Evaluation of the Prevalence and Clinical Characteristics of Intrapulpal Cracks Utilizing a Novel Classification System

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Evaluation of the Prevalence and Clinical Characteristics of Intrapulpal Cracks Utilizing a Novel Classification System

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Dentistry at Virginia Commonwealth University.

by

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Few studies have investigated cracks involving the pulp chamber walls or floor. The purpose of this study was to create a classification system for intrapulpal cracks and analyze associated clinical characteristics. Retrospective analysis included 52 teeth confirmed to have an intrapulpal crack. The classification system describes the crack based upon its location within the pulpal walls and floor. Documentation consisted of demographic, subjective and objective examination data. Chi-squared analysis tested associations with the intrapulpal crack classifications. Ninety-two percent of intrapulpal cracks run M-D, 75% involve one pulp chamber wall, and 84% terminate at the floor-wall junction or extend into an orifice without involving the pulpal floor. There was a statistically significant ($P<0.05$) relationship between the location of the intrapulpal crack and tooth type as well as between the classification system and bite test and transillumination. The classification system was adaptable to clinical practice and provides insight into these challenging situations.
Introduction

As the number of those retaining their natural dentition continues to climb, the cracked tooth is quickly becoming a more commonly diagnosed causative factor of pulpal and periapical pathosis (1). The cracked tooth has been the subject of expert opinion papers and case series since the latter half of the 1950s. In 1957, Ritchey et al. (2) first described an incomplete fracture in the long axis of the molar tooth and involving the pulp chamber as responsible for irreversible pulpitis symptoms. They noted that patients with this incomplete fracture would commonly experience vague pain on chewing and sensitivity to cold. In 1964, Cameron (3) described this incomplete fracture with pain on pressure and unexplained thermal sensitivity as the “Cracked Tooth Syndrome” or CTS. He defined this crack as a more centered mesial to distal crack that propagates apically within the tooth along the lines of the dentinal tubules. He also stressed the importance of early detection of these cracks to prevent further propagation and pulp exposure. In a study by Hiatt (1) in 1973, 100 teeth were evaluated within a general dentistry practice diagnosed with incomplete crown-root fractures. He confirmed the findings by Cameron (3) in that the most common symptoms were chewing pain and sensitivity to thermal insults. Hiatt also evaluated the prevalence of these cracks and noted that the most commonly involved teeth for these incomplete fractures were the mandibular second molars, followed by the mandibular first molars, maxillary premolars and maxillary first molars. It is important to note that this was the first study to address the prevalence of these fractures in unrestored or minimally restored teeth.
He found that 74% of these incomplete crown-root fractures occurred in unrestored or minimally restored teeth. Ehrmann and Tyas (4) supported this finding in recommending that we should suspect CTS in any patient presenting with abrupt onset of pain in a non-restored or shallowly restored tooth. In a follow up study to his original work on CTS, Cameron (5) noted that almost all centrally located cracks extend in a mesial to distal direction, with occasional buccal to lingual cracks in mandibular molars.

The etiologies of the cracked tooth have been extensively discussed in the literature. Hiatt (1) was the first to propose predisposing factors of an incompletely fractured tooth. He noted that parafunctional habits (i.e.: clenching/grinding), eccentric interferences, the wedging effect of cusp/fossa relations in posterior teeth, the lack of protection of transverse ridges in mandibular molars, and the lever principle of greater forces located more posteriorly near the fulcrum of the temporomandibular joint (TMJ) as factors that may predispose a tooth to fracture. Talim and Gohil (6) further elaborated on possible etiological factors of cracks including trauma and accidental biting on hard objects. In his review of the cracked tooth syndrome, Rosen (7) discussed possible causes and predisposing factors of CTS, including: masticatory accidents, steep cusps/deep fossae, premature occlusal contacts, radical tooth preparation, threaded pins, improperly seated posts/cores/inlays, parafunctional habits, and teeth prepared as abutments. Others have reiterated these factors as possible reasons for the eventual development of a cracked tooth (8, 9, 10).

Diagnosis of a cracked tooth can be difficult because of the variety of presenting clinical signs and subjective symptoms. In 1954, Gibbs (11) first shed light on this phenomenon, noting that patients were often dismissed for lack of obvious etiology to their symptoms of pain during mastication. The tooth would initially appear clinically sound and radiographically normal on
examination. Ritchey et al. (2) verified this difficulty in their case series in 1957. Identifying the “incomplete tooth fracture” in patients who had been incorrectly diagnosed by previous clinicians, they observed that often times the patient would go through several occlusal adjustments, replacement of restorations, and extension of restorations with a concomitant progressive increase in the frequency and occurrence of pain. In both of his studies on the CTS, Cameron (3, 5) noted that the symptoms were not always consistent and the problematic tooth not always easily identifiable. The abrupt onset of unexplained thermal sensitivity was stressed as an important clue in the diagnostic evaluation. In his 1983 review, Abou Rass (8) noted that we “should suspect a cracked tooth in all examples of obscure etiology and mixed chronic symptoms”. However, he also stressed that not all cracks are symptomatic.

In the 1997 AAE Colleagues of Excellence “Cracking the Cracked Tooth Code”, Simon (12) reviewed this variability of presenting signs and symptoms. He stated that the dental history may include a history of chronic undiagnosed pain, repeated occlusal adjustments without relief of symptoms, and a possible history of other cracked teeth. Radiographically the crack is rarely visible. The difficulty to diagnose these cracks is compounded by their ability to mimic other orofacial disorders. These can include acute rhinosinusitis, TMJ disorders, headaches, ear pain, atypical facial pain, and other non-odontogenic related pain conditions (13, 14). Abbott and Leow (15) confirmed the clinical difficulty in diagnosing cracked teeth with their finding that only 9% of patients are correctly identified with a cracked tooth by the referring general practitioner. In another study by Braly and Maxwell (16), 20% of patients referred to the endodontic practice with diagnostic uncertainties were diagnosed with incomplete tooth fractures. These difficulties in identifying clinical signs and symptoms and the confusing dental histories highlight the complexity of diagnosing and correctly treating these cracked teeth.
Greater understanding of the variability and complexity of these symptoms has led to a re-evaluation of the term “cracked tooth syndrome”. Abbott and Leow (15) suggested that a syndrome is defined by symptoms occurring together and characterizing a specific disease. Because a crack is not a disease and is associated with a number of symptoms that vary considerably from case to case, the term “cracked tooth syndrome” was inappropriate. As the symptoms are dependent on the extent of the underlying disease process, there existed a significant variability in the symptoms that a patient may present with clinically. Cohen et al. (17) noted that the determination of a cracked tooth is not routinely objective therefore the term syndrome should be avoided. Often times, the determination is more of a prediction then a definitive diagnosis. Given these findings, the validity of the term “cracked tooth syndrome” is questionable (15, 17, 18, 19).

Compounding this variability is the plethora of clinical terminology used by researchers to describe the cracked tooth. These terms include: cuspal fracture odontalgia (11), incomplete tooth fracture (2), greenstick fracture (20), cracked tooth syndrome (3), vertical incomplete fracture (6), tooth structure crack (8), and vertical root fracture (17, 21). The terms crack and fracture are often used synonymously in the literature to describe a crack line within a tooth. With these vastly different terms, one can understand the confusion amongst clinicians in describing a crack within the crown of a tooth. However, the term “cracked tooth” is used to denote a more centered fracture extending apically from the occlusal surface as described by Cameron (3). This will be discussed in greater depth in a later section.

As clinical signs and subjective symptoms of cracked teeth can vary, the diagnosis must rely heavily upon diagnostic testing and clinical examination to identify the crack. Exclusion of other known etiologies for the pulpal and periradicular pathoses must always be attempted first
Diagnostic tests and clinical examination methods utilized to identify the cracked tooth include: bite test, cold test, probing depths, transillumination, direct visualization, microscopic evaluation, and staining with dye. Radiographic evaluation of the cracked tooth is of little value, as the mesial-distal orientation of the crack line cannot typically be seen radiographically (8, 9, 12). Only when the fracture is more severe and oriented in a buccal-lingual plane can the radiograph detect the fracture (9). Cone beam computed tomography (CBCT) is also limited in its ability to detect crack lines (18, 23). With current technology, the available resolution is not sensitive enough to detect the small size of the fracture line. The surrounding structures will also impede the ability to detect the crack (23). It is important to note that the finding of a cracked tooth is not a diagnosis in itself but rather an etiology of pulpal and periradicular disease (8, 15).

Cameron (3) first identified most of the commonly utilized diagnostic tests: bite test, cold test, transillumination and staining. He proposed the use of a wooden toothpick for the bite test to elicit pain on biting. Hiatt (1) recommended a burlew wheel for the same purpose. In a follow-up work by Cameron (5), he noted that pain might also occur on release of biting pressure. Rosen (7) and Abou Rass (8) both suggested that this momentary increase in pain as pressure is released is a strong indicator of a cracked tooth. In his work on the hydrodynamic theory, Brannstrom (24) noted that biting pressure may cause fluid movement within the crack and associated dentinal tubules, eliciting this painful reaction. Ehrmann and Tyas (4) were the first to recommend the use of the Tooth Slooth® (Professional Results, Inc., Laguna Niguel, CA), the now commonly used diagnostic tool for eliciting pain on biting or release. The Tooth Slooth® allows accurate location of the specific cusp(s) associated with the centrally located crack (25).

Brannstrom (24) first theorized the mechanism of the sharp painful response to cold in a cracked tooth. He identified this acute pain response was a result of the hydrodynamic theory of
outward dentinal fluid flow within the crack and adjacent dentinal tubules. Depending on the depth of the crack and proximity to the pulp chamber, the cold test may elicit a response associated with a normal pulp, reversible pulpitis, irreversible pulpitis, or even pulpal necrosis (12). This response to cold is based upon the histopathologic status of the pulpal tissues.

Probing depths are used as a diagnostic tool to evaluate the periodontal involvement of the associated crack. When the crack extends below the gingival margin and involves the periodontal ligament, the bacteria within the crack may elicit an inflammatory reaction and subsequent crestal bone loss (1, 7). Therefore, the presence of an isolated narrow defect may be an indicator of an adjacent crack line (7, 8, 9, 22). However, caution must be utilized in evaluating probing depths. Other periodontal conditions may mimic a deep probing depth and often times a crack may be present with no associated probing defects (8, 22).

Transillumination with a fiber optic light was recognized by Cameron (5) as a way to visualize the crack. The light beam is placed directly against the tooth surface and positioned perpendicular to the plane of the suspected crack. When a crack is present, the crack will disrupt the light transmission and the tooth structure proximal to the crack will appear brighter than the tooth structure distal to the crack. In structurally sound teeth and those with shallow cracks limited only to the enamel surface, the light will transmit through the entire crown (12). Abou Rass (8) recognized transillumination as one of the “most valuable diagnostic techniques”. He recommended the use of fiber optics to view the fracture with and without staining. Gutmann and Rakusin (26) suggested using the fiber optic light to determine the direction and extent of the fracture. In a comparative study of transillumination and dyes in resected root end cracks, Wright et al. (27) found that transillumination with or without staining with methylene blue was the most accurate method for diagnosing root end dentinal cracks.
Direct visualization of the crack has been recommended for evaluation of the extent of the fracture. This visualization should be done by removing the restoration and followed by staining of the cracked surface with methylene blue. Cameron (5) recommended the use of methylene blue. If the crack was still not visible, he recommended sealing the methylene blue within the tooth for 2-3 days with a cotton pellet and zinc oxide eugenol temporary restorative material. Abou Rass (8) recommended applying the methylene blue with a cotton pellet for a several minutes and suggested that the speed of absorption of the dye indicated the depth and width of the crack. Transillumination of the stained area provided even more information on the crack. While the use of staining and transillumination aids the clinical detection of cracks, there are drawbacks as well. According to Clark et al. (28), methylene blue may obscure cracks by staining decalcified enamel and dentin as well as plaque. They also suggested that prolonged exposure to sodium hypochlorite might result in excessive absorption of dye, thus obscuring the crack. Therefore, they recommended the application of the methylene blue immediately following access of the pulp chamber and canal system. Microscopic evaluation allows the use of higher magnifications to visualize the crack. Clark et al. (28) recommend the use of 14-18x magnifications with maximum lighting intensity for visualization of dentinal cracks.

Once identified through diagnostic testing, the crack should be classified based upon location and extent of the fracture. Several authors have attempted to classify the types of cracks commonly observed. The first classification system, proposed by Silvestri and Singh (29), developed categories based on the vertical and horizontal dimensions of the crack. Clark and Caughman (30) postulated another classification system based on the prognosis of the treatment for the crack. Their proposed categories for the classification system were Excellent, Good, Poor, and Hopeless. They stated that vertical fractures into the pulp should be categorized as
Poor and vertical fractures through the pulp and onto the root surface should be considered Hopeless. These categories were based on empirical evidence alone. In 1997, the AAE published a “Cracking the Cracked Tooth” guide that adopted the currently used classification system for tooth cracks (12). These 5 categories are: Craze lines, Cuspal fractures, Cracked teeth, Split teeth, and Vertical root fractures. Rivera and Williamson (32) further described these cracks as “Longitudinal” based on their ability to extend in both a vertical direction and over a period of time.

Understanding the definitions of and differences between the 5 categories of tooth cracks is paramount to correct diagnosis. Craze lines are seen on anterior teeth as well as crossing marginal ridges and buccal and lingual surfaces in posterior teeth. They are superficial in nature, affecting only the enamel surface, and are not a concern beyond aesthetics. Cuspal fractures are complete or incomplete fractures involving one or more cusps and extending in a peripheral oblique direction from the occlusal surface to the cervical third of the crown or root surface. They typically terminate at or slightly below the gingival margin and will commonly have two directional components: buccal-lingual and mesial-distal. The cracked tooth is the more centrally located incomplete fracture that is typically oriented mesiodistally and involves one or both marginal ridges. Being well centered, the fracture is capable of propagating apically into the pulp chamber or root of the tooth and therefore more destructive in nature than a cuspal fracture. The split tooth is the complete fracture resulting from the apical extension of a cracked tooth. The more centered, apically directed complete fracture terminates in the middle or apical third of the root, typically towards the lingual surface. The split tooth may or may not be restorable depending on where the complete split occurs on the root surface. The vertical root fracture (VRF) is the complete or incomplete fracture initiated within the root at any level and typically
oriented in a buccal-lingual direction. The fracture is located only within the root and may or may not extend to both buccal and lingual surfaces. Vertical root fractures are typically seen in root canal treated teeth and may present with minimal symptoms. They are difficult to diagnose as they mimic other conditions including endo-perio lesions, endodontic failure, and periodontal disease. Craze lines, cuspal fractures, cracked teeth, and split teeth originate in the crown of the tooth, while vertical root fractures originate in the root of the tooth (12, 31). The cracked tooth will be the focus of the discussion within this research paper.

Many investigators have discussed the treatment of the cracked tooth. Treatment is primarily dictated by the extent of the crack within the coronal tooth structure and the progression of any pulpal or periradicular disease processes. The goal of treatment is to protect the cusps, bind the cracked segments together, and prevent further propagation of the fracture (4, 5, 8-11). Additionally, the crack should be sealed to prevent further bacterial contamination and progression of any pulpal or periradicular inflammatory processes (1, 10, 29). Early investigators, including Gibbs (11) and Cameron (3, 5), were the first to emphasize early diagnosis and full coverage crown treatment for the cracked tooth. They suggested that only full coverage restoration of the cracked tooth would protect the tooth from further propagation of the fracture. Since that time, many review articles and expert opinion papers have continued to recommend the use of a full coverage crown to treat the cracked tooth (4, 8, 9, 22, 33). When preparing the tooth for a full coverage crown, the location of the crown margin has been suggested to be important for the prevention of crack propagation. Ideally, the margins of the crown should be placed 2 mm below the termination of the fracture line to encompass the crack and prevent propagation (9). Other restorative suggestions have included the use of ¾ crowns, indirect bonded restorations, and direct bonded restorations with amalgam or composite cuspal
coverage (10, 34-37). These treatment modalities have been traditionally based on empirical studies and expert opinions.

