Literature review and critical analysis of research articles related to a topic in endodontics

Evaluate the following statement

"Non-vital teeth that present with no apical or lateral radiolucencies require only "aseptic" endodontic treatments whilst non-vital teeth that present with apical or lateral radiolucencies require "antiseptic" endodontic techniques"

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In this essay I will attempt to compare and contrast the aetiology, pathogenesis and treatment of non vital teeth with and without periradicular radiolucencies using historical and contemporary scientific papers.

Before embarking on a discussion of the above statement, let us define the crucial phrases and words- (A dictionary of Dentistry 2010, Oxford University Press)

Periapical adj. Surrounding the apical area of a tooth root. A periapical granuloma (apical granuloma) consists of a mass of inflammatory cells, fibroblasts, and collagen at the apex of a tooth root, usually caused by disease progression from the pulp of an associated tooth root; it is frequently asymptomatic although the soft tissue over the apex may be tender to pressure. If left untreated it can develop cystic change. Radiographically it appears as a well-defined area of radiolucency. Periapical periodontitis refers to inflammation of the periapical tissues; it may be either acute or chronic. Acute periapical periodontitis is associated with increased blood flow and the formation of oedema in the periapical tissues, and the tooth is very tender to pressure; there are minimal radiographic changes, although widening of the periodontal ligament may be seen; it may resolve or develop into chronic periapical periodontitis (periapical granuloma). See also periodontitis. Periapical tissue consists of the periapical alveolar bone and the periodontal membrane in the region of the apex of the tooth, which interfaces between the tooth root and the alveolar bone.

Radiolucent adj. Having the property of being transparent to x-rays. Radiolucent materials appear dark on an x-ray.

Non-vital- Describing a tooth in which the pulp has undergone degenerative change, necrosis not responding to thermal or electrical stimulation.

Aseptic technique- A procedure that is free from contamination by any bacteria, fungi, virus, or other microorganisms.

Antiseptic technique- The use of a substance that destroys (bactericidal) or inhibits (bacteriostatic) the growth of bacteria or other microorganisms.

For the purpose of this essay we are assuming that a correct diagnosis has been made regarding the lack of blood supply to the tooth, and an accurate diagnosis of a periapical/lateral (periradicular) area that definitively requires root canal therapy. More recent diagnostic aids such as Laser Doppler Flowmetry and Cone Beam Computed Tomography have helped in diagnosis. The use of radiography and thermal or electrical stimulation when aiding in making a definitive diagnosis can sometimes be art rather than an exact science. Table one describes the differential diagnosis for a radiolucent periapical area.

What is aseptic technique in endodontics, and what is the gold standard?

Exclusion of the access cavity and pulpo-dentinal complex from the oral environment and its commensal bacteria is mandatory. Rubber dam plus the use of plugging agents providing a saliva and crevicular fluid tight seal, and the use of sterile single use endodontic files is essential. We are attempting ***not*** *to introduce microbiota into the pulpo-dentinal complex. Further steps such as use of pumice and iodine on the tooth surface prior to access, sterilisation of the operating field including the dam, use of sterile single use burs maybe considered. The use of surgical drapes and cross infection procedures used for the placement of implants maybe superfluous. However with the advent of HTM 01-05 and the discovery of the prion, political pressure may dictate future protocols.*

Table 1. Lesions of the jaws that may present as 'periapical pathosis' and should be considered as part of the differential diagnosis of such pathoses.

