The Influence of Full Coverage Restorations On Pulp Vitality: A Ten-Year Retrospective Study

Abby Yavorek
Marquette University

THE INCIDENCE OF ROOT CANAL THERAPY AFTER FULL-COVERAGE RESTORATIONS: A TEN-YEAR RETROSPECTIVE STUDY

by

Yavorek, Abby DMD

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ABSTRACT
THE INCIDENCE OF ROOT CANAL THERAPY AFTER FULL-COVERAGE RESTORATIONS: A TEN-YEAR RETROSPECTIVE STUDY

Yavorek, Abby DMD

Marquette University, 2019

INTRODUCTION: The incidence of pulpal disease after delivery of a full-coverage crown has been previously described as between 2-19%. The objective of this study was to identify and analyze the factors that contribute to the incidence of NS-RCT after the delivery of single-unit full-coverage restorations.

MATERIALS AND METHODS: Insurance claims from 88,409 crown placements in the Delta Dental of Wisconsin Insurance database were analyzed from the years 2008 to 2017. The Cox Regression model was used to analyze the effect of the predictor variables on the survival of the tooth. Untoward events were defined as NS-RCT, tooth extraction, retreatment of root canal, or apicoectomy as defined by the Code on Dental Procedures and Nomenclature.

RESULTS: Out of 88,409 crowns placed, 8.86% were all-metal, 41.40% were all-porcelain and 49.64% were metal and porcelain. The majority of all untoward events for all groups consisted of non-surgical root canal therapy. The probability of survival of all teeth with crowns placed was 90.41% after 9 years. Porcelain fused to metal (PFM) crowns exhibited a higher rate of untoward events than all-metal crowns, and lower rate than all-ceramic crowns. Crowns placed on individuals between 50 years of age and younger had higher rates of untoward events than those placed on individuals ages 51 years and above.

CONCLUSION: The risk of endodontic treatment after the placement of crowns is low. This risk increases with the placement of all-porcelain or metal-and-porcelain crowns and as age decreases

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INTRODUCTION

For centuries, full-coverage restorations have been used to support and protect teeth after extensive caries removal, root canal therapy, or cracks. Crowns have become routine procedures for dental practitioners and, recently, much research and development has been directed towards making these procedures more convenient, faster, and more predictable.

However, the procedure to prepare a tooth for a crown and subsequently restore it leaves many opportunities for pulpal irritation. Crown preparations open dentinal tubules to the oral environment, opening a pathway for microbes to enter the pulp chamber. This problem is exacerbated by ill-fitting provisional crowns, which can expose tubules for days to weeks until the final crown has been synthesized. Excessive heat generated from high-speed handpieces can inflict irreversible damage to pulp tissue. Other irritants, such as cements, can impact pulpal health as well.

Teeth presenting with symptomatic irreversible pulpitis during the provisional period or after crown placement are not uncommon. While past studies have demonstrated that crowned teeth have a probability of 8-15.6% of pulpal pathology after 10 years, these studies were limited by small sample sizes and poor follow-up (1, 2). Another study found that younger age and greater amount of coronal tooth destruction were significant predictors of RCT after crown placement(3). However, there have been no other studies corroborating these results or conducting further analysis into other predictors of pulpal pathology after crown placement.

In order to further evaluate the likelihood of root canal therapy after crown placement and related predictors, an insurance database study was completed. This type of study provides a real-word evaluation of treatment being rendered in a private practice environment, and supplies a large population to garner meaningful results. Delta Dental of Wisconsin provided electronic insurance claims record and enrollment data encompassing a ten-year period from 2008 to 2017. These claims were analyzed to predict survival rates for individual teeth after receiving full-coverage restorations. Variables such as patient age, tooth location, and crown material were examined to define predictors for adverse events.

LITERATURE REVIEW

The Pulp-Dentin Complex

The human tooth is a complex structure consisting of four types of tissue – enamel, dentin, cementum, and pulp. Enamel is the hardest and most outer part of the tooth and functions to support and protect the tooth from external forces. Dentin lies under enamel and has a higher water content, making it less calcified than enamel. It acts to support enamel's function. Cementum covers the outer surface of the tooth root and provides attachment for collagen fibrils to anchor the tooth to the periodontium. Dental pulp exists within the center of the tooth and comprises the vital structures of dentition. Together, dentin and pulp form a functional unit, which affects the tooth's development, innervation, metabolism, and immune response (4).

