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Endodontic Radiolucency on a Mature Permanent Tooth in the Pediatric Population: Can the Tooth be Vital?

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Endodontic Radiolucency On A Mature Permanent Tooth In The Pediatric Population: Can The Tooth Be Vital?

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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Abstract

ENDODONTIC RADIOLUCENCY ON A MATURE PERMANENT TOOTH IN THE PEDIATRIC POPULATION: CAN THE TOOTH BE VITAL?

By Erika Sloane Lentini, DMD

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

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Thesis Director: Karan Replogle, DDS, MS
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The objective was to determine the prevalence of vital teeth requiring non-surgical root canal therapy (NSRCT) that present with radiolucencies in mature permanent teeth in a pediatric population. A retrospective electronic dental chart review of children treated with NSRCT at VCU's School of Dentistry between November 30, 2009 and March 1, 2013 was conducted. The presence or absence of a periapical radiolucency was determined from digital radiographs by three calibrated dentists. Specific characteristics of each tooth were collected. Statistical analysis using logistic regression was completed on all teeth with radiolucencies and vital teeth with radiolucencies. NSRCT was completed on 551 teeth. Radiolucencies were present in 246 teeth. Vitality data on access was only available in 184 teeth. In these, the prevalence of vital teeth with radiolucencies was 45.1% (n=83). Significant differences were noted for tooth type, gender, and puberty. Post-pubertal subjects had more vital teeth with radiolucencies than pubertal subjects.

Introduction

A non-vital tooth, or necrotic tooth, is defined as one in which the blood supply is nonexistent and the nerve supply is nonfunctional. Typically these teeth will not respond to electric pulp tests or cold stimulation. Necrotic pulps sustain prolonged bacterial growth that extend beyond the apical foramen. This can result in periradicular changes and subsequent radiographic lesions of pulpal origin.[1] Historically in dentistry, with the understanding of the disease process that occurs in non-vital teeth, it has been taught that any tooth with a suspected radiographic lesion of pulpal origin is likely non-vital. Most dental textbooks and research studies teach to this norm. In a study by Kaffe in 1988, he stated that a radiographic pathosis of a widening or break in the lamina dura is the most consistent radiographic finding when a tooth is non-vital.[2] The 2001 Spring/Summer *ENDODONTICS: Colleagues for Excellence* insert titled *Systematic Pulpal Diagnosis* states, "If a radiolucency is in the periradicular region of a tooth with a vital pulp, it cannot be of pulpal origin and will be either a normal structure or another type of pathosis."[3] Dental textbooks explain the diagnosis of periapical disease, classifying teeth with widened lamina dura or periradicular radiolucencies, as asymptomatic (chronic) periradicular periodontitis, acute periradicular abscess, and chronic periradicular abscess and associate the diagnosis with teeth that will not respond to pulp vitality tests. This indicates that these teeth are non-vital.[1] However, it is also known and supported that necrosis may be partial or complete, involving only a portion of the pulp/root canal system in a multirooted tooth.[1] If this is the case, then a tooth could present with a radiolucency and still be vital.

When studying periapical radiolucencies of pulpal origin and their association with tooth vitality, there are multiple factors to be considered. First is the anatomical features of the normal pulp and the disease processes that lead to pulpal inflammation and necrosis. Second is the pulpal response to dental pulp testing, allowing the dentist to form a diagnosis and prior to initiating treatment and without performing a biopsy. Third is the manner in which radiographically evident bony lesions are formed. Finally, it is important to understand the inherent healing potential of the dental pulp. All of the aforementioned factors have been studied significantly, especially in the adult population, and these studies have allowed dentists to understand the disease process of a carious tooth.

The dental pulp is a unique soft tissue of mesenchymal origin that is comprised of an intricate network of specialized cells, nerve fibers, vascular blood supply, connective tissue, and ground substance that interact intimately with the surrounding dentin and environment.[1] The pulp is divided into many layers, each of which serves a function and is host to a different component of the pulp anatomy. Important layers for this discussion are the cell-rich zone, which is home to many cell types including undifferentiated mesenchymal cells or stem cells, and the pulp proper, which contains the nerve and vascular supply as well as fibroblasts or pulpal cells.[1]

The many cells of the normal, healthy pulp include odontoblasts, pulp fibroblasts, macrophages, dendritc cells and lymphocytes.[1] Mast cells are typically found in chronically inflamed pulps.[3] Odontoblasts are the most characteristic cell of the pulp because they are responsible for the continued formation of the dentin in both the developing tooth and the mature tooth. The most numerous cells are the pulp fibroblasts. They are tissue-specific cells that can give rise to other cells, that when given the proper signal can undergo differentiation, which is

important in wound healing of the pulp. The fibroblasts allow a mature pulp to maintain a resemblance to embryonic connective tissue and to supply a rich source of stem cells.[1] Macrophages perform many functions, one of them being to participate in immune reactions by processing antigens and interacting with specific receptor sites on memory T cells.[5,6] During inflammatory reactions, macrophages can produce interleukin-1, tumor necrosis factor, growth factors, and other cytokines, all of which are important mediators in pulpal infections and the process of developing periapical bony lesions. Other accessory cells related to immune responses are the dendritic cells. They are important in the induction of T cell-dependent immunity. Lymphocytes, specifically T-lymphocytes, are found in the normal pulps.[6] These cells, along with macrophages and dendritic cells, indicate the pulp is prepared for an immune response which is necessary during pulpal inflammation.[4,7]

The nervous supply of the pulp is as complex and diverse as the cells the make up the pulp. The maxillary nerve in the upper jaw and the mandibular nerve in the lower jaw are branches of the fifth cranial nerve, the trigeminal nerve, and supply sensory innervation to all teeth. The result is a highly innervated dental pulp. These nerve fibers enter the pulp through the apical foramen and are distributed along the path of the blood supply.[8,9,10] There are two types of sensory nerve fibers in the pulp, the myelinated A fibers, with 90% being A-delta and 10% being A-beta, and the unmyelinated C fibers. The A fibers are found in the coronal portion of the pulp at the pulp-dentin border, mainly in the pulp horns. The C fibers are located mainly in the core of the pulp.[11,12] The A fibers are responsible for fast, sharp pain that is easily localized, while C fibers generate slow pain, typically labeled as dull and aching.[11] Clinically, it is important to note that since nerve fibers' cell bodies are located in ganglia outside the pulp, these nerve fibers may

be resistant to necrosis.[13] Also of importance is the fact that C fibers can survive in a hypoxic environment[14], possibly explaining the presence of pain in a necrotic tooth.[15]

As stated earlier, both the majority of the vascular supply and the nerve fibers themselves enter the tooth through the apical foramen, with some smaller vessels entering the tooth through lateral or accessory canals. The vascular network supplies the odontoblasts with a rich source of metabolites, which is especially important and present in young teeth where the capillaries commonly reach into the odontoblastic layer.[1] The coronal portion of the pulp has nearly twice the capillary blood flow as the root portion.[16] Even more specifically, the pulp horns have the greatest blood flow when compared to any other region of the pulp.[17] The regulation of pulpal blood flow is controlled by many systems, but it is more important to understand the unique environment of the pulp. It is rigidly encased within dentin, making it a low compliance environment. This means that vasodilation and increased vascular permeability, common during inflammatory reactions, lead to increased hydrostatic pressure.[18]

The connective tissue and ground substance components of the pulp will not be discussed in great detail. A few key characteristics of these parts, however, will be mentioned. The connective tissue of the pulp is comprised of collagen and elastin. Odontoblasts synthesize type I collagen and fibroblasts synthesize type I, III, V, and VII collagen. Type III collagen is a fetal form found in the dental papilla and in the mature pulp. Ground substance, or extracellular matrix (ECM), is a gel like structure that is responsible for the water-holding properties of the connective tissues. In young pulps, the water content is very high, meaning that the ground substance can form a cushion, enabling it to protect the cellular and vascular components of the tooth. The ground substance also allows for cell metabolites, nutrients, and wastes to pass

between cells and blood vessels. It also can alter osmotic pressure by excluding osmotically active molecules.[1]

As mentioned earlier the dental pulp is a unique connective tissue. Being surrounded by a hard, unyielding structure and having limited collateral circulation lead it to be very susceptible to injury, such as injury experienced following the onset of dental caries. These features also complicate its ability to regenerate. The rich neurovascular supply promotes and supports the effect of inflammation, which may, in turn, lead to rapid degeneration and necrosis.[1] The initial response of the pulp following insult by acidic byproducts of the carious process is for the release of bioactive molecules to initiate tertiary dentin formation.[19] The caries causing microorganisms, their metabolic byproducts, toxins, enzymes, and disintegration products directly affect not only the pulp, but also periapical tissues.[20,21] Kakehashi proved such a relationship in a classic study where mechanical pulp exposures in rats led to infection by bacteria in the oral cavity and subsequent periapical lesions.[22] While the tertiary dentin formed can provide a physical barrier against further insult, there is still a pulpal immune response to the invading pathogens.[1] This immune response, triggered by the irritants acting like antigens, is of the humoral and cell-mediated type and can further the progression of the inflammatory periapical disease process.[23,24] T cell immunoregulatory mechanisms may be more involved in periapical lesion pathogenesis, with T helper cells being the predominate cell in an actively expanding lesion. Macrophages, which are activated by T cells and bacterial components, play an important role in bone destruction by magnifying bone resorptive mediators.[25] These microorganisms and their products move from the infected root canals through the apical, lateral, or furcation foramina, through dentinal tubules without an external cementum covering and directly affect the surrounding periodontal tissues and can lead to pathology in these tissues.[26]

Total inflammation or necrosis of the pulp does not have to occur prior to the radiographic observation of an endodontic periapical radiolucency.[23,24,27] It has been shown through biopsies of pulps that necrosis can be isolated only to specific portions of the tooth like the coronal portion or a particular root.[28] A study by Yamasaki in 1994 utilized the Kakehashi model of inducing periapical lesions in rats and suggested that periapical inflammation began prior to pulpal necrosis. A bacterial infection and bacterial mediators leaking past the apex was enough to begin an inflammatory response in the periapical tissues.[29] Langeland et al. and Lin and Langeland have also shown that vital pulp tissue can be present in a tooth long after a periapical pathosis has developed.[24,27]

Initial evaluation of a patient includes the gathering of thorough medical and dental histories, an extraoral exam, and an intraoral exam. With the intraoral exam, the dental clinician is looking for signs of swelling, sinus tracts, or any other abnormalities. The clinician will also perform the initial endodontic tests by palpating the alveolar hard tissue, percussing the dentition, and checking each tooth for mobility.[1] Positive percussion tests indicate an inflammatory process that has moved into the periodontium, which is rich in proprioceptors.[8] Neither percussion, palpation, nor tooth mobility are indicators of pulp vitality, instead, they indicate a compromise in the periodontal attachment apparatus.[1]

Even though a hard encasing of dentin and enamel surrounds the soft tissue pulpal structure, it remains acutely responsive to the outside environment and stimulation.[1] Endodontic diagnostic tools utilize this relationship between the pulp and the outside environment. They aid the dental clinician by providing valuable diagnostic and treatment planning information. Many endodontic pulp tests rely on the distribution, large diameter size, conduction speed and myelin sheath of A-delta fibers.[11,14] While cold testing is the most common pulp testing method for

today's dental clinician, to be reliable it should be used in conjunction with the electric pulp test so that one test can verify the findings of the other test.[1] Rapid temperature changes, like those employed by cold testing, result in immediate painful sensations in teeth with viable nerves by 'hydrodynamic forces' causing excitation of the A-delta fibers.[30,31] The electric pulp test also utilizes the A fibers in the pulp. The A fibers appear relatively late in pulpal development explaining why the electric pulp test is not reliable in young teeth.[32] If a mature, untraumatized tooth does not respond to either the cold test or the electric pulp test, it should be considered non-vital.[33] Pulp vitality testing should assesses the pulp's supply of blood, but most of the current tests labeled as vitality tests do not directly assess the pulp's vascularity. Laser Doppler Flowmetry and pulse oximetry are two techniques being evaluated for use in diagnosing pulp status, but neither is used commonly in dental practices and both require future research to improve their diagnostic reliability and accuracy.[34] Considering all the available pulp tests, it should be noted that a retrospective evaluation of five studies showed that results of diagnostic testing were more likely to be accurate in disease-free teeth rather that in teeth with pulpal disease.[35]