EPITHELIAL CYSTS

Developmental odontogenic
- Odontogenic keratocyst
- Dentigerous cyst
- Lateral periodontal cyst
- Glandular odontogenic cyst

Non-odontogenic
- Nasopalatine duct cyst
- Nasolabial cyst

NEOPLASMS AND OTHER TUMORS

Odontogenic

BENIGN
- Ameloblastoma
- Squamous odontogenic tumor
- Calcifying epithelial odontogenic tumor
- Clear cell odontogenic tumor
- Ameloblastic fibroma
- Ameloblastic fibrodentinoma
- Odontoamelobastoma
- Adenomatoid odontogenic tumour
- Calcifying odontogenic cyst
- Odontogenic fibroma
- Odontogenic myxoma
- Benign cementoblastoma

CARCINOMAS
- Malignant ameloblastoma
- Primary intraosseous carcinoma
- Malignant variants of other odontogenic tumors
- Malignant changes in odontogenic cysts

SARCOMAS
- Ameloblastic fibrosarcoma
- Ameloblastic fibrodentinosarcoma
- Odontogenic carcinosarcoma

Non-Odontogenic

BENIGN
- Cemento-ossifying fibroma
- Neurofibroma
- Neurilemoma
- Osteoid osteoma
- Osteoblastoma
- Chondroma
- Idiopathic histiocytosis

MALIGNANT
- Ewing's sarcoma
- Chondrosarcoma
- Osteosarcoma
- Neurogenic sarcoma
- Carcinoma of the maxillary sinus
- Malignant neural tumors
- Burkitt's lymphoma
- Metastatic carcinoma
- Primary lymphoma of bone
- Plasma cell neoplasms
  - Solitary plasmacytoma
  - Multiple myeloma
- Malignant salivary gland tumors

NON-NEOPLASTIC BONE LESIONS
- Fibrous dysplasia
- Cemento-osseous fibroma and cemento-osseous dysplasias (including periapical cemental dysplasia and florid osseous dysplasia)
- Cherubism
- Central giant cell lesions
- Central hemangioma of bone
- Aneurysmal bone cyst
- Simple (traumatic/solitary/hemorrhagic) bone cyst

INFLAMMATORY LESIONS
- Radicular cysts (of pulpal origin)
  - Apical: true, pocket
  - Lateral
  - Residual
- Paradental cysts-including:
  - Inflammatory collateral cyst
  - Mandibular infected buccal cyst
- Periapical granuloma
- Condensing osteitis (idiopathic bone sclerosis)
- Periapical abscess
- Osteomyelitis
- Tuberculosis

METABOLIC DISEASES
- Paget's disease (initial phase)
- Hyperparathyroidism

How does antiseptic and aseptic technique differ in endodontic treatment?

As described above a basic level of asepsis is mandatory in endodontic procedures. The difference between aseptic and antiseptic technique is that the operator is introducing protocols into the endodontic procedure to remove and or inactivate microbiota, and their by-products, from the anatomy of the pulpo-dentinal complex. Most commonly this involves the use of combinations of medicaments that remove or inactive the microbiotal complex. For the purposes of orthograde endodontic therapy antiseptic technique encompasses aseptic technique and is an extension of it.

It is essential that we now describe the scientific evidence behind the cause of periapical periodontitis and is possible sequela.

The pathogenesis of the radiolucent area.

While physical and chemical stimuli can cause irritation and even necrosis of the pulp, it is now widely accepted that disease of the pulpo-dentinal complex is mainly due to microbiota (Miller 1894, Kakehashi et al 1965). Excluding cases of complicated crown fracture, micro-leakage of the hard surfaces of the tooth allows influx of bacteria into the dentinal tubules, and a dynamic balance between host response and bacterial loading determines the outcome. A healthy pulps immediate response is inflammatory in nature, greater blood flow to the pulp increasing the flow of dentinal fluid outwards, which removes the bacteria through hydrostatic pressure. This flushes bacterial toxins and bacteria away from the pulp (Maita et al 1991; Nagaoka et al. 1995).

In the case of a compromised pulp, bacteria are able to invade the tubules. Of the approximately 800 different bacteria taxa able to survive in the oral cavity (Paster et al 2006), few manage to invade the dental tubules.