More recently, several longitudinal studies have focused on this issue of restoration of the cracked tooth. Krell and Rivera (33) evaluated treating cracked teeth with reversible pulpitis utilizing full coverage crowns. All teeth (n=127) were evaluated at 1-year recall unless nonsurgical root canal treatment (NSRCT) was needed before this date. After 6 months, approximately 21% of these teeth required NSRCT due to diagnoses of irreversible pulpitis or pulpal necrosis. The authors took into account that many of these teeth may have required NSRCT due to the crown preparation and not the crack itself. Opdam et al. (34) evaluated the long-term efficacy of restoring painful cracked teeth with direct composite restorations. Two groups were included, one with cuspal coverage (n=21) and one without cuspal coverage (n=20). The symptoms were consistent with reversible pulpitis. After 7 years, the group with cuspal coverage composite restoration had no failures, while the group without cuspal coverage had a mean annual failure rate of 6.0%. This difference was statistically significant. In a similar study by Signore et al. (36), cracked teeth with reversible pulpitis symptoms were treated with indirect composite onlays and evaluated at 1 week, 4 weeks, and every 6 months for up to 6 years duration. The overall 6-year success rate was approximately 93%. These studies provide stronger evidence to suggest that there may be several ways to restore the cracked tooth when the symptoms indicate reversible pulpitis.

Another question in the treatment of cracked teeth is whether or not the crack line should be chased or removed entirely from the involved tooth. While most investigators agree that any restoration should be removed to properly assess the extent and location of the crack, they do not agree on the removal of the crack line itself (3, 8, 9, 13, 25, 26, 38). Those who believe the crack
line should be traced and removed in its entirety suggest that only in removing the crack will the clinician be able to prevent further propagation of the crack and eliminate potential pathways for bacterial penetration (8, 15, 38). Abbott and Leow (15) considered the crack as a biologic problem, rather than a mechanical problem, and therefore the crack has the potential to cause pulpal and periradicular diseases in the same manner as caries does by allowing bacterial penetration of the pulp. The aim then should be to completely remove the crack, in much the same way the aim in restorative treatment of caries is to remove all of the carious process. On the other hand, there are those investigators who believe that no attempt should be made to remove the crack in its entirety (4, 9, 12, 25, 31). In the 2008 AAE Colleagues of Excellence guide, Rivera and Walton (31) recommended the removal of the fracture line only in the area of the cavity floor that would be included in an ideal endodontic access. The practitioner should not chase the entire extent of the crack, as the crack becomes invisible long before it terminates and sound dentin would be lost in the process. They also recommended against the removal of the fracture line on the proximal surfaces below the level of the cementoenamel junction. Doing so would remove significant tooth structure from the supporting marginal ridges and would render the tooth non-restorable. Based upon the literature available, the question of chasing or removing the entire crack is still unanswered with the prudent practitioner attempting to balance removal with preservation of sound tooth structure.

In 1978, Silvestri and Singh (29) introduced the idea of initial stabilization of the cracked tooth. Focusing on the need to control microleakage within the crack to prevent periodontal or pulpal inflammation, they recommended the use of copper bands, arch wires, preformed aluminum or stainless steel crowns for temporary splinting of the tooth. This initial treatment would prevent further propagation of the crack until definitive full coverage crown treatment
could be completed. Ehrmann and Tyas (4) also promoted this idea of immediate stabilization of the fracture. In their review paper on the cracked tooth syndrome, they recommended the use of stainless steel bands to splint the cracked tooth for a period of 2-4 weeks. If no further pain persisted, they recommended full coverage crown treatment. If pain persisted following splinting, they recommended NSRCT and full coverage crown treatment. Many recent articles have reinforced these ideas of immediate stabilization with orthodontic bands or full coverage acrylic provisional crowns (9, 10, 12, 15, 22, 31, 38, 39). The NSRCT can be completed with the band in place to protect the tooth from wedging forces and the possibility of propagating the fracture (9, 10, 12, 22, 31, 39).

Several investigators have proposed a systematic protocol for treatment of cracked teeth. This approach utilizes flow charts for treatment protocols that are dependent on symptoms, location and extent of the fracture (9, 10, 39). In a recent review by Ailor (9), he proposed a flow chart with two major categories for treatment: 1) Asymptomatic or newly symptomatic teeth and 2) Non-vital or long-term symptomatic teeth. For those that were Asymptomatic or newly symptomatic, the treatment of choice was placing a temporary full coverage crown and observing for symptoms. If symptoms persisted, NSRCT was recommended, but if symptoms subsided, permanent crown fabrication would commence. In those that were Non-vital or long-term symptomatic, the treatment of choice was stabilization with an orthodontic band and NSRCT. Evaluation of the extent of the fracture within the pulp chamber was recommended upon access of the pulp chamber, followed by permanent crown fabrication. Utilizing a similar methodology, Tan et al. (39) recommended two major categories for treatment: 1) Vital pulp and 2) Irreversible pulpitis. In contrast to Ailor (9), they placed orthodontic bands on all cracked teeth for initial stabilization. Following this initial stabilization, those teeth within the category of
Vital pulp were treated with full coverage crowns. Those within the category of Irreversible pulpitis underwent NSRCT, evaluation of the extent of the fracture within the pulp chamber, and subsequently restored with full coverage crowns. Patients that did not wish to have full coverage crowns placed were treated with amalgam cores and orthodontic bands. While the investigators differ in certain aspects of the protocol, the main concepts of treatment are similar in these models: initial stabilization, assessment of the extent of the crack within the pulp chamber (when NSRCT is needed), and full coverage crown treatment (9, 39).

While many studies have evaluated the cracked tooth and proposed characteristics, diagnosis, and treatment methods, very little research has focused on the pulpal aspects of these longitudinal cracks. As the crack is left to propagate untreated, the pulpal structures eventually become involved. This propagation results in inflammatory changes in both the pulpal and periradicular structures usually necessitating NSRCT. The author of this thesis has chosen herein to refer to all cracks propagating into the pulp chamber as Intrapulpal Cracks. The intrapulpal nature of these cracks refers to their location within the pulp chamber walls and floor of the tooth. Upon access of the pulp chamber and removal of the pulp chamber roof dentin, any crack extending longitudinally within the pulp chamber structure will be referred to as an intrapulpal crack.

Bacteria associated with intrapulpal cracks are the causative factors in crack associated pulpal and periradicular pathoses. Hiatt (1) was one of the first to discuss that these fracture lines permit bacterial invasion into the pulpal or periodontal structures. Abou Rass (8) noted that a crack could be an etiology of pulpal disease, extending into the pulp chamber and irritating the pulp physically and through microleakage of bacteria and bacterial byproducts and toxins. Brannstrom (24) further described the pulpal aspects of this process in his classic work on the
hydrodynamic theory of dentinal sensitivity in 1986. He observed that cracks are permeable to dentinal fluid and bacteria, allowing bacterial metabolic byproducts and endotoxins to diffuse towards the pulp inciting an inflammatory process. As the crack progresses apically towards the pulpal tissues, so does the inflammatory response within the pulpal complex. This concept of bacterial penetration of cracks and invasion of the root canal system was confirmed by Love (40). Utilizing an ex vivo model, he demonstrated that following the creation of enamel/dentin cracks, bacteria could penetrate the root canal system. These studies clearly elucidate that intrapulpal cracks and the bacteria within them are the etiology of pulpal and periradicular pathoses. Therefore a better understanding of the prevalence, prognosis, and treatment of these cracks must be sought.

The prevalence of intrapulpal cracks has not been extensively evaluated. In earlier studies, such as those by Ritchey et al. (2), Cameron (3), and Silvestri and Singh (29), the investigators only briefly discussed the concept of propagation of cracks within teeth and the eventual pulpal involvement if not recognized and treated early. There was no attempt to evaluate the prevalence of intrapulpal cracks. In more recent studies, investigators have evaluated the prevalence of cracked teeth requiring NSRCT (34, 42, 43). Eakle et al. (41) evaluated an undergraduate dental clinic population over 1.5 years and examined 191 patients with 206 complete or incomplete fractures of posterior teeth. Complete fractures were defined as cuspal fractures while incomplete fractures were defined as centrally located incomplete fractures. They noted that 18% (n=37) of those fractured teeth involved the pulp. In another study by Roh and Lee (42), over a 1-year period within a hospital dental clinic, 42.4% (n=65) of cracked teeth required NSRCT. The 2007 study by Krell and Rivera (33) also evaluated cracked teeth requiring NSRCT. They found that approximately 21% of cracked teeth diagnosed with
reversible pulpitis and treated with full coverage crowns would become irreversibly symptomatic or necrotic and require NSRCT within 6 months. While the latter two studies highlight the significance of cracks and the need for NSRCT, they do not discuss the extent of the crack line within the tooth. No study to date has evaluated the prevalence of cracks in relation to their location with respect to the pulp chamber wall, floor, and orifice(s). Future studies must be completed to address this paucity of clinical data and to better answer the question of the prevalence of intrapulpal cracks and their location within the pulp chamber.

The lack of a standardized method of classifying these intrapulpal cracks leaves the clinician with no objective manner in which to discuss the extent and location of the cracks within the pulp chamber. In the 2008 AAE Colleagues for Excellence, “Cracking the Cracked Tooth Code: Detection and Treatment of Various Longitudinal Fractures” Bonus Material, Rivera (43) proposed a classification system based upon hypothesized prognoses for longitudinal cracks. This system included the following intrapulpal classifications: I. One marginal ridge extending from crown to root surface, II. Two marginal ridges extending from crown to root surface, III. Marginal ridge(s) and into canal orifice(s), IV. Marginal ridge(s) and pulpal floor, and V. Furcation involvement. Another classification system proposed by Tan et al. (39) included the groups: I. Coronal (confined within the wall of the pulp chamber), and II. Radicular (extended beyond the orifice of the root canals), as well as noting the walls involved. Both of these classifications are helpful in describing the location of the intrapulpal cracks within the chamber, however they do not give the clinician a full understanding of the complete extent of crack along the pulp chamber wall(s), pulp chamber floor, and orifice(s) involvement. A classification system that could objectively describe the complete extent of each intrapulpal
crack would provide a systematic way to record these findings as well as follow them in longitudinal outcome studies.

Recently, two articles have been published evaluating the success of intrapulpal cracks and NSRCT. The first, a case series by Liu and Sidhu (38), evaluated 6 patients diagnosed with irreversible pulpitis or pulpal necrosis requiring NSRCT due to cracks communicating directly with the pulp chamber. These cracks involved the pulp chamber walls but not the pulpal floor. In the recall range of 1 - 3.5 years, all 6 cases remained successful with no periodontal destruction, periapical healing, and asymptomatic. The second, a retrospective outcome study of root filled cracked teeth by Tan et al. (39), evaluated 49 patients diagnosed with irreversible pulpitis and cracks extending into the pulp chamber. The location (mesial, distal, buccal, lingual), extent (Coronal, Radicular), and number of cracks were identified. Crack lines extending beyond the orifices of the root canals were classified as Radicular. Both Coronal and Radicular types of cracks were treated with NSRCT. The survival rate at the 2-year recall period was 85.5%. In evaluating pre-operative predictors of survival, they found that the extent and location of the crack did not affect the survival of the tooth. However, they did note that future long term prospective studies would be needed to evaluate the treatment outcome of cracked teeth. While both of these articles are limited by their sample size, classification methods, evaluations of success, and recall time, they serve as a guide for future longitudinal studies on treatment of intrapulpal cracks. They begin to elucidate the need for further clinically based studies on the assessment of treatment outcomes of teeth with intrapulpal cracks.

The prognosis of teeth with intrapulpal cracks is currently based on empirical evidence alone. No longitudinal prospective clinical trials exist to determine the prognosis of treatment for
these cracks. Therefore, the empirical evidence is presented here to establish a basis of current thought on prognosis of intrapulpal cracks.

Earlier researchers proposed that any crack involving the pulp chamber would be associated with a poor prognosis (5, 7, 11, 21, 28). Cameron (5) suggested that the closer the crack was to the pulp, the poorer the prognosis would be for the tooth. However, there was no evidence provided to substantiate his claims. More recent studies have attempted to evaluate certain characteristics of the intrapulpal crack when assigning prognosis and treatment recommendations (6, 7, 9, 12, 26, 29, 30). In the 1997 AAE Colleagues of Excellence guide (12), cracks visible partially across the chamber floor were deemed restorable and protected with a band or temporary crown and root canal treated, while cracks involving the full width of the pulp chamber floor were given a Poor prognosis and those with a deep periodontal defect were Hopeless. In another study on the management of incompletely fractured molars, Gutmann and Rakusin (31) created similar prognoses based on the extent of the crack within the pulp chamber. A “Best” prognosis was assigned to fractures limited to one marginal ridge not penetrating apically more than 2-3 mm below the periodontal attachment. A “Guarded” prognosis was assigned to fractures involving both marginal ridges that did not communicate through the pulp chamber floor. And a “Poor” prognosis was assigned to fractures involving both marginal ridges and communicating through the pulp chamber floor. Per Rivera and Walton (31), “prognosis is more variable with cracks than with other types of longitudinal fractures. Determining the position and extent may help in determining when to recommend extraction”. Although several authors have attempted to examine intrapulpal cracks by describing their extent, differing terminology, opinions, classifications, and a lack of clinical research have left clinicians to base their treatments of these intrapulpal cracks on speculative and empirical evidence at best.
Due to this paucity of clinical research, the classification, treatment, and prognosis of intrapulpal cracks is currently based on subjective clinical impressions and lower levels of evidence. Currently endodontists treat patients who have teeth with intrapulpal cracks that are symptomatic and based upon pulpal and periapical diagnoses require NSRCT. At this point in clinical treatment, evidence-based endodontics does not exist from which to base endodontic treatment, restorative treatment, or to predict outcomes. This void in endodontic research remains a major problem for clinical endodontic treatment.

To address this complex dilemma, intrapulpal cracks must be described, defined, and classified. A commonly accepted description and definition of intrapulpal cracks would allow for application in a universal clinical setting. If intrapulpal cracks could be documented with respect to their pattern of extent and location within the pulp chamber, then predictive patterns for the extent and location of these cracks could emerge. These predictive patterns could aid the treating clinician when evaluating teeth for longitudinal cracks as well as in locating these cracks within the pulp chamber structures. If the extent and location of intrapulpal cracks could also be correlated with treatment outcomes, then future treatment of these cracks could be provided in a more predictable manner with respect to prognosis. With these goals in mind, the classification system was created to begin objective analysis of the types of intrapulpal cracks seen within the pulp chamber. The author was aware that it would be necessary for the classification system to be simple, yet robust enough to provide adequate detail for the emergence of predictive patterns. The classification system would need to convey the longitudinal manner of the intrapulpal crack from pulpal wall to possibly the pulpal floor and to or beyond the pulpal orifice(s). Ease of use of the classification system was a goal.
The following classification system for intrapulpal cracks was developed utilizing analysis of images and data collected during documentation of intrapulpal cracks in a pilot study at VCU School of Dentistry Graduate Endodontic Practice as well as the proposed classification systems by Rivera (43) and Tan et al. (39). The patterns recognized for intrapulpal cracks during this analysis were: 1) cracks traversing down only one wall, 2) cracks traversing down two walls, 3) cracks traversing down one wall and into a canal orifice, 4) cracks traversing down two walls and into a canal orifice from one wall while terminating at the floor-wall junction at the other wall, 5) cracks traversing down one wall and partially across the pulpal floor, 6) cracks traversing down two walls and partially across the pulpal floor from one wall while terminating at the floor-wall junction at the other wall, 7) cracks traversing down one wall and running entirely across the pulpal floor, 8) cracks traversing down two walls and running entirely across the pulpal floor, 9) cracks traversing down two walls and into canal orifices on both sides, 10) cracks traversing down two walls and into an orifice from one wall while running partially across the floor from the other wall, 11) cracks traversing down two walls and into an orifice from one wall while running the entire length of the pulpal floor from the other wall. Based on these patterns, the classification system was divided into two categories: Pulp chamber walls and Pulp chamber floor. The classifications within each category are encompassing of the various cracks existing within pulp chambers.