Dental Pulp:

Dental pulp is a mass of mesodermal tissue that vascular in nature. It consists of odontoblasts, fibroblasts, mesenchymal cells, immune cells, and connective and neural tissues. This specialized tissue has three main functions. It is the location of nerve endings, allowing sensory information to be passed from the tooth to the central nervous system. It is responsible for the deposition of dentin during the lifespan of a tooth. Lastly, the dental pulp supplies the necessary vasculature needed to support all tooth functions and provide immune mediators(5)

Odontoblasts are cells located in the outer periphery of the pulp chamber. The odontoblast is a fully differentiated cell with no division potential -- its lifespan and the lifespan of the

dental pulp will coincide. Odontoblasts have long processes that extend into dentinal tubules which help in depositing peritubular dentin Odontoblastic processes are living appendages of the odontoblastic cells, and serve as a direct communication to the dental pulp. Any time a dentinal tubule is exposed, there is risk of pulpal contamination(6).

The depth at which odontoblastic processes penetrate into the dentinal tubules is controversial. Scanning electron microscopy (SEM) studies have shown odontoblastic processes limited to the inner third of dentin. However, these results may be influenced by shrinkage during fixation and dehydration (7, 8). Other studies have theorized the odontoblastic processes penetrate much further into dentin, and, in some cases, all the way to the dentino-enamel junction (DEJ)(9, 10). Injury to the odontoblastic process can have an adverse impact on the health of the dental pulp.

Dentin:

Dentin comprises the layer of the tooth directly inferior to enamel. By weight, it consists of 70% mineral phase, 20% organic matrix, and 10% water. This makes dentin less calcified than enamel, but more calcified than human bone. It acts to support enamel, protect the constituents of the dental pulp, and house odondotblastic processes(11).

Dentin can be classified into three types. Primary dentin is tubular dentin formed before eruption. Secondary dentin is circumferential dentin formed after tooth eruption. Secondary dentin contributes to a net increase in dentinal thickness over a lifetime(12).Tertiary dentin is irregular and formed in response to stress on the tooth such as trauma, caries, and restorative materials(13). If a carious lesion approximates the pulp space, or when dentinal tubules are exposed via fracture or wear, the pulp will deposit a

layer of tertiary dentin over the tubules of the primary or secondary dentin. This mechanism allows the pulp to retreat below an improved protective barrier when threats to pulpal vitality occur(14). Recent studies have concluded that this barrier is not impermeable, and that many new tubules still communicate with primary dentinal tubules to allow access from the cemento-enamel junction (CEJ) to the pulp(15).

Dentin has variable permeability dependent on location within the tooth, age of the individual, and vitality of the pulp. Tubular dentin diameter ranges from 0.7 μm at the DEJ to 3 μm at the pulp(7, 16). Dentinal tubules in teeth with vital pulps, however, will have 5-10% less functional diameter, due to the presence of odontoblastic processes(17). Tubule density also increases closer to the pulp. Pashley estimates that the total surface area occupied by tubule lamina at the DEJ is 1%, while at the pulp interface it reaches 22%(16). As a tooth ages, peritubular dentin thickens, decreasing the luminal diameter of tubules, and therefore, their permeability. Dentinal permeability can also decrease due to insult from caries, which causes a demineralization, followed by a remineralization of the surrounding dentin. This process produces caries crystals within tubules which can increase the density of dentin below the affected area(5, 6).

(18).

Pulpal Response to Injuries:

Since dental pulp is solely responsible for secondary and tertiary dentin formation, the tooth loses its capability to regenerate new tissue and fight foreign antigens without vital pulp tissue. Secondary and tertiary dentin formation causes a reduction in the pulp chamber volume and pulp mass as teeth age. However, age also brings a decrease in the

density of pulp cells, including odontoblasts. Reduced vascularity is another sequelae of aging, and limits the tooth's immune response(5). These changes challenge the pulp's ability to heal after insult.

The low-compliance environment of the pulp chamber and its significance on inflammation and healing has been controversial. Previous studies supported a "self-strangulation theory" to describe the pulp's hindered ability to recover after injury. The theory stated that increased pressure from inflammation of the dental pulp had limited ability to spread and diffuse, as the walls of dentin surrounding the pulp chamber were rigid. Increased pressure of the pulp chamber would ultimately impinge upon the vasculature entering the tooth from the apical foramen, leading to a depletion of nutrients and other vascular constituents needed for the pulp to survive(19).