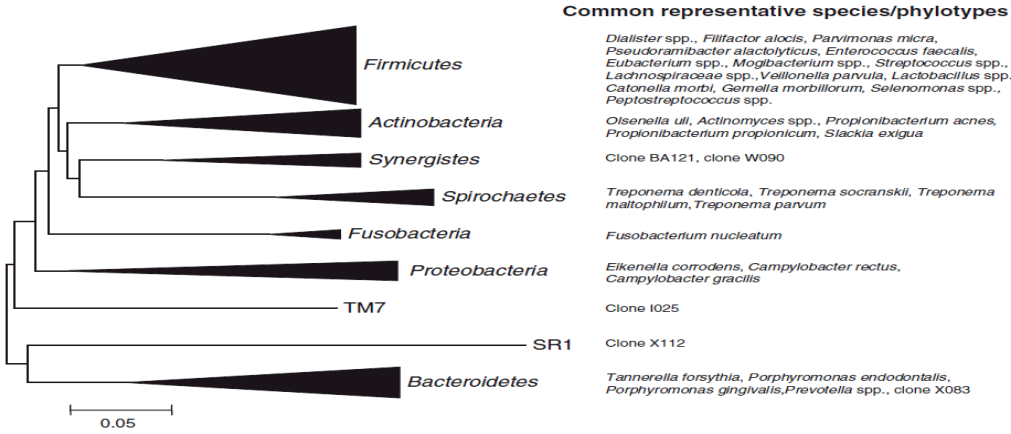


Fig 1 Bacterial phyla that have representatives in endodontic infections. On the right, example species or phylotypes for each phylum are presented.

The most common is Streptococcus this maybe due to its ability to express multiple surface protein adhesins (Hasty et al., 1992) and its ability to recognise components present within dentinal tubules, such as collagen type I, which stimulate bacterial adhesions and intra-tubular growth (Love, McMillan 1997). Specific interactions of other oral bacteria with invading streptococci may then facilitate the invasion of dentine by select bacterial groupings. As a result of this bacterial ingress a pulpal immune and inflammatory response is produced. Again this is a dynamic process. In the early stages of pulpal infection the microbiota are dominated by aerobic and facultative anaerobic bacteria (Nair et al 1997; Sundqvist et al 1994). Obligate anaerobes will also be present but in smaller numbers (Fabicius et al 1982). As the infection progresses the number of obligate and facultative anaerobes increase at the expense of the aerobic bacteria, this is due to the change in redox potential, pH and availability of nutrients (Fidgor and Sundqvist 2007). These bacteria survive by metabolising the remnants of the necrotic pulp tissue and exist as planktonic bacteria a prerequisite to organising themselves in biofilms (Bowden et al 1998). The formation of biofilms, and its attachment to root canal walls, is critical as it provides the participating bacteria up to 1000 times more resistance to antimicrobials than their planktonic forms (Gilbert et al 1997). It is also thought to make the

bacteria more pathogenic than those in the planktonic state. Bacterial cell in biofilm 15% by volume embedded in non random extracellular matrix 85% by volume. Dental biofilms can reach 300 cells thick (Socranskey et al 2000). Nair, in 1987, was possibly the first to identify biofilm structures in infected root canals with the use of transmission electron microscope.