**Pulp chamber wall classification:**

I. One wall involved

II. Two walls involved
Pulp chamber floor classification:

A. Terminates at floor-wall junction, not involving orifice(s)

B. Extends into orifice, not involving floor

C. Traverses partially across floor, not involving orifice(s)

D. Traverses across entire floor, not involving orifice(s)

The proposed classification system for intrapulpal cracks provides an objective means to evaluating the prevalence and characteristics associated with intrapulpal cracks. No clinical research has focused on the prevalence or specific characteristics associated with these cracks, nor have they attempted to correlate them with the extent of the crack within the pulp chamber system. Determining the pre-operative variables to be evaluated in treatment of intrapulpal cracks, Tan et al. (39) recorded the following characteristics: the number of cracks (single or multiple), location of the crack (mesial, distal, buccal, or palatal/lingual), the extent of the crack (Coronal or Radicular), the presence of periodontal pocketing, the patient’s age and gender, the location of cracked tooth, the type of tooth, and location of the tooth in the arch. Using their methodology as a preliminary guide, a number of variables were selected for evaluation and inclusion in this study.

This analysis will provide not only valuable information on the prevalence of the individual intrapulpal crack classifications but also on intrapulpal cracks as a whole. Furthermore, the involved orifices, walls, and floor can be determined for individual teeth to determine patterns for intrapulpal cracks. Additional data analysis can be completed for the purposes of understanding possible relationships of characteristics of intrapulpal cracks. Pre-operative
variables, specifically demographics, diagnostic findings, and pulpal and periapical diagnoses, can be correlated with the various classifications of intrapulpal cracks. These findings will provide a greater insight into the nature of intrapulpal cracks and uncover possible significant relationships between pre-operative variables and the extent of the intrapulpal crack. Subjective evaluation of the classification system and ability to locate and classify intrapulpal cracks will also be evaluated. The future goal of this research endeavor, once a reliable classification system has been implemented and evaluated, is endodontic treatment of these teeth and prospective longitudinal outcome studies. Longitudinal outcome studies will provide sound scientific evidence upon which to base clinical decision-making for intrapulpal cracks.

Therefore, the purposes of this retrospective study were: 1) Analyze the prevalence, demographics, and descriptive characteristics of intrapulpal cracks, 2) Evaluate the associations between certain clinical and diagnostic findings for intrapulpal cracks and the proposed classification system, and 3) Assess an operator’s ability to utilize the classification system and current limitations of evaluating intrapulpal cracks.
Materials and Methods

The study population comprised all patients confirmed to have an intrapulpal crack after access preparation for endodontic treatment in the Graduate Endodontic Practice at VCU School of Dentistry (Richmond, VA). Patients were referred for evaluation and treatment from the VCU School of Dentistry undergraduate student clinics as well as the AEGD residency, Faculty Practice, and Richmond metropolitan private dental practices. Endodontic treatment included non-surgical root canal treatment and orthograde retreatment. Not all patients were initially referred for evaluation of a suspected cracked tooth. A total of 52 patients confirmed to have an intrapulpal crack between August 15, 2012 and August 15, 2013 were included in the study. All patients had one or more teeth with a confirmed intrapulpal crack upon visual microscopic evaluation. The involved teeth were mandibular molars (n= 25), maxillary molars (n=21), maxillary premolars (n=6), and mandibular premolars (n=0).

The inclusion and exclusion criteria were determined for selection of cases to include in this research study. All posterior teeth with intrapulpal cracks visible with or without staining at any magnification were included in this study, irrespective of the ability of the digital camera to document the crack line. All pulpal and periapical diagnoses necessitating root canal treatment were included. Not all teeth diagnosed with intrapulpal cracks were initially suspected to have a crack. Teeth with incomplete documentation of the diagnostic testing were included (i.e.: missing bite test or transillumination results). However, patients with incomplete documentation of the intrapulpal crack were excluded from the study. Also excluded were all teeth that did not have a fracture extending into the pulp chamber of the tooth visible under microscopic
magnification with or without staining. This included those crack lines extending through the enamel and into the dentin but not propagating beyond the pulp chamber roof. Anterior teeth, craze lines, cuspal fractures, split teeth, and vertical root fractures were also excluded from this study.

Graduate endodontic residents completed the initial evaluations of all patients including collecting patient subjective and objective information, clinical findings, diagnostic testing results and pulpal and periapical diagnoses. This information was recorded in the electronic patient record. Treatment options were reviewed with the patients and included: 1) Perform NSRCT or Retreatment with endodontic access and evaluation of the pulp chamber for the presence and location of an intrapulpal crack, 2) Extraction of the tooth and replacement with other prosthesis (i.e.: fixed partial denture, implant and crown, or removable partial denture), 3) No further treatment. The advantages, disadvantages, risks, benefits, and cost of these treatment options were discussed with the patient. All posterior teeth were evaluated for intrapulpal cracks upon access of the pulp chamber, regardless of whether or not a longitudinal crack was identified during the initial evaluation. If an intrapulpal crack was identified microscopically upon access, the crack was documented with the use the VCU Graduate Endodontics Intrapulpal Cracked Tooth Template (See Appendix A) and the findings recorded in the electronic record. The intrapulpal crack was then classified based upon the use of the novel classification system for intrapulpal cracks. The patient was informed of the findings and given the treatment options of either continuing with the NSRCT or Retreatment procedure or Extraction of the tooth. The treatment was not part of this research project.

The VCU Graduate Endodontics Intrapulpal Cracked Tooth Guidelines and Template were created previously for data gathering and documentation purposes as part of the Graduate
Endodontic policy for treatment of any tooth with an intrapulpal crack. All residents were instructed and calibrated on the use of the guidelines and the template for data collection. The treating clinician followed these guidelines and all documentation was recorded in the patient’s electronic chart (AxiUm) or the paper Intrapulpal Cracked Tooth Template. All residents were also previously calibrated on the use of the novel classification system for intrapulpal cracks and utilized this system during documentation.

Initial examination of the patient utilized both questions directed towards understanding the patient’s chief complaint and any history related to a possible intrapulpal crack as well as assessment of the tooth in question. The subjective symptoms were recorded based upon questions asked routinely during clinical evaluation of a patient. The diagnostic testing was completed using the following: Refrigerant Spray – 1,1,1,2 TetraFluoroEthane, mouth mirror, Tooth Slooth® (Professional Results, Inc., Laguna Niguel, CA), fiber optic transilluminator, and periodontal probe. Radiographic evaluation of the patient was assessed using periapical radiographs taken with Dexis CCD digital sensors (Dexis LLC, Hatfield, PA).

Initial examination of the patient would include the following data recorded in the patient’s electronic record:

Subjective Symptoms: Pain to Cold, Pain to Heat, Pain on Biting or Release, Localizable or Diffuse Pain

Diagnostic Testing: Cold test, Bite test (Biting and Release), Percussion test, Transillumination, Mobility, and Probing Depths

Radiographic Evaluation: Presence or Absence of a Periapical Radiolucency, Size of the Periapical Radiolucency, Periodontal Defects present (Isolated, Generalized, or Vertical)

Diagnosis:
Pulpal: Symptomatic Irreversible Pulpitis, Asymptomatic Irreversible Pulpitis, Pulpal Necrosis, Previously Treated


Following clinical examination and assessment of the tooth, the resident would review their findings, discuss treatment options, risks, benefits, cost, and anesthetize the patient. If the intrapulpal crack was identified upon access, then the template would be retrieved at that time. All documentation was collected, organized, and maintained in the Database by MSD and utilized for this retrospective study.

The image documentation was completed using two sources: 1) Sony 3-chip digital cube camera (Sony Corp. of America, New York, NY) and 2) Canon T4i DSLR camera (Canon USA, Arlington, VA). Both sources were integrated with the OPMI pico dental microscope (Carl Zeiss Meditec, Jena, Germany) utilizing a 50/50 beam splitter, T2 adapter ring, photo adapter, and Vello FreeWave wireless remote shutter release (Gradus Group LLC, New York, NY). The light sources used were Xenon and Halogen illumination. The OPMI Pico microscope provides 5 magnification settings: 0.4, 0.6, 1.0, 1.6, and 2.5, which correspond to the following magnifications, depending on the focal length of the objective: 250 nm: 3.40x, 5.10x, 8.50x, 13.60x, 21.25x; 300 nm: 2.83x, 4.25x, 7.08x, 11.33x, 17.71x.

Magnifications were calculated using the following formula:

\[ TM = \left( \frac{f_{T}}{f_{O}} \right) \times EP \times MV \]

TM: Total Magnification

f_{T}: Focal length telescope

f_{O}: Focal length objective
EP: Eyepiece Power

MV: Magnification Value

Images were taken with and without the use of Vista-Blue methylene blue dye (Vista Dental, Racine, WA) for staining purposes. Images were stored in the Intrapulpal Cracked Tooth Database located in a secure network drive accessible only by the investigators in this study.

The retrospective study design was independently reviewed and approved by the VCU Institutional Review Board (IRB #: HM20000027). Results were summarized with counts and percentages or with means and standard error, as appropriate. Associations were tested using likelihood-ratio chi-square analysis. All statistical analyses were performed using SAS software (JMP pro 11, SAS Institute Inc., Cary NC). Statistical significance was declared at alpha=0.05.
Results

The results of the study are presented in three parts based upon the characteristics and associations of intrapulpal cracks being evaluated:

I. Descriptive Characteristics

II. Associations and Predictive Trends: Tooth Type vs Location

III. Associations and Predictive Trends: Classification vs Clinical Examination

Part I: Descriptive Characteristics

Of the 52 patients who met the inclusion criteria, 62% were female and 69% were identified as Caucasian (Table 1). The average age was 56.5 years (SD = 9.49). The range of ages was 31-75 years.

Table 1. Demographics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>32</td>
<td>62</td>
</tr>
<tr>
<td>M</td>
<td>20</td>
<td>38</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>9</td>
<td>17</td>
</tr>
<tr>
<td>Asian</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Caucasian</td>
<td>36</td>
<td>69</td>
</tr>
<tr>
<td>Hispanic</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>(unspecified)</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>
Next, in evaluating the prevalence of intrapulpal cracks based on tooth type, there was approximately the same number of mandibular and maxillary teeth diagnosed with intrapulpal cracks (Table 2). The involved teeth were predominantly first and second molars (86%). Overall, only 12% of the cases were premolars, of which all were maxillary premolars. Within this study group, the mandibular 2\textsuperscript{nd} molar was found to be the most frequently diagnosed with an intrapulpal crack (27%), followed by the maxillary 1\textsuperscript{st} molar (23%), and the mandibular 1\textsuperscript{st} molar (19%).

<table>
<thead>
<tr>
<th>Type of tooth</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mandibular 1st molar</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>Mandibular 2nd molar</td>
<td>14</td>
<td>27</td>
</tr>
<tr>
<td>Mandibular 3rd molar</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Total mandible</td>
<td>25</td>
<td>48</td>
</tr>
<tr>
<td>Maxillary 1st premolar</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Maxillary 2nd premolar</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Maxillary 1st molar</td>
<td>12</td>
<td>23</td>
</tr>
<tr>
<td>Maxillary 2nd molar</td>
<td>9</td>
<td>17</td>
</tr>
<tr>
<td>Total maxillary</td>
<td>27</td>
<td>52</td>
</tr>
</tbody>
</table>

The status of the restoration present was next assessed. The majority of cases had existing amalgam restorations (54%). Almost one out of five cases were discovered upon access through a full coverage crown (17%). Only 2% of all cases were non-restored, while 33% contained one surface restorations and 31% contained two surface restorations (Table 3). Therefore, a total of 66% of all teeth diagnosed with intrapulpal cracks were non-restored or minimally restored (1-2 surface restorations).
Table 3. Restorative Status

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Existing Restoration</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amalgam</td>
<td>28</td>
<td>54</td>
</tr>
<tr>
<td>Composite</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>Crown</td>
<td>9</td>
<td>17</td>
</tr>
<tr>
<td>Temporary</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td><strong>Surfaces restored</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>1 surface</td>
<td>17</td>
<td>33</td>
</tr>
<tr>
<td>2 surfaces</td>
<td>16</td>
<td>31</td>
</tr>
<tr>
<td>3 surfaces</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>4+ surfaces</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Crown</td>
<td>9</td>
<td>17</td>
</tr>
</tbody>
</table>

Following assessment of the restorative status, the results of certain diagnostic tests were evaluated within the study group (Table 4). During the clinical examination, 79% of the cases were positive to transillumination and 73% were positive to percussion. Patients subjectively reported pain on biting 69% of the time whereas only 23% of patients reported pain on release. Following bite testing with the Tooth Slooth®, 68% of patients reported pain. Thirty-seven percent of cases had deep pockets (5mm or more). Transillumination, percussion, pain on biting, and bite test with Tooth Slooth® all tested positive in greater than 60% of patients with intrapulpal cracks (Figure 1).
### Table 4. Clinical Examination

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transillumination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>–</td>
<td>7</td>
<td>21</td>
</tr>
<tr>
<td>+</td>
<td>26</td>
<td>79</td>
</tr>
<tr>
<td>Percussion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>–</td>
<td>14</td>
<td>27</td>
</tr>
<tr>
<td>+</td>
<td>38</td>
<td>73</td>
</tr>
<tr>
<td>Pain on Biting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>16</td>
<td>31</td>
</tr>
<tr>
<td>Y</td>
<td>36</td>
<td>69</td>
</tr>
<tr>
<td>Pain on Release</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>20</td>
<td>77</td>
</tr>
<tr>
<td>Y</td>
<td>6</td>
<td>23</td>
</tr>
<tr>
<td>Bite Test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>–</td>
<td>12</td>
<td>32</td>
</tr>
<tr>
<td>+</td>
<td>25</td>
<td>68</td>
</tr>
<tr>
<td>Probing Depth (mm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 5 mm</td>
<td>33</td>
<td>63</td>
</tr>
<tr>
<td>5–10 mm</td>
<td>19</td>
<td>37</td>
</tr>
</tbody>
</table>

![Figure 1. Clinical Characteristics](image-url)
Next, certain questions were analyzed with respect to the use of the microscope, staining, and digital imaging in visualizing and classifying intrapulpal cracks. Using the specified protocol, intrapulpal cracks were evaluated under the dental microscope. The microscopic magnification values for initial visualization and classification of the intrapulpal crack were recorded. Subjective questions related to staining and evaluation of the crack were also analyzed. The results are shown in Table 5. The average magnification necessary for visualization was 4.3X (SD = 1.3X) and for classification of the intrapulpal crack was 7.0X (SD = 2.3X). On average, the classification magnification was 1.7x the visualization magnification, a statistically significant difference ($P < .0001$). Analysis of the ability of staining to enhance visualization or diagnosis demonstrated that in the majority of cases the answer was “yes” (65% visualization and 57% identification). The responses to these two questions were identical except for 3 cases (out of 37) where visualization=Yes and identification=No.
Table 5. Microscopic Findings

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At what magnification were you able to 1st visualize the crack? X</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.8</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>3.4</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>4.3</td>
<td>18</td>
<td>49</td>
</tr>
<tr>
<td>5.1</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>7.1</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td><strong>At what magnification did you feel you could classify the crack? X</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.4</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>4.3</td>
<td>10</td>
<td>27</td>
</tr>
<tr>
<td>5.1</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>7.1</td>
<td>9</td>
<td>24</td>
</tr>
<tr>
<td>8.5</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>11.3</td>
<td>8</td>
<td>22</td>
</tr>
<tr>
<td><strong>Did staining enhance your ability to visualize the crack in the scope?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>9</td>
<td>24</td>
</tr>
<tr>
<td>Equal</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Yes</td>
<td>24</td>
<td>65</td>
</tr>
<tr>
<td><strong>Did staining enhance your ability to diagnose the crack in the scope?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>12</td>
<td>32</td>
</tr>
<tr>
<td>Equal</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Yes</td>
<td>21</td>
<td>57</td>
</tr>
<tr>
<td><strong>Is what you saw under the scope the same as what you expected to see before you accessed the tooth?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>28</td>
<td>76</td>
</tr>
<tr>
<td>Yes</td>
<td>9</td>
<td>24</td>
</tr>
<tr>
<td><strong>Were you able to capture with your digital images everything you were able to see under the microscope?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>23</td>
<td>62</td>
</tr>
<tr>
<td>Yes</td>
<td>14</td>
<td>38</td>
</tr>
</tbody>
</table>

Finally, the prevalence of intrapulpal cracks with respect to the classification system was assessed. The location of the intrapulpal cracks was classified by pulpal wall and pulpal floor.

