A clinical classification of the status of the pulp and the root canal system

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Abstract

Many different classification systems have been advocated for pulp diseases. However, most of them are based on histopathological findings rather than clinical findings which leads to confusion since there is little correlation between them. Most classifications mix clinical and histological terms resulting in misleading terminology and diagnoses. This in turn leads to further confusion and uncertainty in clinical practice when a rational treatment plan needs to be established in order to manage a specific pathological entity. A simple, yet practical classification of pulp diseases which uses terminology related to clinical findings is proposed. This classification will help clinicians understand the progressive nature of the pulp disease processes and direct them to the most appropriate and conservative treatment strategy for each condition. With a comprehensive knowledge of the pathophysiology of pain and inflammation in the pulp tissues, clinicians may accomplish this task with confidence.

Key words: Dental pulp, classification, pulp disease, inflammation, necrosis, infection.

INTRODUCTION

Many different classification systems (Table 1) have been advocated for pulp diseases[1-13] although most of them are based on histopathological findings. Many authors and clinicians have attempted to correlate the histological conditions of the pulp to the clinical signs and symptoms.[1-13] These classifications mix clinical and histological terms resulting in many misleading terms and diagnoses for the same clinical condition (Table 1). This in turn leads to confusion and uncertainty in clinical practice when a rational treatment plan needs to be established in order to target a specific pathological entity. Confusion in diagnosis can also arise when using a classification with a wide selection of overlapping conditions.

One of the primary purposes of establishing a diagnosis is to determine what clinical treatment is required. If an incorrect diagnosis is made, then incorrect or inappropriate treatment may be instigated and this may result in various consequences ranging from mild to severe. Diseases of the pulp and periapical tissues are, in general terms, either inflammatory in nature or due to infections. An infection will also be associated with inflammation of the adjacent tissues. Hence, if the condition is inflammatory then the treatment regime adopted by the clinician should be targeted at removing the inflammatory stimuli and reducing the inflammation in the tissues – in such a case, an anti-inflammatory agent would be indicated as part of the local treatment (e.g., within a root canal medicament) or as part of any systemic medications used. On the other hand, an infection will require the use of an anti-microbial agent (e.g., antibiotic or anti-septic) within the root canal medicament or, in certain circumstances, systemically. Infected canals cause inflammation within the adjacent periapical tissues and therefore an anti-inflammatory agent combined with the anti-microbial agent in the root canal medicament should also be considered. Hence an accurate diagnosis is essential to ensue appropriate treatment is provided.

A further purpose of a classification of diseases is to enable communication between teachers, students, clinicians and researchers. Hence any classification needs to be useful, easily understood and readily applied to the clinical environment. The use of terminology that is inappropriate or that cannot be applied clinically is counter-productive to these aims. For example, if a histological examination is required since histological terms are used, then this is impractical in a clinical situation where an immediate diagnosis is required before treatment is instigated; in this scenario, a diagnosis is required in order to dictate what treatment regime should be followed.

A clinician's ability to diagnose accurately is dependent on having a thorough understanding of the disease processes involved as well as having a thorough understanding of the diagnostic procedures and tests being used and their limitations (see below).

The progression of disease in the dental pulp is similar to changes in other connective tissues. Typically, the tissue and disease progresses through the following stages: normal, inflammation (i.e., pulpitis), necrosis, infection and loss of pulp tissue (i.e., pulpless canals). Inflammatory changes may be acute, chronic, reversible or irreversible, and chronic conditions may have acute exacerbations at any time. The classification presented

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Table 1. Comparative terminology and classifications of pulp diseases used by various authors and organisations[1-13]

World Health Organization[1]	Weine[2]	Ingle[3]	Seltzer & Bender[4]	Cohen & Burns[5]	Tronstad[6]
(NOTE: Normal pulp not mentioned)	(NOTE: Normal pulp not mentioned)	Healthy pulp	(NOTE: Normal pulp not mentioned)	**Within normal limits** Normal pulp Calcific metamorphosis	Healthy pulp
Pulpitis Initial (hyperaemia) Acute Suppurative (pulpal abscess) Chronic Chronic ulcerative Chronic hyperplastic (pulpal polyp) Other unspecified pulpitis Pulpitis unspecified	**Pulpitis** Hyperalgesia (reversible pulpitis) Hypersensitive dentine Hyperaemia Painful pulpitis Acute pulpalgia (acute pulpitis) Chronic pulpalgia (subacute pulpitis) Nonpainful pulpitis Chronic ulcerative pulpitis Chronic pulpitis (no caries) Chronic hyperplastic pulpitis (pulp polyp)	**Pulpitis** Hyper-reactive pulpalgia Hypersensitivity Hyperaemia Acute pulpalgia Incipient Moderate Advanced Chronic pulpalgia Hyperplastic pulposis	**Pulpitis** Incipient form of chronic pulpitis Acute pulpitis Chronic partial pulpitis with partial necrosis Chronic total pulpitis with partial liquefaction necrosis Chronic partial pulpitis (hyperplastic form)	**Pulpitis** Reversible Irreversible Asymptomatic irreversible pulpitis Hyperplastic pulpitis Internal resorption Canal calcification Symptomatic irreversible pulpitis	**Pulpitis** Asymptomatic pulpitis Symptomatic pulpitis
Necrosis of the pulp	**Pulp necrosis**	**Pulp necrosis** Liquefaction Sicca	**Pulp necrosis**	**Necrosis** Partial Complete	**Necrotic pulp**
Pulp degenerations Denticles Pulpal calcification Pulpal stones	**Pulp degeneration** Atrophy Dystrophic calcification	**Pulp degeneration** Atrophic pulposis Calcific pulposis	**Pulp degeneration** Atrophic pulp Dystrophic mineralization		
Abnormal hard tissue formation in pulp Secondary or irregular dentine	**Internal resorption**	**Internal resorption**			

American Association of Endodontists' Glossary[7]	Harty[8]	Walton & Torabinejad[9]	Grossman[10]	Castellucci[11]	Stock[12]	Bergenholtz[13]
Normal pulp not mentioned	Normal pulp	(NOTE: Normal pulp not mentioned)	(NOTE: Normal pulp not mentioned)	Healthy pulp	Normal pulp	Pulpa sana
Pulpitis Reversible pulpitis Irreversible pulpitis	**Pulpitis** Reversible pulpitis Irreversible pulpitis	**Pulpitis** Reversible pulpitis Irreversible pulpitis Hyperplastic pulpitis	**Hyperaemia** **Pulpitides** Acute pulpitis Chronic ulcerative pulpitis Chronic hyperplastic pulpitis	**Pulpitis** Hyperaemia Pulpitis irreversible	**Concussed pulp** Reversible pulpitis Irreversible pulpitis	**Pulpitis**
Pulp necrosis	**Necrosis**	**Pulpal necrosis** Pulp calcification Internal (intracanal) resorption	**Necrosis** **Pulp degeneration** Calcific Fibrous Atrophic Internal resorption	**Necrosis**	**Pulpal necrosis** Internal resorption	**Necrosis pulpae**

below and in Table 2 has been proposed previously by Abbott.[14-16] It is a simple yet comprehensive clinical diagnostic system that utilizes terminology relating to the clinical findings (i.e., signs and symptoms) and it is based on the progression of pulp diseases through the various stages shown in Fig 1. It also includes "normal" pulp tissue which is an entity that should be diagnosed and given recognition when there are no signs of disease.

Signs and symptoms of pulp and root canal conditions

Diseases of the pulp tissues are dynamic and progressive in nature (Fig 1). Each disease condition may progress to other conditions if left untreated. Hence, the signs and symptoms will vary depending on the stage of the disease at the time the patient presents for treatment. In addition, the reaction to and the perception of pain will vary between individual patients and is influenced by the individual's emotional status and the coping strategies used to manage the pain. Many of the signs and symptoms overlap between the various pulp conditions due to the dynamic interactions and the progressive nature of the disease process. Therefore, the following descriptions of pulp diseases are based on the *typical* presenting complaints and clinical findings. When making a diagnosis, clinicians should be aware of the

Table 2. Clinical classification of the status of the pulp and root canal conditions proposed by Abbott[14,15] and used in the School of Dentistry at The University of Western Australia[16]

Clinically Normal Pulp (based on clinical examination and test results)

Reversible pulpitis – Acute
– Chronic

Irreversible pulpitis – Acute
– Chronic

Necrobiosis (Part of pulp necrotic & infected; the rest is irreversibly inflamed)

Pulp necrosis – No sign of infection
– Infected

Pulpless, infected root canal system

Degenerative changes
- Atrophy
- Pulpal canal calcification – partial
  – total
- Hyperplasia
- Internal resorption – Surface
  – Inflammatory
  – Replacement

Previous root canal treatment
- No sign of infection
- Infected
- Technical standard (based on the radiographic appearance)
  – Adequate
  – Inadequate
- Other problems - e.g. perforation, missed canals, fractured instrument, etc.

dynamic nature of the pulp disease process whilst also taking into consideration the limitations of assessing the true state of the pulp's blood supply with current pulp testing and examination techniques.

