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Editorial Notices

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Contributions for inclusion in the Journal should be sent to the Editor, Dr Robert Love, PO Box 647, Dunedin. Deadline for inclusion in the May or November issue is the first day of the preceding month.

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Information for Authors

The Editor welcomes original articles, review articles, case reports, views and comments, correspondence, announcements and news items. The Editor reserves the right to edit contributions to ensure conciseness, clarity and consistency to the style of the Journal. Contributions will normally be subjected to peer review.

It is the wish of the Editor to encourage practitioners and others to submit material for publication. Assistance with word processing and photographic and graphic art production will be available to authors.

Arrangement

Articles should be typewritten on one side of A4 paper with double spacing and 3cm margins. The author’s name should appear under the title and name and postal address at the end of the article. If possible, the manuscript should also be submitted on computer disc, either Macintosh or PC compatible.

References

References cited in the text should be placed in parenthesis stating the authors’ names and date, eg (Sundqvist & Reuterving 1980). At the end of the article references should be listed alphabetically giving surnames and initials of all authors, the year, the full title of the article, name of periodical, volume number and page numbers.

The form of reference to a journal article is:

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Illustrations

Illustrations should be submitted as clear drawings, black & white or colour photographs and be preferably of column width. Radiographs are acceptable. However a black & white photograph is preferred. Illustrations must be numbered to match the text and bear the author’s name and an indication of the top edge on the back. Legends are required for all illustrations and should be typewritten on a separate page.
Introduction

Not all root canal treatments are successful. Success rates vary according to the criteria for evaluation and the multitude of different factors constituting success or failure. In clinical practice success or failure is based on the findings of clinical and radiographic examinations.

In the field of endodontics many diagnostic and treatment decisions are based on radiographic findings. Radiographs are clinical aids that enable visualisation of the periapical area. A particular requirement of endodontic radiography is that this region must be demonstrated clearly and accurately. The paralleling technique is the method of choice for endodontic radiography, as it can result in significantly less distortion than the bisecting angle technique. The use of film holders can overcome problems caused by the presence of rubber dam, clamps, frames, and endodontic instruments. Furthermore the use of holders and beam alignment devices for periapical radiography standardises the x-ray examination, ensuring the set-up is reproducible by taking the guesswork out of beam alignment (Walton, 1996).

Radiographs perform essential functions in three areas: diagnosis, treatment and recall; each requires its own special approach for radiographs to be used to their best advantage. Diagnosis involves not only identifying the presence and nature of pathosis, but also determining root and pulpal anatomy and differentiating other normal structures. Working radiographs are taken during the treatment phase and have applications in determining working length, locating canals and evaluating obturation.

Treatment success is ultimately verified at recall follow-ups. Since failure frequently occurs without clinical signs or symptoms, radiographs are essential to evaluate the periapical status (Zakariasen et al., 1984). At the recall appointment, a radiograph of the treated tooth is taken and compared with radiographs taken at the time of treatment. Changes in radiographic images accompany periapical bone changes. On this basis, clinicians seek to determine whether healing has occurred where expected. In radiographic observation one should be aware that a difference in projection angulation from one radiograph to another may change the size of the lesion’s radiographic image (Goldman, et al., 1972). Therefore an attempt should be made to obtain the same cone angulation on the recall as on the post-operative radiograph. The decision regarding success or failure is important because it will determine the subsequent treatment of the case.

The use of the radiograph depends on an understanding of its advantages and limitations. The advantages are obvious in that it allows a privileged look inside the jaw, information of which is essential and cannot be gleaned from other means. One of the major limitations of radiographs is that the image is limited to two dimensions and often the third dimension needs to be visualised. Perhaps the greatest shortcoming of conventional radiographs comes from classic bone studies, which have demonstrated that a significant amount of osseous destruction must occur before a lesion is detectable radiographically. Bender and Seltzer (1961a) created artificial lesions in the mandibles and maxillae of human cadavers. They concluded that the lesions, which originated from cancellous bone, could not be detected radiographically until they extended into the cortical bone or to the junction between cortical and cancellous bone. Moreover, Shoha et al. (1974) found that the true lesions were always larger than their radiographical images. This suggests that it is impossible to detect from radiographs alone the existence and/or extent of periapical lesions if the lingual or buccal cortical plate is not involved. This factor must be considered in evaluating teeth that become symptomatic, but show no radiographic changes since inflammation and resorption affecting the cortical plates must be sufficiently extensive before a lesion is visible radiographically. In contrast to these findings are those of Pitt Ford (1984), who investigated the radiographic detection of periapical lesions in dogs and found that cortical plate involvement was not necessary.

Studies involving more recent radiographic techniques such as Digital Imaging and Radio-

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visiography (RVG) have demonstrated varying results in early detection of lesions. Kullendorff et al. (1988) found that subtraction radiography using digitised radiographs improved the detection of small lesions in the periapical area, but for deeper lesions there was no advantage over the conventional technique. More recently, Tirrell et al. (1996) compared conventional radiography to digital imaging in detecting chemically created lesions in human cadaver jaw specimens. It was found that digital radiography was more effective in demonstrating lesions when they were contained by the lamina dura and the cancellous bone. However, to date the majority of studies have shown digital imaging to confer no real advantage (Furkart et al., 1992), thus this discussion will be limited to conventional radiography.

**Criteria for success and failure**

Numerous studies have been published evaluating success and failure of endodontic treatment reporting success rates varying from 74-94% (Strindberg, 1956; Ingle, 1965; Kerekes & Tronstad, 1979; Sjögren et al., 1990; Ray & Trope, 1995). The wide range in success rate depends on differences in experimental design and clinical procedures, criteria for evaluation of periapical healing, and the length of post-operative observation period. Other variables such as observer bias (with varying criteria for success), bias in interpreting radiographs, varying levels of patient compliance to return to recalls, subjectivity of patient response and host variability in responding to treatment make interpretation of different studies difficult. Therefore, studies cannot be compared and each case should be assessed individually with consideration given to all relevant factors.

