electron microscopic follow-up study. J Endod. 1990;16(12):580-8.
76. Nair PN, Sjögren U, Figdor D, Sundqvist G. Persistent periapical radiolucencies of root-filled human teeth, failed endodontic treatments, and periapical scars. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;87(5):617-27.
77. Ramachandran Nair PN, Pajarola G, Schroeder HE. Types and incidence of human periapical lesions obtained with extracted teeth. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996;81(1):93-102.
78. Ramachandran Nair PJET. Non-microbial etiology: foreign body reaction maintaining post-treatment apical periodontitis. 2003;6(1):114-34.
79. Calişkan MK. Pulpotomy of carious vital teeth with periapical involvement. Int Endod J. 1995;28(3):172-6.
80. Langeland K, Rodrigues H, Dowden W. Periodontal disease, bacteria, and pulpal histopathology. Oral Surg Oral Med Oral Pathol. 1974;37(2):257-70.
81. Hume W, Massey WJP, Mosby RoTSL. Disease dynamics of the dental pulp. 1998:37-44.
82. Andreasen FM. Transient apical breakdown and its relation to color and sensibility changes after luxation injuries to teeth. Endod Dent Traumatol. 1986;2(1):9-19.
83. Andreasen J. The effect of excessive occlusal trauma upon periodontal healing after replantation of mature permanent incisors in monkeys. Swed Dent J. 1981;5(3):115-22.
84. Kvinnsland S, Kristiansen AB, Kvinnsland I, Heyeraas KJ. Effect of experimental traumatic occlusion on periodontal and pulpal blood flow. Acta Odontol Scand. 1992;50(4):211-9.
85. Shi Y, Wang J, Cao C. Clinical studies on pulpitis and periapical periodontitis caused by traumatic occlusion. Zhonghua Kou Qiang Yi Xue Za Zhi. 1997;32(1):23-5.

Please cite this paper as: Abbott PV. Pulp, Root Canal and Peri-radicular Conditions: The Need for Re-classification. Iran Endod J. 2024;19(3): 158-74. *Doi: 10.22037/iej.v19i3.44394.*