Clinically normal pulp

The term "clinically normal pulp" is used to classify a pulp that has no signs or symptoms to suggest that any form of disease is occurring. The term "clinically" is used since such a pulp may not be histologically normal and/or may have some degree of fibrosis (scarring) as a result of previous injury or stimuli.

A clinically normal pulp is asymptomatic. It produces a mild and transient response to various stimuli but the nature and severity of the response may vary according to the age and state of the tooth. As long as there has been no calcification of the coronal pulp space, a clinically normal pulp will react to cold stimuli with mild pain that lasts for no more than 1–2 seconds after the stimulus is removed. A clinically normal pulp does not respond to heat stimuli. Percussion and palpation tests will not elicit any tenderness. Radiographic examination will demonstrate normal appearance of the pulp chamber, root canals and periapical tissues.

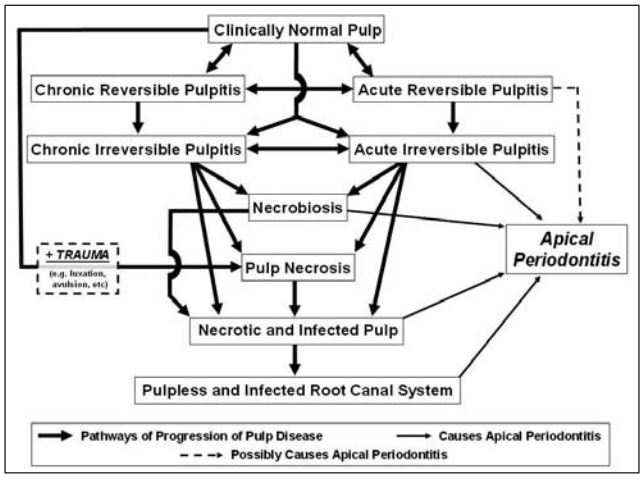


Fig 1. The progression and interaction of the various stages of pulp and root canal conditions and their relationships to apical periodontitis.

Fig 2. Tooth 35 has chronic reversible pulpitis due to a crack undermining the buccal cusp. There is pain to biting pressure. Transillumination of the tooth clearly demonstrates the presence, position and extent of the crack.

Dentine is usually sensitive when exposed to irritants hence dentine sensitivity should be distinguished from pulp inflammation.

Dentine sensitivity

When pain occurs with thermal, chemical, tactile, or osmotic stimuli associated with exposed dentine, the diagnosis is dentine sensitivity. The pain is consistent with an exaggerated response of the normal pulpo-dentinal complex, and it is severe and sharp on application of the stimulus to the exposed dentine. Nonetheless, there is no lingering discomfort once the stimulus is removed.

When there is a specific factor (other than exposed dentine) causing the sensitivity, such as caries, fractures, open restoration margins or recent restorative treatment, teeth may exhibit symptoms that are identical to dentine sensitivity. When symptoms develop in these situations, a diagnosis of reversible pulpitis is appropriate.

Reversible pulpitis

A pulp with reversible pulpitis has mild inflammation and it is capable of healing once the irritating stimulus has been removed. Pain is only felt when a stimulus (usually cold or sweet foods but sometimes heat) is applied to the tooth, and the pain ceases within a few seconds or immediately upon removal of the stimulus. The pain is short and sharp in nature but not spontaneous. There are no significant radiographic changes evident in the periapical region, and the only radiographic findings of note may be the cause of the problem, such as caries, a deep restoration, etc. Usually more extreme temperatures are required to induce the pain rather than mild changes (e.g., ice cream rather than tap water).

If there is pain to biting pressure as well as the above symptoms, then this may indicate a crack in the tooth (Fig 2) or restoration. While reversible pulpitis is usually acute, it may also be an acute exacerbation of a chronic condition. Here the terms "acute" and "chronic" are not used as histological terms but are based on the clinical symptoms: that is, acute means painful and chronic means no pain or only mild discomfort. Conservative pulp therapy in conjunction with the removal of the cause and the pathway of the irritation will result in resolution of the pulp inflammation and the return of the pulp to a clinically normal state.

A diagnosis of reversible pulpitis should always be considered as a "provisional diagnosis" since it is impossible to be completely certain about any individual pulp's ability to recover. This will depend on many factors – such as previous problems, previous inflammation, the degree of fibrosis, the true status of the pulp, etc. – and these are generally impossible to assess clinically. Hence once the diagnosis of reversible pulpitis has been provisionally made, the tooth can be managed in a conservative manner (as above) and then arrangements must be made to review the status of the pulp to determine whether it has actually returned to a clinically normal state. If it has, then it should be free of symptoms, have no signs of pulp or periapical diseases and should respond normally to pulp sensibility tests. The pulp status should be reviewed after several weeks (assuming there are no postoperative symptoms) although a three-month interval is generally considered to be more reliable as healing may take some time or necrosis (if it occurs) may take some time to become evident. At the review appointment, the provisional diagnosis can be confirmed as reversible pulpitis if the pulp has returned to a clinically normal state. However, if symptoms have persisted or if pulp necrosis has occurred, then the original provisional diagnosis will need to be altered to that of "irreversible pulpitis".

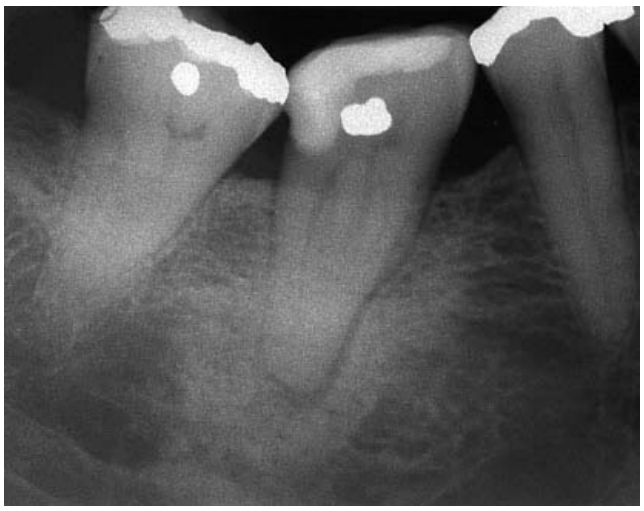
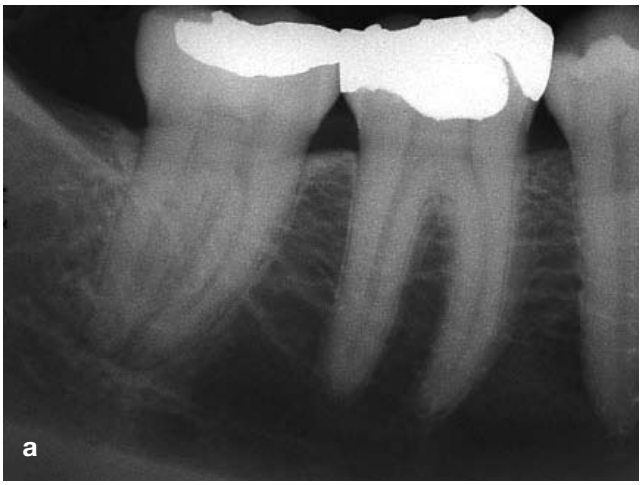


Fig 3. Tooth 46 has acute irreversible pulpitis with primary acute apical periodontitis due to marginal breakdown of the composite resin restoration.