The detection of a radiographic bony lesion is greatly affected by the relative location of the root of the tooth and its orientation in respect to the cortical and cancellous bone.[1] Previous research has shown that bony lesions can be detected on an ordinary intraoral radiograph only when there is (a) a perforation, (b) extensive destruction of the outer surface of the bone cortex or (c) erosion of the cortical bone from the inner surface. Those lesions confined solely in cancellous bone cannot be detected on the same radiographs. This was illustrated by Bender and Seltzer, who created artificial lesions in cadaver bone and evaluated them radiographically for evidence of a radiolucency.[21] Radiographic lesions are seen according to the contrast in bone

densities, meaning that a visual radiographic lesion depends on how much mineral is lost from the calcified tissue. There is more mineral content per unit volume in cortical bone than in cancellous bone, thus demonstrating that resorptive or demineralization changes in the more highly calcified tissue, like cortical bone, will result in a more rapid appearance of a radiographic lesion. Since cancellous bone is not as highly calcified as cortical bone, there is not enough mineral loss in cancellous bone alone to exhibit a radiographic lesion.[36] Numerous studies show that 30% to 50% of mineral loss must occur in bone before a lesion can be visualized radiographically.[36,37,38] A follow up to this study also theorized that certain teeth are more likely to exhibit radiographic bony change due to their anatomic location and the relationship of the apex of the tooth to the cortical-cancellous bone junction. Anterior teeth and premolars have apices close to this bone junction, thus exhibiting periapical pathosis earlier than molars, which have their roots more centrally located within cancellous bone. Also, in molars the lesions must expand a greater distance to reach the cortical-cancellous bone junction.[39]

Looking beyond what is occurring below the tooth in the periradicular bone, it should be noted that changes in the angulation of the x-ray beam can increase, decrease, or even eliminate the presence of a radiographic lesion.[40,41] Since it is the percent of mineral loss within the path of the central x-ray beam perpendicular to the object, and not the size of the lesion, that produces a visual radiographic lesion, then a change in angulation in the x-ray or a change in position of the object can alter the appearance of the radiographic lesion.[36]

It is well understood that the dental pulp has a great potential to heal itself. Utilization of the components naturally found in the normal pulp occurs in many ways. For instance, macrophages begin the healing process by debriding the injured tissue. The vascular supply is of critical importance because it transports inflammatory mediators into the area of the injury and

supplies nutrients to fibroblasts as they build new collagen. Odontoblasts are responsible for laying down reparative dentin or reactionary dentin. The new tubules are formed in response to growth factors released from collagen. These growth factors include transforming growth factor (TGF)-beta, insulin-like growth factor (IGF)-I and –II, bone morphogenetic proteins (BMPs), vascular endothelium growth factor (VEGF), and other growth factors that cause the attraction, proliferation, and differentiation of mesenchymal stem cells.[1]

As shown, the tooth is a unique and complex part of the human body, especially when one considers the dental pulp. The dental pulp's components- cells, tissues, nerves, and blood vessels- all greatly contribute to the pulps daily function, response to trauma and insult, and regenerative potential. Ideally, for the dentist to truly understand the complexity and vitality of the pulp, examination of histologic sections of the pulpal tissue is necessary. However, this is impractical and not feasible in a clinical setting. Instead, the dentist must rely on the collection of historical data given by the patient, as well as clinical exams, specialized tests, and radiological studies. This collection of both subjective and objective data, for the most part, while being supported as evidence based techniques, come with limitations and are at times proven to be no longer fact-based. One such example is the previous thought that any tooth with a suspected pulpally derived periapical radiolucency is non-vital. Many researchers and their studies have proven, even if indirectly, that this is not always the case.[1,24,27,29] The purpose of this study, which is a retrospective chart and radiograph review, is to evaluate this shift in paradigm by looking at the prevalence of radiolucent lesions associated with vital, mature permanent teeth in a pediatric population under the age of 19.

Materials and Methods

Design: This is a retrospective electronic dental chart and digital radiograph review focusing on the diagnostic clinical notes, diagnostic radiographs, and day of treatment clinical notes to determine the prevalence of vital teeth requiring non-surgical root canal therapy (NSRCT) that present with radiolucencies in mature permanent teeth in a pediatric population. The Institutional Review Board of Virginia Commonwealth University, Richmond, Virginia, approved this study. (IRB #: HM15164)

Electronic Dental Charting and Digital Radiographs: Virginia Commonwealth University's School of Dentistry (VCU SoD) employs the use of Axium for its electronic dental charting and MIPACS for its digital radiographs. All residents and faculty members at VCU SoD have access to the electronic dental charts and digital radiographs of patients of record.

Sample Selection: The initial population was all patients who presented for dental treatment at the Virginia Commonwealth University's School of Dentistry. The population was reduced to 551 subjects by querying the electronic dental recording system (Axium) for patients between the ages of 8 and 18 who had received non-surgical root canal therapy (NSRCT) under the ADA codes 3310, 3320, and 3330 between the dates of November 30, 2009 and March 1, 2013. This reduced population was made up of 252 males and 299 females. This population was refined even further by evaluating the diagnostic radiographs of the tooth associated with the completed

ADA code 3310, 3320, and 3330 of each of the 551 subjects to determine the presence or absence of a periapical radiolucent lesion. Of the 551 subjects that presented for NSRCT, 246 were determined to have a periapical radiolucency. These 246 subjects had the clinical notes evaluated, specifically looking for information on the pulpal tissue at the time of access. Of the 246 clinical notes evaluated, only 184 subjects had the information, thus the prevalence of vital teeth requiring non-surgical root canal therapy (NSRCT) that present with radiolucencies in mature permanent teeth in a pediatric population was determined from these 184 subjects.

Data collection: All data collected was recorded in an Excel Spreadsheet. All 551 subjects from the query of Axium had a number 1-551 assigned to them. The number assigned was keyed to the subjects dental record number which was kept separately so as to avoid HIPAA violations. These 551 subjects had their ADA code treatment completed, date of birth, gender, insurance type, and tooth number recorded. This information was available through the results of the query and were easily transferred to the Excel Spreadsheet. The date of birth and date of diagnostic radiograph, which was obtained and recorded through evaluation of the MIPACS system, was used to calculate the age at the time of the diagnostic radiograph. This age was recorded in the Excel Spreadsheet. All 551 subjects had one diagnostic radiograph separately labeled as "pedo/endo" in MIPACS for evaluation. The evaluation of the digital radiograph was carried out by two calibrated dentists, Drs. Erika Lentini (Pediatric Dental Resident) and Claudia Colorado (Endodontic Dental Resident). Initially, Drs. Erika Lentini and Claudia Colorado viewed each "pedo/endo" radiograph on the computer, through MIPACS with no enhancements. Each evaluator determined if the tooth had a periapical radiolucency present or not, and if present, which root was associated with the lesion. The subjects that resulted in a disagreement of the

presence or absence of a periapical radiolucency had the same radiographs evaluated by Dr. Karan Replogle (Advanced Specialty in Endodontics, Program Director) who made the final decision. The presence or absence of the periapical radiolucency and the root with which it was associated with was recorded in the excel spreadsheet. There were 246 subjects determined to have a periapical radiolucent lesion. The remaining data was collected by Dr. Erika Lentini. These 246 subjects had their electronic diagnostic notes and treatment notes reviewed for the following information: responses to palpation, percussion, EndoIce, and pulp testing, as well as the pulp and periapical diagnosis and condition of pulpal tissue upon access. When available, this information was recorded in the Excel Spreadsheet. Of the chart notes reviewed, 184 of the 246 had information on the condition of the pulpal tissue upon access. If the pulpal tissue was noted as hemorrhagic at the time of access, even if only in one canal, it was considered vital. Dr. Al Best, a statistician, then preformed a statistical analysis of this study by looking at two groups, all the teeth with radiolucencies and the teeth with radiolucencies that were also determined to be vital. These two groups were evaluated for significance in gender, insurance, age, puberty, and tooth characteristics, including arch, anterior versus posterior, tooth type (incisor, canine, premolar, molar), and root association. The groups were then compared using logistic regression.

Results

The results of the study are shown in multiple sections. The initial section discusses the calibration and consistency between Drs. Erika Lentini and Claudia Colorado in terms of reviewing the radiographs for the presence or absence of a periapical radiolucency. In the next sections, teeth with radiolucencies and teeth with radiolucencies that are vital are examined separately by evaluating demographic characteristics, tooth characteristics, and endodontic pulpal testing. The last sections of the results explain the multivariable analyses of the teeth with radiolucencies and the teeth with radiolucencies that present as vital.

Agreement Statistic

When reviewing radiographs for the presence or absence of a periapical radiolucency, Drs. Erika Lentini and Claudia Colorado agreed 88% of the time (Kappa=76.5%, P<0.001). With the 70 cases in which there was a disagreement, Dr. Karan Replogle reviewed the radiographs and made the final decision on a periapical radiolucency. The following results are based on teeth determined to have a periapical radiolucency by the three dentists.

Demographic characteristics

A total of 551 cases met the inclusion criteria for the study. Fifty-four percent of the patients were female, 83% were Medicaid patients, and 70% were post pubertal, with puberty being defined as males ages 9 through 14 and females ages 8 through 13 (Table 1). Overall, the prevalence of radiolucency was 44.6% (246/551, 95% CI = 40.5% to 48.8%). There was no evidence for a relationship between sex and radiolucency; that is, in females 44% showed

radiolucency and in males 45% (P>0.7). Similarly, there was no difference in radiolucency depending upon insurance (P>0.3). There was a significant difference in radiolucency depending upon puberty. Fifty-two percent of pubertal children displayed radiolucency, while only 42% of post-pubertal children did (P=0.04). There was also a significant difference with radiolucency depending on age. The average age of children with teeth with radiolucencies was 14.6 years versus 15.2 years teeth without radiolucencies (t-test P value = 0.0106).

Vitality was assessed in teeth with radiolucencies. These results were available for 184 of the 246 teeth presenting with radiolucencies. Overall, 54.9% of teeth were non-vital (Table 2). The prevalence of vital teeth was 45.1% (95% CI = 38.1% to 52.3%). That is, in children with teeth presenting with a radiolucency, the prevalence of actual vitality was significantly greater than zero (P < 0.0001). Forty-five percent were vital upon visual inspection of the pulp.

The right hand portion of Table 1 shows the relationship between the demographic characteristics and tooth vitality. In female teeth, 49% were vital and in males 62% were vital, a non-significant difference (P > 0.07). There was no evidence for a difference depending upon insurance status or age, but there was a difference in vital teeth depending upon pubertal status. Post pubertal children had more vital teeth than did pubertal children (53% vs. 31%).

Table 1. Demographic characteristics

	Radiolucency			*P*	Vitality			*P*
Tooth	Yes	No	%Yes	value	Not vital	Vital	%V	value
Sex				0.7975				0.0735
Female	132	167	44		50	52	51	
Male	114	138	45		51	31	38	
Insurance				0.3187				0.2556
Medicaid	204	252	45		77	70	48	
Private	19	16	54		13	5	28	
No insurance	23	37	38		11	8	42	
Puberty				0.0401*				0.0027*
Prepubertal	0	1	0		0	0		
Puberty	87	80	52		47	21	31	
Post puberty	159	224	42		54	62	53	
Age (years)				0.0106*				0.1112
8	1	1	50		1	0	0	
9	12	6	67		6	3	33	
10	7	7	50		2	4	67	
11	10	13	43		6	1	14	
12	15	21	42		7	5	42	
13	22	19	54		13	6	32	
14	34	31	52		13	11	46	
15	42	41	51		19	15	44	
16	44	61	42		15	16	52	
17	33	66	33		7	13	65	
18	26	39	40		12	9	43	
Mean	14.64	15.16			14.29	14.87		
SD	2.43	2.30			2.53	2.35		

* There was a significant difference between the groups at ($P < 0.05$) using a likelihood ratio chi-square test or t-test, as appropriate.

Table 2. Vitality Results in Teeth with Radiolucencies

Vitality	N	Percentage	95% CI	
Not vital	101	54.9	47.7	61.9
Vital	70	38.0	31.3	45.2
Both	13	7.1	4.2	11.7
Total	184			

Abbreviation: CI = confidence interval, Both = in a multi-rooted tooth, one root was vital and the other was not.