There is a lack of consensus for assessing success or failure. Radiographic analysis forms only part of the evaluation process. What constitutes endodontic success or failure is based on histological, clinical and radiographic criteria.

**HISTOLOGICAL EVALUATION**

Histological evaluation is the only accurate means of assessing success. The ideal criteria for success of the endodontically treated tooth is absence of any inflammation around the root and the reconstitution of periapical structures. This is indicative of healing. At present histological examination is the only means to detect inflammation. Brynolf (1967) investigated post-mortem specimens in an evaluation of periapical healing and suggested that histological analysis is the most stringent assessment of post-treatment healing. She found that inflammation was totally absent in only 7% of cases. This suggested that most endodontic treatment did not result in perfect healing, i.e. no inflammation. Because this study involved cadaver specimens, there was no pre-operative or clinical history and the quality of the root fillings was unknown. Furthermore, it is an older study; rubber dam may not have been used during treatment and the importance of coronal leakage was probably not fully appreciated. Thus the high incidence of inflammation may not accurately reflect modern endodontic techniques. In a later study, Andreasen and Rud (1972) used histological examination as a tool for investigating the reasons for failure in root-filled teeth. They operated on endodontically treated cases with persistent periapical radiolucent lesions and analysed biopsy specimens by histobacteriology. They reported that the presence of bacteria in the root canal or dentinal tubules was related to an inflammatory response in the periapical tissues. This confirms the aetiology of inflammatory periapical disease of endodontic origin.

Routine histological evaluation of periapical tissues for patients is unethical and impractical; thus clinical and radiographic criteria are the only means of assessing success and failure.

**CLINICAL EVALUATION**

Clinical criteria for success often rely on subjective findings, such as pain or discomfort on percussion, that are subject to individual variation. Criteria as outlined by Bender et al. (1966) include the absence of pain, disappearance of sinus tract, no loss of function, and no evidence of soft tissue destruction, including periodontal probing defects and abnormal mobility. Clinical failure is usually associated with acute signs and symptoms associated with infection. However, many clinically asymptomatic teeth may have histopathological changes at the root apices along with minimal or extensive radiographic changes. Even in the presence of an apparently normal radiographic appearance, a clinically asymptomatic tooth may exhibit histopathologic changes in the periradicular tissues (Bender et al., 1966).

As stated above, the tooth should be intact with a functional restoration. For most posterior teeth that have been endodontically treated, full cuspal coverage has been recommended to limit cusp
flexion and subsequent cusp fracture (Sorensen and Martinoff, 1984). However, a tooth does not necessarily require a crown to be functional, e.g. an over-denture abutment. Restorative aspects are important since many root canals fail either because of coronal leakage, which ultimately compromises the root canal seal (Torabinejad et al., 1990) or because of teeth that have fractured due to inadequate restorations (Sorensen and Martinoff, 1984). Ray and Trope (1995) evaluated the relationship of the quality of coronal restorations and of the root canal obturation on the radiographic periapical status of endodontically treated teeth. They examined 1010 root-filled teeth and concluded that the technical quality of coronal leakage, which ultimately compromises the root canal seal (Torabinejad et al., 1990). However, a tooth does not necessarily require a crown to be functional, e.g. an over-denture abutment. Restorative aspects are important since many root canals fail either because of coronal leakage, which ultimately compromises the root canal seal (Torabinejad et al., 1990) or because of teeth that have fractured due to inadequate restorations (Sorensen and Martinoff, 1984). Ray and Trope (1995) evaluated the relationship of the quality of coronal restorations and of the root canal obturation on the radiographic periapical status of endodontically treated teeth. They examined 1010 root-filled teeth and concluded that the technical quality of the coronal restoration was significantly more important than the technical quality of the endodontic treatment for apical periodontal health. Thus it is important to employ restorative techniques that provide a coronal seal and protect remaining tooth structure.

**RADIOGRAPHIC EVALUATION**

Radiography is the other criteria in judging success. The radiographic image should be evaluated for signs of bone lesions or root resorption. Success is indicated when a radiolucent bone lesion of endodontic origin is diminished in size or completely resolved, or does not develop after endodontic treatment. However, a decrease in size of a lesion does not necessarily indicate success. The timeframe for healing must be considered (Ørstavik, 1996). Often criticised is the Washington Study by Ingle (1965). This was a non-standardised study, open to misinterpretation. The authors examined a series of radiographs of root-filled teeth associated with failure. Success rates of 91.5% were reported. However, since treatment was performed by different groups and only a two-year follow-up period was employed, the results of this study should be viewed with caution. This study has been further censured for attributing the main cause of failure to incomplete obturation and root perforation. In no case was retained root canal infection listed as the primary cause of failure. Undoubtedly in most failure cases bacteria are ultimately the cause of failure (Sjögren et al., 1990). In the Washington study it is possible that bacteria may have been left in the canal, as canal sterility was not assessed, and that microleakage via the crown of the tooth could have occurred. This important aspect of endodontic treatment has only relatively been recognised (Torabinejad et al., 1990).

Many practitioners rely solely on radiographic analysis as a determinant of success. Resorting only to radiographic evaluation may allow pathosis that is clinically evident, but produces no radiographic manifestation to be overlooked, thus both should be assessed together. In a study of surgical healing, Andreasen and Rud (1972) suggested that periapical inflammation may be characterised radiographically by a periapical radiolucent area possessing any one of the following features: 1) width more than twice the periodontal ligament; 2) a circular or semi-circular outline with a funnel-shaped extension into the periodontal space; and 3) a collar-shaped increase of the lamina dura lateral to the root surface with normal bone trabeculation outside the radiolucent area.