Only one wall (Type I) was involved in 75% of cases (Table 6). Pulpal Floor classifications were predominantly Type A, terminating at the floor-wall junction, (44%) and Type B, extending into
an orifice, (40%). Overall, 84% of the intrapulpal cracks did not physically involve the pulpal floor. Three cases with an intrapulpal crack involving two walls (Type II) had two different Pulpal Floor classifications, i.e.: Terminating at the floor-wall junction at one wall and extending into an orifice at the other wall. For these cases, the more severe classification was chosen, making an assumption that was not based upon any clinical evidence.

**Table 6. Intrapulpal Crack Classifications**

<table>
<thead>
<tr>
<th>Pulpal Floor Classification</th>
<th>Pulpal Wall Classification</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>II</td>
</tr>
<tr>
<td>A</td>
<td>19</td>
<td>4</td>
</tr>
<tr>
<td>B</td>
<td>19</td>
<td>2</td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>D</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
<td>13</td>
</tr>
</tbody>
</table>

Abbreviations: I=one wall involved, II=two walls involved, A=crack terminates at floor-wall junction and does not involve orifice(s), B=extends into orifice(s) and does not involve the floor, C=traverses partially across the floor and does not involve the orifice(s), D=traverses entire floor and does not involve orifice(s).

Part II: Associations and Predictive Trends: Tooth Type vs Location

One of the specific aims of this study was to analyze possible associations and predictive trends for the location of intrapulpal cracks based upon individual tooth types. Identifying associations and trends for tooth types may help in clinical identification and prediction of the location and extent of the intrapulpal crack.

The first analysis evaluated the relationship of tooth type and intrapulpal classification (Table 7). There was no obvious relationship identified between the intrapulpal classifications and tooth type. Overall, 73% of mandibular molars, 67% of maxillary molars, and 84% of
maxillary premolars were classified as Type I-A, one wall and terminating at the floor-wall junction, or Type I-B, one wall and extending into an orifice. The predominant pulpal wall classification for each tooth type was Type I, only wall involvement. The predominant floor classification differed based upon tooth type. In mandibular molars, Type B, extending into an orifice, (54%) was the most prevalent classification followed by Type A, terminating at the floor-wall junction, (27%). In maxillary molars and maxillary premolars, Type A (52%; 84%) was the most prevalent classification followed by Type B (29%; 17%).

### Table 7. Tooth Type vs Classification

<table>
<thead>
<tr>
<th>Arch, Tooth</th>
<th>Mandibular Molar</th>
<th>Maxillary Molar</th>
<th>Maxillary Premolar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classification</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I–A</td>
<td>27%</td>
<td>38%</td>
<td>67%</td>
<td>4</td>
</tr>
<tr>
<td>I–B</td>
<td>46%</td>
<td>29%</td>
<td>17%</td>
<td>1</td>
</tr>
<tr>
<td>I–C</td>
<td>4%</td>
<td>0%</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>II–A</td>
<td>0%</td>
<td>14%</td>
<td>17%</td>
<td>1</td>
</tr>
<tr>
<td>II–B</td>
<td>8%</td>
<td>0%</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>II–C</td>
<td>0%</td>
<td>5%</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>II–D</td>
<td>12%</td>
<td>14%</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Chi-square $P = 0.1587$

### Arch, Tooth

<table>
<thead>
<tr>
<th>Pulpal Wall Classification</th>
<th>Mandibular Molar</th>
<th>Maxillary Molar</th>
<th>Maxillary Premolar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>77%</td>
<td>67%</td>
<td>83%</td>
<td>75%</td>
</tr>
<tr>
<td>II</td>
<td>19%</td>
<td>33%</td>
<td>17%</td>
<td>25%</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>21</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Chi-square $P = 0.5162$

### Arch, Tooth

<table>
<thead>
<tr>
<th>Pulpal Floor Classification</th>
<th>Mandibular Molar</th>
<th>Maxillary Molar</th>
<th>Maxillary Premolar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>27%</td>
<td>52%</td>
<td>83%</td>
<td>44%</td>
</tr>
<tr>
<td>B</td>
<td>54%</td>
<td>29%</td>
<td>17%</td>
<td>40%</td>
</tr>
<tr>
<td>C</td>
<td>4%</td>
<td>5%</td>
<td>0%</td>
<td>4%</td>
</tr>
<tr>
<td>D</td>
<td>12%</td>
<td>14%</td>
<td>0%</td>
<td>12%</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Chi-square $P = 0.1789$
The relationship between tooth type and crack direction was next evaluated (Table 8).

Overall, 92% of all intrapulpal cracks occurred in the mesiodistal direction. No significant associations for any specific tooth type were discovered.

Table 8. Tooth Type vs Crack Direction

<table>
<thead>
<tr>
<th>Direction</th>
<th>Mandibular Molar</th>
<th>Maxillary Molar</th>
<th>Maxillary Premolar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>B-L</td>
<td>8%</td>
<td>10%</td>
<td>17%</td>
<td>8%</td>
</tr>
<tr>
<td>M-D</td>
<td>92%</td>
<td>90%</td>
<td>83%</td>
<td>92%</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Chi-square $P = 0.5301$

Next, analysis of the marginal ridge involved with the intrapulpal crack and the tooth types revealed significant associations and predictive trends. The marginal ridge involvement varied by tooth type (Table 9 and Figure 2). The DMR involvement seemed to predominate in mandibular molars (46%) and was less in maxillary molars (19%) and premolars (0%). Statistical analysis confirmed that intrapulpal cracks significantly more often involved the DMR in mandibular molars than in maxillary premolars and molars ($P = 0.0097$). Intrapulpal cracks more often involved the MMR in maxillary premolars (33%) and molars (29%) than mandibular molars (0%), but not significantly ($P = 0.0522$). Combined MMR and DMR involvement was similar in mandibular molars and maxillary molars.
### Table 9. Tooth Type vs Marginal Ridge Involvement

<table>
<thead>
<tr>
<th>Marginal Ridge(s) Involved</th>
<th>Arch, Tooth</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mandibular Molar</td>
<td>Maxillary Molar</td>
<td>Maxillary Premolar</td>
<td>Total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DMR</td>
<td>46%</td>
<td>19%</td>
<td>0%</td>
<td>0%</td>
<td>33%</td>
<td>17</td>
</tr>
<tr>
<td>MMR</td>
<td>0%</td>
<td>0%</td>
<td>29%</td>
<td>6%</td>
<td>33%</td>
<td>2</td>
</tr>
<tr>
<td>MMR, DMR</td>
<td>23%</td>
<td>4%</td>
<td>24%</td>
<td>5%</td>
<td>23%</td>
<td>12</td>
</tr>
<tr>
<td>n/a</td>
<td>15%</td>
<td>4%</td>
<td>24%</td>
<td>5%</td>
<td>33%</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>12%</td>
<td>5%</td>
<td>1%</td>
<td>17%</td>
<td>10%</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>52</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Chi-square $P = 0.0097$

Abbreviations: DMR=Distal Marginal Ridge, MMR=Mesial Marginal Ridge, n/a=not applicable (i.e.: Crown, MOD restoration), None=No crack noted in Marginal Ridge

![Figure 2. Tooth Type vs Marginal Ridge Involvement](image-url)
Following analysis of the marginal ridge involvement, the pulpal wall involvement was next assessed. Similar to the marginal ridge findings, the pulpal wall involvement also varied by tooth type (Table 10 and Figure 3). Overall, the distal wall predominated and this seemed to especially be the case in mandibular molars, where 69% of the cases involved only the distal wall. Mesial wall involvement seemed to be more prevalent in maxillary molars (38%) and premolars (50%). Intrapulpal cracks significantly more often involved the distal wall in mandibular molars than in maxillary premolars and molars ($P = 0.0025$). Intrapulpal cracks significantly more often involved the mesial wall in maxillary premolars and molars than mandibular molars ($P = 0.0015$). Mesial and distal wall involvement seemed to favor maxillary molars (33%) compared to maxillary premolars (17%) or mandibular molars (15%). However, this relationship was not statistically significant.

Table 10. Tooth Type vs Pulpal Wall Involvement

<table>
<thead>
<tr>
<th>Pulpal Wall(s) Involved</th>
<th>Arch, Tooth</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mandibular Molar</td>
<td>Maxillary Molar</td>
<td>Maxillary Premolar</td>
<td>Total</td>
</tr>
<tr>
<td>M</td>
<td>8%</td>
<td>2</td>
<td>38%</td>
<td>8</td>
</tr>
<tr>
<td>D</td>
<td>69%</td>
<td>18</td>
<td>19%</td>
<td>4</td>
</tr>
<tr>
<td>M, D</td>
<td>15%</td>
<td>4</td>
<td>33%</td>
<td>7</td>
</tr>
<tr>
<td>B</td>
<td>0%</td>
<td>0</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>L</td>
<td>0%</td>
<td>0</td>
<td>10%</td>
<td>2</td>
</tr>
<tr>
<td>B, L</td>
<td>4%</td>
<td>1</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Chi-square $P = 0.0025$

Abbreviations: M=Mesial, D=Distal, B=Buccal, L=Lingual
Significant associations were next assessed for tooth type and pulpal orifice involvement. The orifice involvement again varied by tooth type (Table 11). In 56% of all cases there was no orifice involvement. The percentage of intrapulpal cracks with no orifice involvement was much higher for maxillary premolars (83%) and maxillary molars (67%) than for mandibular molars (40%). However, this difference was not statistically significant ($P = 0.1290$). Within the mandibular molar types, involvement of the distal orifice was significantly more common than involvement of any other orifice ($P = 0.0013$).

**Figure 3. Tooth Type vs Pulpal Wall Involvement**
Table 11. Tooth Type vs Pulpal Orifice Involvement

<table>
<thead>
<tr>
<th>Pulpal Orifices Involved</th>
<th>Arch, Tooth</th>
<th>Mandibular Molar</th>
<th>Maxillary Molar</th>
<th>Maxillary Premolar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>MB, D</td>
<td></td>
<td>8%</td>
<td>2</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>MB1</td>
<td></td>
<td>0%</td>
<td>0</td>
<td>10%</td>
<td>2</td>
</tr>
<tr>
<td>MB2</td>
<td></td>
<td>0%</td>
<td>0</td>
<td>5%</td>
<td>1</td>
</tr>
<tr>
<td>D</td>
<td></td>
<td>36%</td>
<td>9</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>DB</td>
<td></td>
<td>4%</td>
<td>1</td>
<td>5%</td>
<td>1</td>
</tr>
<tr>
<td>DL</td>
<td></td>
<td>12%</td>
<td>3</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>B</td>
<td></td>
<td>0%</td>
<td>0</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td>0%</td>
<td>0</td>
<td>14%</td>
<td>3</td>
</tr>
<tr>
<td>n/a</td>
<td></td>
<td>40%</td>
<td>10</td>
<td>67%</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>6</td>
</tr>
</tbody>
</table>

Chi-square $P = 0.0013$

Part III: Associations and Predictive Trends: Classification vs Clinical Examination

Another specific aim of this study was to evaluate the proposed intrapulpal classification system. To address this aim, the associations between the individual classifications of the intrapulpal crack and certain clinical and radiographic examination findings were tested. Each analysis addresses three aspects of the association: the association with the 8-category classification system, the association with the pulpal wall classification (i.e.: I vs II), and the association with the pulpal floor classification (i.e.: A vs B vs C vs D).

The relationship between the subjective symptom of pain on biting and the classification system was first assessed. Within the 52 cases included in the study group, there were 38 classified as Type I–A, one wall and terminating at the floor wall junction, or Type I–B, one wall and extending into an orifice. Overall, the ratio of pain on biting Yes to No was approximately 2 to 1. As may be seen in Table 12, this ratio was also evident in the predominant classifications. However, there was no association between the overall classification of the intrapulpal crack and pain on biting ($P = 0.2495$). Furthermore, there was no evidence for a statistically significant
association with either the pulpal wall classification \((P = 0.1197)\) or the pulpal floor classification \((P = 0.8857)\).

### Table 12. Classification vs Pain on Biting

<table>
<thead>
<tr>
<th>Pain on Biting</th>
<th>I–A</th>
<th>I–B</th>
<th>I–C</th>
<th>II–A</th>
<th>II–B</th>
<th>II–C</th>
<th>II–D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>6</td>
<td>7</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>16</td>
</tr>
<tr>
<td>Y</td>
<td>13</td>
<td>12</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>19</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

*Overall chi-square \(P\)-value = 0.2495*

*Wall classification \(P\)-value = 0.1197*

*Floor classification \(P\)-value = 0.8857*

Next, bite testing was evaluated for any significant association with the various classifications for intrapulpal cracks (Table 13). There was insufficient evidence for a significant association between the overall classification of the intrapulpal crack and the bite test result \((P = 0.0580)\). However, in evaluating only the pulpal wall classification, 10 of 25 teeth with a positive bite test result were classified Type II, two walls involved, \((40\%)\) whereas only 1 of 13 teeth with a negative bite test result were classified Type II \((7.7\%)\). This difference was statistically significant \((P = 0.0250)\). Similarly, in evaluating only the pulpal floor classification, 9 of 25 teeth with a positive bite test result were classified as Type A, terminating at the floor-wall junction, \((36\%)\) whereas 8 of 12 teeth with a negative bite test result were classified as Type A \((67\%)\). This difference was also statistically significant \((P = 0.0356)\).
Table 13. Classification vs Bite Test

<table>
<thead>
<tr>
<th>Bite Test</th>
<th>I–A</th>
<th>I–B</th>
<th>I–C</th>
<th>II–A</th>
<th>II–B</th>
<th>II–C</th>
<th>II–D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>–</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>+</td>
<td>6</td>
<td>9</td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>12</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>37</td>
</tr>
</tbody>
</table>

Overall chi-square $P$-value = 0.0580

Wall classification $P$-value = 0.0250

Floor classification $P$-value = 0.0356

The relationship between transillumination and the intrapulpal crack classifications was next analyzed (Table 14). There was insufficient evidence for a significant association between the overall classification of the intrapulpal crack and the transillumination result ($P = 0.1254$). However, in evaluating only the pulpal wall classification, 10 of 26 teeth with a positive transillumination result were classified Type II, two walls involved, (39%) whereas 0 of 7 teeth with a negative transillumination result were classified Type II (0%). This difference was statistically significant ($P = 0.0172$). In evaluating only the pulpal floor classification, this difference was no longer significant ($P = 0.3101$).
Table 14. Classification vs Transillumination