Tooth characteristics

The tooth characteristics were considered next. Table 3 shows that a radiolucency was more common in mandibular teeth than in maxillary teeth (52% vs 37%). Anterior teeth had a higher percentage of radiolucencies as compared to posterior teeth (57% vs 41%). However, when looking at individual anterior teeth, the incisors had twice the prevalence of radiolucency than did canines (although the number of canines is too small for a statistical comparison). Within the posterior teeth, there appears to be a difference between radiolucency in premolars (24%) versus molars (44%). Further evaluation of differences between individual teeth show the highest prevalence of radiolucency in L1 (centrals) at 63%, which is higher than the 48% in M1 (first molars) ($P = 0.01$). These two individual teeth were significantly higher than all the others ($P < .0001$), however they were not significantly different from one another.

The right hand portion of the table shows the relationship between the tooth characteristics and vitality in teeth with radiolucencies. Mandibular teeth had a higher percentage of vitality (55%) than did maxillary (31%). Posterior teeth had a higher percentage of vitality (55%) than anterior teeth (13%), with molars being vital 57% of the time as compared to other tooth types.

The specific root associated with the radiolucency was only described in teeth with radiolucency. Twenty-nine percent of the teeth with radiolucencies were single rooted. The teeth

with multiple roots presented with a radiolucency on the mesial root 52% of the time, and 47% on the distal root. There was a significant relationship between vitality and whether the root was single rooted ($P < .0001$). Vital teeth were more likely in the mesial or distal root of multi-rooted teeth.

Table 3. Tooth Characteristics

	Radiolucency			*P*	Vitality			*P*
Tooth	Yes	No	%Yes	value	Not vital	Vital	%V	value
Arch				0.0007*				0.0010*
Mandibular	146	137	52		49	60	55	
Maxillary	100	168	37		52	23	31	
Anterior vs Posterior				0.0029*				<.0001*
Anterior	64	48	57		39	6	13	
Posterior	182	257	41		62	77	55	
Tooth Type				0.0004*				<.0001*
Incisor	63	45	58		39	6	13	
Canine	1	3	25		0	0		
Premolar	12	38	24		7	3	30	
Molar	170	219	44		55	74	57	
Tooth				<.0001*				<.0001*
L1	55	32	63		33	6	15	
L2	8	13	38		6	0	0	
C	1	3	25		0	0		
P1	2	10	17		1	1	50	
P2	10	28	26		6	2	25	
M1	150	163	48		52	62	54	
M2	20	56	26		3	12	80	
Single rooted								<.0001*
Yes	71				43	8	16	
No	175				58	75	56	
Mesial root								0.0003*
Yes	127				41	56	58	
No	119				60	27	31	
Distal root								0.0001*
Yes	115				35	52	60	
No	131				66	31	32	
Palatal root								0.8994
Yes	23				8	7	47	
No	223				93	76	45	
Buccal root								0.7807
Yes	22				11	8	42	
No	224				90	75	45	

* Groups were significantly different by a likelihood ratio chi-square test ($P < .05$).

Vitality Testing

Endodontic diagnostic testing performed on treated teeth was intermitantly recorded in the electronic health record. Those with a positive palpation were significantly less likely to be vital. Results from percussion were unrelated to vitality. Those with no response to cold testing were significantly more likely to be non-vital, as were those who were 80/80 on the EPT.

Table 4. Viability testing

	Vitality			*P*
Testing	Not vital	Vital	%V	value
Palpation				0.0096*
Positive	45	53	54	
Negative	54	29	35	
Percussion				0.7159
Positive	24	18	43	
Negative	75	64	46	
Cold testing				<.0001*
No response	87	11	11	
Hyperresponsive	6	33	85	
Prolonged	2	28	93	
Normal response	1	6	86	
EPT				0.0230*
80/80	64	8	11	
other	8	5	38	

* Groups were significantly different by a likelihood ratio chi-square test ($P < .05$).

Endodontic diagnoses before treatment

Pulpal and periapical diagnoses were recorded before treatment (Table 5).

Table 5. Pulpal and Periapical Diagnoses

	Vitality		
Diagnosis	Not vital	Vital	%V
Pulpal diagnosis			
normal pulp	0	1	100
reversible pulpitis	0	1	100
irreversible pulpitis	1	9	90
asymptomatic irreversible pulpitis	1	11	92
symptomatic irreversible pulpitis	2	43	96
previously initiated	10	3	23
necrotic pulp	87	15	15
Periapical diagnosis			
asymptomatic apical periodontitis	39	23	37
symptomatic apical periodontitis	45	57	56
acute apical abscess	7	2	22
chronic apical abscess	10	1	9

Multivariable analyses: Radiolucency

A number of factors were individually related to the presence or absence of radiolucency and in this section, they are considered together. A multiple logistic regression was used to analyze the four demographic characteristics in Table 1 and none were significant (P = 0.0825). When the tooth characteristics (arch, anterior vs posterior, tooth type) are considered, the arch differences depend on tooth type (P = 0.0363). The arches had different prevalence of radiolucency only within the first molars (P = 0.0065) and second molars (P = 0.0391, Table 6). In molars, radiolucency is more likely in the mandible. And so, the prevalence of radiolucency may be grouped into the anterior teeth 57% (64/112), premolars 24% (12/50), mandibular first molars 54% (107/199), maxillary first molars 38% (43/114), mandibular second molars 33% (19/57), and maxillary second molars 5% (1/19).

Table 6. Radiolucency: Arch Differences within Tooth Type

	Mandibular			Maxillary			
	Radiolucency			Radiolucency			*P*
Tooth	Yes	No	%Yes	Yes	No	%Yes	value
L1	15	0	100	40	32	56	0.9850
L2	2	1	67	6	12	33	0.2947
C	0	0		1	3	25	ND
P1	0	1	0	2	9	18	0.9964
P2	3	5	38	7	23	23	0.4236
M1	107	92	54	43	71	38	0.0065*
M2	19	38	33	1	18	5	0.0391*

* Arch difference testing within each tooth. ND=significance test not done.

Multivariable analyses: Vitality

The primary outcome variable in teeth presenting with radiolucencies was vitality. A tooth was found to have a periapical radiolucency and be non-vital upon visual inspection only 55% of the time. In 45% of the cases a tooth with a periapical radiolucency was still vital. The demographic characteristics in Table 1 were individually found to be significantly related to vitality. When all of the demographic characteristics were considered in a multiple logistic regression, there was a significant relationship (P = 0.0075) with only puberty remaining significant (P = 0.0148). What this implies is a relationship with age and sex and redoing the analysis with these two variables indicates that older females and younger males are more likely to have teeth with radiolucencies that are still vital (Figure 1).

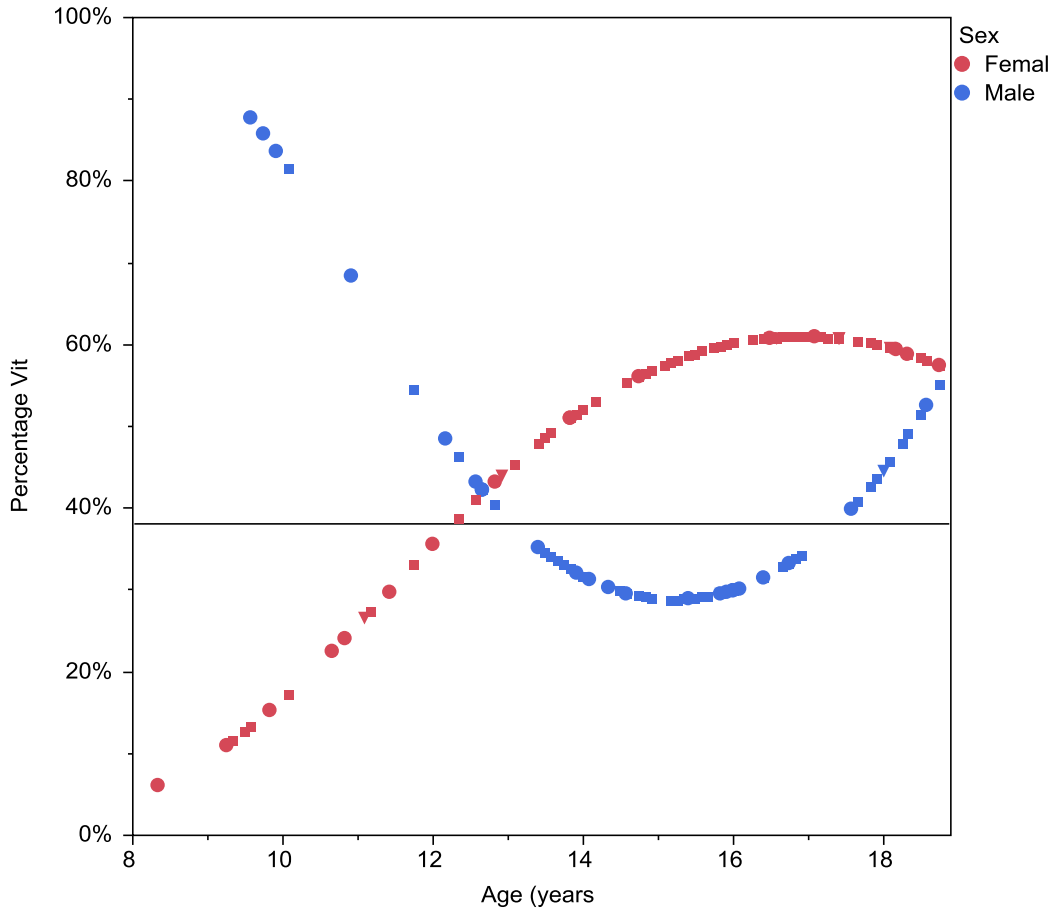

Figure 1. Vitality Relationships with Age and Sex

The results in Table 3 indicated that tooth characteristics were individually related to tooth vitality. In a multivariable logistic regression comparing mandibular versus maxillary, anterior versus posterior, and the four tooth types (incisor, canine, premolar, molar), it was found the relationship with vitality was significant ($P < .0001$).

Discussion

As detailed above in the introduction, a review of literature revealed an abundance of studies pertaining to the formation process of periapical lesions and their detection on radiographic film in the adult population. There are also multiple case studies that examine the disease process and vitality in an immature, permanent tooth. Few studies and literature references exist, however, that evaluate specific patient demographics and tooth characteristics in terms of their relationship to the presence of a radiographic periapical lesion and vitality for both a mature, permanent tooth and the pediatric population. This study focused on such a population, pediatric patients with mature, permanent teeth. It was found that relationships with both demographics and tooth characteristics and the presence of a radiolucency exist, as well as relationships in terms of demographics and tooth characteristic and vitality or non-vitality of the tooth. The shortage of past research places some limitations on understanding the findings of this study, however, the data that is available can begin to offer insight into the results.

While this study's main goal was to determine the prevalence of teeth presenting with radiolucencies that are vital, it first analyzed the characteristics of the teeth presenting with a radiolucent lesion, with no consideration for vitality. For comparison, a past study, by Sadeghi and Dibaei was analyzed. It looked at the prevalence of odontogenic sinus tracts in endodontically treated teeth. Sadeghi and Dibaei's study differed from this study in two aspects. This first difference is obvious in that the previous study focused on the presence of an odontogenic sinus tract, and not a radiographic periapical lesion.[42] An odontogenic sinus tract is

a secondary manifestation of a chronic dental infection and generally indicates the presence of a necrotic pulp, chronic apical abscess, and sometimes a periodontal abscess.[1] Clinically, sinus tracts often present in combination with a peripical radiolucency. This holds true in the Sadeghi study, as it was noted that a sinus tract was always associated with a tooth that presented with a periapical radiolucency.[42] For the sake of comparison, since all teeth with sinus tracts had radiographic lesions, the term sinus tract will be considered synonymous with periapical radiolucency and the findings of the Sadeghi and Dibaei study will be superficially compared to this study. The second difference results from the age discrepancy. This study's population was of patients under the age of 18, which only partially resembles the population of the Sadeghi and Dibaei study, which looked a population between the ages 10 and 69. When these allowances were made for the differences in the focus of the two studies, it was found that there were similarities in the results.