Strindberg (1956) provided strict radiographic criteria for success, which have been used in later studies (Sjögren et al., 1990). In this classical study, Strindberg (1956) radiographically followed up teeth he had root-filled for up to 10 years after treatment. Treatment was successful when: 1) the width and structure of the apical lamina dura was normal; or 2) the periodontal contours were widened only around an excess of filling material. All cases in which these criteria were not fulfilled were judged unsuccessful. These criteria question the claims of Bender and Seltzer (1961a) regarding the need for cortical bone involvement for lesions to be visible. In this situation cortical bone is not lost. A thickened periodontal ligament space is seen which is unrelated to cortical bone. Histological confirmation of the radiographic criteria developed by Strindberg (1956) was substantiated by Brynolf (1967), who compared radiographs and histology. She tested the reliability of x-ray records to distinguish between healthy and diseased periapical tissues. The results showed that 98% of cases with periapical lesions that were histologically identifiable could be detected by examining the continuity of the lamina dura and the shape and width of the periodontal ligament.

Strindberg’s work provided fundamental data on healing patterns and the minimum length of observation period required to evaluate the outcome of endodontic treatment. Strindberg (1956) suggested that healing tendency is a sign that the balance between the irritants and the host defence is tipped positively in favour of the latter. However, he also said that a reduction in the radiographic size of the apical lesion is no guarantee for eventual complete healing. Most lesions resolved within 4-5 years after therapy, although some cases may take as long as 10 years to heal. The size of the pre-operative lesion had no influence on the outcome of treatment except if it was a retreatment. Late failures
cannot be discounted, and thus studies with observation periods less than four years may include cases which have not attained a stable periapical condition, and so the results may be erroneous. Later studies by Bryström et al. (1987), Sjögren et al. (1990) and Ørstavik (1996) support the 4-5 year observation period for healing. Furthermore, Ørstavik (1996) went on to suggest that the process of healing could be diagnosed earlier. He reported that 88% of roots with pre-operative chronic apical periodontitis healed after endodontic treatment and that radiographic evidence of healing for most cases was detectable at one year.

Factors other than root canal infection may contribute to chronic apical periodontitis later than one year after treatment. A more recent study, with a similar design as Strindberg (1956), was undertaken by Sjögren et al. (1990). This group looked at factors affecting the long-term results of endodontic treatment. Their results were in agreement with Strindberg's original observation that the results of treatment were directly dependent on the pre-operative status of the pulp and periapical tissues with a higher rate of success for those cases having no pre-operative radiolucency. Furthermore, they concluded that the possibility of instrumenting the root canal to its full length and the level of the root filling significantly affected the outcome of treatment. Chronic apical periodontitis, as diagnosed radiographically, dominates the evaluation of endodontic success or failure. Significantly, evidence of complete radiographic regeneration of periapical structures does not always occur. An apical radiolucency that remains unchanged in size or increases in size indicates failure. Only a few studies have investigated the possible aetiology of such failures, and these reports have revealed that there are four factors that may contribute to chronic failure.

1. Intraradicular infection. Nair et al. (1990b) investigated therapy-resistant periapical lesions with light and electron microscopy and found that the major cause of root canal treatment failure was micro-organisms persisting in the apical part of root canals. Enterococcus faecalis has been commonly recovered from root canals of teeth where the previous treatment has failed (Molander et al. 1993).

2. Extraradicular infection. Bacteria can prevent healing of periapical lesions by establishing themselves in the periapical tissues. Two species, Actinomyces israelii and Propionibacterium propionicum (formally Arachnia propionica) are known to have the ability to establish such extraradicular infections in the form of periapical actinomycosis (Nair and Schroeder 1984, Sjögren et al. 1988). The presence of infected dentine or cementum chips in the periapical tissues has also been shown to be associated with impaired healing (Holland et al., 1980).

3. Foreign body reaction. In some cases, no micro-organisms can be detected, and treatment failures can be attributed to a foreign body reaction of periapical tissue to remnants of excess filling material or contaminants such as talcum powder associated with gutta percha (Nair et al., 1990a & b).

4. Cysts. Results of recent studies indicate that intrinsic factors such as the presence of a true radicular cyst, sometimes containing cholesterol crystals, can adversely affect healing, resulting in a lesion refractory to conventional endodontic therapy (Nair et al., 1993, 1996).

The radiographic healing pattern after periapical surgery differs from that observed after conservative endodontic treatment by containing the so-called 'scar tissue' group associated with bicortical bone loss. This group is characterised by a decreasing rarefaction with an irregular outline extending angularly into the periodontal space. Further healing may include the formation of a lamina dura around the apex, separating the rarefaction from the tooth (Rud et al., 1972). Molven et al. (1996) followed 24 cases treated by periapical surgery and classified radiographically as incomplete healing. At the conclusion of the study, 22 of the cases were still recorded in the same healing group, but with a reduction of the defect in bone. The group concluded that cases clearly showing features of incomplete healing (scar tissue) at the regular follow-up one year after surgery can be regarded as successes and don’t require periodic clinical and radiographic evaluation. Very few cases will fail later; they will either heal or remain in the scar tissue group. This conclusion differs from Rud et al. (1972) and Gutmann and Harrison (1991), who recommend that cases with incomplete healing one year after surgery should be followed regularly because late signs and symptoms of inflammation may occur in some cases.

The importance of radiographic evaluation in determining endodontic success or failure cannot be over-emphasised. However, despite being such a universal tool in the assessment of treatment results, the limitations of visual interpretation must always be considered. Observer bias influences the interpretation of radiographs. Goldman et al. (1972,
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Radiographs are indispensable to endodontic treatment and determination of success. However, their limitations must always be considered. Radiographic analysis is not the ultimate technique, but an adjunctive tool that is open to variable interpretation and observer bias. Radiographers should be used for the evaluation of success and failure in combination with clinical criteria. Because it is not usually feasible to histologically evaluate the presence of periapical inflammation, long-term radiographic evidence of healing with longer follow-up periods is recommended.

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Endodontic Treatment Complicated by Atypical Odontalgia

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Introduction

Atypical odontalgia (AO) manifests as a chronic, moderate to severe pain that mimics toothache. It is not alleviated by dental intervention and may be unaffected by local anaesthetic (Bates and Stewart, 1991). The difficulty of diagnosing AO is compounded by a lack of clinical or radiographic evidence supporting a physical cause for the pain. Pharmacologically, AO has been linked to sympathetically maintained pain suggesting that it is a neuropathic condition. Relief of pain from AO has been achieved with tricyclic antidepressants and more recently with capsaicin (a capsicum extract) which acts on afferent C fibres through substance P.