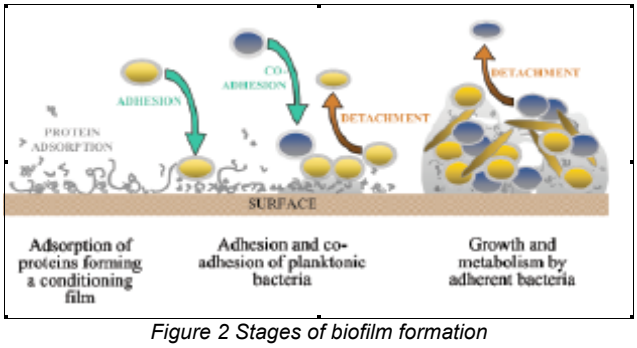


Figure 2 Stages of biofilm formation

The preconditions for the formation of biofilms in root canals will depend on the cause of the pulpal breakdown. Caries exposure, will produce an inflammatory lesion front receding in bursts towards the apex, this provides a fluid medium that allows the planktonic bacteria to multiply and attach to the root canal walls. Apical ramifications, lateral canals, and isthmuses connecting main root canals have all been shown to harbour bacterial cells, which are also frequently organized in biofilm-like structures. In addition, biofilms adhered to the apical root surface (extraradicular biofilms) have been reported and regarded as a possible cause of post treatment periapical periodontitis (Ricucci and Siqueira, 2008, 2010).

Although fungi, archaea, and viruses contribute to the microbiotal diversity in endodontic infections, bacteria are the most common micro-organisms occurring in these infections. (Figure 1). Cultural and molecular analysis has identified endodontic infections consisting of 10 to 30 species per canal (Siqueira and Rocas 2005). Total bacterial counts vary fro 10,000 to 1,000,000,000 cells per infected canal (Siqueira et al 2007). The bacterial profiles vary from individual to individual (Sakomoto et al 2006). Therefore, no particular species can be identified as the main pathogen. See page 8 for tables illustrating bacterial diversity.

Once infection has reached the area near the apex, an inflammatory response of the spongeosa attempts to maintain the infection intraradicularly. This usually means that microbiota are restricted from the periradicular area. However, the inflammatory and immune response elicited, produce chemicals which destroy the alveolar bone surrounding the root.

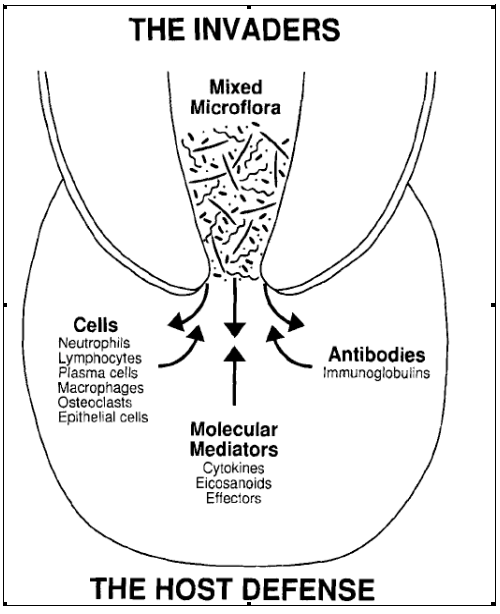


Figure 3. Simplified diagram of inflammatory and immune response at the periapex.

Experiments by Moller et al in 1981 on monkeys have shown that unless there are bacteria within the pulpo-dentinal complex, apical periodontitis does not occur and radiolucent area cannot form. There is also evidence that the greater the number of bacterial species in the infected canal, and the greater number of bacterial cells the larger the size of the radiolucent area.

There is therefore no doubt that apical periodontitis leading to radiolucent areas at the periapex of non vital teeth, where no previous endodontic treatment has been undertaken, is caused by the microbiota residing within the pulpo-dentinal complex. (Kakehashi et al 1965, Sundqvist 1976, Moller et al 1981).