<table>
<thead>
<tr>
<th>Classification</th>
<th>I–A</th>
<th>I–B</th>
<th>I–C</th>
<th>II–A</th>
<th>II–B</th>
<th>II–C</th>
<th>II–D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transillumination –</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>+</td>
<td>5</td>
<td>10</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>26</td>
</tr>
<tr>
<td>n/a</td>
<td>7</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
<td>16</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>6</td>
<td>45</td>
</tr>
</tbody>
</table>

Overall chi-square $P$-value = 0.1254

Wall classification $P$-value = 0.0172

Floor classification $P$-value = 0.3101

Probing depths and classifications were also analyzed for significant associations. As can be seen in Table 15, there was insufficient evidence for any significant association between the intrapulpal crack classifications and probing depth. Overall, 33 of 52 (64%) cases had probing depths < 5 mm. Based upon the $P$-values calculated, no significant association could be made between the location of the intrapulpal crack and the probing depth.
Next, the ability of staining to enhance the diagnosis was assessed for any significant relationship with the various intrapulpal crack classifications. As can be seen in Table 16, there was no evidence of any significant association between the intrapulpal crack classifications and staining to enhance the diagnosis. While all but Type II-C, two walls and traversing partially across the floor, demonstrated a greater number of Yes values for staining enhancement, there seemed to be no overall relationship between any individual classification and staining enhancement. Based upon the $P$-values noted, there could be no significant association identified between the location of the intrapulpal crack and staining for enhancement of diagnosis of the crack.
Table 16. Classification vs Staining

<table>
<thead>
<tr>
<th>Staining Enhanced Diagnosis</th>
<th>I–A</th>
<th>II–A</th>
<th>I–C</th>
<th>II–B</th>
<th>I–D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>4</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Equal</td>
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<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Yes</td>
<td>6</td>
<td>1</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>21</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>15</td>
<td>0</td>
<td>4</td>
<td>1</td>
<td>4</td>
</tr>
</tbody>
</table>

Overall chi-square $P$-value = 0.4043

Wall classification $P$-value = 0.9709

Floor classification $P$-value = 0.5558

The existence of a periapical radiolucency was next analyzed for any relationship with the intrapulpal crack classifications (Table 17). Analysis of the combined pulpal wall and floor classifications demonstrated a statistically significant ($P = 0.0232$) relationship between the overall classification and the presence of a periapical radiolucency. Evaluating the number of cases with and without periapical radiolucencies within each overall classification group revealed several patterns. Type I-A, one wall and terminating at the floor-wall junction, and I-B, one wall and extending into an orifice, groups had a greater number of cases with periapical radiolucencies whereas Type II-A, two walls and terminating at the floor-wall junction, and Type II-B, two walls and extending into an orifice, groups had a greater number of cases without radiolucencies. In the opposite manner, Type I-C, one wall and traversing partially across the floor, and Type I-D, one wall and traversing across the entire floor, groups had a greater number of cases without periapical radiolucencies while Type II-C, two walls and traversing partially across the floor, and Type II-D, two walls and traversing across the entire floor, groups had a
greater number of cases with periapical radiolucencies. However when evaluating the individual classifications for the location of the intrapulpal crack within the pulpal walls or floor alone, no statistically significant relationship existed with the presence or absence of a periapical radiolucency.

Table 17. Classification vs Periapical Radiolucency

<table>
<thead>
<tr>
<th>Periapical Radiolucency</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I–A</td>
</tr>
<tr>
<td>N</td>
<td>8</td>
</tr>
<tr>
<td>Y</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
</tr>
</tbody>
</table>

Overall chi-square $P$-value = 0.0232
Wall classification $P$-value = 0.4752
Floor classification $P$-value = 0.4412

Next, the relationship between the pulpal diagnosis and the location of the intrapulpal crack as classified by the novel classification system was analyzed. As can be seen in Table 18, there was no evidence of a significant association between the intrapulpal crack classifications and the pulpal diagnosis. Although there were a greater number of cases with a diagnosis of necrotic pulp within almost all classifications, this was not statistically significant. Based upon the $P$-values noted, there could be no significant association made between the location of the intrapulpal crack and the pulpal diagnosis for the tooth.
Table 18. Classification vs Pulpal Diagnosis

<table>
<thead>
<tr>
<th>Pulpal Diagnosis</th>
<th>I–A</th>
<th>I–B</th>
<th>I–C</th>
<th>II–A</th>
<th>II–B</th>
<th>II–C</th>
<th>II–D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital</td>
<td>5</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>17</td>
</tr>
<tr>
<td>Previous Treatment</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Necrotic</td>
<td>8</td>
<td>10</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>26</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>19</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Overall chi-square P-value = 0.7693
Wall classification P-value = 0.1305
Floor classification P-value = 0.6414

Finally, the relationship between the periapical diagnosis and the location of the intrapulpal crack as classified by the classification system was analyzed. As can be seen in Table 19, the diagnosis of Symptomatic Apical Periodontitis was the predominant periapical diagnosis for every overall classification group with the exception of Type II-C, two walls and traversing partially across the floor, which accounted for one of fifty-two cases. In evaluating the pulpal wall or floor classifications individually, there seemed to be no relationship between any of the classifications and the periapical diagnosis. Based upon the P-values noted, there could be no statistically significant association established between the location of the intrapulpal crack and the periapical diagnosis of the tooth.
<table>
<thead>
<tr>
<th>Periapical Diagnosis</th>
<th>I–A</th>
<th>I–B</th>
<th>I–C</th>
<th>II–A</th>
<th>II–B</th>
<th>II–C</th>
<th>II–D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic Apical Periodontitis</td>
<td>15</td>
<td>13</td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>4</td>
<td>37</td>
</tr>
<tr>
<td>Asymptomatic Apical Periodontitis</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Acute Apical Abscess</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Chronic Apical Abscess</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Normal Periapical Tissues</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>19</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>6</td>
<td>52</td>
</tr>
</tbody>
</table>

Overall chi-square $P$-value = 0.712

Wall classification $P$-value = 0.9318

Pulpal classification $P$-value = 0.5726
Discussion

The cracked tooth has been defined by the AAE as the more centrally located fracture that extends apically and may cause pulpal and periapical pathosis (31). Rivera and Williamson (32) denoted the longitudinal nature of this crack in reference to both the directional and time components. Extensive research has been pursued in an attempt to understand the diagnosis, treatment, and prognosis of this unpredictable phenomenon. Unfortunately, most of this research has focused on the external and coronal aspects of the cracked tooth, with little emphasis on the internal pulpal or radicular structures. When reference is made to pulpal and radicular structures, most authors have empirically stated that cracks involving these structures have a poor or hopeless prognosis and therefore should be extracted. The treatment option for NSRCT has not been considered to be predictable long term.

More recently, several studies attempted to answer the question of treatment of cracks involving the pulpal or radicular studies. Liu and Sidhu (38) evaluated 6 cases treated for pulpal disease with an etiology of direct communication of a longitudinal crack with the pulp chamber. All 6 cases remained asymptomatic and demonstrated periapical healing at 1-3.5 years post treatment. Tan et al. (39) evaluated 49 patients diagnosed with irreversible pulpitis and cracks communicating with the pulp chamber and possibly involving the radicular structures. At 2-year recall, 85.5% of all treated teeth were considered to be functional and successful by survival criteria. While these studies were small in sample size and utilized variable classification methods, success criteria, and recall time, they were important in emphasizing the need for
extensive clinical evaluation and study of cracks extending beyond the coronal tooth structure and involving the internal pulpal and radicular structures.

Intrapulpal cracks, as defined by the author, are the longitudinal cracks extending beyond the coronal tooth structure and involving the pulpal and radicular structures of the tooth. With no previous classification system to describe the pulpal and radicular aspects of these cracks and no clinical studies to evaluate the prevalence, characteristics, or associations of these cracks, this study was the first in attempting to address some of these grey areas in the endodontic literature. The first of several goals of this study was to understand the characteristics of intrapulpal cracks. Descriptive characteristics included: demographic findings, the effectiveness of certain diagnostic testing methods and microscopic examination, prevalence data with respect to tooth type and restorative status, and the overall prevalence of intrapulpal cracks utilizing the proposed classification system.

Fifty-two cases of intrapulpal cracks were documented over a 1-year period in the Graduate Endodontic practice. The overall prevalence of intrapulpal cracks within the practice population (n=843) was 5.9%. This percentage contrasts with previous studies that attempted to quantify the prevalence of cracked teeth. Eakle et al. (41) evaluated fractures (n=206) in posterior teeth in an undergraduate setting and noted that 18% of the incomplete fractures involved the pulp chamber. The specific involvement of the pulp chamber structures was not discussed. Krell and Rivera (33) found that over a 6-year period (n=8175), 9.7% of cases evaluated in an endodontic private practice population were diagnosed as cracked teeth. It is important to note that these studies primarily assessed cracked teeth and not intrapulpal cracked teeth. There were no previous studies that assessed the prevalence of intrapulpal cracks within a clinic population. In addition,
the current study was over a one-year period of time. Future studies may incorporate a larger group of patients to re-evaluate the prevalence of these cracks.

The demographic results indicated that 62% of patients were female. This predilection for female patients with intrapulpal cracks agrees with Homewood (35), Abbott and Leow (15), and Cameron (5), who all suggested that cracks or CTS may be more common in female patients due to better dental education or were more likely to seek care when a tooth is symptomatic. The most common age group found was the 50-59 year age group (48.1%). When including the 60-69 year age group, these groups accounted for 78.8% of the intrapulpal cracked teeth cases. This finding is in agreement with several previous studies on cracked teeth and CTS, and could indicate a greater proportion of older patients seeking care in a specialty practice, the retention of teeth that may become less hydrated as they age and thus more susceptible to fracture, as well as teeth that sustain repeated occlusal stresses over time and a longitudinal time component with these cracks (5, 7, 42). The mechanism or mechanisms responsible for the greater proportion of the patients within the 50-59 and 60-69 year categories has not been determined at this point in time.

Several previous studies have evaluated the prevalence of tooth types associated with cracks and suggested the mandibular 2nd molar is the most commonly cracked tooth (1, 4, 5, 33). In our study, we found that intrapulpal cracks seem to follow the same pattern, with mandibular 2nd molars the most prevalent tooth type followed by the maxillary 1st molar and mandibular 1st molar respectively. These findings would be expected, as the intrapulpal crack is a direct extension of the longitudinal crack into the pulp chamber, further involving the pulp chamber walls and possibly floor. It has been suggested that mandibular molars are more susceptible to fracture due to structural defects in the central grooves, lack of a transverse ridge, the plunger
cusp effect of the maxillary molar palatal cusp, as well as the bite force generated in molars due to the proximity to the temporomandibular joint (1, 7, 44).

The restorative status of the cracked tooth has been discussed in several studies. While both Hiatt (1) and Roh and Lee (42) found that the majority of cracks occurred in teeth with no restoration or minimally restored, others have determined the opposite and argued that the larger restoration meant a greater susceptibility for crack propagation (5, 35, 44). In our study, we found that the greatest percentage of intrapulpal cracks occurred in teeth with a 1 surface restoration (33%) and nearly two-thirds of all cases had only 1 or 2 surfaces restored (64%). Interestingly, 17% of all intrapulpal cracks were located in teeth with full coverage crowns. These results seem to indicate that the majority of teeth with intrapulpal cracks were less heavily restored. The susceptibility to propagation of the longitudinal crack may be due to weakening of the marginal ridges or coronal tooth structure by intra-coronal restorations (44). While restorations have been suggested to deflect cracks in an oblique direction and result more often in cuspal fracture, it may be that restorations also weaken the tooth and permit extension of the fracture (1, 5, 44). The majority of teeth (54%) were restored with amalgam, which may play a role in the propagation of the longitudinal crack. Amalgam has a higher thermal expansion coefficient compared with teeth and may be more likely to propagate a fracture (1, 5, 19, 44). The full coverage crown with cuspal coverage may protect the tooth from mechanical forces and may prevent propagation of the fracture. In the case of the 17% found to have an intrapulpal crack upon access through a full coverage crown, it may be that one reason for the placement of the crown was to protect the tooth against an existing longitudinal crack. However, no historical evidence in the dental records was given to confirm or deny this theory.
In evaluating the diagnostic tools used for determination of the presence of an intrapulpal crack, the same tools were utilized as those recommended for cracked teeth. Transillumination, bite test, probing depths, staining and microscopic evaluation were all evaluated for positive or negative response in aiding diagnosis of these intrapulpal cracks. Findings indicated that the majority of teeth with intrapulpal cracks tested positive to transillumination (79%) and bite test (68%). The majority of patients also described pain on biting (69%). Figure 1 demonstrates the positive findings of these tests. These findings are in agreement with previous studies on cracked teeth (1, 2, 4, 5, 7-10, 44). Abou Rass (8) has previously stressed the use of transillumination with a fiber optic light source for diagnosis of a cracked tooth as one of the most valuable diagnostic techniques. Ehrmann and Tyas (4) as well as others (9, 10, 26) have recommended the use of a Tooth Slooth® for diagnostic bite testing as a primary tool to identify a cracked tooth. The Tooth Slooth® applied to individual cusps of the affected tooth will typically result in a painful response when occlusal forces are increased and relief occurs once the pressure is withdrawn. However, others have demonstrated pain on withdrawing of the bite forces to the affected tooth (4, 5, 7, 8, 10). Ritchey et al. (2) were the first to suggest that pain on biting was associated with incomplete fractures in the long axis of molars and involving the pulp chambers. Cameron (3, 5) reiterated this finding in his work on the CTS. Pain on biting has been repeatedly reinforced as a common symptom in the cracked tooth (1-5, 7).

The subjective finding of pain on biting as well as positive results for the bite test and transillumination of intrapulpal cracks could be expected as this crack is a direct extension of the crack line to involve the pulpal and possibly radicular structures. However, no evaluation of these testing results for the intrapulpal crack has been previously studied to confirm these expectations. In their analysis of factors associated with the cracked tooth, Seo et al. (44) found
that 51% of patients experienced bite pain and 82.2% responded positively to the bite test. These values are comparable to this study’s finding of 69% of patients experiencing bite pain and 68% responding positively to the bite test. These findings suggest a predictive nature of these subjective symptoms and clinical tests for the presence of intrapulpal cracks. However, without direct comparison of intrapulpal-cracked teeth to either non-cracked teeth or teeth with cracks limited to the coronal tooth structure, we can not make a statement with regard to the predictive nature of these diagnostic tests.

Interesting to note was that 73% of all cases of intrapulpal cracks were percussion positive to axial inclined forces, which could suggest the periradicular extension of pulpal inflammation or lateral PDL inflammation secondary to crack extension. Rosen (7) suggested that as the crack progresses apically, the crestal epithelial attachment and proprioceptive A-beta fibers within the PDL are sensitized and results in pain to percussion. This mechanical allodynia is also seen commonly with irreversible pulpitis and pulpal necrosis due to apical progression of inflammation (45). In their study on longitudinal cracked teeth, Seo et al. (44) found that 43.9% were percussion positive. The greater percentage of percussion positive cases in our study could be explained by the confirmed presence of intrapulpal cracks in our study. As all cracks have extended to include the pulpal structures, more severe pulpal and periapical inflammatory responses would be expected. This could account for the greater mechanical allodynia associated with the periapical tissues in our study.