"Self-strangulation" theory has been disregarded in recent years due to an influx of literature supporting the pulp's ability to self-regulate rising pressure. Van Hassel found that mean intrapulpal pressure was approximately 25mmHg, and could reach 40mmHg during inflammation. After this rise, however, intrapulpal pressure gradually decreased back to 25mmHg(20). In a review article years later, Tonder elucidated mechanisms which counteract "self-strangulation", including capillary filtration and lymphatic drainage (21). These studies support the notion that pulpal death occurs from a circumferential spread of a localized area of necrosis at an initial site of injury.

Apical Periodontitis and Pulpal Pathology

Apical periodontitis describes inflammation of the periodontium in the apical portion of the a tooth's root(22). It is a disease most commonly caused by bacterial infiltration through the pulp space of a tooth, which incites inflammatory changes in the surrounding periodontal tissues. Kakehashi found that this periradicular inflammation occurred in every case after a rat's tooth with pulp necrosis was exposed to oral microorganisms, and in no cases when necrosed teeth were germ-free(23). After numerous replications of this study in other organisms, it is now largely accepted that bacteria is the most important etiologic factor in the development of apical periodontitis(24, 25).

If left untreated, apical periodontitis can lead to pain, swelling, loss of tooth, or bacteremias. The findings of Kakehashi and others aided Schilder to develop new mechanical and pharmacological techniques to clean and debride the root canal system(26). These techniques allowed predictable success after root canal therapy and are still being used today. Without the contributions of Kakehashi, Schilder, and others, extraction would be the only feasible treatment option for a tooth inflicted with apical periodontitis.

Pulp necrosis is necessary for the development of apical periodontitis. However, the etiologic factors responsible for apical periodontitis may or may not have had a role in the development of pulpal necrosis. While caries can cause both pulpal necrosis and apical periodontitis, there are other irritants that can encourage pulpal inflammation and the subsequent necrosis that can leave the tooth more susceptible to periradicular inflammation. In addition to caries, trauma, heat, desiccation, and chemical irritation have also been implicated as etiologic factors of pulpal necrosis.

Caries remains the largest contributor to pulpal pathology. Odontoblasts, as the most peripheral cells in the pulp, act as a first line of defense. These cells initiate the innate immune response, leading to production of cytokines, chemokines, and peptides that act to recruit other immune effector cells and facilitate targeted responses towards bacterial cells(27). As bacterial organisms penetrate further into dentinal tubules, pulp tissue evokes a humoral immune response, releasing T-helper cells, B-cells, neutrophils and macrophages. This response is directly proportional to the depth of the carious lesion within the tooth(28). As the lesion grows, inflammatory mediators become more densely populated in the more coronal areas of the pulp, ultimately migrating into the tubules beside the odontoblastic processes(29). If caries progression is unhindered by restorative intervention, complete destruction of the pulp tissue will occur due to these immunopathologic mechanisms.

Pulp necrosis can develop after traumatic injuries, even when dentinal tubules are not exposed. If a trauma to a tooth is applied with enough force, a disruption of the vasculature can occur. This is followed by pulpal tissue infarct and coagulation necrosis. If no bacteria are present in the root system, a sterile necrosis can occur. However, this tooth will be at a greater risk of developing apical periodontitis in the future, as any exposure of the dentinal tubules will allow unimpeded access for bacterial to enter the pulpal space(30).

Heat, desiccation, and chemical injuries can contribute to pulpal pathology. Heat, often associated with high-speed handpieces, causes loss of odontoblasts and deposition of collagenous tissue into dentinal tubules. These events lead to subsequent degeneration of pulpal tissue and localized necrosis(31). Desiccation during dental procedures causes

displacement of odontoblastic nuclei, followed by pulpitis and necrosis(32). Hydrogen peroxide, sodium hypochlorite, calcium hydroxide, and other chemical components used in dentistry can exert toxic effects on the pulp if within a close proximity and may induced localized necrosis(33). If any of these precipitating factors are severe, chronic inflammation can occur within pulp tissue and total necrosis is seen.