Clinical

A 39-year-old female patient presented with a six month history of continuous dull pain in tooth 16 and a history of sharp pain of short duration (minutes) in tooth 26, particularly with hot/cold stimuli. The patient expressed a strong conviction to retain the teeth, despite a deep periodontal pocket associated with tooth 26. Radiographically, there was a slight rarefaction at the apex of the palatal roots of teeth 16 and 26 but these teeth were not tender to percussion. Analgesia was attained using a posterior superior dental block together with intraosseous delivery of local anaesthetic. After two appointments the canals in teeth 16 and 26 were clean and free of any sign of infection or inflammation. However, the pain was unabated and after a visit to her general medical practitioner the patient was taking 60mg morphine sulphate twice per day.

The patient was diagnosed as having AO and accepted advice to start taking amitryptiline 25mg at night and increase to 50mg. At review after 9 days, the patient was taking 50 mg amitryptiline daily. Improvement was reported in the control of pain and morphine intake had been reduced to 60mg at night. At this stage topical capsaicin 0.025% w/w (Zostrix, Knoll Australia Pty Ltd.) was provided. Surface anaesthetic was first achieved with 5% Xylocaine ointment. The patient was instructed to place topical anaesthetic in the buccal sulcus adjacent to the upper first molars, four times daily, then rub capsaicin over the area and to increase the amitryptiline to 75mg daily. Within two days of increasing the amitryptiline dosage and applying capsaicin topically, the patient stopped taking morphine. After twelve days using capsaicin and 21 days of amitryptiline therapy, the pain was reported to be completely resolved and the patient discontinued all medication. Completion of root canal treatment has proceeded uneventfully.

Summary

This report describes the clinical presentation and treatment of a patient with atypical odontalgia. While patients undergoing endodontic treatment may experience some inter-appointment pain, it is expected that this will last only two to three days and should be controlled by conventional analgesics. The persistence and severity of pain in the absence of an identifiable cause warrants consideration of non-endodontic causes such as atypical odontalgia. Capsaicin acts to deplete substance P and has been effective in the treatment of post-herpetic neuralgia. While definitive double-blind studies will be needed to establish the efficacy and long-term success of treatment of AO with capsaicin alone, this treatment presents the possibility for non-invasive, cost-effective, localised therapy. The recent paper by Vickers et al. (1998) provides a comprehensive report on the diagnosis and treatment of AO.

References


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Discuss how the progression of endodontic knowledge has dismissed the theories of focal infection

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This paper was placed second in the 1998 Australian Society of Endodontics undergraduate essay contest. See Society news for further details of the competition. Editor.

Introduction

In the early 20th century, oral foci of infection, or oral sepsis, was thought to cause many systemic diseases. This belief was based on focal infection theory. It was an old theory but both medical and dental communities felt its impact at the time. William Hunter, a British physician, was largely credited for the introduction and application of the focal infection theory to dental diseases (Hunter, 1918). He based his arguments largely on clinical observations and little on scientifically determined fact (Grossman, 1976, 1982a, 1982b; Dussault and Sheiham, 1982). Hunter argued that during and following restorative dental treatment, bacteria ended up in the dentinal tubules and eventually leaked toxins and spread into other parts of body via blood or lymphatic drainage. His list of disease caused by focal infection included eye infection, arthritis, heart disease, endocrine and gastro-intestinal disorders, mental illness and pregnancy complications (Hunter, 1918; Burket and Burn, 1976; Dussault and Sheiham, 1982).

In 1910, Hunter addressed the medical faculty of McGill University, Montreal, Canada, on the septic potential of diseased teeth. In the lecture, Hunter criticised dental restorations as “a perfect gold trap of sepsis”. Although the lecture was addressed before the medical faculty, it was also published in a popular journal at the time. Interviews with Hunter appeared in newspapers with banner headlines. Soon the alarmed public rushed to their physicians to inquire if their bad teeth were the cause of whatever ills they had, and rushed to their dentists to have them out (Grossman, 1976).

By 1920, focal infection theory had a significant number of dental and medical professionals accepting part or all of it as fact. This popularised theory resulted in countless extractions of diseased teeth in order to cure a multitude of diseases (Dussault and Sheiham, 1982; Silver, 1997). As a consequence, the growth of conservative dentistry, as well as endodontics, was stunned by the focal infection theory.

Some began to question the validity of the arguments that supported the focal infection theory. Extensive and controlled experiments failed to reproduce the similar data that supporters of focal infection theory published.

In order to discuss how the progress of conservative dentistry, especially endodontics, dismissed the focal infection theory, it is essential to understand how such theory was developed with respect to the scientific and social history.

Social history of focal infection theory

Hunter (1918) criticised the dentists who practised conservative dentistry and accused them of “conserving sepsis under gold crowns”. This promoted extensive scientific investigations in the United States, partly because conservative dentistry was largely practised in the United States. In Britain, however, conservative dentistry was underdeveloped and most dental treatment consisted of extractions and fitting of dentures. Perhaps because of this, the work of Hunter was more readily accepted in Britain than in the United States. Dussault and Sheiham (1982) report that Hunter’s theory in Britain coincided with a time when competition in dentistry was increasing and qualified practitioners campaigned for recognition and hoped for prohibition of unregistered practice. It was convenient that the theory of focal infection provided justification for more qualified dentists. It also fuelled the claim
that dentistry should be regarded as an important branch of medicine. Only when Parliament prohibited the practice of unregistered dentistry and allocated enough funds for the training of qualified dentists, did British dentists turn to confront the focal infection theory.

**Scientific history of focal infection theory**

Physicians and scientists during mid-19th century extended Pasteur’s germ theory on disease to explain the causes of many different systemic diseases (Burnett et al., 1976). They also observed that a focus of infection, located anywhere in the body, could pass into the blood and spread to other parts of the body. This meant that infected teeth, tonsils, prostate glands or any localised foci of infection were responsible for a wide range of systemic diseases (Grossman, 1976). Hunter (1918) emphasised the importance of oral sepsis as focus of infection. He criticised gold and root filled teeth as the focus of oral sepsis. Following that, most dentists extracted diseased teeth regardless of their vitality with a view to eliminate oral sepsis and to prevent systemic diseases (Easlick, 1951).