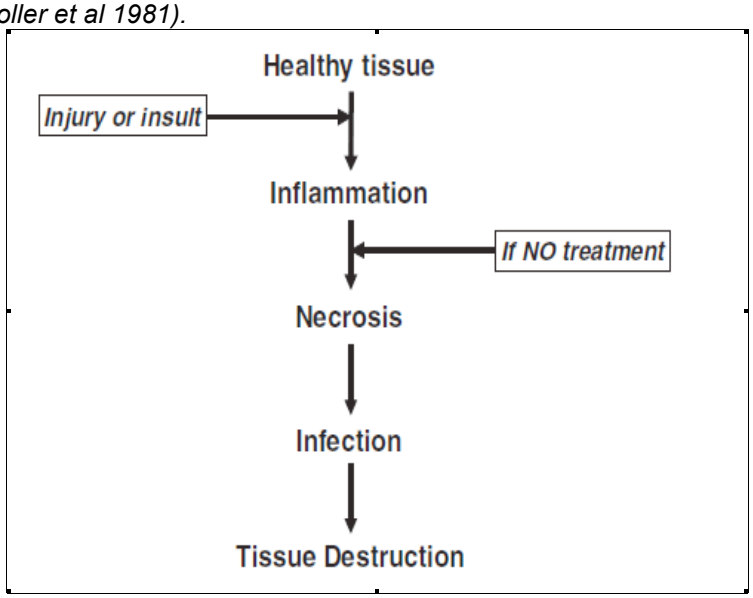


Figure 4 Disease process in tissue if untreated.

If the above statement is factual then treatment of periapical periodontitis, leading to the periradicular radiolucency, must be the removal of the causal agents i.e. the microbiota. In practice this is achieved surgically by either orthograde or retrograde root canal therapy as antibiotic therapy if ineffective in reducing the microbiotal load from the pulpo-dentinal complex due to the reduced blood supply. The treatment of choice is firstly orthograde therapy as it is generally the easiest and the treatment with a higher success rate. (Farzaneh M et al 2004.)

Appropriate treatment of teeth with periradicular radiolucent areas.

We must now discuss if more effective removal of the microbiota can be achieved by aseptic or antiseptic orthograde techniques. As discussed previously the difference between the two approaches is the use of combinations of medicaments that reduce the microbiotal loading in the dentino pulpal complex. Although many different chemicals have been used historically, a combination of sodium hypochlorite, chlorohexidine gluconate, EDTA and Calcium Hydroxide are generally accepted to be the medicaments of choice at present. Antiseptic technique for orthograde root canal therapy would involve exactly the same protocols as for aseptic technique; however, the operator would use the chemicals above instead of inert materials such as saline solution. Studies by Bystrom and Sundqvist, in 1981, treated 17 single rooted teeth, with periapical areas with the use of stainless steel files and sterile saline solution. They found a 100 to 1000 fold reduction in bacterial count. This could be explained by the introduction of oxygen into the canal system reducing the number of strict anaerobes, also the removal of the intraradicular components from the coronal and middle thirds of the canal system. The burnishing effect of the files however produced a smear layer which saline could not remove. They also discovered that the bacterial count increased, nearly to the original numbers, if no intra canal medicament was used between visits. They discovered that only in 20-43 % of cases showed complete elimination by mechanical instrumentation alone. In 1983 Bystom discovered that chemo mechanical debridement with the use of 0.5% sodium hypochlorite produced complete disinfection in 40-60% of cases. Later Bystrom 1985 and Sjogren 1991 showed that chemo mechanical debridement with 0.5% sodium hypochlorite and one week with Calcium Hydroxide complete disinfection in 90-

100% of cases. These and numerous later studies proved that aseptic technique is less effective than antiseptic technique at reducing bacterial loading.

In summary,

A non-vital tooth without a radiographic area requires antiseptic endodontic technique rather than aseptic techniques because:

- Present diagnostic techniques in general dental practice are insufficient to definitively identify the presence of periradicular radiographic areas in a proportion of cases. Bender and Seltzer in 1961 showed that even considerable loss of the spongious bone may be radiographically invisible, depending on the density or thickness of the overlying compact bone. In simple terms, just because an area is not present on a radiograph does not mean that there is no pathology present. In 1974 Goldman et al showed that radiological diagnosis varied considerably between operators. So there may or may not be a lesion there and if there is may or may not be diagnosed!
Until the use of CBCT becomes more practical as a diagnostic aid in endodontics, a dentist's skill in interpreting radiographs in conjunction with a meticulous history and examination and use of special tests will remain the standard procedure. Table 2.