In this study a significant relationship existed between the presence of a periapical radiolucency associated with a tooth and the pubertal status of the patient. The overall prevalence of radiolucent lesions was 44.6% (246/551, 95% CI=40.5% to 48.8%), with 52% of radiolucent lesions being found in pubertal children and 42% in post-pubertal children (P=0.4). Again, puberty was defined as males, ages 9 through 14 and females ages 8 through 13. Looking beyond simply pubertal status, there also existed a significant relationship between the presence of a radiolucency and the chronological age of the patient. The average age of a child who had a tooth presenting with a radiolucency was 14.6 years versus the age of children who had teeth with no associated radiolucency, which was 15.2 years (t-test P value=0.0106). In the Sadeghi and Dibaei study, pubertal status was not considered. Instead, the Sadeghi study chose to examine specific chronological age, finding that the highest prevalence of sinus tracts was in the 10-19 year age

group.[42] Even though their study examined a population age range much greater than this study, the age groups affected by the pulpal changes and subsequent sequelae, periapical lesion or sinus tract, are similar. To understand the possible reason behind the prevalence of radiographic lesions and sinus tracts in this age population, it is prudent to consider the bony changes that occur during growth.

No research could be found that evaluated the cortical and cancellous bone specifically in the maxillary and mandibular jaw. However, a study by Mora evaluated the age-related changes in cortical and cancellous vertebral bone density in growing girls. It was found that throughout childhood and adolescence, cortical bone density increases progressively, whereas cancellous vertebral bone density changes little with age in pre-pubertal girls, decreases slightly in early puberty, increases in late puberty, and reaches maximum values at sexual maturity. The results of the Mora study suggested that, in females, hormonal and/or metabolic factors were more important in the regulation of cancellous bone density during the first two decades of life, than were mechanical considerations.[43] Understanding that a lesion must cause at minimum 30% to 50% of bone loss for a lesion to be visualized radiographically[36,37,38] and that growing children have innately less dense bone[43] helps to explain the age that more commonly presented with a periapical radiolucency. In both this study and the Sadeghi and Dibaei study there was no evidence of a relationship between gender and the presence of a radiolucency or sinus tract.[42]

The study herein, as well as the Sadeghi and Dibaei study on a more limited scale, evaluated tooth characteristics, like tooth type and root anatomy, and their relationship to the presence of a periapical radiolucency or sinus tract. The Sadeghi and Dibaei study found that the prevalence of sinus tracts in the mandible was significantly higher than in the maxilla (P=0.011) and higher in anterior teeth as compared to posterior teeth (P=0.001).[42] Similarly, the results of

this study showed that periapical radiolucencies were more commonly associated with mandibular teeth than maxillary teeth (52% vs. 37%) and that anterior teeth had a higher percentage of radiolucencies as compared to posterior teeth (57% vs. 41%). The Sadeghi and Dibaei study, unlike this study, did not evaluate specific teeth or roots and their association with odontogenic sinus tracts, but this study did evaluate specific teeth. When evaluating the results of this study the highest prevalence of radiolucency was found in central incisors (63%) and first molars (48%). The prevalence of radiolucencies in these two teeth, while significantly higher than other teeth ($P<0.0001$), were not significantly different from one another. When all factors are considered together as to how they relate to the presence or absence of a radiolucency, it was found that the prevalence of a tooth with a radioluceny among this population can be grouped into anterior teeth 57% (64/112), mandibular first molars 54% (107/199), maxillary first molars 38% (43/114), mandibular second molars 33% (19/57), premolars 24% (12/50), and maxillary second molars 5% (1/19).

By both recalling that periacpial lesions of pulpal origins are a result of inflammation from a dental infection and understanding the normal eruption pattern and anatomy of the permanent dentition and its connection to caries formation, support is given to the finding of more peripical lesions being found in anterior teeth and first permanent molars. Typically the central incisors and the permanent first molars are first to erupt. This is relevant because the length of time the teeth are in the oral cavity is directly related to the exposure to cariogenic bacteria and susceptibility to carious infections due to poor oral hygiene and diet.[44,45] When one considers the anatomy of the molars and their incompletely coalesced occlusal pits and fissures at the time of eruption, it is easy to understand why these teeth are more prone to caries.[45] Knowing that caries are an initiator of pulpal inflammation, it becomes clearer as to why these

teeth more commonly present with radiographic periapical lesions. In terms of anatomy of the incisor, lingual pits are also vulnerable to rapidly proceeding carious lesions. Of equal importance in the discussion of maxillary incisors is the issue of crowding, which is common in the mixed dentition of children aged 6-12. The crowding inhibits natural cleansing during mastication and causes difficulty for the patient in at home hygiene. This leaves the teeth more susceptible to carious lesions[45], and potentially pulpal infections and periapical lesions.

Further evaluation of tooth characteristics and associated radiolucencies showed that 71% of teeth with radiolucencies were multi-rooted teeth, mainly molars. In a study by Demirci that assessed the prevalence of caries on individual permanent tooth surfaces, it was found that the first and second molars contributed most significantly to caries frequency, from 52.7% to 66.3%. The study also compared individual tooth surface caries rates among gender and age groups, and it was found that the prevalence of caries experience was highest among individuals between the ages of 17 and 25, with females showed a higher incidence of caries (59.1%) than males (40.9%).[46] This last finding is similar to the present study where the percentage of females (132/246, 54%) and males (114/246, 46%) who presented to the Virginia Commonwealth University Endodontic Clinic had a periapical radiolucency. With the knowledge that the periapical lesions evaluated in this study were a result of a pulpal infection caused by a carious infection, it could be expected that the findings from the aforementioned study would explain why the same teeth commonly present with a periapical radiolucency.

In a multi-rooted tooth, the radiolucency was more commonly associated with mesial roots (52% of the time) rather than distal, palatal, or buccal roots. This finding was described in a study by Bender, which found that when examining the mandibular first molar the mesial root

has a higher incidence of a detectable radiographic lesion than the distal root due to the distal root having its apex located more in the central portion of cancellous bone.[36]

After evaluating the characteristics of the teeth presenting with a radiolucency, this present study analyzed the vitality in all teeth determined to have periapical radiolucencies. A periapical lesion, also commonly termed a periapical abscess, periapical granuloma, or periapical cyst, is typically thought to be a sequelae of pulpal necrosis secondary to the inflammation process and defense mechanism of the pulp.[47,48,49] As such, many clinicians expect to find a non-vital pulp upon access of the pulp chamber on any tooth with a carious process and radiographic lesion. This is the paradigm under which most dental clinicians have been practicing. This study aims to dispute this belief and offer a fresh perspective on the vitality of pulpal tissue of mature, permanent teeth in a pediatric population.

In this study, a tooth was labeled vital or non-vital if the electronic dental health record had documentation of the pulpal tissue upon access of the pulp chamber. Teeth with pulpal tissue that was hemorrhagic upon endodontic access were defined as vital. The documentation of pulpal tissue was available for 184 of the 246 teeth presenting with periapical radiolucencies. The results of the study show a significant prevalence of vital teeth that present with a periapical radiolucency at 45.1% (95% CI = 38.1% to 52.3%). This finding was significantly greater than zero ($P<0.0001$) and thus begins to dispel the previous notion that periapical radiolucencies of pulpal origin are only associated with non-vital teeth. While the Sadeghi and Dibaei study mentioned previously did not specifically examine the prevalence of vital teeth associated with a periapical radiolucency, the results of that study showed that 192 of 348 (55%) teeth had a radiographic lesion and a preoperative diagnosis of vitality.[42]

It is also important to consider that studies, such as the one led by Kontogiannis in Athens, Greece, proved some periapical lesions are not the result of pulpal necrosis, even when they are diagnosed as such. The aim of that retrospective study was to record the incidence of periapical lesions that are not secondary to pulpal necrosis, but that originally presented to the clinic with a diagnosis of necrosis. The results of that particular study showed periapical abscesses in 0.32%, periapical granulomas in 31.28%, radicular cysts in 64.91% and various non-pulpal necrosis lesions in 3.42%, including developmental cysts, odontogenic tumors, and other lesions.[50] This knowledge gives insight into potential bias of the study herein, since no histological sections were taken on the periapical region of the subjects, here in, to verify the diagnosis of pulpal origin, thus a true diagnosis was not always available.

To understand the physiology behind how the results of this study could go against the previous paradigm of periapical radiolucencies of pulpal origin being associated with only non-vital teeth, the concept of self-strangulation of the pulp should be explored. This concept garnered support from the work by Van Hassel. It recognized, like other tissue, that pulpal tissue responds to injury with inflammation. Because the pulp is surrounded by the rigid walls of the dentin, enamel and cementum, the normal response to the inflammation is impossible. Instead there is a marked increase in the pulpal tissue pressure, which can cause compression of blood vessels and nerves. Eventually, this can result in ischemia, hypoxia, and potentially necrosis. In the last decades, though, the concept of complete self-strangulation during pulpal inflammation has changed.[51,52,53] Several studies have shown that the pressure increase during pulpal inflammation may be localized only and does not necessarily involve the entire pulp.[51,53,54] This would imply that a tooth could show necrotic changes and periapical involvement, but still have vital tissue present in the pulp.

As mentioned earlier, limited information is available on the relative amounts of cortical and cancellous bone in children and how it relates to aging.[43,55] What is known is that there is a progressive increase in the amount of cortical and cancellous bone for both genders throughout childhood and adolescence.[43,56] It is important to keep this in mind when attempting to understand the results of this study in regards to vitality. While the results showed there was no evidence for a difference depending on age, there were differences in vital teeth associated with periapical radiolucencies and pubertal status. Post-pubertal children had more vital teeth than did pubertal children (53% vs. 31%). When a multivariable analyses was completed to evaluate all characteristics of vital teeth associated with periapical radiolucencies it was found that only pubertal status had a significant relationship with vitality and the presence of a radiolucency (P=0.0148). However, there still existed a relationship with age and gender. Upon further analysis using only these two variables it was found that older females and younger males were more likely to have teeth with radiolucencies that are still vital.

The results of the previously mentioned Mora study that show cortical bone density increasing steadily, and cancellous bone not increasing until late puberty[43] do not completely explain the above findings, but do offer some explanation as to why children of any pubertal status can maintain tooth vitality in the presence of a periapical lesion. If this finding holds true for the cancellous bone in the jaws for both girls and boys, one would expect a periapical lesion to progress more rapidly with minimal inflammation from the pulp. The less dense cancellous bone would enable a quicker destruction of cortical bone, thus allowing for an earlier detection of a radiographic lesion and a greater chance for vital tissue to still be present within the tooth.

To better recognize how puberty could be significantly related to the presence of a radiolucency in a vital tooth, an understanding of the pattern of maturing immune systems in the

adolescent is imperative. As mentioned in the introduction, the dental pulp has macrophages and lymphocytes that respond to carious infections. This is similar to the human body as whole, in which the immune system is noted to change rapidly during puberty. Puberty is characterized by an increase in secretion of both androgens and estrogens in boys and girls.[57] Many studies support the concept that immunological responses and sex steroid hormoes are linked at a physiological level and a cellular level. At the time of puberty and immediately following puberty, children are at an increased risk for autoimmune disorders, which strongly suggests the immune system function is affected by the sex hormones.[58] Both T-cell lymphocytes and macrophages have intra- and extracellular receptors for androgens and estrogens, implying that they have a direct effect on the immune system.[59] There is evidence that supports the role of testosterone as a suppressor for a stress response to infection[60], which could explain the prevalence of more vital teeth in a younger population that typically are not vital in an older population. Pre-puberty and puberty are times of increased testosterone production. Testosterone, as a stress response suppressor, could potentially decrease the amount of pulpal inflammation following a carious insult, therefore, likely increasing the chance of maintaining vitality. The ages that these hormonal changes occur varies for boys and girls and could explain the difference in age found between genders.

The study again evaluated tooth characteristics, like tooth type and root number, focusing this time on their relationship to tooth vitality in the presence of a periapical radiolucency. The results showed that mandibular teeth had a higher percentage of vitality (55%) than did maxillary (31%), with posterior teeth being vital more often (55%) than anterior teeth (13%). More specifically, molars were found to be vital more often than any other tooth type (57%). A basic understanding of the anatomy of teeth helps to decipher the meaning behind

this finding. First, molar teeth are generally larger than any other tooth in the mouth. This implies a larger pulp with a greater capacity to either heal itself or maintain some area of vitality following inflammation. Molars, especially first permanent molars, are known to possess accessory canals, which would also add to pulpal volume. The number of accessory canals can range from one to over 20. Mandibular molars have a higher incidence of accessory canals (56%), than do maxillary molars (48%)[1]. The presence of these canals could possibly offer greater surface area of pulpal tissue and may play a role in maintaining vital tissue.