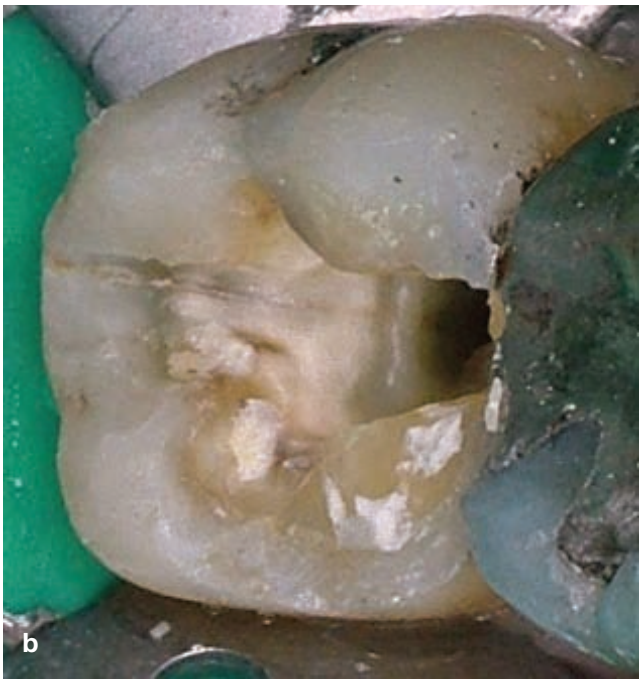


Fig 4. (A) Tooth 47 has acute irreversible pulpitis with primary acute apical periodontitis due to a vertical crack on the distal aspect of the tooth. (B) The extent of the crack can be seen following investigation of the tooth by removing the restoration – it extends into the tooth root and communicates with the pulp chamber and root canals.

Irreversible pulpitis

One of the classic symptoms of irreversible pulpitis is lingering pain induced by thermal stimuli. Only mild

temperature changes are required to induce the pain (e.g., tap water, breathing cold air). The initial reaction is a very sharp pain to hot or cold stimuli and it then lingers for minutes to hours after the stimulus is removed. The lingering pain is usually a dull ache or a throbbing pain. Spontaneous (unprovoked) pain, which may wake the patient at night and may become worse when lying down, is another hallmark feature of irreversible pulpitis. Patients with irreversible pulpitis often need strong analgesics and may have difficulty locating the precise tooth that is the source of the pain. They may even confuse the maxillary and mandibular arches (but not the left and right sides of the mouth) because of the extensive branching of dental nerve axons[17,18] and perhaps fewer proprioceptive fibres in the pulp.[19] Pulps afflicted with irreversible pulpitis without periapical pathosis may sometimes be difficult to diagnose; information provided by the patients and the results of pulp sensibility tests become particularly helpful in these cases. When the periapical tissues are involved (Figs 3 and 4), the tooth will usually be tender to pressure and/or percussion. Dynamic changes occur in the irreversibly inflamed pulp and it may move from being chronic and asymptomatic to having acute pain within a matter of just a few hours.

Acute irreversible pulpitis

Acute irreversible pulpitis usually has a sudden onset that may wake the patient at night. The pain is spontaneous with moderate to very severe intensity, and it lingers in response to temperature changes (heat and/or cold). The pain may be intensified by posture changes such as when lying down or bending over. Common analgesics are rarely effective in controlling the pain. In most cases, radiographs are not useful in diagnosis but they can be helpful in identifying the possible cause of the disease (e.g., deep caries, an extensive or fractured restoration, pins etc.). The tooth may be tender to biting pressure and/or percussion and, if present, this usually indicates spread of the inflammatory process to the periapical tissues (Figs 3 and 4). In some cases, the biting or percussion pain may indicate a crack in the tooth (Fig 4) and this is particularly noticeable when the crack is undermining a cusp.

Chronic irreversible pulpitis

Chronic irreversible pulpitis will have similar signs and symptoms but they will be much less severe than those in the acute cases. Patients may complain of moderate pain, which is more intermittent rather than continuous and it may be controlled by common analgesics. In the early stages of the disease process, the diagnosis may be difficult because the tooth may not show any demonstrable periapical change on the radiographs or a definitive sign of tenderness to percussion. However, information from the patient and pulp sensibility tests should be useful. As the disease progresses to involve the periapical tissues, periapical changes are more likely to be evident radiographically and/or clinically.

Necrobiosis

A tooth with necrobiosis has both inflamed and necrotic (usually infected) pulp tissue.[20] Many dentists use the term "partial necrosis" for this stage of the disease process; however, "necrobiosis" was suggested by Grossman[20] because it more accurately indicates the condition – the key factor to the spread of the disease process is the presence of bacteria within the necrotic part of the pulp rather than the necrosis itself of part of the pulp. The necrotic tissue may be in the coronal portion of the pulp (e.g., pulp chamber) with the inflamed tissue apically, or the different tissue states may exist in different canals of a multi-canal tooth. Teeth with this condition can be quite difficult to diagnose since they usually present with a mixture of the signs and symptoms of both pulpitis and necrosis with infection. The symptoms may be mild with intermittent painful episodes over many weeks or months. Pulp sensibility test results are mixed and frequently inconclusive or inconsistent with the patient's description of symptoms. Teeth with necrobiosis may also have apical periodontitis with radiographic evidence of a widened periodontal ligament space, which may be unexpected because the patient has reported sensitivity to hot and/or cold stimuli.

Pulp necrosis

A necrotic pulp should be suspected when the tooth does not respond to pulp sensibility tests. However, this will not always be the case since teeth with pulp canal calcification, previous root fillings or pulpotomies will also not respond to pulp sensibility tests. Likewise, some teeth or patients just do not respond to such tests for no apparent reason. When pulp necrosis is present, the history may reveal past trauma, previous episodes of pain or history of restorations and caries. Radiographically, a tooth with a necrotic pulp may have signs (such as untreated caries, an extensive restoration, previous pulp capping) or there may be no such signs (e.g., following trauma). Trauma to a tooth may cause pulp necrosis as a result of severing the apical blood supply if the tooth has been displaced from its normal position (e.g., luxations, avulsion) or if there has been significant damage and inflammation to the apical periodontal ligament (e.g., subluxation).

No significant radiographic changes are evident at the root apex unless there is also periapical involvement, and this only occurs once the necrotic tissue becomes infected. It is important to realize that a necrotic pulp *per se* does not cause apical periodontitis unless it is infected.[21-23] Pain usually does not present unless the periodontal ligament is affected. In this situation, the canal is infected and leads to apical periodontitis.

Occasionally, patients will complain of a dull continuous ache that is exacerbated by heat but relieved by cold water being held over the tooth. The reason is unknown although it has been speculated that micro-organisms producing gases may infect the

necrotic pulp and these gases contract with application of the cold water which relieves pressure on the nerve endings that may still be functioning, particularly in the apical portion of the canal. However, the relief from the cold water is only temporary and the pain returns as soon as the tooth warms up again and the gases expand. This situation occurs since the apical part of the pulp nerves are the last part of the pulp to necrose. It is likely that the apical nerves maintain some viability through blood supply to their more medial portions for a short time after the rest of the pulp has died.

Once a necrotic pulp is invaded by bacteria, then the bacteria will spread throughout the entire root canal system. The necrotic pulp acts as a source of nutrients for the bacteria which ingest the necrotic tissue and render the tooth pulpless. This may occur within 1–2 months of the initial invasion by the bacteria.[24]

Pulpless and infected root canals

Pulpless canals will always be infected. There will be no pain from the tooth itself when it has a pulpless canal, although some patients may give a history of occasional vague discomfort over a period of time. However, pain may arise from the periradicular tissues that become inflamed because of the presence of bacteria in the pulp space. The only clinical sign suggesting the condition may be the lack of response to pulp sensibility tests. No significant changes can be detected on the radiograph in the early stages of this condition but within 2–10 months there will be a radiolucency suggesting periapical involvement.[24] As there is the potential for erroneous pulp sensibility test responses, corroborating the clinical findings, radiographs and patient information is necessary for a definitive diagnosis.