**Microbiological experiments**

The initial microbiological investigations supported the focal infection theory. Rosenow (1915), using animal experiments, suggested that streptococci isolated from oral septic foci demonstrated ‘elective localisation’ – a theory that bacteria from oral sepsis were likely to infect certain tissues in other parts of the body. The significance of this idea was that streptococci from oral sepsis were likely to cause infection in certain parts of body such as heart, joints and kidneys causing systemic diseases. The experiments were mainly conducted by culturing whole extracted teeth in growth media. Many authors (Fish and Maclean, 1936; Easlick 1951) criticised the culturing of extracted teeth as a diagnostic test and warned of the possibility of culturing normal mouth flora. Microbiological experiments conducted from late 1930’s and onwards produced some conflicting results. Fish and Maclean (1936) showed that non-infected extracted teeth also demonstrated streptococci and concluded that it was due to streptococcal contamination from gingival sulcus during the extraction procedure. Gunter et al. (1937) showed that extracted root surface of vital sterile pulps always showed contamination. Burket and Burn (1938) showed that by contaminating the gingival sulcus with non-pathogenic organisms, they were able to recover these organisms in blood following tooth extraction. These investigators suggested bacterial contamination from gingival sulcus as the cause of pulp contamination. They also proved conclusively that the pulps of normal, vital teeth were always sterile and that the earlier reports based on culture of extracted teeth had to be discarded. Sommer and Crowley (1940) developed a technique to obtain uncontaminated cultures from root canals of teeth in situ and reported that out of 14 teeth that showed radiographic evidence of periapical granuloma, 10 of these were sterile. Ostrander and Crowley (1948), using the same technique as Sommer and Crowley, reported that 46 out of 119 periapical lesions were sterile.

Easlick (1951) thoroughly evaluated the effects of dental foci of infection on health and concluded that the early correlation of endodontically treated teeth as foci might be due to imprecise research protocols and poor aseptic techniques. Grossman (1982a, 1982b) summarised three major flaws associated with the earlier experiments: 1) There was no proper control. 2) The doses of bacteria injected into the experimental animals were too massive. 3) The teeth involved in the experiments were contaminated with normal oral bacteria during the extraction procedure. He concluded that the association of non-vital teeth with systemic disease, as stated by the supporters of the theory, was unjustified. Torabinejad et al. (1981) also confirmed that there was no significant difference in the serum concentration of immune complexes and complement between patients with periapical lesions and those without.

**Understanding of pulp pathology**

Rosenberg (1962) demonstrated that streptococcal infection of necrotic pulp in the cat resulted in the increased antibody titre to the bacteria. Kakehashi et al. (1965) exposed the dental pulp of conventional and germ-free rats to their oral flora, and reported pulpal and periradicular lesions in conventional rats, but not in germ-free rats. Moller et al. (1981) investigated the significance of bacteria in the development of periradicular lesions by sealing noninfected and infected pulps in monkeys. After 6 to 7 months, clinical, radiographic, and histological examination of teeth sealed with non-infected pulps showed an absence of pathology.
in periapical tissues, whereas teeth sealed with necrotic pulps containing certain bacteria showed periapical inflammation. Lin et al. (1991) reported that bacterial infection of the root canal might arise from bacteria originally present, or introduced during instrumentation; defective seal or exposed dental tubules; or bacteria entering the canal through bacteraemia (anachoresis – spreading of viable streptococci from the oral focus). Yamasaki et al. (1994) demonstrated that pulp necrosis progressed gradually from upper portion of pulp to the apex. They observed that necrotic pulp was not able to eliminate the bacteria but temporarily slowed the spread of the infection; and reported that when the bacteria or bacterial products from the necrotic pulp diffused into the adjacent bone, periapical lesion developed. Currently it is understood that a periapical lesion (granuloma) is an accumulation of inflammatory and reactive elements (inflammatory and endothelial cells) which result in bone resorption and replacement by chronically inflamed tissue (Soames and Southam, 1996). Stashanko (1990) suggested that periapical granuloma was a host defence mechanism that stopped the spread of bacteria and bacterial components into the adjacent bone. This was based on the hypothesised role of cytokines and osteoclast-produced coupling factors as inhibitors of reparative bone formation. He speculated that the resorption of bone prevented direct hard tissue invasion and development of osteomyelitis.

**Sequelae to periapical granuloma**

Soames and Southam (1996) described six possibilities with untreated periapical granuloma. 1) The granuloma will grow without any symptoms. 2) It will display symptoms of acute periapical periodontitis. 3) It will form an acute periapical abscess with clinical symptoms such as rapid onset of pain and swelling. 4) It will irritate the epithelial rests of Malassez, causing proliferation and development of radicular cyst. 5) Osteosclerosis. 6) Hypercementosis. It was suggested that if the cause of the abscess was left untreated either by extraction of teeth, endodontic or antibiotic treatment, the pus was likely to spread into the adjacent bone, periodontal ligament space or tissue spaces (Soames and Southam, 1996). This contrasts to the original focal infection theory that stated metastasis of septic foci via blood or lymph to distant organs.

**Sub-acute bacterial endocarditis**

Hunter (1977) attempted to extend the focal infection theory to the pathogenesis of sub-acute bacterial endocarditis. He suggested it may be due to 1) anachoresis, 2) spread of bacterial products and components via circulation, 3) anaphylactic hypersensitivity, or 4) autoimmune mechanisms. He reported that sub-acute bacterial endocarditis patients were hypersensitive to streptococcal products (eg. streptolysin O) and demonstrated a common antigen between streptococci and mammalian heart. This is thought to be due to cross-reaction of the antibodies against myocardial cells following rheumatic fever (Kumar et al., 1992). The host would then become susceptible to bacterial endocarditis following transient bacteraemia caused by dental procedures (Kumar et al., 1992; Sonis et al., 1995). Transient bacteraemia is known to occur following dental procedures such as endodontic procedure (Debelian et al., 1994), and antibiotic prophylaxis is indicated for patients with history of rheumatic fever. Other diseases such as rheumatoid arthritis have not been proven to be associated with root filled teeth. No clear aetiology had been found and some suggested a humoral immune mechanism as a possibility (De Clerk, 1995). Bacteraemia is expected with any dental instrumentation, however, no study so far has shown that it could be due to the presence of non-vital or root filled teeth (Silver, 1997).