A review of literature returns an abundance of research involving adults, however, research involving children is typically less prolific. Extrapolating the results from adult focused studies to application with children is unjust due to the physiological and behavioral differences between the two age groups. However, the extrapolation of young child and adult findings to the more adolescent population is still employed. This extrapolation of findings from adults to children is especially true in regards to research on pulp testing, and is the reason behind possible post-publication bias.[61] While it is understood that the electrical pulp test and most thermal tests are not reliable in the primary and young, permanent dentition it can be a useful diagnostic aid in the mature, permanent dentition.[1,44,45] Still, there is little research on any pulpal tests in the mature, permanent dentition of the pediatric population. What is known is that the response of children to any pulp test can be unreliable due to the child's apprehension associated with the test itself. Because some of the testing can be uncomfortable for the child, having consistent results on repeated tests can be difficult to obtain.[45]

The most accurate way to determine the pulp status is by examination of a histological section to directly visualize the extent of inflammation or the presence of necrosis. Unfortunately in the clinical setting this is not feasible, and instead the clinician must rely on pulp tests to aid in

diagnostic purposes.[34] The two most common endodontic diagnostic tests, cold and electrical pulp tests are often referred to as vitality tests. They would be more accurately termed pulpal sensitivity tests because they are not a direct measure of pulpal vitality, but instead rely on a subjective response of the patient to an external stimulus on the nervous supply.[34,62,63] Clinicians are able to use the results of the pulpal sensitivity tests, which are nothing more than "qualitative sensory manifestations," to extrapolate and predict the vitality of the pulp.[64] If there is a response from the pulp to the stimulus, or test, it is assumed the pulp has innervation, and therefore blood supply, and is typically diagnosed by the clinician as being vital/healthy or vital/inflamed, depending on other clinical signs and symptoms.[34] It has been shown that there are some associations with the histological appearance of the pulp and the quality sensory manifestation of the pulpal sensitivity tests[65], which is why their use is still employed in the clinical setting.

Other pulpal sensitivity tests include palpation and percussion. The results of this study showed that teeth with positive palpation were more likely to be non-vital (P=0.0096). As stated above, palpation is not a vitality test, so its relationship with vitality will have to be considered in regards to the known physiology behind a painful palpation. The purpose of palpation is to detect any soft tissue swelling or bony expansion.[1] In typical situations, soft tissue swelling and bony expansions are not noted by a clinician's touch or by sensitivity on the patient's part until a more advanced, chronic infection has been established.[1] Pretreatment pulpal and periodontal diagnosis were made and compared to the vitality findings. Teeth diagnosed as having a chronic apical abscess more commonly present with soft tissue changes, bony changes, and sensitivity to palpation. In this study those teeth diagnosed as chronic apical periodontitis only had a 9% chance of vitality. Results from percussion were found to be unrelated to vitality. This finding is supported in the endodontic literature, as it is taught that a diagnosis of vital versus non-vital

cannot be made with a painful response to percussion. It can only indicate inflammation in the periodontal ligament.[1] The teeth in this study that had no response to cold testing were significantly more likely to be non-vital, as were those who scored an 80/80 on the EPT, which is supported by findings in other studies.[1]

False-positive responses (i.e. non-vital teeth responding positively to testing) and false-negative responses (i.e. vital teeth responding negatively to testing) in pulp testing were reported in this study. In regards to the false-positive responses, there were some teeth in the study herein that scored other than an 80/80 on the electrical pulp test but were determined to be non-vital (8/13) and some teeth that responded to cold but that presented as non-vital (9/76) (see Table 4 in Results). There are many possible explanations for a false- positive response, but one possibility is inherent in this population study. Pulp tests are dependent on the patient's response, and many times anxious or young patients who are anticipating an unpleasant sensation may give a premature or false response.[66,67,68] Another suggested reason is that the breakdown and necrosis of tissue can be localized to only a part of the pulp, therefore conduction can occur throught the remaining viable nerve tissue resulting in a false-positive response.[66,69] Another possible explanation for a false-positive response exists in regards to multi-rooted teeth where the necrosis may be located in only one root allowing conduction through the nerves in another root.[66,70] An additional possibility for a false-positive response is due to conduction through a metal restoration or on an inadequately dried tooth.[66,71]

In regards to false-negative responses, there were some teeth in the study herein that did not respond to cold (11/98) and who had a 80/80 on their EPT recordings (8/72), but that presented as vital (see Table 4 in the Results). Even though the goal of this study was to evaluate mature, permanent teeth, or teeth with closed apices, detection of such closure is not always

accurate on radiographic evaluation. It is known that teeth with incomplete root formation may have a higher threshold to testing and therefore will elicit a false-negative response.[66,72] The fact that teeth erupt and become functional before the neural development is complete is the explanation for this phenomenon.[66,73,74] Another suggestion behind false-negative responses is the belief that cold tests are not as reliable in teeth with obliteration of coronal pulp, due to the reparative dentin acting as an insulator.[66,75] False-negative responses also result from changes that would be inherent to the population studied, due to the typical age that patients seek orthodontic treatment. There is a marked change of responses to electrical pulp tests and thermal test following orthodontic treatment. This false-negative response may be attributed to a reduction in blood flow and possible anoxia of A delta fibers.[66,76,77]

It is imperative to remember that previous studies have shown that the clinical signs and symptoms, like those determined by diagnostic testing, do not always precisely reflect the pulp condition.[51,78] Unfortunately, few studies have been completed to evaluate the dependence of diagnostic accuracy on the character and severity of pulpal changes. Studies evaluating the specificity and sensitivity of pulpal diagnostic tools are working under the presumption that the population is homogeneous and that all patients have the same clinical spectrum in terms of their pulpal status, signs and symptoms, which is not the case in a clinical setting.[62]

In this study, the pulpal and periodontal diagnosis prior to initiation of endodontic treatment was documented for each tooth presenting with a radiolucency (when it was available) in the electronic dental record. It should be noted that the diagnosis of each tooth was made prior to the initiation of treatment and that each diagnosis was dependent upon the clinical findings during the initial appointment. Knowing that there are false-positive responses and false-negative responses associated with the pulpal tests, as mentioned above, it would be

expected that there would be some discrepancies in the initial pulpal and periodontal diagnosis and actual tooth vitality. The most common pulpal diagnosis for teeth presenting with a radiolucent lesion regardless of vitality was "necrotic pulp". However, of the one hundred and two teeth diagnosed as "necrotic pulp," 15% were found to be vital (Table 5, Results). The most common periapical diagnosis for teeth presenting with a radiolucent lesion was "symptomatic apical periodontitis". However, one hundred and two teeth were diagnosed as having "symptomatic apical periodontitis" with vitality being found in 56% of the teeth.

There are multiple factors that need to be considered in the present study. This study was a retrospective chart review, and thus, less powerful than a prospective study. The subjects used for this study were patients who presented to the Virginia Commonwealth University Department of Endondontics. This implies that these patient were seeking on their own or from referrals the specific diagnosis and treatment of an endodontist. The study limited its subject by not evaluating patients of other Virginia Commonwealth University, School of Dentistry departments (pediatric dentistry, pre-doctoral, oral surgery, advanced education in general dentistry (AEGD), faculty practice, and periodontics). It is not beyond reason to assume that most departments would consult with the endodontic department when a periapical radiolucency was diagnosed on a pediatric patient, however departments such as AEGD, the pre-doctoral department and faculty practice do perform non-surgical root canals outside of the endodontic department. Teeth that presented to these clinics with radiolucencies could have changed the results of this study. This is especially true when considering that many pediatric patients who present to the pediatric department only have bitewing radiographs taken on a regular basis and that generally periapical radiographs are only taken when there are clinical signs and symptoms.

Therefore, the diagnosis of a periapical radiolucency could have been delayed until a more chronic infection manifests or could be missed completely.

Another area of bias and weakness for the study herein is due to the subjective definition of "vitality." Teeth with pulpal tissue that was hemorrhagic upon endodontic access were defined as vital. A more objective approach to defining "vitality" could have strengthened the study. This could have been accomplished by using more objective parameters, like electric pulp test and cold test results only, to define vitality. There were also limitations due to missing documentation of pulpal tissue upon access in the electronic dental record that excluded 62 subjects from the 246 subjects noted to have a periapical radiolucent lesion. The exclusion of these possible subjects could have resulted in wrong sample size bias and could have allowed for missed differences in the result.[61] However, excluding the subjects who did not have proper documentation in the electronic dental record did eliminate the possibility of missing clinical data bias.[61]

No consideration was given to race or medical status of the subjects, and both have been shown to alter a patient's endodontic traits. In regards to race it is noted that specific types of canal morphology occur in different racial groups. African American patients, in comparison to Caucasian patients, have a higher number of extra canals in both the mandibular first premolar (32.8% versus 13.7 %) and the mandibular second premolar (7.8% versus 2.8%).[1,79] Asian patients have been noted to have different percentages of canal configurations when compared to Caucasian and African populations[1,80] and have been shown to have a higher incidence of singe-rooted and C-shaped mandibular second molars.[1,81] Analyzing teeth dependent on race could offer a clearer understanding of the reasons behind radiographic lesions and vitality associated with specific teeth. With regard to medical conditions, many of these conditions have oral

manifestations that must be considered when properly forming an accurate diagnosis, especially when those medical conditions have clinical presentations that can mimic oral pathologic lesions.[1,82] Patients who are immunocompromised and who have poorly controlled diabetes mellitus may present with recurring abscesses in the oral cavity that must be differentiated from abscesses of dental origin.[1,83] Patients with sickle cell disease typically have loss of trabecular bone patter on radiographs, which may be mistaken for a radiographic lesion of pulpal origin[1]. Knowledge of the medical conditions of the patient could aid in reducing the bias due to misdiagnosed lesions and vital teeth.

This study also looked for the presence of bleeding alone as a diagnosis for vitality and not specific bleeding quality. Inflammation of the pulp leads to a local increase in the interstitial fluid pressure and an increase in the blood flow.[84] Prolonged bleeding of pulpal tissue may indicate a tooth that is severely inflamed and is in the active process of necrosis.[1] Thus, the tooth would not be a candidate for any vital pulp therapy. Instead, it would be considered a non-vital tooth, and therefore might not be accurately labeled "vital." Subjective interpretations were also made when the pulp was determined to be partially necrotic. In this study, if there was bleeding present, if only in one area of the pulp or one root, the tooth was diagnosed as "vital." This decision could have resulted in a higher prevalence recording of vital teeth with radiolucencies. Also, no histological studies were completed on periapical tissue to validate whether the periapical lesion was of pulpal or non-pulpal origin. Although rare, periapical lesions from non-pulpal origins can mimic those extending from the pulp.[50] This is a possible area of mimicry bias.[61] Many of the lesions that mimic pulpal lesions are developmental cysts like odontogenic keratocysts, but can also be odontogenic tumors like ameloblastomas or malignant lesions. Knowing this, it may be prudent when possible to perform a histopathological exam on tissue

harvested from periapical lesions, especially when the tooth also presents as vital.[50] As an example in the study herein, one tooth was found to have a radiolucency and be defined as normal pulp and acute apical periodontitis on initial evaluation, but following treatment it was determined to have a periapical cyst (Subject #90).

Another potential area of bias, is the concern that some patients do not present for care in a timely manner due to dental fear or do not present until the symptoms are no longer bearable. This reluctance of the patient to seek treatment may result in a starting time bias.[61] This is, of course, under the assumption that if radiographic lesions were detected earlier, more teeth would be in early stages of inflammation and, therefore, could potentially have vital tissue still present within the pulp space.