Clinically, it is not possible to distinguish whether a tooth has a "necrotic pulp without infection", the early stage of a "necrotic pulp with infection" or the early stage of a "pulpless, infected root canal system". These disease entities can only be differentially diagnosed when a periapical radiolucency develops or if there are other clinical signs of periapical involvement. A "necrotic pulp without infection" will not have apical periodontitis but a "necrotic pulp with infection" will have apical periodontitis, as will a "pulpless infected root canal system". If a tooth has a "necrotic pulp with infection", this implies that it is in the early stage of the infection process since the necrotic tissue has yet to be ingested by the bacteria. Hence, it is more likely that there will be no periapical radiolucencies evident.[24] The periapical changes (i.e., bone resorption) take 2–10 months to be evident even though the necrotic pulp remnants are removed by the bacteria within 1–3 months after invasion.[24] Hence, a tooth with periapical radiolucency is extremely likely to have a "pulpless, infected root canal system" (Figs 5 and 6).

Tenderness to biting pressure and/or percussion is a typical classical sign of apical periodontitis and this may be present before the radiolucency is evident if the periapical response is rapid. Hence, such tenderness without a radiolucency would indicate either a "necrotic pulp with infection" or the early stages of a tooth with a "pulpless, infected root canal system".

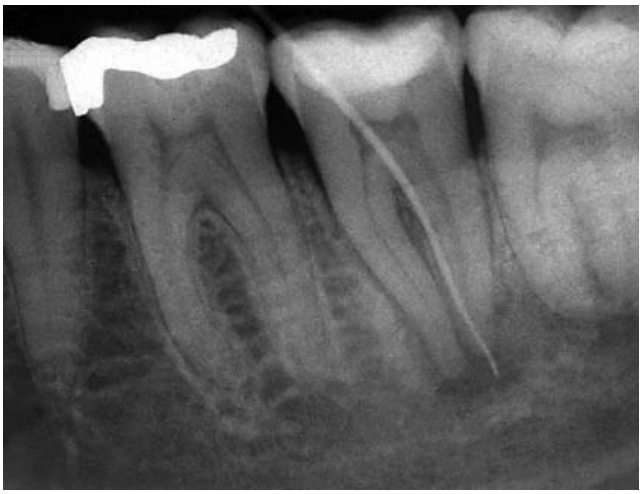


Fig 5. Tooth 37 has a pulpless, infected root canal system with a chronic periapical abscess due to marginal breakdown of the composite resin restoration. A gutta percha tracer point has been placed in the draining sinus to trace the origin of the sinus tract.

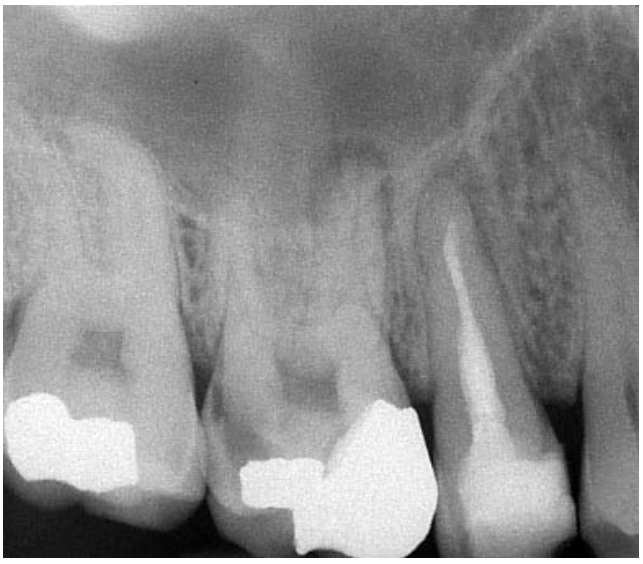


Fig 6. Tooth 16 has a pulpless, infected root canal system with secondary acute apical periodontitis due to the presence of caries on the distal surface and marginal breakdown of the amalgam restoration. Tooth 15 has an infected root canal system with chronic apical periodontitis due to breakdown of the composite resin restoration. The root canal filling is technically unsatisfactory.

Degenerative changes

The pulp will usually respond to noxious stimuli by becoming inflamed, but it may also respond by degeneration which includes atrophy and fibrosis, calcification, root resorption, or hyperplasia.

Atrophy

Atrophy is a normal physiologic process that occurs with age and is asymptomatic. Pulp sensibility tests responses may be normal or delayed. No significant

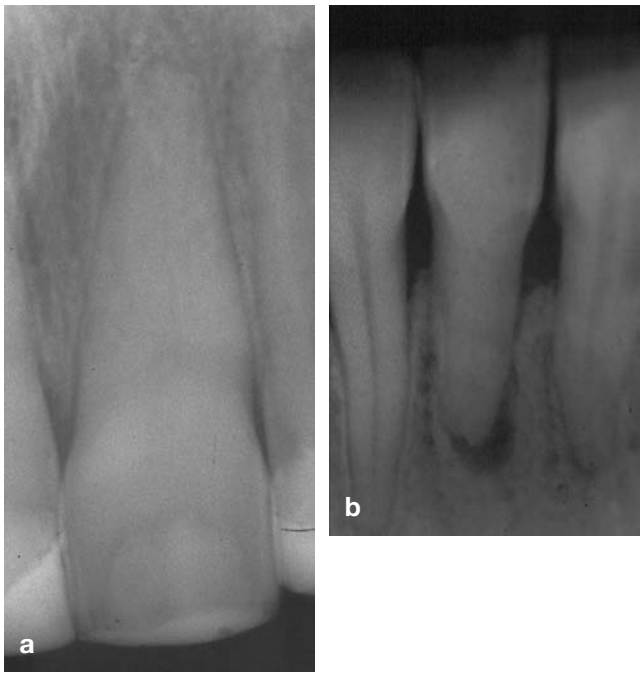


Fig 7. Teeth with pulp canal calcification. (A) Tooth 21 has a clinically normal pulp with no evidence of periapical pathosis but there are radiographic signs of pulp canal calcification. Endodontic treatment is not required for this tooth. (B) Tooth 41 has a pulpless, infected root canal system with chronic apical periodontitis. There is radiographic evidence of pulp canal calcification. Endodontic treatment is required for this tooth.

radiographic or clinical signs are present. As the pulp atrophies, there will also likely be fibrosis of the pulp tissue and the extent of this will be largely determined by the number of irritant episodes suffered by that particular pulp throughout its history. The size of the pulp chamber may be reduced. No treatment is required.

Pulp canal calcification

Teeth with pulp canal calcification (commonly referred to as pulp canal obliteration or PCO) may or may not have any symptoms – if the pulp tissue within the calcified or calcifying canal is normal then there will be no symptoms (Fig 7a); on the other hand, however, if the canal is pulpless and infected, then this will cause apical periodontitis with symptoms eventually developing as the apical periodontitis progresses (Fig 7b). There are usually no responses to thermal tests (e.g., heat, cold) but an electrical test may elicit a normal or delayed response. Radiographically, there is no evidence of the usual pulp chamber outline and the root canal may appear narrow or it may not be evident at all. Pulp canal calcification may appear radiographically to be partial or complete but it is not possible to assess the extent of calcification from a clinical or radiographic examination. It is feasible, and likely, that the canal may only be a few cells wide and therefore will not be evident radiographically, but may still contain viable and healthy tissue. Hence, the term "obliteration" is not ideal since this term implies that there is no canal remaining and instead the term "calcification" is preferred.