- If there is a definitive diagnosis of the absence of a periradicular area, this does not mean that pathology will not develop. Indeed cases of acute periodontal abscesses are often seen where there is no periradicular pathology visible radiographically. It is therefore imperative that the cause of the possible pathology is removed, ie the microbiota of the pulpo-dentinal complex. The reason being "prevention is better than cure". As discussed previously aseptic technique is insufficient to reduce bacterial loading of a tooth to a level that is sufficient to ensure levels of microbiota are non pathogenic in a healthy individual.

- A non vital tooth has lost its defence mechanism, a healthy vital pulp. Dentinal tubules remain patient and are a portal for the ingress of microbiota, which may lead to the development of periradicular pathology. There is nothing as efficient as a healthy pulp in defending the periapex of a tooth from infection. However, with current endodontic techniques a well obturated pulpo-dentinal system provides a physical barrier which entombs bacteria in the pulpo-dentinal complex and prevents further invasion. As current systems for root canal obturation are broadly bacteriostatic after 7 days rather than bactericidal, antiseptic technique is needed to achieve this.

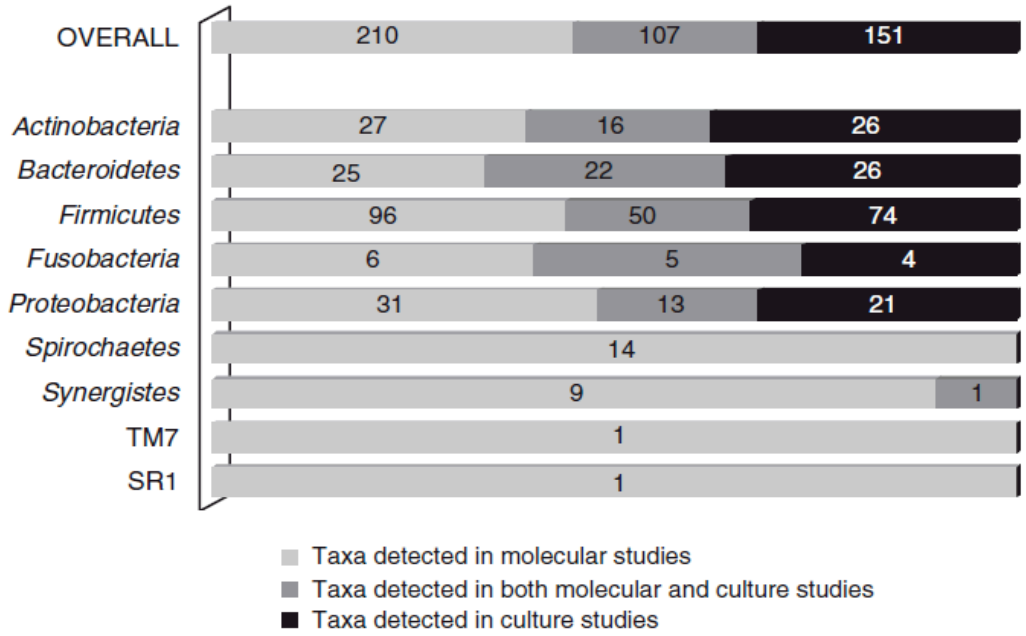
-Technology has not yet produced a diagnostic aid that can identify the absence of bacteria within the pulpo-dentinal complex, more importantly its proximity to the periradicular area. There is no way of knowing if a non vital tooth is sterile or non sterile, it is therefore logical to assume it is infected and requires therapy.

A tooth with a periradicular area requires antiseptic technique because as described above, periradicular periodontitis is a disease of microbiotal origin, predominantly bacterial origin. Antiseptic technique reduces the bacterial loading more effectively than aseptic technique.