The majority of current dental literature on this subject focused on disease and treatment in the adult dentition and suggested that a radiographic periapical lesion associated with a carious tooth was the result of total necrosis. The present study showed that, for the pediatric population, a mature, permanent tooth with a carious lesion can present with a periapical radiolucency and still be at least partially vital. Vitality of the dental pulp is essential in the long term survivability of a tooth. It is also necessary for the complete formation and maturation of the tooth. Even though this study examined mature, permanent teeth (meaning that the apex of the tooth has completely formed), it is also known that the tooth continues to mature internally and reaches a maximum number of myelinated nerve fibers in the pulp-dentin border at the plexus of Rashkow for up to five years.[36] When possible, maintaining a vital pulp is superior to completing a non-surgical root canal due to the protective resistance to masticatory forces that the pulp offers.[51,85] Vital pulp therapy is a treatment option that aims to maintain healthy pulp tissue that has been compromised but not destroyed by caries, trauma, or restorative procedures. It does this by

eliminating bacteria from the dentin-pulp complex. There are several different treatment options for vital pulp therapy, ranging from pulp capping to pulpotomy procedures. All of these treatments rely upon an accurate assessment of the pulp status, and careful management of the remaining pulp tissue.

In conclusion, the presence of a periapical radiolucency associated with a tooth having a carious lesion can only inform the clinician that periapical inflammation is present. It cannot predict the vitality of the tooth.[28] However, this study does show that in the pediatric population, there exists mature, permanent teeth that can present with a radiographic periapical radiolucency and still be vital. Recognizing all the areas of possible bias and limitations in this study, further research is needed to fully understand this phenomenon. The focus should be on developing tools to aid in the assessment and diagnosing of the condition of the pulp and the degree of inflammation present. Clinical research should aid in furthering the reliability and acceptance of use of known vitality pulp tests. Such tests include laser doppler flowmetry, Transmitted Laser Light (TLL), and spectrophotometry, to name a few.

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Appendix

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
1	3330	F	15	M	3	Y	MB	P	N	NR		N	SAP	NV
2	3320	F	17	M	5	N								
3	3310	M	12	M	9	Y	S	N	N	NR	64/80	N	SAP	NV
4	3330	F	16	M	14	Y	MB	N	N	H		SIP	SAP	V
5	3330	M	16	M	3	N								
6	3310	M	14	M	8	N								
7	3330	F	17	M	3	Y	MB							
8	3310	F	8	M	8	Y	S	N	N	NR		N	AAP	NV
9	3330	M	13	M	19	N								
10	3330	M	16	N	18	Y	M,D	P	P	NR	80/80	N	SAP	NV
11	3330	M	16	N	14	N								
12	3330	F	15	N	31	N								
13	3330	M	12	M	30	N								
14	3330	F	18	P	30	N								
15	3330	M	13	M	14	Y	M	N	N	NR	80/80	PI	AAP	NV
16	3330	M	13	M	30	Y	M,D	N	N	H		AIP	AAP	V
17	3320	F	17	M	13	N								
18	3310	M	14	M	8	Y	S	P	P	NR		N	CAA	
19	3330	F	18	N	31	N								
20	3330	M	17	M	30	N								
21	3330	F	15	M	30	Y	M	P	N	N	32/80	AIP	SAP	V
22	3330	F	17	M	19	N								
23	3330	F	10	M	30	N								
24	3310	F	12	N	8	N								
25	3330	F	15	M	30	N								
26	3310	F	17	M	9	Y	S					IP	SAP	V
27	3330	F	18	M	2	Y	M	N	P	P	14/80	IP	SAP	V
28	3330	F	18	M	30	Y	M,D	P	N	P		IP	SAP	V
29	3330	M	16	M	31	N								
30	3330	F	11	M	19	N								
31	3310	M	16	M	10	Y	S							
32	3310	M	16	M	7	N								
33	3330	F	9	M	19	Y	M	N	N	H	80/80	N	AAP	NV
34	3330	F	9	M	30	Y	M	P	P	NR	80/80	N	AAA	NV
35	3330	M	13	M	14	N								
36	3330	M	10	M	19	Y	M,D	N	N	H		SIP	AAP	
37	3330	M	11	M	19	Y	MB,D	P	P	P		SIP	SAP	V
38	3330	M	16	M	3	N								
39	3330	M	17	M	14	Y	DB	N	N	NR		N	SAP	NV
40	3330	F	16	M	31	Y	M,D	P	P	P		SIP	SAP	V

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
41	3310	M	14	M	9	Y	S	N	N	NR	80/80	N	AAP	NV
42	3330	F	17	M	14	N								
43	3330	F	15	M	3	Y	MB	P	N	P		SIP	SAP	V
44	3330	F	17	M	18	N								
45	3330	M	14	M	19	Y	M,D	P	N	NR	80/80	N	SAP	NV
46	3330	F	18	M	19	N								
47	3320	F	17	M	13	N								
48	3330	F	16	M	30	N								
49	3330	F	15	M	14	Y	M	P	N	P		SIP	SAP	
50	3330	F	17	M	3	Y	P,M,D	P	N	NR	80/80	N	SAP	
51	3330	M	16	M	19	N								
52	3330	M	13	M	30	Y	M	P	N	NR		N	CAA	NV
53	3330	F	16	N	30	Y	M,D	P	P			PI	SAP	V
54	3330	M	18	M	19	Y	M,D	N	N	NR	80/80	N	CAA	NV
55	3330	F	14	M	30	N								
56	3330	M	15	N	30	Y	M,D	N	N	NR	80/80	N	AAP	NV
57	3330	F	14	M	19	Y	D	P	N	H		SIP	SAP	V
58	3330	F	14	M	3	Y	P	N	N	H		SIP	AAP	V
59	3330	M	15	M	30	N								
60	3330	F	16	P	15	N								
61	3330	F	16	M	30	N								
62	3330	M	18	P	18	Y	M,D	P	P	NR	80/80	PI	SAP	NV
63	3330	M	17	M	18	N								
64	3330	M	16	M	2	N								
65	3330	F	18	M	19	N								
66	3330	F	17	M	31	N								
67	3330	F	14	M	14	N								
68	3330	F	16	M	18	Y	M,D	P	N	H		SIP	SAP	V
69	3330	M	15	M	30	Y	M,D	P	N	H	70/80	SIP	SAP	
70	3310	M	16	M	9	N								
71	3320	M	18	M	13	N								
72	3330	M	16	M	3	N								
73	3330	F	15	M	30	Y	M	P	N	NR	80/80	N	SAP	NV
74	3330	F	16	M	30	N								
75	3330	F	9	M	19	Y	M,D	N	N	H		IP	AAP	V
76	3330	F	16	M	14	Y	P	N	N	NR	80/80	N	CAA	NV
77	3330	F	18	M	18	N								
78	3330	F	15	P	18	Y	D	N	N	H		RP	AAP	V
79	3330	M	18	N	3	N								
80	3330	M	18	M	14	N								
81	3310	M	13	M	24	Y	S	N	N	N		NP	AAP	V
82	3310	M	12	M	8	Y	S	N	N	NR	56/80	N	AAP	NV
83	3310	F	17	M	10	N								
84	3310	F	16	M	8	N								

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
85	3310	M	12	M	8	Y	S	N	N	NR	80/80	N	AAP	NV
86	3310	M	14	M	25	Y	S	P	N	NR		N	SAP	NV
87	3320	F	18	M	4	N								
88	3330	F	15	M	30	Y	M,D	N	P	H		SIP	SAP	V
89	3330	M	15	M	30	Y	M	N	N	N		AIP	AAP	
90	3330	F	16	M	2	N								
91	3320	F	16	M	12	N								
92	3330	F	16	M	19	N								
93	3310	F	16	M	7	N								
94	3330	M	16	M	18	Y	D	P	P	P		SIP	SAP	
95	3330	F	17	M	30	Y	M,D	P	N	P	80/80	N	SAP	V,NV
96	3310	F	17	N	10	N								
97	3330	M	15	M	14	N								
98	3330	F	18	P	3	N								
99	3310	M	13	M	24	Y	S	P	N	NR	80/80	N	SAP	
100	3330	F	18	M	14	N								
101	3330	F	15	M	2	N								
102	3330	M	15	M	31	N								
103	3330	M	16	M	30	N								
104	3310	M	10	M	8	Y	S	P	N	P		SIP	SAP	V
105	3330	F	14	M	19	Y	M,D	P	N	P		SIP	SAP	V
106	3330	M	15	P	14	Y	M,D	N	P	NR	80/80	N	SAP	NV
107	3330	M	13	M	19	N								
108	3330	F	18	M	31	N								
109	3330	F	17	M	18	N								
110	3330	M	17	M	3	N								
111	3310	F	10	M	9	Y	S	P	N	NR	80/80	N	SAP	NV
112	3310	F	14	M	9	N								
113	3320	M	17	M	29	N								
114	3320	M	15	M	5	N								
115	3330	F	14	M	19	Y	M,D	P	N	H		SIP	SAP	V
116	3330	M	16	M	14	Y	P	N	N	H		SIP	SAP	V
117	3330	M	15	M	14	N								
118	3320	F	11	M	4	Y	S	P	P	NR		N	SAP	NV
119	3330	F	15	M	30	N								
120	3330	F	15	M	18	N								
121	3330	F	10	M	19	Y	D	P	N	H		PI	SAP	V
122	3310	F	14	M	9	N								
123	3330	F	15	M	3	N								
124	3330	F	16	P	14	Y	P,B	N	P	NR	80/80	N	SAP	NV
125	3310	F	18	N	26	N								
126	3320	F	17	M	5	Y	S	P	N	P		SIP	SAP	V
127	3330	F	18	N	3	N								
128	3330	F	14	M	19	Y	M,D	P	N	NR		PI	SAP	

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
129	3330	F	15	M	30	N								
130	3320	F	18	M	29	N								
131	3310	F	10	M	8	Y	S	N	N	NR	80/80	N	AAP	NV
132	3320	F	15	P	13	N								
133	3321	F	17	M	31	N								
134	3322	F	15	M	19	Y	M,D	P	N	H		SIP	SAP	V
135	3323	F	13	M	30	Y	M	P	N	H	25/80	AIP	SAP	NV
136	3324	M	15	M	30	Y	M	N	N	P		SIP	AAP	V
137	3325	F	18	M	15	N								
138	3326	F	17	M	3	Y	M,D	N	P			AIP	SAP	V
139	3327	M	17	M	8	Y	S	N	N	NR	80/80	N	AAP	NV
140	3328	F	11	M	9	N								
141	3329	F	17	M	30	N								
142	3330	F	17	M	30	N								
143	3331	F	18	P	4	N								
144	3332	M	14	M	30	Y	M	N	N	NR		PI	AAP	NV
145	3335	M	16	N	15	N								
146	3336	F	12	M	14	Y	M	N	N	N		AIP	AAP	V,NV
147	3337	F	15	N	18	Y	M,D	P	P	H	64/80	SIP	SAP	V
148	3339	M	15	M	9	N								
149	3330	F	13	M	19	Y	D	P	P	NR	80/80	N	AAA	V,NV
150	3310	M	16	M	8	Y	S	N	N	N	48/80	N	AAP	
151	3310	M	14	M	23	Y	S	P	P	NR	80/80	N	AAA	NV
152	3330	F	13	M	15	N								
153	3330	F	11	M	30	Y	M,D	N	N	NR		N	AAP	NV
154	3330	M	18	M	19	Y	D	P	N	NR		N	SAP	
155	3330	F	15	M	30	Y	M,D	P	P	NR	80/80	N	CAA	NV
156	3330	M	16	M	14	N								
157	3330	F	17	M	31	N								
158	3310	F	13	M	7	N								
159	3310	F	13	M	25	Y	S	N	N	NR	80/80	N	AAP	NV
160	3330	M	16	M	14	Y	D	N	N	H		AIP	AAP	
161	3310	M	14	M	25	Y	S	N	N	NR	80/80	N	AAP	
162	3330	F	16	M	14	Y	P,B	P	P	NR		N	SAP	
163	3320	F	14	M	4	N								
164	3330	F	16	M	19	Y	D	N	N	N		NP	AAP	
165	3330	M	15	M	3	N								
166	3330	M	14	M	3	N								
167	3330	M	16	M	19	Y	M,D	N	N	P		SIP	AAP	V
168	3330	M	10	M	14	N								
169	3310	F	9	P	8	Y	S	N	N	NR	80/80	N	AAP	NV
170	3330	F	17	M	19	Y	M,D	N	N	NR	80/80	N	AAP	NV
171	3330	F	16	M	3	Y	P,B	N	N	NR	80/80	N	AAP	NV
172	3310	M	15	M	9	Y	S	N	N	NR	80/80	N	AAP	NV