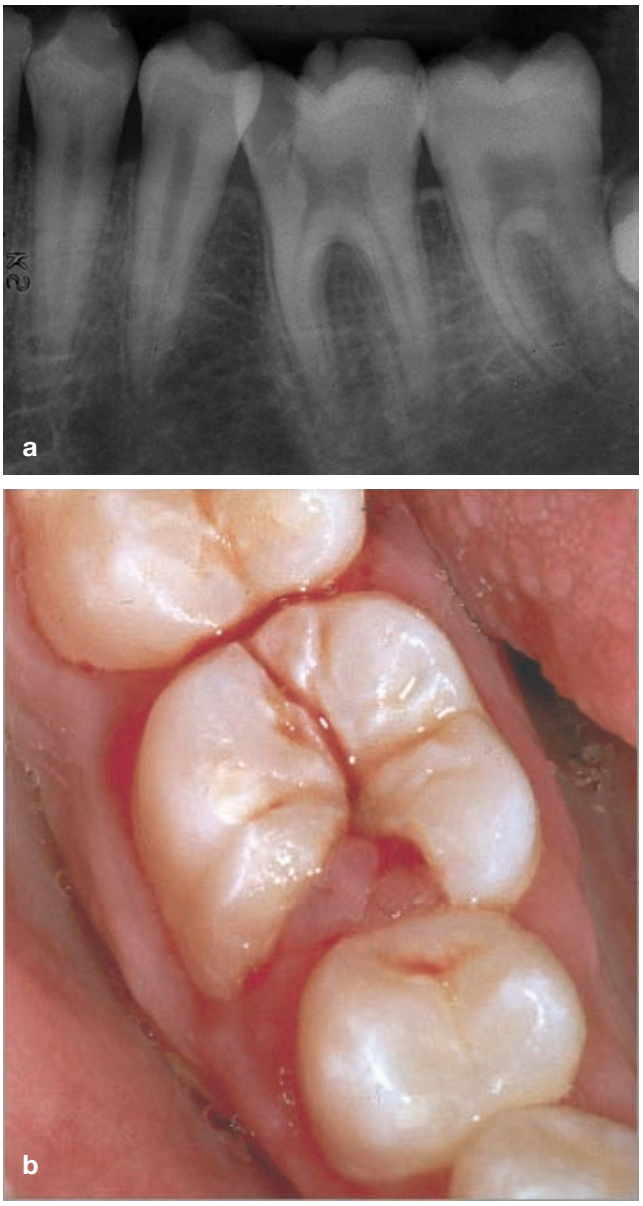


Fig 8. Tooth 36 has acute irreversible pulpitis and primary acute apical periodontitis due to a vertical crown:root fracture and an extensive carious lesion. (A) The radiographic appearance also suggests that condensing osteitis has been present for some time. (B) Some of the gingival tissue has grown into the carious cavity and this overgrowth of gingival tissue should not be mistaken for hyperplastic pulpitis.

Hyperplasia

Hyperplasia of the pulp occurs almost exclusively in young teeth with an abundant blood supply and a large carious lesion. It is essentially an overgrowth of granulation tissue and may result in the development of a pulp polyp. The hyperplasic tissue is relatively insensitive to touch due to having only a few nerves present. It may bleed easily depending on the degree of the tissue vascularization and the degree of ulceration. Occasionally it may cause mild discomfort during mastication. 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 vasodilation and minimal chronic inflammation. The tooth will respond to pulp sensibility testing but often

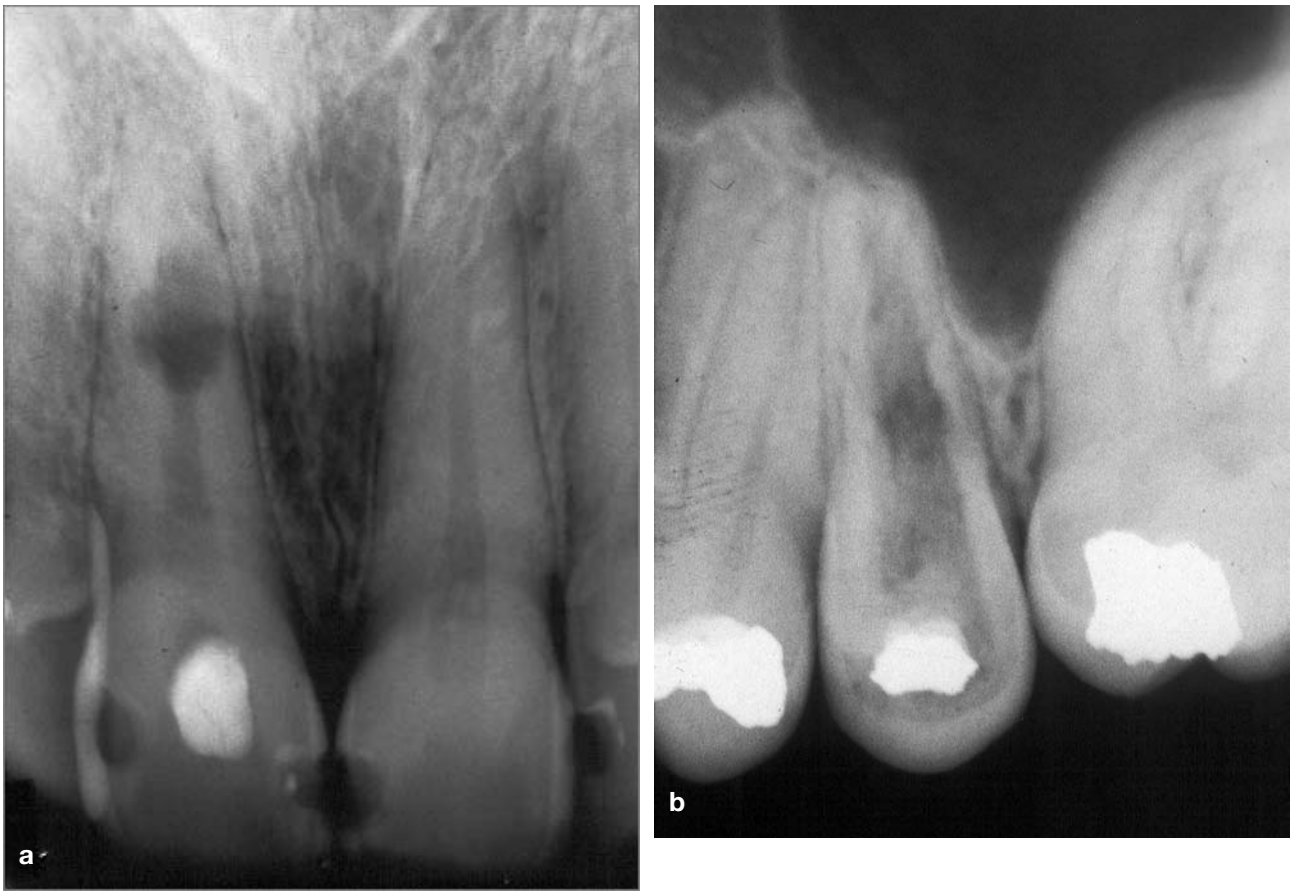


Fig 9. (A) Tooth 11 has an arrested lesion of internal inflammatory resorption. Originally, there would have been pulp necrobiosis that stimulated the resorption but now the tooth has a pulpless, infected root canal system and chronic apical periodontitis, as evidenced by no responses to thermal and electric pulp sensibility testing, a very slight widening of the apical periodontal ligament space and a "different" sensation on percussion. (B) Tooth 25 has internal replacement resorption. The pulp has been replaced by bone-like hard tissue. The periradicular tissues are normal.

with an exaggerated response to cold tests. No significant radiographic changes (except for the cause of the problem – for example, caries, fractured restoration, etc.) are evident unless there is also periapical involvement presenting as a radiolucency or radiopacity.[25]

Sometimes, the gingival tissues may grow into the carious lesion and this may be suggestive of a hyperplasic pulpitis (Fig 8b). In these cases the distinction may be made by careful examination of the tissue mass, so as to determine if it is connected to the pulp or to the gingivae.

Internal resorption

There are three forms of internal resorption – surface, inflammatory and replacement. Internal *surface* resorption is unlikely to ever be diagnosed since it is defined as minor areas of resorption of the surface of the root canal wall. It is unlikely to be noticed radiographically and there will be no clinical signs or symptoms. No treatment is required, which is fortunate since it cannot be clinically or radiographically diagnosed.

Internal *inflammatory* resorption can occur at any point along the length of the pulp space – that is, coronally within the pulp chamber or within the root canal. It is believed to occur in teeth with necrobiosis and it is probably due to a metaplastic change or activation of dentinoclasts within the inflamed pulp tissue that is in contact with the coronal pulp which is necrotic and infected. This condition is usually asymptomatic and often only recognized during a routine radiographic examination. If symptoms are present, they are usually indicative of acute apical periodontitis due to an infected canal, or pain when perforation of the crown occurs and the metaplastic tissue is exposed to oral fluid and bacteria. Initially, part of the pulp (i.e., the apical portion) is alive and still contains nerve fibres so a response to pulp sensibility testing is possible. However, as the lesion progresses, the entire pulp becomes necrotic and infected, and over time the canal will become pulpless and infected – hence there will be no response to pulp sensibility testing. Radiographically, internal inflammatory resorption will have an oval-shaped increase in the size of part of the root canal system. Periapical changes may be noted after the canal has become infected and apical periodontitis develops (Fig 9a).