**Progress in endodontic procedures**

Grossman (1976, 1982b) and Spangberg (1998) credited the technical advancement of endodontics as a progression of endodontic knowledge. Grossman (1976, 1982b) described the advent of periapical radiographs, local anaesthesia, sterilisation, rubber dam and precision endodontic instruments as the attributes of successful endodontic treatment. He also noted the change in the clinical practice of root canal cleaning and shaping, and obturation techniques using biocompatible materials as being one of the important changes that increased the success rate. He also emphasised the importance of research in endodontics as it allowed better understanding of the interaction of the materials and the living tissue. The use of calcium hydroxide intra-canal dressing, in addition to mechanical
cleaning and irrigating, has also helped to improve the sterility of the root canal during endodontic treatment (Sjogren et al., 1991).

**Endodontics and other disciplines of dentistry**

The advances in other disciplines of dentistry also assisted the progress in endodontic knowledge. For example, it was shown that periodontal disease influenced the condition of the pulp (Lindhe, 1996). The bacterial products and substances released by inflammatory processes in the periodontium were shown to gain access to the pulp via exposed accessory canals, apical and furcal foramina and dentinal tubules. It was also shown that bacterial products could leak through apical or accessory canals to the periodontium from unfilled spaces in the root canal and cause breakdown of periodontal tissues. Strong antiseptic drugs used for root canal disinfection and pulp devitalization could cause severe damage if they leaked out into the periodontium. Hence, combined endodontic and periodontal treatment strategy is recommended in recent texts when there is a doubt as to whether a lesion was of endodontic or periodontal origin, or both (Lindhe, 1996; Walton and Torabinejad, 1996). Recent articles suggest that an established chronic periodontal disease provided a burden of endotoxins and inflammatory cytokines that initiated and exacerbated atherosclerosis (Beck et al., 1996). They also reported that majority of severe periodontitis were associated with a select few microbial species. The authors stressed that the bacterial virulence induced a substantial systemic effect that led to thrombosis. Loesche et al. (1998) reported on the relationship between dental diseases and coronary heart disease in elderly U.S. veterans, and pointed to a variety of oral health variables, including periodontal health, as risk indicators for coronary heart diseases. Although this finding has implicated periodontitis as an oral septic focus, there appears to be no direct relationship between the two conditions.

**Modern endodontic principles**

Bacterial culture studies of periapical lesions show that endodontic therapy, with proper aseptic technique, does not result in root canal contamination. They also showed that many cultures were sterile. These findings prompted the sterilisation of endodontic instruments and the use of antimicrobial chemicals in the root canal as well as chemomechanical instrumentation (Sjogren et al., 1991). Hence, the modern endodontics is based on extensive research and understanding of the aetiology of pulp pathology. Strict adherence to this principle showed that proper aseptic technique was enough to lower the chances of bacterial infection of the pulp (Al-Kandari et al., 1994).

Better knowledge of the tooth and pulp anatomy has also contributed to success. For example, modern endodontic textbooks described that the root canals were not uniform and that sometimes they had smaller lateral canals and apical deltas (Walton and Torabinejad, 1996). Bacteraemia used to be a risk factor in endodontics, especially with patients who had a history of rheumatic fever, but it was shown that it could be reduced or avoided with proper antibiotic prophylaxis, sterilised instruments and confining all endodontic instrumentation to within the canal (Erhmann, 1977). Endodontic treatment is now considered as a safer option for some patients with medical problems. Dentists now refer such patients for endodontic treatment, rather than exodontia, because of reduced trauma and haemorrhage associated with properly root filled teeth (Erhmann, 1977; Walton and Torabinejad, 1996). Sjogren et al. (1990) reported an over 90% success rate of modern endodontic treatment. They reported a slightly lower success rate for the roots with previous periapical lesions but this was thought to be due to improper root canal instrumentation where dentine and cementum chips or root filling material were left in the lesion. The report suggested that properly root filled teeth could not be a source of infection.

**Conclusion**

The focal infection theory was the architect of its own destruction, simply because it was built upon weak scientific facts. As Grossman (1976) noted, this was the difference between the empirical observations of the past and current scientifically determined knowledge. The demise of the theory prompted both the medical and dental professions to develop well controlled and conducted scientific experiments. Supporters of the old theory still refer to old, outdated research to support their claim that root canal treatment is potentially hazardous. George Meinig’s book, Root Canal Cover-Up (1994), as reviewed by Silver (1997), was described as an example of such ignorance. It is also important for both medical and dental professions to be
able to critically evaluate current reports that support the focal infection theory. In order to end the confusion surrounding the validity of the theory, there is a need for more current experiments. With the advances in molecular microbiology, it is now possible to measure quantitative variables such as serum concentrations of antibody levels. Such experiments would yield statistically significant data that many clinicians would accept. The focal infection theory was dismissed by progress in scientific research and clinical procedures. However, it should be credited for prompting dentists to use more precise and aseptic procedures in the placing of any dental restoration in, or over, a diseased tooth.

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LETTER TO THE EDITOR

Problem-Solving in Endodontic Radiography: Clinical Hints

Dear Sir,


Since designing the Snapex film holder in 1976 and writing the manual that has accompanied the kits since 1988, I have confronted many clinical challenges similar to the one described by Chandler and Anderson and have developed some additional techniques that might be of assistance in similar cases.

In Chandler and Anderson’s article, illustrations 2 and 4 (pre-operative and post-obturation respectively) are acceptable films. Regarding the pre-operative film, the authors note that there is insufficient detail of the buccal canals. When this occurs, the film should be repeated, but with an increased exposure time. It is a clinical finding that a standardised exposure time will not give a satisfactory result for all patients.