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
173	3330	M	15	M	30	N								
174	3330	M	13	M	31	N								
175	3330	F	15	M	19	Y	M,D	N	N			N	AAP	V
176	3330	F	12	M	19	N								
177	3330	F	18	M	30	Y	M,D							
178	3310	M	16	M	8	Y	S	N	N	NR	80/80	N	AAP	NV
179	3330	F	17	M	15	N								
180	3330	M	12	M	19	N								
181	3310	M	15	N	25	Y	S	P	N	H		AIP	SAP	
182	3330	F	16	M	31	N								
183	3310	F	17	M	9	N								
184	3330	F	16	M	19	N								
185	3320	M	16	M	29	Y	S	N	N	NR		PI	AAP	
186	3330	M	16	M	18	N								
187	3330	F	15	M	31	N								
188	3310	F	14	M	9	Y	S	P	P	NR		N	CAA	
189	3330	F	15	P	19	Y	M,D	P	P	NR		N	CAA	NV
190	3310	M	16	N	7	N								
191	3330	M	13	M	18	Y	M,D	N	N	NR		N	AAP	NV
192	3330	M	15	M	19	Y	M,D	N	N	NR		N	AAP	
193	3330	F	17	M	18	N								
194	3330	M	16	M	18	N								
195	3330	F	17	M	3	N								
196	3330	F	18	M	15	N								
197	3330	F	17	M	14	N								
198	3310	M	9	M	8	Y	S	P	P	NR	80/80	N	SAP	V
199	3330	F	12	M	30	N								
200	3330	M	17	M	31	N								
201	3320	M	16	M	13	N								
202	3330	F	18	P	3	N								
203	3330	M	14	M	19	N								
204	3320	F	16	M	5	N								
205	3330	F	16	M	18	N								
206	3310	F	11	M	9	Y	S	P	N	NR	80/80	N	SAP	NV
207	3330	M	13	M	30	N								
208	3330	M	15	M	3	N								
209	3330	M	15	M	19	Y	M,D	N	N	NR	80/80	PI	AAP	NV
210	3310	F	12	M	8	Y	S	N	N	NR	80/80	N	AAP	NV
211	3330	M	16	M	2	N								
212	3310	F	17	M	7	N								
213	3310	F	10	M	9	Y	S	P	N	NR	80/80	N	SAP	V,NV
214	3330	F	13	M	30	Y	D	P	N	NR	70/80	N	SAP	NV
215	3330	M	16	M	30	Y	M,D	P	N	NR		N	SAP	NV
216	3330	F	14	M	14	N								

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
217	3330	M	17	M	19	N								
218	3320	M	18	P	13	Y	S	P	N	NR		N	AAA	NV
219	3310	M	16	M	8	Y	S	N	P	NR	80/80	N	AAP	NV
220	3330	M	16	M	31	N								
221	3310	M	11	M	9	N								
222	3320	M	17	M	12	N								
223	3310	F	16	M	9	N								
224	3330	F	18	M	3	N								
225	3330	M	14	P	14	Y	B	P	P	NR		N	SAP	NV
226	3330	M	14	N	3	N								
227	3330	F	16	N	3	N								
228	3330	M	18	M	30	Y	M,D	N	N	H		SIP	AAP	V
229	3320	M	16	N	13	N								
230	3330	F	11	P	30	N								
231	3330	M	14	M	19	N								
232	3330	F	18	N	19	N								
233	3310	F	11	M	8	N								
234	3320	M	17	N	13	N								
235	3310	M	12	M	9	Y	S	N	N	H	60/80	NP	AAP	
236	3310	M	14	M	24	Y	S	N	N	NR		N	AAA	
237	3330	M	17	M	14	N								
238	3310	M	13	M	9	Y	S	N	N	P		SIP	AAP	NV
239	3310	M	15	M	9	N								
240	3320	F	12	M	12	Y	P,B	P	N	NR	80/80	N	SAP	NV
241	3330	M	15	M	3	Y	M,D,P	N	N	P	41/80	N	SAP	NV
242	3330	F	17	N	31	Y	M,D	P	P	H		IP	SAP	V
243	3330	M	18	M	30	Y	M,D	P	N	P		IP	SAP	
244	3330	F	18	N	18	N								
245	3330	M	15	M	19	N								
246	3330	M	15	N	3	Y	B	N	N	NR		N	AAP	NV
247	3330	M	15	M	14	Y	P,M	P	N	P		SIP	SAP	
248	3330	M	15	M	18	N								
249	3330	F	16	M	30	Y	M	P	N	NR	80/80	N	SAP	NV
250	3330	M	14	M	19	N								
251	3330	F	17	M	30	Y	D	P	P	NR		N	SAP	
252	3330	F	17	M	30	Y	M,D	P	N	NR		PI	SAP	
253	3330	M	14	M	15	N								
254	3330	F	12	M	14	N								
255	3330	F	16	M	19	Y	D	N	N	H		AIP	AAP	V
256	3330	M	11	M	30	N								
257	3330	M	12	M	3	Y	P	P	N	N		AIP	SAP	V
258	3330	M	12	M	30	N								
259	3330	F	15	N	14	N								
260	3330	F	15	N	30	Y	M,D	P	N	H		SIP	SAP	V

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
261	3330	F	17	M	14	Y	M,D,P	P	P	NR	77/80	N	SAP	
262	3330	F	13	M	30	Y	M,D	P	N	NR		N	SAP	
263	3310	M	14	M	8	N								
264	3310	M	10	M	8	N								
265	3310	F	13	M	10	N								
266	3330	F	16	M	14	N								
267	3330	F	18	M	19	N								
268	3330	M	17	M	3	N								
269	3330	M	17	M	3	N								
270	3310	F	17	M	8	Y	S	N	N	NR	80/80	N	AAP	
271	3330	M	15	M	30	N								
272	3330	M	16	M	18	Y	M,D	P	N	P		IP	SAP	V
273	3330	F	14	M	14	N								
274	3310	M	9	M	9	Y	S	N	P	NR	80/80	N	SAP	NV
275	3330	M	16	M	19	Y	D	P	N	H		IP	SAP	V
276	3330	F	16	P	30	Y	M,D	P	P	P	40/80	IP	SAP	V
277	3310	M	12	M	8	N								
278	3330	M	15	M	14	N								
279	3330	M	17	M	18	N								
280	3310	M	13	P	10	N								
281	3330	F	16	M	19	Y	D	P	N	P		SIP	SAP	V
282	3330	F	16	M	3	N								
283	3330	F	11	M	19	N								
284	3310	M	9	M	8	N								
285	3330	M	13	P	30	Y	M	N	N	NR		N	AAP	V,NV
286	3330	F	17	M	19	Y	M	P	N	H		SIP	SAP	
287	3330	F	17	M	14	N								
288	3330	M	18	P	19	Y	M	N	N	P		SIP	AAP	V
289	3330	F	12	M	3	Y	M,P	N	N	NR	80/80	N	AAP	NV
290	3330	M	15	M	30	Y	M,D	N	N	NR		N	CAA	NV
291	3330	M	15	M	19	Y	M,D	N	N	NR		N	CAA	NV
292	3330	F	15	M	30	Y	M,D	P	N	NR	80/80	N	AAA	NV
293	3330	M	12	M	19	N								
294	3330	F	14	M	30	N								
295	3310	M	16	M	25	Y	S	P	N		80/80	PI	SAP	NV
296	3330	F	15	P	19	Y	M,D	P	N	NR	80/80	N	SAP	NV
297	3330	F	16	M	19	Y	M,D	N	N	H		SIP	AAP	V
298	3330	F	13	P	19	Y	F,D	P	P	NR	80/80	PI	SAP	NV
299	3330	F	14	M	19	Y	M,D	P	N	H		SIP	SAP	V
300	3330	F	17	M	19	N								
301	3310	F	17	M	10	N								
302	3320	M	15	M	20	Y	S	P	N	P		SIP	SAP	V
303	3330	F	15	M	30	Y	M,D	P	P	NR	80/80	N	AAA	NV
304	3330	M	17	M	19	Y	M,D	N	N	N		AIP	AAP	

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
305	3310	M	9	N	8	N								
306	3330	M	16	N	19	N								
307	3330	F	12	N	30	Y	D	P	N	H		SIP	SAP	V
308	3330	F	13	M	19	Y	M,D	P	P	NR		N	CAA	
309	3320	F	17	M	4	Y	B	P	P	NR		N	SAP	
310	3330	F	15	M	2	N								
311	3330	F	16	M	2	N								
312	3330	M	17	M	3	Y	B	P	N	N		SIP	SAP	V
313	3330	M	14	N	14	N								
314	3310	F	14	M	9	Y	S	N	N	NR	80/80	N	AAP	NV
315	3330	M	14	M	30	Y	M,D	P	N	H		SIP	SAP	
316	3320	F	15	N	4	N								
317	3310	F	13	M	26	Y	S	N	N	H	80/80	N	AAP	NV
318	3330	F	11	M	30	Y	M,D	P	N		80/80	N	SAP	NV
319	3310	M	16	M	8	Y	S	N	N	NR	80/80	N	AAP	
320	3330	M	14	M	3	Y	P	P	N	P		SIP	SAP	V,NV
321	3330	F	14	M	30	Y	M	P	N	H		SIP	SAP	V
322	3330	F	16	M	31	Y	M,D	P	N	P		IP	AAP	V
323	3330	M	18	M	14	N								
324	3330	F	16	M	31	N								
325	3330	F	14	M	19	Y	M,D	N	P	NR		N	SAP	
326	3310	M	11	M	9	Y	S	P	N	NR		N	SAP	
327	3310	M	12	N	8	N								
328	3330	F	15	N	31	Y	M	P	N	NR	80/80	N	SAP	
329	3330	F	16	M	18	N								
330	3330	M	16	M	18	N								
331	3330	F	12	M	30	N								
332	3330	F	17	M	30	Y	M	P	N	H		AIP	SAP	V,NV
333	3310	F	9	M	8	N								
334	3320	F	17	M	13	N								
335	3330	F	17	M	30	Y	M,D	P	N	P		AIP	SAP	V
336	3310	M	14	N	24	Y	S	N	N	NR		N	AAP	NV
337	3330	F	15	M	3	N								
338	3330	F	17	M	14	Y	M,D,P	N	P	NR		N	SAP	NV
339	3330	M	17	M	18	N								
340	3330	F	18	M	30	Y	M	P	N	NR	75/80	N	SAP	NV
341	3330	F	18	M	18	N								
342	3330	M	17	M	3	N								
343	3330	F	17	M	18	N								
344	3310	M	15	P	9	N								
345	3320	M	16	N	13	N								
346	3330	F	18	N	3	Y	MB	P	N	H	54/80	SIP	SAP	NV
347	3330	M	17	M	18	N								
348	3330	M	17	M	30	N								

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
349	3330	F	12	M	30	N								
350	3310	F	18	N	9	Y	S	N	N	NR	80/80	N	SAP	
351	3330	M	16	M	19	N								
352	3330	F	18	N	14	N								
353	3330	M	12	M	30	Y	M,D	P	N	H		SIP	SAP	V
354	3310	F	10	M	8	N								
355	3310	M	15	M	7	Y	S	N	N	NR	80/80	N	AAP	NV
356	3310	M	16	P	9	Y	S	P	P	NR	80/80	N	SAP	NV
357	3330	M	13	M	30	Y	M,D	N	N	NR	80/80	N	CAA	NV
358	3330	M	16	M	15	N								
359	3320	M	16	M	13	N								
360	3330	M	13	N	19	N								
361	3320	M	16	N	13	N								
362	3330	M	15	N	19	Y	D	N	N	P		SIP	AAP	V
363	3320	M	18	P	20	N								
364	3330	M	12	N	30	N								
365	3310	M	9	M	8	Y	S	P	P	NR	80/80	N	CAA	
366	3330	M	16	M	19	N								
367	3310	M	16	M	10	N								
368	3310	M	14	M	25	Y	S	P	N	NR	80/80	N	SAP	NV
369	3320	M	15	M	4	N								
370	3330	M	13	M	3	Y	M	P	N	H		SIP	SAP	V
371	3330	F	15	M	19	Y	M	P	P	NR		N	SAP	
372	3330	M	17	M	3	Y	M,D	N	N	NR	80/80	N	AAP	NV
373	3330	M	10	M	30	Y	M,D	P	N	H		SIP	SAP	V
374	3330	F	12	M	19	Y	D							
375	3310	F	9	M	9	N								
376	3330	F	14	M	31	Y	M,D	P	N	P		SIP	SAP	V
377	3330	M	14	M	19	N								
378	3330	M	14	M	30	Y	M,D	N	N	NR	80/80	N	AAP	NV
379	3320	F	16	M	29	N								
380	3330	M	16	M	14	N								
381	3330	M	14	M	14	N								
382	3330	F	16	M	19	Y	M,D	N	N	NR		N	AAP	V
383	3330	F	10	M	30	N								
384	3320	M	13	M	4	N								
385	3330	F	15	M	31	N								
386	3330	M	12	M	19	N								
387	3330	F	18	M	18	Y	M,D	P	P	P		SIP	SAP	V
388	3330	M	15	M	14	Y	B	N	N	NR	80/80	N	CAA	NV
389	3310	F	11	M	7	N								
390	3330	F	14	M	30	N								
391	3320	M	17	M	4	N								
392	3330	F	17	M	30	N								