Probing depths were found to be greater than 5 mm in 37% of patients in this study. This finding is within the range of several other studies that evaluated probing depths of cracked teeth. Seo et al. (44) noted that 48.6% of the patients in their study had probing depths of < 3 mm, while 19.6% had probing depths > 6 mm. Tan et al. (39) found that 34% of the cracked teeth
with confirmed pulpal and possible radicular involvement had probing depths > 3 mm. Those teeth associated with deeper probing depths could suggest an extension of the intrapulpal crack into the PDL as the crack propagates further apically into the pulpal and radicular structures. Hiatt (1) first discussed this periodontal breakdown due to the propagation of cracks apically. The propagation of the crack may lead to inflammation associated with the periodontal ligament and/or the crestal epithelial attachment (1, 7). Whether or not these probing depths were associated with the intrapulpal crack or another etiology of periodontal inflammation and breakdown was not evaluated in this study. Future studies evaluating the periodontal component of teeth with intrapulpal cracks would be necessary to confirm this theory. It is also difficult to accurately measure probing depths on the direct mesial or distal surfaces of the tooth with an intrapulpal crack unless there are no adjacent teeth. This difficulty may underestimate or overestimate the percentage of significant probing defects.

Microscopic findings evaluated the clinician’s ability to diagnose and classify the intrapulpal crack. The subjective nature of evaluating these types of cracks was assessed as the clinician recorded the magnifications required to visualize and classify the intrapulpal crack. As can be seen in the results section (Table 5), the average magnification required of the clinician to adequately classify the intrapulpal crack (7.0X) was significantly greater than the magnification required to identify the intrapulpal crack (4.3X). The average magnification for classification was 1.7 times the magnification for visualization (P < .0001). This could be explained by the difficulty in assessing the apical extent and location of the intrapulpal crack. Current limitations with illumination sources could also complicate the ability to visualize the complete extent of the fracture. These complications in visualizing crack lines within teeth have been stressed by
Ehrmann and Tyas (4) and others (9, 10). Future studies utilizing different illumination and microscopic systems would be advantageous in further assessing this clinical difficulty.

Additional subjective questions investigated staining and the image capture of intrapulpal cracks. The majority of clinicians felt that staining enhanced their ability to visualize and diagnose the intrapulpal crack. Staining with methylene blue was utilized and has been previously indicated for the diagnosis of cracked teeth (2, 5, 8, 9, 38). Others have concluded that staining did not enhance their ability to diagnose the crack (4, 46, 47). Ehrmann and Tyas (4) have argued that complete visualization of cracks is not possible and not necessary for treatment. These studies were conducted without the aid of microscopes and did not investigate cracks within the pulp chamber specifically. Furthermore, we evaluated the ability to document the crack with digital capture technology. The findings suggested that the current technology for digital imaging documentation of intrapulpal cracks with the microscope is inadequate. Most clinicians felt that what could be seen visually within the optics of the microscope could not be replicated in digital images. This could be a result of the current limitations with the microscopes, lighting sources, or digital image capture technology. Future studies comparing different microscope systems, illumination sources, and digital image capture technologies would be pertinent to address these shortcomings.

Based upon the microscopic findings, the clinician was able to visualize and classify the intrapulpal crack with the current microscopic and illumination technology. While the magnification required for classification of the crack was significantly greater, the treating clinician was able to adequately diagnose the crack with the proposed classification system. The ability to capture the intrapulpal crack digitally was the shortcoming identified in this evaluation. While this data is subjective in nature, it provides support to the clinician’s ability to visually
identify and diagnose the intrapulpal crack with current microscopic technology. Future studies comparing subjective and objective identification of the intrapulpal crack through in vivo and ex vivo methods would be helpful in assessing the validity of our findings.

Another purpose of this study was to understand the characteristics of intrapulpal cracks. To this effect, evaluating the prevalence of these cracks with respect to the pulpal walls, pulpal floor, and pulpal orifices was important in beginning to understand the nature of intrapulpal cracks. The previously described classification system was created in an attempt to simplify an all-encompassing categorization of the various intrapulpal cracks that could be seen internally within the pulp chamber. The pulpal wall classifications were I: 1 wall and II: 2 walls. The pulpal floor classifications were A: Terminates at floor-wall junction, not involving orifice(s), B: Extends into orifice, not involving floor, C: Traverses partially across floor, not involving orifice(s), D: Traverses across entire floor, not involving orifice(s). The results of the study found that 75% of the intrapulpal cracks were classified as Type I, or involving only one wall of the pulp chamber. Only 25% of the intrapulpal cracks were Type II, or involving two walls of the pulp chamber (i.e.: M and D walls). Within the limitations of the study, these results would seem to indicate that the majority of cracks extending into the pulp chamber would only be involving one wall of the pulp chamber. It could be hypothesized that intrapulpal cracks involving more walls of the tooth would predispose the tooth to a worse prognosis. To this effect, Tan et al. (39) found that teeth involving multiple cracks were predisposed to a worse prognosis. However, the effect of multiple wall involvement on treatment and prognosis for the intrapulpal crack is unknown at this time.

In regards to the pulpal floor and orifices, the results found that 44% of the intrapulpal cracks were classified Type A and 40% were classified Type B. Only 4% of the intrapulpal cracks were
classified Type C and 12% Type D. These findings indicate that the majority (84%) of intrapulpal cracks do not involve the pulpal floor. An almost equal number of cracks terminated at the pulpal floor-wall junction (Type A, 44%) as extended into a pulpal orifice without involvement of the pulpal floor (Type B, 40%). Within the limits of our study, these findings indicate that almost half of the intrapulpal cracks did not involve an orifice or the floor. These findings are in agreement with Tan et al. (39), who found that Coronal cracks (not involving the floor) were more prevalent than Radicular cracks (extending beyond the orifices). Krell and Rivera (33) also found that 100% (n=27) of the teeth with cracks within the pulp chamber upon access for NSRCT did not involve the pulpal floor.

The involvement of the pulpal floor or orifices would seem to indicate that the intrapulpal crack has a radicular component and a worse prognosis then the intrapulpal crack terminating at the floor-wall junction. In their work on vertical fractures, Clark and Caughman (30) stated that vertical fractures through the pulp and into the root were hopeless. Lynch and McConnell (10) also recommended that cracks running through the pulpal floor or below the level of the alveolar bone should be considered hopeless and the tooth should be extracted. While these empirical based studies would recommend extraction of a tooth with a fracture below the level of the pulpal floor, there are no higher levels of evidence to support extraction of these teeth. Tan et al. (39) evaluated the survival of teeth with intrapulpal cracks and found that the extent of the crack did not significantly affect the survival of the tooth at 2-year recall. It would be an assumption to state that intrapulpal cracks involving the pulpal floor or orifices are more severe than those terminating at the floor-wall junction. Future research should be aimed at long term outcome studies for these types of cracks to support or contradict these assumptions.
Another purpose of this study was to evaluate the existence of significant associations and predictive trends for intrapulpal cracks with regards to specific tooth groups. The intrapulpal cracks were evaluated for their overall classification, crack direction, marginal ridge involvement, pulpal wall involvement, and pulpal floor involvement with regards to specific tooth groups. The tooth groups included mandibular molars, maxillary premolars, and maxillary molars. Statistical analysis using chi-square evaluated the tooth groups for statistically significant findings that may indicate associations or predictive trends for intrapulpal cracks. These trends may help clinicians when evaluating teeth for intrapulpal cracks and understanding future prognoses.

With regard to the overall classification of intrapulpal cracks, there were no statistically significant findings to indicate any definitive association for specific tooth groups. However, the results suggested several trends for the directional nature of intrapulpal cracks with respect to their pulpal wall and floor locations. Assessing only the pulpal wall component of these cracks, the majority of intrapulpal cracks were classified as Type I, or only one wall involvement, within each tooth group. This is in agreement with the descriptive characteristics that found 75% of intrapulpal cracks were involving only one wall of the pulp chamber. In contrast, the pulpal floor location was split between mandibular and maxillary tooth groups. In mandibular molars, the predominant classification was Type B, or extending into an orifice without involvement of the pulpal floor. In maxillary premolars and molars, the predominant classification was Type A, or terminating at the floor-wall junction without involving an orifice. These findings, although not statistically significant, would suggest that upon access of a tooth with a suspected intrapulpal crack, a clinician would more often find a one wall involved crack that either terminates at the floor-wall junction or extends into an orifice, depending on the tooth type that is being accessed.
While preliminary, these findings begin to elucidate the nature of intrapulpal cracks with respect to their locations and provide areas of focus for further study to substantiate these trends.

Analysis of the direction of intrapulpal cracks also demonstrated no statistically significant findings. However, further evaluation of the data suggested a trend in crack direction that was non-specific to any one tooth group. Overall, 92% of all intrapulpal cracks were mesio-distal in direction (mandibular molars 92%, maxillary premolars 83%, maxillary molars 90%). This predominance of directionality suggests a predictive trend for the mesio-distal direction of intrapulpal cracks. This directional nature is in agreement with previous studies evaluating the mesio-distal nature of coronal cracks (2, 4, 5, 9, 12, 42). Bucco-lingual cracks were noted in all tooth groups, accounting for only 8% of the overall cases. This bucco-lingual direction has been discussed in previous studies on coronal cracks, with mandibular molars being the most common tooth group to contain a crack in this direction (9, 12). Whether the restorative status of the tooth has an influence on the direction of the crack is questionable. Cracks are typically oriented in a mesio-distal direction due to tooth anatomy, structural defects in the pits, fissures, and grooves of molars due to failure of the calcification of dentin, as well as the direction of occlusal forces (1, 7, 9, 42). Assuming that the longitudinal nature of coronal cracks would account for progression of cracks to involve the pulpal structures, the overall majority of intrapulpal cracks possessing the same directional nature as coronal cracks would be expected and was confirmed by this analysis. Future studies with a larger group of cases may substantiate this finding statistically.

When evaluating the specific marginal ridge involvement of the intrapulpal crack, several associations and trends were discovered within the individual tooth groups. With respect to the distal marginal ridge, analysis revealed that mandibular molars were statistically more likely to have distal marginal ridge involvement of the intrapulpal crack than maxillary premolars and
molars ($P < 0.05$). With respect to the mesial marginal ridge, analysis suggested that maxillary molars and premolars were more likely to have mesial marginal ridge involvement of an intrapulpal crack than mandibular molars. However, within the limits of this study, this mesial marginal ridge involvement was not statistically significant. Combined mesial marginal ridge and distal marginal ridge involvement was less frequently encountered and was seen almost equally in all tooth groups (See Figure 2). These results suggest that some mechanism inherent to mandibular molars would promote the involvement of the distal marginal ridge in longitudinal crack initiation and propagation communicating with the pulp chamber. Whether this is related to occlusal forces, defects in the anatomical structure of the marginal ridge or underlying dentin, or lingual inclination of the crown is uncertain. The same should be evaluated in maxillary premolars and molars as to whether the mesial marginal ridge is more susceptible to crack initiation and propagation. Future studies with greater numbers of cases may provide reinforcement to these findings statistically.

This study also identified significant associations between specific tooth groups and the pulpal wall involved with propagation of the intrapulpal crack. Statistical analysis revealed the following significant associations: 1) Distal wall involvement was statistically more likely associated with mandibular molars than the other tooth groups and 2) Mesial wall involvement was statistically more likely associated with maxillary molars and premolars than mandibular molars ($P < 0.05$). The mosaic plot (see Figure 3) for pulpal wall involvement clearly depicts the differences in wall involvement based upon the tooth type. These associations lend support to the trends discovered in analyzing the marginal ridge results and suggest that when evaluating teeth for intrapulpal cracks, it will be more common to find the distal marginal ridge/wall involved in mandibular molars and the mesial marginal ridge/wall involved in maxillary posterior teeth.
Again, the etiology of these trends is unknown and could be contributed to various causes including tooth anatomy or occlusal forces.

These trends for location of intrapulpal cracks are helpful for the clinician in diagnosis and clinical evaluation of intrapulpal cracks. The question then becomes what, if any, effect does the location of the intrapulpal crack have on the prognosis of the cracked tooth? In Tan et al. (39), the authors evaluated the pulpal wall involved with cracks propagating into the pulp chamber and whether this was a predisposing factor for survival of the treated tooth at two years. While they did not evaluate this wall involvement for specific tooth groups, they did determine that the location of the crack did not significantly affect the survival of the cracked tooth. There have been no other studies that have evaluated the location of the crack within the pulp chamber and its effect on the survival and/or prognosis. At this point, the answer to this question remains unknown and only with future outcomes research can this be investigated.

Evaluation of the specific tooth groups and the pulpal orifice involvement of the intrapulpal crack also revealed significant associations and predictive trends. Overall, all tooth groups trended towards having no pulpal orifice involvement for intrapulpal cracks, with 40% of mandibular molars, 67% of maxillary molars, and 83% of maxillary premolars having no orifice involved. When orifice involvement was present, the results suggested that the distal orifice was most commonly involved in mandibular molars (36%). This was a significant finding per chi-squared analysis ($P < 0.05$). In evaluating maxillary molars and premolars, no trend for a specific orifice involvement could be substantiated. These findings suggest that mandibular molars with a single distal canal will commonly have a distal marginal ridge, distal wall, and distal orifice oriented crack. In the opinion of this author, this is often the case when accessing mandibular molars with suspected cracks. The wide distal canal orifice, when only one distal canal is
present, predisposes the tooth to crack propagation through this orifice. Maxillary premolars and
molars, with several different orifice orientations and no centrally located M or D orifices,
tended to have less involvement of specific orifices with intrapulpal crack propagation. While the
initial thought was that a MB or MB2 orifice may be more likely involved with a mesio-distal
crack in maxillary molars, this was not demonstrated in our results. Whether future studies with a
larger group of maxillary molars may elucidate this thought is unknown. Furthermore, how this
affects the prognosis is uncertain at this time. In their survival analysis study on cracks involving
the pulp chamber, Tan et al. (39) found that the radicular extension of a crack into an orifice did
not affect the survival of the tooth at 2-year recall. However, the authors did not evaluate the
specific orifice involved with the intrapulpal crack. No study has accounted for the specific
orifice involved with cracks propagating into the pulp chamber or how this affects the prognosis.

In summary, these analyses of tooth type and intrapulpal crack location revealed several
significant associations and predictive trends. The significant associations identified included:
Mandibular molars: distal marginal ridge, distal pulpal wall, and distal orifice involvement,
Maxillary molars: mesial marginal ridge involvement, and Maxillary premolars: mesial marginal
ridge involvement. The predictive trends suggested by the results included the mesio-distal
direction of intrapulpal cracks in all tooth groups, one pulpal wall (Type I) involvement in all
tooth groups, mandibular molars associated with a Type B pulpal floor classification (extending
into an orifice, not involving the floor) while maxillary molars and maxillary premolars
associated with a Type A pulpal floor classification (terminating at the floor-wall junction),
mesial wall involvement in maxillary molars and premolars, and no specific orifice involvement
in maxillary molars and premolars. These associations and trends will help to identify intrapulpal
cracks clinically. Cracks are often very difficult to identify, even with current microscopic
technology and diagnostic tools. Predictors can help to locate which areas are most likely to have intrapulpal cracks and provide more reliability in identifying these cracks. Understanding the size of the study was limited with only fifty-two cases, these trends can be evaluated in future studies to confirm their predictive value.

Another purpose of this study was to evaluate associations between clinical examination findings and the location of the intrapulpal crack. These associations would help in identifying clinical predictors for the location or severity of the intrapulpal crack during examination. Specific testing results that indicate the presence of an intrapulpal crack or the location of the crack would aid the clinician in determining the intrapulpal crack as the etiology of pulpal and periradicular disease as well as determining treatment recommendations based upon the location of the crack. The clinical examination data that was evaluated in this study included the subjective patient symptom of pain on biting, clinical results for the bite test with Tooth Slooth®, transillumination with fiber optic light, probing depths, staining with methylene blue, the radiographic presence of a periapical radiolucency, as well as the pulpal and periapical diagnoses. The intrapulpal crack location was evaluated based upon the individual pulpal wall and floor classifications as well as the overall classification of the crack.