In conclusion

***All** endodontic treatments, including those presenting with or without periradicular areas, require adequate aseptic technique to ensure there is absolutely no exogenous microbiota introduced into the pulpo-dentinal complex. This is a basic premise upon which successful endodontic treatment is based.*

Teeth definitively diagnosed with a periradicular area of endodontic origin MUST be treated by antiseptic technique, as studies have unequivocally proved that periapical periodontitis is a disease of microbiotal origin, primarily bacterial. Further studies have shown that effective treatment of periapical periodontitis can only be achieved by the use of chemo mechanical debridement, using a combination of bactericidal medicaments.



Distribution of bacterial species/ phylotypes found in endodontic infections according to the detection method. Data are given overall and for the 9 phyla that have endodontic representatives.

Phyla	Taxa	As-Yet-Uncultivated Phylotypes	Taxa Detected by Molecular Studies	Taxa Detected by Culture Studies
Firmicutes	184	69	131	98
Bacteroidetes	69	24	42	48
Actinobacteria	54	11	31	39
Proteobacteria	44	11	32	21
Fusobacteria	14	5	9	9
Spirochaetes	14	4	14	0
Synergistes	10	10	10	1
TM7	1	1	1	0
SR1	1	1	1	0

Bacterial species/ phylotype (taxa) Richness in Primary Endodontic Infections

Bacterial species/Phylotype (taxa) Richness in Extraradicular Infections

Phyla	Taxa	Taxa Detected by Molecular Studies	Taxa Detected by Culture Studies
Firmicutes	47	16	41
Bacteroidetes	18	10	12
Proteobacteria	12	6	8
Actinobacteria	10	7	8
Fusobacteria	3	3	2
Spirochaetes	2	2	0

Procedure	*Result*
1. History and discussion with patient Medical history Dental history Description of presenting complaint Details of any previous treatment of presenting complaint	→ Provisional diagnosis of presenting condition
2. Clinical Examination Extra-oral signs Intra-oral signs Individual tooth assessment Restoration assessment	→ Assess possible causative factors → Provisional diagnosis of tooth status
3. Clinical Tests Pulp sensibility tests	→ Provisional diagnosis of the status of the pulp and/or the root canal system
Percussion, mobility, palpation	→ Provisional diagnosis of the periapical status
4. Radiographic Examination	→ Confirm/assess causative factors → Provisional diagnosis of periapical status
5. *Correlation of the history, clinical, radiographic and test findings*	→ *DEFINITIVE DIAGNOSIS* - Pulp, root canal and periapical status - Cause(s) of the diseases
6. TREATMENT PLAN Investigation/restoration removal	→ Confirm the definitive diagnosis and cause(s)
Reassessment of the tooth and its prognosis	→ Finalize and continue the treatment plan

Table 2 Summary of the examination and diagnostic processes for the assessment of the status of the pulp and periapical tissues.

References: In the order they appear in the essay.

Miller WD (1894)- An introduction to the study of the bacterio-pathology of the dental pulp. Dental Cosmos 36, 505–28.

Kakehashi,S, Stanley,H.R.. & Fitzgerald, R.J. 1965- The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. Oral Surg Oral Med Oral Pathol, 20, 340-9

Maita, E., Simpson, M.D, Tao, l., Pashley, D. H- 1991 Fluid and protein flux across the pulpo-dentine complex of the dog in vivo. Archs oral biol; 36: 103-110.

Nagaoka, S., Miyazaki, Y., Liu, H. J., Iwamoto,Y., Kitano,M. & Kawagoe, M. 1995- Bacterial invasion into dentinal tubules of human vital and non vital teeth. J Endod, 21, 70-3.

Paster B.J, Olsen I, Dewhirst F.E 2006-. The breadth of bacterial diversity in the human periodontal pocket and other oral sites. Periodontol 2000 42:80-87

Hasty D.I, Ofek I, Courtney H.S, and Doyle R.J 1992- Multiple adhesins of streptococci. Infection and Immunity, 2147-2152 A mini review.