Internal *replacement* resorption is an uncommon condition that occurs when the pulp undergoes metaplastic changes and the dentine is resorbed and replaced by bone-like hard tissue. The cause of internal

replacement resorption is usually unknown but is not due to the presence of bacteria in the pulp. This condition is asymptomatic, and usually only recognized during routine radiographic examination. Radiographically, internal replacement resorption will have an irregular enlargement of the pulp space that is filled with bone-like hard tissue (Fig 9b). Periapical changes do not usually occur with internal replacement resorption.

Endodontically-treated teeth

Many teeth that are examined by dentists will have had previous pulp therapy or endodontic treatment. Such treatments include pulp capping, partial pulpotomy, pulpotomy, partial pulpectomy and pulpectomy. These teeth may or may not have signs or symptoms, and they may or may not have apical periodontitis associated with them. The technical standard of the previous treatment may also vary considerably and this can only be partly assessed by the radiographic appearance of the material(s) placed in the pulp space or root canal(s). Essentially, the radiographic appearance of a root filling only indicates the radiodensity of the material and where the material has been placed. Radiographs do not provide any indication of whether or not the root canal system was disinfected during the previous treatment, how effectively the canal was sealed, whether the seal has been maintained, whether any bacteria survived the previous treatment and whether any new bacteria have entered the root canal system since the treatment was completed.

When assessing a tooth that has had previous conservative pulp therapy that was designed to preserve the pulp (such as pulp capping, partial pulpotomy, pulpotomy and partial pulpectomy), the key decision to make is whether or not the remaining pulp is healthy. In contrast, when assessing a tooth that has had previous endodontic treatment (i.e., pulpectomy), the key decision is whether the root canal system is infected or not (Fig 5). If the root canal system is infected, then the decision is usually based on the presence of some form of apical periodontitis, an apical abscess or facial cellulitis. In such cases, the radiographic appearance (i.e., the technical standard) of the root canal filling is irrelevant as the root filling will need to be removed in order to manage the infection and periapical condition.

The presence of a widened periodontal ligament space or a periapical radiolucency is an important and sometimes the only indicator of apical periodontitis. However, the cause of a radiolucency is not necessarily just an infection in the apical part of the root canal system.[26] In some cases, the radiolucency may indicate a periapical true cyst, an extra-radicular infection, a foreign body reaction or a periapical scar.[27] The radiolucency may also be an indication of periapical tissues that are still healing when assessing teeth that have had recent root canal treatment, because some periapical lesions may take 2–5 years to heal.[28]

Technical inadequacy of a root canal filling may be seen on a radiograph but it may often be obscured due to the two-dimensional nature of radiographic images. Assessment of a previously treated tooth should also include identification of any other "mechanical" problems such as a fractured file, a root perforation, etc.

Periapical diseases

A discussion about pulp diseases would be incomplete without discussing the periapical tissues and their disease processes since periapical diseases are usually a direct result of pulp diseases. It is beyond the scope of this paper to discuss periapical diseases in detail and such diseases have been fully discussed in other publications.[14,15,27] Unfortunately, there is also considerable confusion amongst authors and clinicians regarding the classifications and terminology used for periapical conditions.[1-15] However, Abbott[14,15] has outlined a classification for periapical diseases that is based on the same principles as the above classification for pulp diseases and root canal conditions and therefore the two classifications can be used concurrently.[14-16]

When a tooth is being examined for pulp disease, the condition of the periapical tissues must also be assessed, and *vice versa*, since apical periodontitis is usually associated with inflammatory conditions of the pulp (i.e., reversible and irreversible pulpitis), or infection of the pulp space following necrosis of the pulp or after previous endodontic treatment. That is, periapical diseases are usually a direct consequence of, and/or a sequel to, interaction with the root canal system.

Examples of some typical diagnoses are:

- Acute irreversible pulpitis with primary acute apical periodontitis due to caries.
- Acute irreversible pulpitis with primary acute apical periodontitis due to breakdown of a restoration (Fig 3).
- Necrotic and infected pulp with primary acute apical periodontitis due to caries and restoration breakdown.
- Pulpless, infected root canal system with secondary acute apical periodontitis due to breakdown of the restoration.
- Pulpless, infected root canal system with a chronic apical abscess due to breakdown of the restoration (Fig 5).
- A pulpless, infected root canal system with secondary acute apical periodontitis due to breakdown of the restoration (Fig 6).
- A root-filled tooth with an infected root canal system and chronic apical periodontitis due to breakdown of the restoration (Fig 6).
- Pulpless, infected root canal system with chronic apical periodontitis due to a crack in the tooth and caries.
- A clinically normal pulp with pulp canal calcification but no signs of apical periodontitis (Fig 7a).

Table 3. Summary of the examination and diagnostic processes for the assessment of pulp, root canal and periapical conditions[14,15]

Stage	Procedure		Outcome
History	Medical history Dental history Description of presenting complaint Details of previous treatment of presenting complaint	➜	Provisional diagnosis of presenting condition
Clinical examination	Extra-oral signs Intra-oral signs Individual tooth assessment Restoration assessment	➜	Identify possible cause(s) Provisional diagnosis of tooth status
Clinical tests	Pulp sensibility tests	➜	Provisional diagnosis of the status of the pulp and/or root canal system
	Periradicular tests	➜	Provisional diagnosis of the periapical status
Radiographic examination	Periapical radiograph(s)	➜	Provisional diagnosis of the periapical status
	Bitewings radiograph (if needed)	➜	Assess/identify/confirm cause(s)
Correlation of all above findings	Combine the history, clinical examination, clinical tests and radiographic examination results	➜	DEFINITIVE DIAGNOSIS Pulp, root canal and periapical status + Cause(s) of the condition(s)
Management plan	Investigation of tooth (i.e. remove restoration, caries, cracks, etc)	➜	Confirm the definitive diagnosis and cause(s)
	Reassess amount of tooth structure and overall prognosis	➜	Finalise the management plan and continue treatment

Examination and diagnostic procedures to assess the status of the pulp and root canals

The importance of gathering all the relevant information for making a correct diagnosis cannot be over-emphasized. An accurate diagnosis is imperative in all cases so appropriate treatment can be provided in a timely manner.

A current medical and dental health history is important, not only for preventing health problems during treatment but also to help reach a thorough diagnosis.[29] For example, pain medication taken within 6–12 hours prior to examination may alter the responses to pulp sensibility tests or other clinical tests. One tablet of pain medication may be sufficient to reduce the pulp or periapical inflammation, or the analgesic may alter the patient's perception of pain by lowering the pain threshold.[30]

Whilst taking the dental history, the clinician should be formulating a provisional diagnosis in his/her mind. The provisional diagnosis should be based on the patient's chief complaint, their description of the symptoms and a thorough history of any relevant problems and any prior dental treatment (Table 3). The provisional diagnosis should be made prior to examining the patient and his/her mouth and teeth. The subsequent clinical and radiographic examinations together with the appropriate clinical tests are used to confirm (or change) the provisional diagnosis. Extra-oral, intra-oral soft tissue and dentition examinations should be performed to establish the patient's overall oral health. Clinical tests include pulp sensibility tests and peri-radicular tests (e.g., percussion, palpation, mobility) that are aimed to reproduce the patient's symptoms. Some periradicular tests (e.g., periodontal probing, mobility) also help to assess the integrity of the attachment apparatus of the tooth and the extent of inflammation of the periodontal ligament resulting from periodontal disease and pulp inflammation. Pulp sensibility tests measure the ability of the pulp's nerve fibres to respond to a stimulus. Among the tests available clinically, the cold pulp sensibility test is the most useful test and the dry ice (carbon dioxide) test is the most reliable and reproducible of the various cold tests available commercially.[31] "Cold sprays" and ice have been shown to be only about 40–60 per cent accurate; therefore they should be considered as unreliable[31] and hence they are not recommended for use. Severe and prolonged response to a cold stimulus is indicative of irreversible pulpitis, whereas no response could indicate pulp necrosis, a pulp confined in an obliterated canal, a previous pulpotomy or a previously filled root canal. Heat tests are not used routinely unless the major symptom reported by the patient is sensitivity to a hot stimulus. An exaggerated and lingering response to heat testing is indicative of irreversible pulpitis. Electric pulp tests can give false negative results (e.g., in some obliterated canals) or false positive results (e.g., with pus in canals, necrobiosis or improper technique). Hence electric pulp sensibility tests are only useful in some cases of coronal pulp canal calcification when cold tests are inconclusive, or as an adjunct during the follow-up of trauma to the teeth. When selecting the appropriate clinical test, one has to remember that no single test is sufficient to make a firm diagnosis of reversible or irreversible pulpitis and therefore multiple tests are required. Periodontal examination of the suspect teeth is essential in all cases because periodontal diseases can mimic endodontic problems and they are often inter-related. Periodontal probing depths also help to determine the prognosis of the tooth. Transillumination of the tooth is a particularly useful examination procedure that can be used to readily detect cracks in teeth (Fig 2), and it should be incorporated into the routine examination of all teeth with pulp or periapical diseases, especially following trauma (Table 4).