Illustration 4 demonstrates the dilemma that despite accurate working lengths obtained with an electronic apex locater, the quality of the root filling is a matter of chance without accompanying working films. Illustration 4 shows incomplete obturation of the distobuccal root. However, the clinician could not verify this until after the treatment was completed. Unfortunately, the authors did not discuss this finding and the possible clinical implications. This could suggest that this result represents an acceptable standard of endodontic treatment. It does not, and I doubt that it was the authors’ intention to suggest otherwise. What additional techniques might have yielded acceptable working films in this case?

Illustration 3, the working film, is described by the authors as the best radiograph taken during the case. Clinicians confronting a similar situation should consider the following points. Working films for posterior teeth like illustration 3 must be stabilised in the mouth to place the film in the most apical position. The best way to achieve this is by having the patient close their teeth together on three cotton rolls bound with dental floss placed anterior to the treated tooth to stabilise the film holder in the most apical position and provide space for the file handles and the clamp bow.
anterior to the tooth in treatment (Figure 1). The cotton rolls also provide space for the file handles and any interference from the bow of the clamp when the patient closes (Figures 2 and 3). Ensure that the patient closes firmly on the cotton rolls with their teeth (unless so instructed, many patients will only close their lips and the film will be cut off apically). In my experience, alternative methods such as the patient holding the Snapex with their hand or closing on the film holder without interocclusal stabilisation will result in a large percentage of unsatisfactory films similar to illustration 3. The cotton roll ‘hint’ will yield a diagnostic film almost every time. Illustration 3 also suggests that the clamp bow may be protruding too far above the occlusal plane and impeding maximum closure. Clamps should be positioned so that the bow is as close as possible to the occlusal plane of the treated tooth (Figure 1).

However, there will be failures in some cases of very long teeth or anatomic variations (Figure 4). Can we compensate in these situations? The answer is yes. Repositioning the film to a vertical configuration will usually give a satisfactory result (Figure 5). An increase in the vertical height of the film holder of several millimetres may be achieved by extending the rod from its normal position of flush with the film holder (Figures 6 and 7).

Figure 4: An example of teeth 35 and 36 that are too long for the conventional horizontal film position.

Figure 5: Teeth 35 and 36 taken with the film in the vertical position.

Figure 6: The 105° aiming rod flush with the film holder.

Figure 7: The 105° aiming rod extended, creating several millimetres of increased vertical height.

be positioned more towards the midline to take advantage of the height of curvature of the palate. The Snapex is designed on the principle of the ‘modified paralleling’ technique. That is, the film is positioned as parallel as possible to the resultant long axis of the tooth and with as little distortion as possible. As Chandler and Anderson state, true parallel radiography in the maxilla is not always possible. It is for this reason that the 105° increase in vertical angulation helps to compensate for the

anatomic variation. In many cases it is necessary to increase this vertical angulation even more for a successful image. Anecdotal data from our endodontic practice suggests that the Root ZX electronic apex locater and the modified paralleling technique give very similar canal measurements. However, this should be the subject of a controlled study to test these impressions.
In cases of a shallow palate or a gagger, the paedodontic film is extremely useful. One’s first reaction to this film is that it is too small for periapical views of posterior teeth in adults. In fact, after allowing for the jaws of the film holder, the ‘working’ part of the film in the horizontal will be 20-22mm, which is more than sufficient for most teeth (Figure 8). The smaller profile of the film makes it ideal for difficult cases (Figures 9-10). The film may also be used in a vertical configuration.

While it is not possible to touch on all aspects of overcoming difficulties in endodontic radiography, I hope the above comments will be of some assistance.

Yours truly,
Steven A. Cohn, Endodontist
60 Park Street, Sydney 2000

Figure 8: The paedodontic film compared to the standard size 2 film.

Figure 9: The 27 is to be treated. The patient has a shallow palate and is a gagger. The standard size 2 film is unsatisfactory.

Figure 10: The obturation of the 27. Paedodontic films in the Snapex holder were used successfully without patient discomfort for all phases of treatment (film enlarged for publication purposes).

REPLY

Dear Sir,

Thank you for inviting me to comment on Dr Cohn’s letter. I welcome his comments, as even in the recent editions of endodontic texts there is a dearth of really practical advice on the problems of endodontic radiography.

The Snapex system has been universally adopted at the School and I remember your correspondent visiting Dunedin about 10 years ago in order to film the ‘Snapex video’ he produced here for the Gunz Company.

Both the video and manual on the Snapex are a core part of our endodontic teaching at undergraduate and postgraduate levels, and the triple cotton roll/floss technique shown in Figures 1 and 2 of the letter should be familiar to readers who graduated this decade. We used this method to take our working film, but it proved to be unsatisfactory. As Dr Cohn states, the cotton roll ‘hint’ will yield a diagnostic film almost every time. Our un-usual patient was the exception; there is always one, and I am pleased to say I haven’t encountered such a problem in the two years since.

I commend the advice in the letter which, with its illustrations, is almost a paper in its own right. I would encourage Dr Cohn to produce a second edition of his 1998 manual to include the additional techniques he has developed, and any reader not using a film holder/beam alignment device as part of their endodontic practice to purchase a Snapex kit.

Nick Chandler
Department of Oral Rehabilitation
News

Student News from the School of Dentistry
Australian Society of Endodontology Essay Competition

Congratulations to Dr Travis Blood, who won first prize in the 1998 competition. Travis’s essay, “Discuss how the progression of endodontic knowledge has dismissed the theories of focal infection”, was written when he was a final year student. He won an inscribed commemorative plaque and $A1000, and the essay will be published in the Australian Endodontic Journal.

Further success for Otago students came from Chong Yun Shin who, as a fourth year student, wrote an essay that was judged second in the competition. The competition is open to all students in the last two years of their courses in Australian and New Zealand Dental Schools. The Society extends congratulations to Travis and Chong on behalf of the Society members.