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
393	3330	M	15	M	19	N								
394	3330	F	16	M	18	Y	D	P	P	P		SIP	SAP	
395	3330	F	15	M	14	Y	MB,DB	P	N	P		SIP	SAP	V
396	3330	F	16	M	19	N								
397	3330	F	9	M	30	N								
398	3330	F	9	M	19	Y	M,D	N	N	NR		N	AAP	
399	3310	M	18	P	10	Y	S	N	N	NR	80/80	N	AAP	NV
400	3330	M	16	M	14	N								
401	3330	F	18	M	19	Y	M,D	P	P	NR		N	SAP	
402	3320	F	18	M	4	Y	B	P	N	NR		N	SAP	V,NV
403	3330	F	9	M	19	Y	D					N	AAA	NV
404	3330	M	15	M	14	N								
405	3330	F	17	M	19	N								
406	3330	F	18	P	30	Y	M	P	N	H		IP	SAP	NV
407	3330	M	18	M	18	N								
408	3330	M	18	P	19	N								
409	3310	F	18	N	25	Y	S	P	P	N	80/80	N	SAP	NV
410	3330	M	17	M	19	N								
411	3330	F	17	M	30	N								
412	3330	M	18	M	14	N								
413	3330	F	14	M	3	N								
414	3330	F	16	M	14	Y	MB	P	P	NR	80/80	N	SAP	V,NV
415	3330	F	18	M	14	Y	D,P	P	N	N		AIP	SAP	V
416	3310	M	16	M	9	Y	S	N	N	NR	80/80	N	AAP	NV
417	3330	F	11	M	30	Y	M,D	N	N	NR		PI	AAP	
418	3330	M	14	M	30	Y	M,D	N	N	NR	80/80	N	CAA	NV
419	3330	M	14	M	19	N								
420	3330	F	14	N	30	N								
421	3330	F	13	N	3	N								
422	3330	F	17	N	19	N								
423	3330	M	12	N	30	Y	M,D	N	N	P		SIP	AAP	V
424	3330	F	14	N	14	Y	M,P	P	N	H		SIP	SAP	V
425	3330	M	18	M	15	N								
426	3330	M	14	M	31	Y	M	N	N	H		AIP	AAP	V
427	3320	F	17	M	5	N								
428	3330	M	16	M	14	Y	P	N	P	NR		PI	SAP	NV
429	3330	M	14	N	3	N								
430	3330	F	15	M	30	N								
431	3330	M	17	M	30	Y	M	N	N		80/80	N	AAP	V
432	3330	F	12	M	30	N								
433	3330	F	17	M	3	Y	M,D,P	P	P	H		SIP	SAP	V
434	3310	F	13	M	24	Y	S	N	N	NR	80/80	N	AAP	NV
435	3330	M	15	M	14	N								
436	3310	M	14	M	24	Y	S	N	N	NR	80/80	N	AAP	

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
437	3330	F	11	M	30	N								
438	3330	F	17	M	14	N								
439	3330	F	17	M	19	Y	M,D	P	P	P		AIP	SAP	V
440	3330	F	16	M	15	N								
441	3330	F	11	M	3	N								
442	3310	M	12	M	9	Y	S	P	P	NR		PI	SAP	
443	3310	F	14	M	6	N								
444	3310	M	15	M	9	N								
445	3310	M	12	M	8	Y	S	N	P	NR		N	SAP	NV
446	3330	F	16	M	19	N								
447	3330	M	10	M	30	N								
448	3330	F	17	M	3	N								
449	3310	M	17	M	6	Y	S	P	N	NR	80/80	N	SAP	
450	3330	M	17	M	3	Y	P	N	N	NR	80/80	N	CAA	
451	3330	F	17	M	31	N								
452	3330	F	16	P	3	Y	P	P	P	P	28/80	IP	SAP	
453	3320	F	17	N	13	N								
454	3330	M	15	N	15	N								
455	3330	M	15	M	30	Y	M	N	N	NR	80/80	N	CAA	V,NV
456	3310	F	14	M	8	N								
457	3330	M	13	M	3	N								
458	3320	M	15	M	29	N								
459	3330	M	13	M	3	N								
460	3330	F	13	M	19	Y	D	P	P	H		SIP	AAA	V,NV
461	3330	F	18	M	30	N								
462	3320	F	16	M	13	Y	S	N	N	NR	80/80	N	AAP	NV
463	3330	F	16	M	19	N								
464	3330	F	15	M	14	N								
465	3310	F	18	N	7	Y	S	P	N	NR	80/80	N	SAP	NV
466	3330	M	17	M	2	N								
467	3330	M	18	P	18	N								
468	3330	F	14	M	19	Y	D	N	N	H		SIP	AAP	V
469	3320	M	18	P	13	Y	B	P	N	NR		N	SAP	NV
470	3310	F	11	M	10	Y	S	P	P	NR	80/80	N	SAP	NV
471	3330	F	17	P	19	Y	M,D	P	N	H		N	SAP	V,NV
472	3330	M	15	M	19	Y	M,D	N	N	NR		N	AAP	V
473	3320	M	17	M	13	N								
474	3330	M	14	N	30	Y	D	P	N	NR	80/80	N	SAP	NV
475	3310	F	18	N	8	Y	S	P	P	NR	80/80	N	SAP	NV
476	3330	M	16	M	3	N								
477	3330	M	13	N	19	Y	D					PI	AAP	NV
478	3320	F	17	M	4	Y	B	N	N	NR	80/80	N	AAA	NV
479	3310	F	13	M	9	N								
480	3320	F	16	M	20	Y	S	P	P		80/80	PI	AAP	NV

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
481	3330	F	11	M	30	N								
482	3330	M	15	P	31	N								
483	3320	F	17	M	4	N								
484	3330	M	17	M	19	Y	M	N	N	NR		N	AAP	
485	3310	M	9	M	25	Y	S	P	P	NR		N	SAP	V
486	3330	F	9	M	19	Y	M,D,F	P	P	NR		N	CAA	
487	3320	F	12	M	13	N								
488	3330	F	18	M	3	N								
489	3330	M	12	M	19	N								
490	3320	F	15	N	5	N								
491	3330	F	12	M	3	N								
492	3330	M	16	M	19	N								
493	3330	F	17	M	31	N								
494	3320	M	18	M	5	N								
495	3320	M	18	P	21	N								
496	3310	F	17	N	11	N								
497	3310	M	11	M	10	Y	S	P	N	NR		N	AAP	
498	3330	M	16	M	19	N								
499	3330	M	16	M	31	Y	M,D	P	P	NR		N	AAA	
500	3330	F	17	M	19	Y	M,D	P	N	NR		N	SAP	NV
501	3330	F	9	M	19	N								
502	3330	M	17	M	31	Y	M,D	P	P	NR		N	SAP	
503	3310	M	12	N	9	N								
504	3330	F	18	M	19	Y	M,D	P	N	P		SIP	SAP	V
505	3330	M	14	M	19	Y	M,D	P	N	NR	80/80	N	SAP	NV
506	3330	F	17	M	3	N								
507	3330	F	18	M	3	N								
508	3330	F	13	M	19	Y	D	N	N	NR	80/80	N	AAP	NV
509	3330	F	14	M	14	Y	P	N		NR		N	SAP	
510	3310	M	14	M	8	N								
511	3330	M	8	M	19	N								
512	3330	F	16	M	3	N								
513	3310	F	11	M	8	Y	S	P	P	NR		N	SAP	NV
514	3330	F	17	M	3	N								
515	3330	F	17	M	30	N								
516	3310	F	10	M	8	N								
517	3330	F	13	P	14	N								
518	3330	M	11	M	30	N								
519	3330	F	17	M	3	N								
520	3330	F	12	M	30	N								
521	3330	M	15	M	19	Y	M,D	N	P	H	70/80	PI	SAP	V,NV
522	3330	F	17	M	30	N								
523	3330	M	15	M	19	Y	M,D	P	N	NR		N	SAP	NV
524	3320	F	18	M	5	N								

ID	IC	Sex	Age	Ins.	T#	PARL	Root	Palp-ation	Percus-sion	Cold testing	EPT	Pulpal diagnosis	Periapical diagnosis	Vital
525	3320	M	16	M	4	N								
526	3330	M	13	M	30	N								
527	3330	M	18	M	19	Y	M,D	P	N	P		SIP	SAP	V
528	3310	F	16	M	6	N								
529	3330	M	18	P	19	N								
530	3330	F	17	N	19	N								
531	3310	M	15	M	9	Y	S	N	N	NR	80/80	N	AAP	NV
532	3330	F	16	M	14	Y	M,D,P	N	N			PI	AAP	
533	3330	M	16	M	3	Y	M,B	P	N	P		SIP	SAP	V
534	3330	M	17	M	3	N								
535	3310	F	9	M	8	Y	S	N	N	H	60/80	N	AAP	NV
536	3310	F	18	N	8	Y	S	P	P	NR	80/80	N	SAP	NV
537	3330	F	16	M	19	Y	M	P	N	P		SIP	SAP	
538	3330	F	14	M	3	N								
539	3330	M	15	M	14	N								
540	3330	F	14	M	30	N								
541	3310	F	16	M	8	Y	S	P	N	NR		PI	SAP	NV
542	3330	F	14	M	14	N								
543	3310	M	17	M	9	N								
544	3310	M	13	M	8	N								
545	3310	F	11	M	8	N								
546	3330	F	17	M	19	N								
547	3330	F	14	M	30	N								
548	3330	F	13	M	19	N								
549	3330	M	17	M	31	Y	D	N	N	NR	80/80	N	AAP	V
550	3330	F	12	M	30	N								
551	3310	M	14	N	24	Y	S	P	P	NR	80/80	N	SAP	

Abbreviations Sex: F=female, M=male

Insurance (Ins.): M=Medicaid, P=private, N=no insurance

Tooth number (T#)

PARL: N=no, Y=yes

Palpation, Percussion: N=negative, P=positive

Cold testing: NR=no response, H=hyperresponsive, P=prolonged, N=normal

Pulpal diagnosis: IP = irreversible pulpitis, N = necrotic pulp, NP = normal pulp, PI = previously initiated, RP = reversible pulpitis, SIP = symptomatic irreversible pulpitis

Periapical diagnosis: AAA = acute apical abscess, AAP = asymptomatic apical periodontitis, CAA = chronic apical abscess, NA = normal apical tissue, SAP = symptomatic apical periodontitis

Vital: NV=not vital, V=vital, V,NV=both

Vita

Dr. Erika Sloane Lentini was born on May 20, 1984, in Jefferson Parrish, Louisiana. She graduated Valedictorian from Sandy Creek High School, Tyrone, Georgia in 2002. She received her Bachelor of Sciences in Biology from University of Georgia, Athens, Georgia in 2006. She then obtained her Degree of Doctor of Dental Medicine from Medical College of Georgia in 2011. Dr. Lentini is a member of OKU and AAPD and will enter private practice in Richmond, Virginia. She will graduate from Virginia Commonwealth University with a Master of Science in Dentistry and a Certificate in Pediatric Dentistry.