Table 4. Summary of the examination procedures that should be performed as part of any routine dental examination and diagnosis but particularly whenever pulp and/or periapical pathosis is suspected[16]

General medical history		As required for all dental procedures
Presenting complaint		Long and short term history, past and current symptoms, past and recent treatment, medications being used (prescribed and self administered) Description of PAIN: Location, onset, nature, duration, stimuli, relief, referred
Clinical examination		Puckering, indentation, draining sinus, facial asymmetry, caries, periodontal diseases, restorations, restoration margins, etc
Clinical tests	Periradicular tests	Percussion, palpation, mobility, periodontal probing
	Pulp sensibility tests	Cold (CO_2 dry ice at -78ºC) Electric (in some cases e.g. when pulp canal calcification) Heat (if required, if patient complains of sensitivity to heat)
Radiographic examination		Periapical radiographs, tube shifts, bitewings, occlusal, panoramic
Other tests		Biting on individual cusps (e.g. with a 'Tooth Slooth') Transillumination of the tooth with a fibre optic light Anaesthetic (e.g. block, infiltration, intra-periodontal ligament, etc.)
Investigation		Remove all restorations, caries, cracks Transillumination of the cavity, cusps, marginal ridges, etc Assess whether, and how, the tooth can be restored again Assess the need for any other treatment (e.g. periodontal) Assess the long term prognosis of the tooth (consider endodontic, periodontal and restorative aspects)

In the above discussion, the term "pulp sensibility test" has been used. It should be noted by clinicians that the older, and sometimes more commonly used term "vitality tests" is no longer recognized as being appropriate since thermal and electric tests do not test the "vitality" of the tooth or the pulp. The term "vitality" refers to the presence of blood supply to the tissues whereas the term "sensibility" is defined as the ability to respond to a stimulus. Hence, "sensibility" is the appropriate term to use since thermal and electric tests are assessing whether the nerve fibres within the pulp are able to respond to the hot, cold or electrical stimulus. If the pulp's nerve fibres do respond, then an assumption is made that there must be a viable blood supply to keep the nerve fibres alive and functioning, and therefore the rest of the pulp tissues and cells are probably also alive and functioning. However, these tests do not necessarily indicate the degree of health or disease within the pulp. It is also important to remember that the nerve fibres are usually the last part of the pulp to undergo necrosis, especially in the apical part of the canal, and therefore a "false positive" response may be obtained from a pulp that is essentially necrotic.

The main functions of pulp sensibility tests are to, firstly, determine whether there is a nerve response and, secondly, to assess the nature of the response. If there is a response, then the pulp may be alive, whereas no response may indicate necrosis, pulp canal calcification, or a tooth that has had a pulpotomy or a root canal filling, as discussed above. This is important when the provisional diagnosis was pulp necrosis or a pulpless canal as here the clinician is searching for the tooth that does not respond. However, if the provisional diagnosis was pulpitis (either reversible or irreversible), then the clinician is searching for the tooth that has an exaggerated response which replicates the pain experienced by the patient – that is, the degree of sensitivity of the pulp (note: sensitivity is defined as an exaggerated response and is different to sensibility). The severity can also help to distinguish between reversible and irreversible pulpitis but the time that the pain lingers after removing the stimulus is usually of more value to distinguish between these two conditions. In this latter scenario of testing for pulpitis, the pulp sensibility tests could be considered as "sensitivity tests" as they are assessing which tooth responds more severely than the others.

Pulp sensibility tests cannot be interpreted without also viewing an accurate and current periapical radiograph of the tooth in question. The results of pulp sensibility tests should correlate with the radiographic appearance of the tooth and the periapical tissues before making a definitive diagnosis. As an example, a tooth that does not respond to a pulp sensibility test could have a necrotic pulp (with or without infection), a pulpless, infected root canal system, or pulp canal calcification. It may also have had a root canal filling, a pulpotomy or a partial pulpectomy, all of which may or may not be infected. All of these conditions can only be fully assessed by viewing a periapical radiograph and therefore the pulp test result could be misinterpreted if a radiograph is not viewed. The radiograph also needs to be a current radiograph since pulp and periapical diseases are progressive in nature and will change over time. Hence, an accurate diagnosis of the current condition cannot be based on a radiograph taken several weeks, months or years earlier, and sometimes even one taken just several days earlier.

Radiography may arguably be the most reliable of all the diagnostic tests although it may not help in the assessment of teeth with pulpitis. However, radiographs will always provide valuable information to assist with diagnosis and treatment.[32,33] A routine radiograph may sometimes be the first indication of the presence of pathosis, such as internal root resorption or chronic apical periodontitis. The cause of the pulp disease (deep caries, deep restorations, open margins, etc.) may also

Table 5. Summary of endodontic treatment strategies for the various pulp and root canal conditions. (Note – recommendations for endodontic treatment and re-treatment are dependent on the tooth having sufficient tooth structure remaining to enable it to be adequately restored again)

Clinically normal pulp		NIL treatment required
	Dentine hypersensitivity	Occlude the exposed dentine (e.g. with potassium oxalate), and/or hyperpolarize the intradental nerves (e.g. with potassium nitrate)
Reversible pulpitis	Acute	Conservative pulp therapy then reassess healing response
	Chronic	Conservative pulp therapy then reassess healing response
Irreversible pulpitis	Acute	Routine endodontic treatment with anti-inflammatory medications then reassess healing response, or extraction
	Chronic	Routine endodontic treatment with anti-inflammatory medications then reassess healing response, or extraction
Necrobiosis		Routine endodontic treatment with anti-inflammatory and anti-microbial medications then reassess healing response, or extraction
Pulp necrosis	No infection	No treatment required but monitor and reassess periapical tissues for signs of periapical inflammation (which would indicate an infected root canal)
	Infected	Routine endodontic treatment with anti-inflammatory and anti-microbial medications then reassess healing response, or extraction
Pulpless, infected root canals		Routine endodontic treatment with anti-inflammatory and anti-microbial medications then reassess healing response, or extraction
Degenerative changes		
Atrophy		NIL treatment required
Pulpal canal calcification	Partial	NIL treatment required unless canal infected with apical periodontitis ➜ then do endodontic treatment and reassess healing response
	Total	NIL treatment required unless canal infected with apical periodontitis ➜ then do endodontic treatment and reassess healing response
Hyperplasia		Routine endodontic treatment with anti-inflammatory medications then reassess healing response, or extraction
Internal root resorption	Inflammatory	Routine endodontic treatment with anti-inflammatory medications then reassess healing response, or extraction
	Replacement	Extraction (eventually) – can monitor initially
Previous root canal treatment		
No sign of infection		No treatment but monitor and reassess unless restoration being replaced ➜ then retreat and reassess healing response
Infected		Orthograde endodontic re-treatment with anti-inflammatory and anti-microbial medications then reassess the need for periapical surgery, or extraction, if the canal is still infected or if there are signs of on-going periapical pathosis
Technical standard	Inadequate	No treatment but monitor unless the restoration is being replaced or the canal is infected ➜ then retreat and reassess healing response
	Adequate	No treatment but monitor unless the restoration is being replaced or the canal is infected ➜ then retreat and reassess healing response
Others	Perforation	Orthograde endodontic re-treatment and repair the perforation from an internal approach if possible; then reassess the need for periapical or periodontal surgery, or extraction, if there are signs of on-going periapical and/or periodontal pathosis
	Missed canal	Orthograde endodontic re-treatment then reassess the need for periapical surgery, or extraction, if the canal is still infected or if there are signs of on-going periapical pathosis

be evident on radiographs. A widened periodontal ligament space or a periapical radiolucency that cannot be "moved" with tube-shift radiography may be diagnostic for periapical pathosis. However, radiography is not a perfect diagnostic tool. Soft tissue diseases of the pulp are not visible on radiographs; only hard tissue changes can be seen on radiographs. Radiographs only provide a two-dimensional representation of three-dimensional structures. Periapical lesions may not be directly evident on the radiographs and their real extent and the spatial relationships to anatomical structures may not be readily visualized. Periapical lesions also take some time (2–10 months) to develop to a size which will be evident on a radiograph.[24]

A pretreatment diagnosis is made after interpretation of all the collected information. This diagnosis should include an assessment of the pulp or root canal condition, the periapical status and the cause of the disease(s).[14,15] Ideally, there should be at least two different signs and/or symptoms present to indicate and confirm the disease(s).[16] Only at this stage can the management of the patient's problem(s) be planned.