The subjective symptom of pain on biting has been suggested as a clinical predictor of the cracked tooth. As previously discussed, many authors have concluded that a patient’s experience of pain with occlusal forces is the primary symptom to indicate the presence of a crack within the tooth (1, 2, 5, 9, 10). Our finding that 68% of patients with intrapulpal cracks experienced pain on chewing is supportive for this symptom being an indicator of the presence of a crack. The association of this subjective symptom and the location of the intrapulpal crack within the pulpal and periradicular structures was therefore evaluated in attempt to discover any
significant association. Following statistical analysis, there was no correlation discovered between the symptom of pain on biting and the location of the intrapulpal crack. The specific location of the intrapulpal crack with respect to the pulpal wall, pulpal floor, and overall classifications did not correlate with the presence of the pain on chewing.

Overall, regardless of the specific classification, there was a predilection for the presence of pain on chewing. This could suggest that most teeth with an intrapulpal crack should have pain to chewing due to the extension of the crack to include the pulpal and possibly periradicular structures. However, without comparison studies, the predictive nature of this test is circumstantial. This pain on chewing is typically produced by movement of fluid within the crack and dentinal tubules associated with the crack as the occlusal forces flex the crack open and close (24). The fluid movement stimulates the A-delta and A-beta mechanoreceptors to activate. As intrapulpal cracks are associated with the pulpal structures, it would be an assumption that the activation of these pulpal nociceptors would be more prevalent. Of further note, it would be interesting to evaluate whether the pulpal status of the tooth has an effect on the symptoms of pain on chewing. However, no correlation was completed in this study.

Bite testing has also been evaluated and recommended as a primary diagnostic tool for identification of cracked teeth (1, 3, 5, 7, 9, 26). Our study found that 68% of those teeth with intrapulpal cracks responded positively to the bite test with the Tooth Slooth®. The correlation between a positive bite test and the location of the intrapulpal crack was evaluated to determine the predictive value for the location of the crack. Statistical analysis found significant associations between a positive bite test result and the location of the intrapulpal crack with respect to the individual pulpal wall and floor classifications. The analysis suggests that cracks involving two walls (Type II) are statistically more likely to produce a positive bite result than
cracks involving only one wall (Type I). The analysis also suggests that cracks extending into a pulpal orifice (Type B) or across the floor (Type C or D) are statistically more likely to produce a positive bite result than cracks involving only the pulpal wall and terminating at or above the floor-wall junction (Type A). These results are important clinically for several reasons. First, within the limits of this study, the results of the bite test may provide a prediction of the location of the intrapulpal crack. This would allow the clinician to more easily locate the crack upon access as well as predict an extent of the crack within the tooth. Secondly, the bite results may allow for prediction of the prognosis of the treatment of the cracked tooth. If the bite results are positive, the prognosis may be less favorable in the long term. This suggestion can only be confirmed with long-term outcome studies on the treatment of these teeth. Regardless, the bite test as a predictor for the location of the intrapulpal crack is an important finding clinically.

Transillumination is a proven method to aid in diagnosis of a cracked tooth (8). In our study, transillumination was found to be predictive of an intrapulpal crack 79% of the time. Unfortunately, transillumination has limitations due to the presence of intra-coronal restorations that replace the interproximal surfaces of teeth as well as full coverage restorations. The question of whether transillumination can aid in the prediction of the location of the intrapulpal crack was analyzed. The analysis suggested that transillumination did not help predict the overall location of the intrapulpal crack or the location with respect to the pulpal floor. However, with respect to the pulpal walls, transillumination was positive statistically more often in teeth with intrapulpal cracks involving two walls (Type II) than those involving only one wall (Type I). This suggests that teeth testing positive to transillumination are more likely to contain cracks down more than one wall, which may aid in assessing the full extent of the crack clinically upon access as well as being predictive for prognosis of treatment in the future. Again, the less favorable prognosis for
intrapulpal cracks involving two walls instead of one can only be assumed at this time. Only with outcome studies can this assumption be confirmed.

It should be noted that the surface or surfaces that tested positive to transillumination were not recorded. Transillumination was only recorded as a positive or negative result by the treating clinician. A correlation between the surfaces recording a positive transillumination result and the presence of a one or two wall crack would be helpful. Even without this information, it is still noteworthy that transillumination may be predictive of the location of the intrapulpal crack with respect to the pulpal walls.

Other clinical examination results analyzed included probing depths, staining enhancement of diagnosis, the radiographic presence of a periapical radiolucency, and the pulpal and periapical diagnoses. Although these findings are part of the clinical examination process and may help in determining the presence of a cracked tooth, there were no statistically significant associations between the presence of deep probing depths (≥ 5 mm), positive staining results, presence of a periapical radiolucency, or pulpal and periapical diagnoses and the location of the intrapulpal crack. No correlations could be determined that would indicate that positive or negative results to these examination findings would help the clinician predict the location of the intrapulpal crack with respect to the pulpal walls or floor of the tooth. In addition, there were no predictive trends that could be elucidated from these analyses.

Overlooking statistical significance and trends, there were several interesting results within these examination findings that were noteworthy. In evaluating the ability of staining to enhance the diagnosis based upon the classification of the intrapulpal crack, the majority of cases indicated a positive enhancement regardless of the classification. While this suggests that there is no predictive capability for staining to differentiate one classification of intrapulpal crack from
another, it may lend to the power of staining to enhance overall identification and diagnosis. However, it must be taken into consideration that the ability of staining to enhance the diagnosis was a subjective assessment recorded by the clinician. This may lead to a certain error based upon a bias by the clinician performing the evaluation.

Additionally, in evaluating the pulpal and periapical diagnoses, intrapulpal cracked teeth regardless of the classification were more likely to be diagnosed with pulpal necrosis and symptomatic apical periodontitis. Whether intrapulpal cracks associated with necrotic teeth have propagated farther than those with vital pulps or have a worse prognosis is unknown. While Berman and Kuttler (18) have stated that such teeth with “fracture necrosis” have an unfavorable prognosis and should be extracted, no clinical study evaluating treatment of these teeth has been attempted. Arguably, this would present its own set of complications and challenges as noted by those authors, but only with long-term outcome studies will the true prognosis for treatment of these teeth be understood. Regarding the pathophysiology of these cracks, the presence of these cracks within the pulpal structures is ultimately the etiology for the bacterial insult, inflammatory response and subsequent irreversible changes that lead to pulpal necrosis. Unfortunately, the pathophysiology of these events with respect to the progression of the intrapulpal crack has not been studied at this time.

In summary, these analyses of clinical examination results and intrapulpal crack location revealed several significant associations and predictive trends. The significant associations identified included: Bite test positive results for two wall cracks (Type II) and cracks extending into an orifice (Type B) or partially (Type C) or entirely across the floor (Type D) as well as Transillumination positive results for two wall cracks (Type II). These significant associations may help the clinician in predicting the location of the intrapulpal crack. As mentioned
previously, cracks are very difficult to identify, even with current microscopic technology and diagnostic tools. These significant associations can help to identify with bite testing and transillumination which areas are more likely to have intrapulpal cracks and provide more reliability in identifying these cracks. Understanding the study was small in size with only fifty-two cases, associations and trends should be re-evaluated in future studies to better assess their possible significance in association with specific classifications.

While much of this study focused on understanding the characteristics of intrapulpal cracks and their correlation with certain diagnostic and clinical examination findings, another purpose of this study was to evaluate the proposed classification system for intrapulpal cracks. Recognizing the void in endodontic literature related to the study of cracks involving the internal pulpal and radicular structures, this study was an attempt to begin to evaluate intrapulpal cracks on a more scientific level. Before any evaluation of these cracks could be completed, a classification system was needed in order to organize them objectively into categories based upon specific locations with respect to the pulp chamber structures. The pulp chamber structures that may be involved with any intrapulpal crack would be the pulpal walls, pulpal floor, and the pulpal orifice system. The classification system needs to be specific enough to describe the intrapulpal crack location in detail but also encompass each individual crack location that may be encountered within the pulpal structures. The classification system also must be applicable to a clinical practice and have significant ease of use. A classification system that is too specific and contains too many parameters and categories would not be easy for a treating clinician to implement in their practice. Therefore, a balance must be struck in creating the classification system for these cracks.

As no universal classification system currently exists for intrapulpal cracks, the proposed
classification system was based upon analysis of previously documented intrapulpal cracks as well as previously utilized systems for analysis of longitudinal cracks involving the pulp chamber (39, 43). Documentation of all intrapulpal cracks within the graduate practice at VCU provided a database of intrapulpal cracks to evaluate. Digital images taken under the microscope provided visual information related to the patterns seen with cracks involving pulpal walls and floor. Additionally, Rivera (43) and Tan et al. (43) have both previously proposed systems for classifying longitudinal cracks that may involve the pulp chamber. In the Bonus material of the AAE’s Colleagues of Excellence “Cracking the Cracked Tooth Code: Detection and Treatment of Various Longitudinal Tooth Fractures”, Rivera (43) proposed a classification system for various patterns of longitudinal cracks that may involve the coronal or radicular structures. Although this system was not specific for those cracks seen within the pulp chamber, it was the first attempt to describe the longitudinal characteristics of these cracks and their propagation to include the pulpal and radicular structures. Rivera’s classification system was as follows:

1. One marginal ridge limited to crown
2. Two marginal ridges limited to crown
3. Marginal ridge(s) and internal proximal cavity wall only
4. Marginal ridge(s) and floor of cavity preparation (may involve restoration removal)
5. One marginal ridge extending from crown to root surface (difficult to visualize)
6. Two marginal ridges extending from crown to root surface (difficult to visualize)
7. Marginal ridge(s) and into canal orifice(s)
8. Marginal ridge(s) and pulpal floor
9. Furcation involvement (only confirmed after exploratory surgery or extraction)
This classification system is an overall view of cracks from superficial to deep. While it addresses certain internal aspects of the pulpal system, i.e.: the canal orifice and pulpal floor, it does not discuss the specific pulpal wall involvement or the extent of pulpal floor involvement. Additionally, this classification system may be difficult to implement clinically due to the amount of categories and variables that may be difficult to visualize.

Rivera (43) hypothesized that the prognosis for the cracked tooth would decrease from questionable to poor as the crack involves more apical structures such as the root surface, canal orifice, pulpal floor, and furcation. Although the prognosis seems to be multi-factorial, involving not just the internal pulpal and radicular structures but also the external periodontal structures, and questionable at best, there is no current clinical evidence to support any assumption related to long-term prognosis for treatment of cracked teeth involving pulpal, periodontal, and radicular structures.

Tan et al. (39) utilized another system to describe cracks propagating within the internal structures of the tooth. In their clinical study, fifty teeth requiring root canal treatment due to cracks were followed for a recall period of two years. They classified the cracks based upon their extent within the internal structures of the tooth. Cracks were classified as follows:

1. Coronal: Crack confined to the walls of the pulp chamber
2. Radicular: Crack extending beyond the orifices of the root canals

While this system simplifies the categorization of internal cracks with a two-category approach, there are several advantages and disadvantages with this type of simplification. The coronal and radicular descriptions allow the clinician to easily categorize the cracks seen within the pulp chamber upon access. This ease of clinical application for the practitioner may translate
into a greater implementation and usage amongst clinicians. However, the disadvantages also lie within this simplification of the classification system. Coronal refers to intrapulpal cracks that are confined to the walls of the pulp chamber, but does not provide additional information relative to the number of walls involved or the location and extent of pulpal floor involvement. The authors appeared to count every involved wall as a separate crack, not establishing any continuity between cracks involving opposite walls, i.e.: mesial and distal or buccal and lingual. Radicular refers to intrapulpal cracks extending beyond the orifices but does not suggest that cracks across the floor would also be categorized as radicular.

Without more specific guidelines, these categories may be too generalized to provide enough information to the treating clinician. A classification system that includes the specifics of the pulpal wall and floor/orifice involvement may provide the practitioner with more information for understanding the location of the intrapulpal crack. Understanding that cracks are oriented in a mesio-distal or bucco-lingual direction and may include one or both walls is also important when evaluating the location. In addition, when evaluating the long-term prognosis for treatment of these cracks, it may be advantageous to specify the location and extent of the intrapulpal crack within the pulpal and radicular structures when assessing whether treatment may be favorable or non-favorable long term. A crack that involves only one wall and terminates at the floor-wall junction of the pulp chamber may have a more favorable prognosis than a crack involving two walls that extends the entire width of the pulpal floor. While treatment and prognosis will only be established in future studies, these are considerations that must be made when creating a classification system.

With these previous classification systems in mind, the proposed classification system was created. The classification system was implemented in this study to evaluate its clinical
application, ease of use, and correlate relationships with other variables related to intrapulpal cracks. The classification system was tested for its ability to comprehensively describe the internal extent of these intrapulpal cracks from pulpal wall to pulpal floor and orifice. The classification system was applied to the fifty-two cases of intrapulpal cracks evaluated in this study. The advantages of the proposed classification system were its ability to separate the pulpal wall and pulpal floor locations and the specificity of the extent of pulpal floor or orifice involvement. Any crack extending longitudinally in a coronal to apical direction can involve one or two walls. As the crack approaches the pulpal floor, the crack may propagate across the floor, either partially or completely, or enter an orifice and continue in a radicular direction. The furcation may or may not be involved as well at this point. Without surgical exposure of the root structure, the crack cannot be visualized externally. As the crack is difficult to predict and the full extent is often not visible, the most information that can be interpreted is helpful to the clinician in making an informed decision on prognosis and treatment. Therefore, the internal aspects of the crack that are visualized must be evaluated for diagnostic purposes.

Assessment of the clinical application of the proposed classification system was verified through subjective questioning on the intrapulpal cracked tooth template (see Appendix A). The ability of the clinician to provide a pulpal wall and floor classification for the specific intrapulpal crack was a measure of the classification’s applicability and ease of use. Overall, 49 out of 52 (94%) intrapulpal cracks could be classified with the proposed classification system. The system proved applicable within this study population. As the classification system involved only two categories to evaluate, the clinician was able to assess and utilize the specific classifications within these two categories.

While there were several advantages to the proposed classification system, there were
also several limitations identified during the evaluation process. The first limitation identified was the inability of the classification system to comprehensively describe the pulpal floor extent of every intrapulpal crack. There were three cases of intrapulpal cracks in which the crack involved two pulpal walls and two separate pulpal floor classifications. In each case, the crack traversed across the floor from one wall and directly into an orifice from the other wall. In two of the cases, the crack traversed only partially across the floor. In the third case, the crack traversed across the entire floor and extended into the same orifice involved with the crack on the opposite wall. The proposed classification system for the pulp chamber floor only included four classifications:

A. Terminates at floor-wall junction, not involving orifice(s)
B. Extends into orifice, not involving floor
C. Traverses partially across floor, not involving orifice(s)
D. Traverses across entire floor, not involving orifice(s)

Therefore, in the former two cases, there would be two separate pulpal floor classifications, Type B and C. However in the latter case, only one of the pulpal floor classifications could be classified with the proposed system. The classification system does not account for a crack extending across the entire pulpal floor and extending into an orifice. These cases highlight two separate problems with the system: 1) how to classify the pulp chamber floor when there are two separate floor classifications for the same crack involving two walls, and 2) how to classify the pulp chamber floor when the proposed system does not describe the full extent of the crack.

Empirically, previous studies have stated that cracks involving the pulpal floor have a worse prognosis (2, 22). However, without long-term outcome studies, we cannot definitively state that
one pulpal floor classification is more severe or has a worse prognosis than the next. Therefore, it would be an assumption to classify the pulp chamber floor as a Type C when both Type B and C are present in the same tooth only because the crack traversing partially across the floor is assumed to be more severe than the crack extending into an orifice. Additionally, in the case with a crack that cannot be classified to its full pulpal floor extent, assigning the classification of the overall pulpal floor as Type B without including the part of the crack running across the floor and into the orifice would be erroneous. In order for the classification system to be applicable in clinical practice, the system must comprehensively describe any intrapulpal crack.