New Zealand Endodontic Society Prize

Congratulations to Fiona Manson, who received the Society’s prize for 1998. This prize is awarded to an undergraduate student who demonstrates high standards in clinical endodontic skills and academic knowledge during the fourth year of the BDS degree. Fiona received a copy of Pathways of the Pulp.
Postgraduate News

Congratulations to Dr Marcelo Polendo on successful completion of his MDS in Endodontics. Marcelo has returned to Mexico, where he plans to set up specialist practice and teach endodontics in a part-time academic position.

Society News

New Zealand Endodontic Society Research Fund

Applications are invited from members for grants to support research in the field of endodontics. The Grants Board will consider applications for funds to support new or on-going projects or for any other purpose that will result in the acquisition and/or dissemination of new knowledge relevant to endodontics. Full details are available from the Secretary.
### Australasian Calendar of Endodontic Events

#### NEW SOUTH WALES

**President:** Dr Peter Duckmanton, Tel. (02) 9293 3284  
**Secretary:** Dr Kim Warby, Tel. (02) 9144 3958

- **Wednesday, 5 May:** Dr Stephen Harlamb – Rotary Instrumentation. Pre-Dinner – Dr John Barbat
- **Wednesday, 28 July:** Dr Ward Massey – Direct Pulp Capping (18th Barry Barker Memorial Lecture)
- **Friday, 12 November:** Joint all day meeting with Perio Society

#### QUEENSLAND

**President:** Dr Don Dellit, Tel. (07) 3209 9686  
**Secretary:** Dr Glen Weston, Tel. (07) 3210 1417

- **Friday, 23 April:** Endodontic specialist panel – *A Night of Herring* — non-endodontic problems that weren’t what they seemed
- **Friday, 11 June:** Drs A. Moule, M. Gentner, G. Weston – Cracks, Splits & Fractures
- **October (date to be advised):** Presidents at Home
- **Friday/Saturday, 12/13 November:** Overseas guest lecturer (to be confirmed)

#### SOUTH AUSTRALIA

**President:** Dr Katina Maiolo, Tel. (08) 8223 3364  
**Secretary:** Dr Ian Trantor, Tel. (08) 8223 1093

- **Tuesday, 27 April:** Dr Geoffrey Heithersay – The most interesting day in my endodontic career – Tuesday, November 1993
- **Tuesday, 17 August:** Drs Paul Heijkioop, Paul Matin, Ian Trantor, Mark Wotzke – Endodontic Table Clinic
- **Tuesday, 21 September:** Various ASE members – Case Presentations and Annual General Meeting

#### VICTORIA

**President:** Dr Vlad Hardi, Tel. (03) 9687 6269  
**Secretary:** Dr Chris Eldridge, Tel. (03) 9650 3946

- **Wednesday, 9 June:** Dr David Cable – Lost & Found – the calcified canal
- **Wednesday, 18 August:** Drs Paul Abbott and Werner Bischof – Selective and Intelligent Use of Antibiotics in Endodontics and Periodontics
- **Wednesday, 17 November:** Drs Phillip Zimet and Robert French – Oh, no – not a root canal!

#### WESTERN AUSTRALIA

**President:** Dr Andrew Hossen, Tel. (08) 9474 2391  
**Secretary:** Dr Paul Mulvay, Tel. (08) 9474 2391

- **Wednesday, 7 April:** Dr David Witherspoon – Problem Solving in Endodontics, half-day meeting
- **Friday, 14 May:** Dr Garry Nervo – Hands-on course for Rotary NiTi instrumentation, half-day meeting
- **Tuesday, 20 July:** Dr John McGeachie – *The Anatomy of Local Anaesthesia*
- **Friday, 8 October:** Dr Luke Moloney – Focal Infection and Instrumentation in Endodontics, half-day meeting
New Zealand Society of Endodontics (Inc.)

Annual General Meeting

Minutes of the Annual General Meeting of the New Zealand Society of Endodontics held at NZDA Headquarters, 3 St Marks Road, Remuera, Auckland, on 3 December 1998.

Meeting Opened: 2000 hours

Present: Dr H. Lala (President), Dr R. Ellis (Treasurer), Dr P. Cathro (Secretary) and four members.

Apologies: None.

Correspondence:
1. Dr R. Love applied for research funds from the Society’s research grant. This would allow him to complete PhD work into the Molecular basis of bacterial invasion of dentinal tubules.
   Request approved.

   Action Point:
   Secretary to write to the editor of the *New Zealand Endodontic Journal* requesting that the availability of the research grant be advertised. The secretary is also to find out what the sum of the research grant is.

2. A letter had been received from the Royal Australasian College of Dental Surgeons requesting nominations for the Board of Studies for Endodontics. Dr S. Manning and Dr R. Love were put forward by the executive.

Finance
1. Dr R. Ellis reported that currently the Society has $100,290.25 in the various bank accounts. After some discussion it was decided to earmark $20,000 for the purchase of endodontic-related items for the Greenlane Hands-On Centre. This could include such items as Root ZX, engine motors etc.

   A further $40,000 to be set aside for an international speaker for the year 2000.

   The trauma booklet will be updated after the March Trauma Conference in Melbourne. It was decided to set aside $10,000.

2. It was agreed that the subscriptions be kept the same for 1999, i.e. $25+GST and $30 for overseas subscriptions.

3. The accounts are to be sent to Spicer and Oppenheimer at the end of June. Richard Ellis to action.

4. The Editor’s stipend of $100 was approved.

General Business
1. Dr H. Lala’s name is to be added to the President’s Board at NZDA Headquarters.

2. Dr Neville McDonald is going to speak at the ADA Conference in Port Douglas, as well as in Auckland, Wellington and a South Island venue. The Executive are to liaise with Dr S. Manning.

3. A copy of *Pathways of the Pulp* has been sent to the Dental School for the fourth year Endodontic Prize. The secretary is to ask Dr Love for the details of the winner so that the president can write a letter.

4. Two students have recently won prizes for the essay competition. The secretary is to ask Dr Love if a photo of these students could be placed in the *Endodontic Journal*.

Meeting Closed: 20.50 hours.