It is not possible to diagnose the histological status of the pulp from the clinical signs and symptoms.[34] Diagnosis of the level of pulp inflammation is difficult but it is relatively easy to determine from clinical findings that a pulp is necrotic or the canal is pulpless. Severity and duration of pain appears to be related to the status of the pulp. Severe lingering pulp pain usually indicates the presence of acute irreversible pulpitis with an increase in pulp tissue pressure. When mild to moderate pain of very short duration is present with no previous history of pain in the tooth, reversible pulpitis may be

occurring. If the tooth is associated with a previous history of pain, irreversible pulpitis is more likely.[35] Sometimes the clinical and radiographic examinations are inconclusive yet the patient has pain. However, it is important not to initiate irreversible treatment without a definite diagnosis. In these cases, the correct procedure is to make a provisional diagnosis. Referral to an appropriate specialist should be considered in such cases, although if not feasible then, guided mainly by the radiographic findings, such as the deepest restoration in the suspect quadrant or caries close to the pulp, the likely tooth can be identified and treated with zinc oxide-eugenol cement that has an excellent local anaesthetic and sedative effect. If necessary, one tooth at a time is treated this way until a definitive diagnosis is established. It is most likely that only one tooth is responsible for acute pain. However, if there is very little evidence that provides definitive information, then either specialist referral or a conservative "wait and see" approach may be necessary in order to allow the vague symptoms to localize to the specific tooth. It is important to understand, and remember, that pulp and periapical diseases are progressive and therefore the symptoms will vary over time, which usually assists the localization and the diagnosis of pain.

Complete patient care must include a discussion of the diagnosis with the patient. It is essential to disclose the examination findings and for the patient to understand his/her presenting condition(s). Details of the recommended treatment, the prognosis and the alternatives to treatment should be discussed as well as the consequences if no treatment is performed.

Management of pulp and root canal conditions

The ultimate decision the practitioner must make is whether to treat or not to treat the tooth, and if treatment is indicated, whether to treat the pulp or the root canal system. The alternative management is to extract the tooth and then to consider a prosthesis to replace it. Accurate diagnosis and identification of the cause(s) of the problem(s) will lead to effective management of the offending tooth. Table 5 summarizes the treatment strategies for the management of the various pulp diseases.

Management strategies vary considerably for the various pulp conditions which emphasizes the need for an accurate diagnosis before considering any treatment. Conditions such as a pulp with atrophy or pulp canal calcification do not require any treatment (unless the pulp has become necrotic and the canal has been infected). A conservative approach should be adopted when dealing with conditions such as pulp necrosis without infection since pulp tests are not entirely reliable and the periapical tissues only become inflamed once the canal is infected, rather than as a response to the necrosis itself. In cases of pulpitis, the key to favourable treatment outcomes is to accurately diagnose the status of the pulp and this means deciding whether the inflammation is reversible (in which case it may be treated conservatively) or irreversible when the pulp (or the tooth) must be removed.

The first principle of managing any diseases is to remove the cause of the problem.[36] Since the presence of micro-organisms is the main cause of pulp diseases, an essential part of the diagnosis and assessment of the tooth is to identify how the bacteria have entered the tooth and the pulp space. In the case of pulp diseases, the cause is often a defective restoration, caries and/or cracks that have allowed bacteria to enter the root canal system. In recent years, Abbott[36] has advocated that all restorations should be removed prior to endodontic treatment in order to remove the causes of the pulp and periapical diseases. A clinical study has shown that signs of bacterial entry via restoration breakdown, caries or cracks were found in only 40 per cent of restored teeth with pulp and periapical disease when they were examined prior to restoration removal but these pathways for bacterial entry were subsequently found in 99 per cent of the teeth following removal of the restorations.[36]

Removal of all the restorations, caries, cracks and any other factors that may be causing the pulp and periapical diseases provides the clinician with an ideal opportunity to fully assess whether the tooth is suitable for further restoration following endodontic or other pulp therapy. One of the key factors affecting the treatment outcome and the long-term survival of endodontically-treated teeth is the quality and longevity of the coronal restoration since breakdown of the restoration provides the most likely pathway of entry for bacteria to enter the tooth again in the future, which then leads to a new lesion of apical periodontitis. Hence, it is essential to assess the amount and quality of remaining tooth structure prior to continuing with endodontic and restorative dental treatment. The removal of the restorations, caries and cracks (Fig 4) can be considered as "investigation" of the tooth[36] and it should be done routinely for every tooth that requires a restoration (Table 3).

When treating teeth with reversible pulpitis, the aim is to preserve the pulp and return it to a clinically normal state. Measures to minimize iatrogenic pulp injury should be incorporated into routine restorative techniques. Since pulp inflammation occurs with polymicrobial infection, it is prudent to avoid the contamination of all cavities with microbial-rich saliva. The use of rubber dam during routine restorative dentistry will limit the number of bacteria in deep cavities to only the cariogenic bacteria, which are weak pathogens to the pulp.[37]

Irreversible pulpitis, pulp necrobiosis and their sequelae (such as pulp necrosis with infection, pulpless infected canals) require root canal therapy or tooth extraction. Likewise, a tooth that has had previous endodontic therapy (such as pulpectomy, pulpotomy) but has remained infected or has become infected again will require endodontic re-treatment or extraction. When root canal treatment (or re-treatment) is the

treatment of choice, the pulp tissue and tissue breakdown products as well as the bacteria and their by-products should be completely eliminated from the root canal system, which is subsequently filled as completely as possible. Coronal restorations should prevent recurrent bacterial entry during and after root canal treatment.[38] These procedures should also be performed under rubber dam isolation. Occlusal reduction may be performed to reduce postoperative pain in patients whose teeth initially exhibit pulp inflammation, tenderness to percussion, and pre-operative pain.[39] After root canal treatment, adequate healing is evidenced clinically by resolution of symptoms and radiographically by bone filling in the radiolucent area at the root apex if it was present prior to treatment. Thereby, a functioning tooth is established with a healthy periodontium.

Internal root resorption is rare in permanent teeth and it can sometimes mimic external invasive root resorption radiographically. Management for internal inflammatory root resorption is usually via endodontic treatment in order to remove the blood supply to the resorbing cells through the apical foramina. Several dressings of calcium hydroxide may be required if the lesion is active, to ensure complete removal of all dentinoclasts and to encourage hard tissue repair on the external surface of the root if there has been a perforation. Currently there is no treatment available to prevent the disease progression of internal replacement resorption. Extraction of the affected tooth is the treatment of choice. However, if the lesion is diagnosed early enough, endodontic treatment with calcium hydroxide dressings may be attempted.

With all pulp diseases, post-treatment follow-up is extremely important due to the diagnostic uncertainties with some conditions and the possible need for further treatment such as periapical surgery when periapical healing is not evident.

CONCLUSIONS

It hurts everyone involved when accessing the pulp space of the wrong tooth, accessing a tooth when pain is of non-endodontic origin, or not intervening when a diseased pulp is causing severe pain. The significance of a thorough diagnosis cannot be overstated. The proposed classification of pulp diseases is simple, yet practical and uses terminology related to clinical findings. This classification will help clinicians understand the progressive nature of the pulp disease processes and will direct them to the most appropriate and conservative treatment strategy for each condition. With a comprehensive knowledge of the pathophysiology of pain and inflammation in the pulp tissues, clinicians may accomplish this task with confidence.

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