Love R.M, McMillan MD, Jenkinson HF (1997). Invasion of dentinal tubules by oral streptococci is associated with collagen recognition mediated by the antigen i/ii family of polypeptides. Infect Immun 65:5157-5164.

Nair P 1997. Apical periodontitis: a dynamic encounter between root canal infection and host response. Periodontology, 2000 13, 121.

Sundqvist G, 1994- Taxonomy, Ecology and Pathogenicity of the root canal flora Oral Surg Oral Med Oral Path ;78:522-30

Fabricius, l., Dahlen,G., Ohman, A E. & Moller, A. J. 1982. Predominant indigenous oral bacteria isolated from infected root canals after varied times of closure. Scand J Dent Res, 90, 134-44.

Figdor, D. & Sundqvist, G. 2007- A big role for the very small–understanding the endodontic microbial flora. Australian Dental Journal, 52, S38-51

Bowden G, Hamilton I 1998- Survival of oral bacteria. Critical reviews in oral biology & medicine 9: 54-85

Gilbert P, Das J and Foley I 1997-Biofilm susceptibility to antimicrobials. Advances in dental research 11: 160-167

Socransky S and Haffajee A- Dental Biofilms: Difficult therapeutic targets. Periodontology 2000. 28. Issue 1, 12-55

Nair P 1987 Light and electronmicroscopic studies on root canal flora and periapical lesions. J.Endod; 13: 29-39.

Ricucci D, Siqueira, J, 2010-Biofilms and apical periodontitis: Study of prevalence and association with clinical and histopathologic findings JOE — volume 36, number 8,

Siqueira J, Rôças I,Paiva s, Magalhães K, Guimarães-pinto T 2007c. Cultivable bacteria in infected root canals as identified by 16s rna gene sequencing. Oral Microbiol Immunol 22: 266–271.

Sakamoto M, Rôças I, Siqueira J, Benno Y 2006. Molecular analysis of bacteria in asymptomatic and symptomatic endodontic infections. Oral microbiol Immunol 21:112–122

Möller, A, Fabricius, I., Dahlén, G., Ohman, A & Heyden, G. 1981. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. Scand J Dent Res, 89, 475-84

Sundqvist G. 1976. Bacteriological studies of necrotic dental pulps. odontological dissertations no. 7. Department of oral microbiology, Umea° University, Sweden.

Farzaneh M, Abitbol S, Friedman S. 2004. Treatment Outcome in Endodontics: The Toronto Study. Phases I to V.JoE

Byström A, Sundqvist G 1981 Bacteriological evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy. Scandinavian journal of dental research 89, 321–8

Byström A, Sundqvist G 1983 Bacteriological evaluation of the effect of 0.5% sodium hypochlorite in endodontic therapy. Oral surgery, Oral medicine and Oral pathology 55, 307–12.

Sjogren U, Figdor D. Spångberg I,,Sundqvist G.1991 The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing. International Endodontic Journal 24:3 119-124

Bender IB, Seltzer S. Roentgenographic and direct observations of experimental lesions in bone: I. J Am Dent Assoc 1961;62:708–16

Goldman M, Pearson A, Darzent M. 1972 Endodontic success who's reading the radiograph? Oral Surgery, Oral Medicine, Oral Pathology 33, 432-7

<u>Figures and tables.</u>

-Abbott. P. 2004- Classicisation, diagnosis and clinical manifestations of apical periodontitis. Endodontic Topics. 8, 36-54
-Tables 1 and 2.
-Figure 4.

-Svensater. G. Bergenholtz.G 2004- Biofilms in endodontic infections. Endodontic topics. 9. 27-36
-Figure 2.

-Siqueira. J. Rocas.I. 2009- Diversity of Endodontic Microbiota Revisited. J Dent Res 88 (11): 969-981
-Figure 1 and all figures on page 7.

-Nair.R.- 2000- Apical Periodontitis: A dynamic encounter between root canal infection and host reponse. Periodontology. 13. 121-148
-Figure 3.