While none of the cases evaluated in this study contained multiple cracks in the same tooth, this is a possible scenario that has not been addressed. The classification system could be applied to both cracks. For example, the mesio-distal crack would have a separate pulpal wall and pulp chamber floor classification than the bucco-lingual crack. Prognosis would be based upon the existence of both cracks. Tan et al. (39) found that multiple cracks in the same tooth decreased the survival of the tooth at 2 years. These authors also classified cracks involving two walls as two separate cracks. This methodology was different than that used in the current study for assessing the continuum of cracks involving two walls. The effect of multiple cracks on treatment and prognosis would need to be evaluated in long-term outcome studies.

Another limitation of the classification system was the number of possible combinations of pulpal wall and floor classifications. Within the two-category system, there were six separate classifications and eight possible combinations for comprehensive classification of the intrapulpal crack. The eight possible combinations may prove to be too cumbersome for clinical practice. Also, the use of Roman and Arabic numerals may be confusing for the practitioner. Future studies evaluating a larger population of intrapulpal cracks may allow for a re-evaluation
of the classification system and possible simplification or renumbering of the classifications.

A final limitation of the proposed classification system was the lack of a periodontal component within the classification system. As any crack involving the internal structures of the tooth may also involve the external surfaces and periodontal structures, a comprehensive classification system should reflect the full extent of the crack. There was no classification included for furcation involvement or reflection of any probing defect associated with the crack. While the focus of this classification system was to describe the internal intrapulpal aspects of the crack that are visualized during access of the tooth, a periodontal component could eventually be evaluated and included. The difficulty would be the ability to visualize and properly diagnose periodontal components of the longitudinal crack. Often times the crack is in a mesio-distal direction and the probing defect cannot be detected. As noted by Rivera and Walton (31) and Berman and Kuttler (18), the full extent of the crack with regards to the radicular and periodontal structures is not visualized until surgical exploration or extraction of the tooth. Also, it has been recognized that the crack may be not be detectable to its full extent, even with microscopic technology (4, 18, 31, 32). Therefore, the ability to classify the intrapulpal crack is limited to what can be visualized. The inclusion of a periodontal component to the proposed classification system may be difficult at this point with current technology.

With these limitations to the classification system in mind, modifications to the classification system should be proposed while maintaining the applicability of the system in clinical practice. One possible solution to the complication of two pulpal floor classifications would be to include both floor classifications in the overall classification instead of excluding one or the other. For example, instead of Type A or B, the classification would be addressed Type A/B. This simple resolution would allow for comprehensive description of the internal
aspects of the intrapulpal crack while maintaining its ease of use. In reference to the crack with only partial classification of the pulp chamber floor, a fifth classification for the pulpal floor could be proposed: “Type E: Traverses across entire floor, extending into an orifice”. As only fifty-two cases were evaluated in this one-year study, future analysis with more cases could highlight additional types of crack involvement within the pulp chamber floor. Re-analysis of the classification system would need to be done to ensure the comprehensive nature of the system.

Another possible solution to the floor classification dilemma would be to change the entire methodology for classifying intrapulpal cracks. Instead of evaluating and classifying the intrapulpal crack as a whole unit, the classification could be broken down into its individual parts. While conceptually any crack may involve either one or two walls due to their mesio-distal or bucco-lingual direction, the classification of a crack having two separate and distinct floor classifications may prove easier by separating them into individual components. To this effect, a Type II intrapulpal crack with two distinct floor classifications, Type A and Type B, would be re-classified as Type I-A and Type I-B. Tan et al. (39) classified cracks in a similar manner by evaluating each wall involved as a separate distinct crack. They did not discuss the possibility of cracks involving two walls. With this new methodology, the pulpal wall classification system would no longer be necessary, as each wall would be classified separately. While these changes would resolve the multiple floor classifications dilemma, they would also eliminate the ability to view intrapulpal cracks as complex longitudinal entities involving multiple internal pulpal structures. In addition, the ability to compare cracks involving one wall versus cracks involving two walls would be eliminated. This proposed methodology is theoretical only and future research would be needed to evaluate how any changes to the classification system would affect our ability to study intrapulpal cracks as well as its applicability in clinical practice.
The possible periodontal component of the classification system is more complicated. Although the proposed classification system evaluates only the internal aspects of the intrapulpal crack, there often may be an external component that affects treatment and prognosis. It would be beneficial to have an all-encompassing classification system for diagnostic and prognosis purposes. As Rivera (43) had proposed a furcation classification, this could be included as another classification for the pulp chamber floor. However, the clinical ease of use must be considered when discussing additional classifications. Including more classifications may prove too confusing for the clinician. In addition, the difficulty in assessing the periodontal components of these cracks must also be evaluated. Future outcome studies for intrapulpal cracks would provide prognosis data for specific classifications of cracks and could address the need for inclusion of furcation involvement as a classification. The furcation involvement of an intrapulpal crack would be assumed to decrease the long-term prognosis for the tooth.

The other periodontal complication not addressed would be periodontal defects detected that are not involving the furcation. To include this variable in the classification system, it would be prudent to understand the relationship between the presence of a periodontal defect and the prognosis. While further research is needed to understand this relationship, this study did evaluate the prevalence of probing defects ≥ 5 mm as well as their relationship with the intrapulpal classifications. Overall, there was a 37% prevalence of probing depths between 5-10 mm. In addition, the presence of a probing depth of this magnitude was not significantly associated with any one classification of intrapulpal crack. While these findings are inconclusive in their significance, they do highlight the common presence of periodontal defects. Highlighting the effect of these periodontal defects, in Tan et al. (39), the authors found that pre-treatment periodontal defects decreased the survival of the cracked tooth at two years follow up. A possible
resolution would be to include a separate periodontal classification for the external component of intrapulpal cracks. Future studies should investigate this periodontal component associated with intrapulpal cracks and evaluate any modifications to the proposed classification system.

Understanding the limitations of the current study is necessary in addressing the future directions of research into intrapulpal cracks. While the aforementioned limitations of the classification system are important to discuss, focus must also be placed on the limitations of the other areas of this study. With this in mind, several areas were discovered following evaluation of the data and analysis. First, there were limitations in documentation and collection of the data set. The ability to document intrapulpal cracks, and cracks in general, is difficult. The technology has improved tremendously with microscopes and illumination, however cracks are often elusive and difficult to assess their apical extent. The microscopic finding that the magnification required to classify the intrapulpal crack was 1.7x greater than the magnification to diagnose the crack is a great example within this study of the difficulty in assessing the apical extent of cracks. This difficulty may result in intrapulpal cracks being missed, mistaken for dead tracts, bur marks, or decalcified areas within the dentin, and eliminated during pulp chamber access and shaping. In addition, the various illumination technologies may play a pivotal role in being able to adequately view the extent of the intrapulpal crack. Improper diagnosis and classification of the crack may occur due to these limitations. Only with future studies assessing these limitations will we better understand their true effect on detection and classification of intrapulpal cracks.

Secondly, the retrospective nature of this study introduces bias and subjectivity into the results. While all residents were trained on documentation of intrapulpal cracks utilizing the intrapulpal cracked tooth template (see Appendix A), there is an innate subject bias when the documentation is not prospective and standardized with only two or three examiners. While this
is a limitation, this could not be eliminated within a large private practice model. The second area of bias is in the documentation by the evaluating clinician. The clinician may inadvertently or purposefully circle the wrong number, classification, or test result. While attempts were made to standardize this data with a template, collected information may not be accurate in all cases. Attempts were made to minimize error by utilizing both the template and the electronic record as sources of data collection and cross checking whenever possible. Unfortunately not all records within the electronic system were complete and many lacked complete results for diagnostic testing. The nature of a retrospective study cannot eliminate these sources of possible error and bias. Only in future prospective studies designed to eliminate subjectivity and standardize the examination and classification process can this be addressed.

Finally, the size of the study was a limitation. As this study only evaluated intrapulpal cracked teeth within the graduate endodontic practice composed of eight endodontic residents over a one-year period, the final n-value of 52 teeth is relatively small. Compounding this limitation was the separation of the cases into the eight possible classification groups. Once separated by classification, many classification categories contained less than 5 to 10 cases. These limitations made assessing statistical significance more difficult. Future studies including a much larger study size would increase the power of the study and allow for better assessment of statistically significant associations and predictors. Inclusion of private practice consortium groups and collaborative efforts by multiple university endodontic residencies in the data collection process would allow for a much more robust and diversified patient population in addition to a much larger sample size. Future emphasis should be placed on establishing these relationships.

With these limitations in mind, there is a vast amount of possible research in the future
with respect to understanding, diagnosing, classifying, and treating intrapulpal cracks. As previously discussed, continued data collection and involvement of consortiums and other university graduate endodontic programs is vital to increasing the study size and diversity. Re-evaluation of the classification system with the inclusion of two floor classifications or the additional pulpal floor classification (Type E), re-evaluation of the classification methodology with respect to two wall intrapulpal cracks, and investigation into the possible inclusion of a periodontal component would all help in refining the classification for use in clinical practice. Comparison studies evaluating illumination and imaging modalities to identify, diagnose, classify, and digitally capture these cracks would be beneficial to understanding our current limitations. Looking further into the future, clinical outcome studies for treatment of the various intrapulpal cracks to evaluate prognosis will be necessary to support treatment or extraction of these teeth. While these outcome studies are invaluable for scientific evidence to support treatment, they must be approached cautiously and methodically as the possible risks and complications to the patient in treating these teeth is a major concern (18).

In conclusion, the newly defined terminology, Intrapulpal Crack, proved useful for referring to all cracks located internally within the pulpal walls, floor, and orifices. The characteristics and predictive associations were beneficial for understanding the nature of intrapulpal cracks and beginning to identify certain predictors based upon tooth type and clinical examination finding. Finally, the internal aspects of cracked teeth could be classified utilizing a novel classification system. For summary purposes, included is a list of the important clinical findings for the descriptive characteristics, significant associations, predictive trends, and novel classification system identified during this study.
Important Clinical Findings

I. Descriptive Characteristics:

1. Prevalence:
   a. Overall (intrapulpal cracks): 5.9%
   b. Tooth Type: Mandibular 2\textsuperscript{nd} molar > Maxillary 1\textsuperscript{st} molar > Mandibular 1\textsuperscript{st} molar > Maxillary 2\textsuperscript{nd} molar > Maxillary 2\textsuperscript{nd} premolar > Maxillary 1\textsuperscript{st} premolar
   c. Existing Restoration: Amalgam > Composite > Crown > No restoration
   d. Surfaces Restored: None to minimally restored (1-2 surfaces) > Heavily restored (2+ surfaces or crown)

2. Magnification: Average magnification required to classify an intrapulpal crack is 1.7x average magnification to visualize an intrapulpal crack

3. Intrapulpal Crack Classifications:
   a. Pulpal Wall Classifications:
      i. Type I (one wall): 75%, Type II (two walls): 25%
   b. Pulpal Floor Classifications:
      i. Type A (terminating at the floor-wall junction): 44%, Type B (extending into an orifice): 40%

II. Significant Associations and Diagnostic Predictors (* = statistically significant):

1. Direction of Intrapulpal Crack: Mesio-distal > Bucco-lingual

2. Floor Involvement:
   a. Mandibular Molars: Type B (extending into an orifice)
   b. Maxillary Molars and Premolars: Type A (terminating at the floor-wall junction)
3. Individual Tooth Type Predictors:
   a. Mandibular Molars: DMR*, D wall*, and D orifice*
   b. Maxillary Molars: MMR, M wall*, and No orifice involvement
   c. Maxillary Premolars: MMR, M wall*, and No orifice involvement

4. Bite Test*:
   a. Wall Involvement: Type II (two walls) intrapulpal cracks are more likely to test positive
   b. Floor Involvement: Type B (extending into an orifice), Type C (traversing partially across the floor) or Type D (traversing across the entire floor) intrapulpal cracks are more likely to test positive

5. Transillumination*:
   a. Wall Involvement: Type II (two walls) intrapulpal cracks are more likely to test positive

III. Classification System:

1. Applicability: Classification system was applicable in 49/52 cases (94%)
References


Appendix A

Intrapulpal Cracked Tooth Template

VCU Graduate Endodontics - Intrapulpal Crack Documentation Template

AxiUm #: ____________________  Resident: ____________________
Tooth #: ___________  Date: ____________________

Microscope - Crack evaluation: (completed by assistant while evaluating under microscope):

Before Staining:
Crack Visible:  Y/N  Y/N  Y/N  Y/N
Magnification:  0.6  1.0  1.6  2.5

After Staining:
Crack Visible:  Y/N  Y/N  Y/N  Y/N
Magnification:  0.6  1.0  1.6  2.5

The following images should be captured:

Before Access: 0.6 (Transillumination)

After Access (per crack): 1 image @ 0.6, 1.0, 1.6, 2.5

After Staining (per crack): 1 image @ 0.6, 1.0, 1.6, 2.5

Orientation of Crack:

Direction:  M – D  B – L  Oblique
Marginal Ridges:  MMR  DMR
Pulpal Walls Involved:  Mesial  Distal  Buccal  Lingual
Pulpal Floor Involved: Yes No
Pulpal Orifices Involved: MB$_1$ MB$_2$ DB P MB ML D B L
Extent of Pulpal Floor:

<table>
<thead>
<tr>
<th>Pulpal Wall(s):</th>
<th>Pulpal Floor:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Terminates at floor-wall junction, not involving orifice(s)</td>
<td></td>
</tr>
<tr>
<td>Extends into orifice(s), not involving floor</td>
<td></td>
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<tr>
<td>Traverses partially across floor</td>
<td></td>
</tr>
<tr>
<td>Traverses across entire floor</td>
<td></td>
</tr>
<tr>
<td>Other: _____________________________________</td>
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Please answer the following questions related to your microscopic evaluation of the intrapulpal crack:

1. At what magnification were you able to 1st visualize the crack? _________
2. At what magnification did you feel you could classify the crack? _________
3. Did staining enhance your ability to visualize the crack in the scope? Y N Equal
4. Did staining enhance your ability to diagnose the crack in the scope? Y N Equal
5. Is what you saw under the scope the same as what you expected to see before you accessed the tooth? _____________________________________
6. Were you able to capture with your digital images everything you were able to see under the microscope? Y N
7. Based upon your microscopic evaluation, what is your classification of the crack?
   - Pulp chamber wall classification: ______
   - Pulp chamber floor classification: ______

Pulp chamber wall classification:
One Wall involved

Two Walls involved

**Pulp chamber floor classification:**

Terminates at floor-wall junction, not involving orifice(s)

Extends into orifice(s), not involving floor

Traverses partially across floor, not involving orifice(s)

Traverses entire floor, not involving orifice(s)
Vita

Dr. Matthew Scott Detar was born on May 11, 1981 in Wilmington, Delaware and is an American citizen. Dr. Detar received his Bachelor of Science in Chemistry from the College of William and Mary in 2003 and Doctor of Dental Surgery from the Virginia Commonwealth University School of Dentistry in 2007. He subsequently received a Certificate in Advanced Education in General Dentistry from the University of Florida College of Dentistry in Seminole, Florida in 2008. Dr. Detar then entered private practice in Dillwyn, VA and completed three years in general dental practice. In 2011, he returned to the Virginia Commonwealth University School of Dentistry for a one-year full-time assistant professorship position in the Department of Endodontics. After completing the professorship, Dr. Detar enrolled in the Advanced Specialty Program in Endodontics at Virginia Commonwealth University School of Dentistry. Dr. Detar is a member of the AAE, ADA, and VDA and will enter private practice in Manassas, VA. He will graduate with a Master of Science in Dentistry and a Certificate in Endodontics.