Full-Coverage Crowns and Their Effect on Pulpal Health

A single-unit, full-coverage crown has been used as a predictable restorative procedure for decades. Crowns can be used to bolster teeth from further break down, provide support after a crack or fracture, or protect endodontically treated teeth from future fracture or bacterial leakage. Single-unit crowns have high success rates, averaging 95% success after 5 years(34).

Definitive single-crowns can be grouped into three main categories based on material: all-metal, porcelain-fused-to-metal (PFM), and all-ceramic. The all-ceramic group can be further broken down into all-zirconia, layered zirconia, lithium disilicate, and leucite-reinforced glass ceramic.

All-metal cast crowns have the longest history in dentistry and remain a gold-standard in fixed-prostheses. These crowns are used most often in posterior teeth for aesthetic reasons, but their usage has been decreasing in recent years with the advent of ceramic crowns(35). They are durable and can withstand occlusal forces in small thicknesses, leading to less reduction in tooth structure. Metal alloys used for crowns have similar wear properties to enamel, leading to less wear on opposing teeth(36).

PFM crowns have been used since the 1960's and provide an aesthetic answer to the limitation of all-metal crowns. These restorations feature a ceramic façade bonded to a metal undercasting. This combination creates a strong and supportive base, hidden behind a more natural appearing veneer. There is more tooth structure removal necessary for these restorations than for all-metal crowns, as ceramics necessitate a greater thickness of tooth reduction. This is vital to provide space for the thicker ceramic façade, in order to compensate for a high susceptibility to fracture(36).

All-ceramic crowns encompass numerous material systems, including all-zirconia, layered-zirconia, lithium disilicate, and leucite-reinforced glass ceramic. Therefore, there is a wide range of properties exhibited within the all-ceramic crown family. All-zirconia crowns exhibit high strength and toughness, and wear at similar rates to natural dentition, making them appropriate choices for posterior teeth(37-39). Layered zirconia, lithium disilicate, and leucite-reinforced glass ceramics appear more translucent than all-zirconia crowns, but are weaker and more susceptible to chipping, fractures, and failure(37, 40, 41). These crowns are largely used in anterior teeth with fewer occlusal forces. Like, PFM crowns, ceramic crowns require a greater bulk of material to compensate for decreased fracture toughness(38).

The preparation depths for crowns vary by material and provider. In all-metal crowns, 1.5mm reduction is necessary for functional cusps. 1.0mm reduction is adequate elsewhere, tapering to 0.5mm at the margin if a chamfer is desired. This depth increases for PFM and all-ceramic crowns, as ceramics need bulk for fracture resistance. For PFM crowns, any porcelain covered functional cusp, including anterior incisal edges, will need at least 2.0mm of reduction. In posterior teeth, this reduction can be minimized if metal

occlusal surfaces are used at the expense of aesthetics. Facial surfaces covered in porcelain need at least 1.3mm of reduction. All-ceramic crowns require 1.5-2.0mm of occlusal reduction, and 1.0mm of clearance elsewhere. A 1.0mm shoulder should be maintained for favorable stress distribution(42). A study by Edelhoff and Soresenson using gravimetric analysis to calculate relative structure removal for preparations using these materials showed that PFM crowns removed 8% more tooth structure than all-ceramic crowns and 20% greater than all-metal crowns(43).

Crown preparation undoubtedly affects pulpal health in vital teeth. Highspeed handpieces with speeds as low as 3-6000 rpm cause localized inflammation and changes in ododntoblast nuclei. These reactions are worsened when air is used as a coolant, or the area is desiccated further. Interestingly, inflammation can be prevented if water coolant is used appropriately and maintains contact between the surface of dentin and the rotating bur(44).

Zach and Cohen found that intrapulpal temperature rise of 5.5 ^{O}C resulted in irreversible pulpitis in 15% of cases. Temperature rise of 11 ^{O}C resulted in irreversible pulpitis in 65% of cases, and temperature rise of 16.6 ^{O}C resulted in irreversible pulpitis in 100% of cases(45). Using a bovine model, Cavalcanti found a temperature rise of 16.4 ^{O}C without cooling, and 11.68 ^{O}C with cooling. This could be lowered to 1.56 ^{O}C with a technique which alternated 2 seconds of drilling with 1 second of rest(46). Similar studies have corroborated these findings(47, 48).

Highspeed handpieces are not the only risks to healthy intrapulpal temperatures. Castelnuovo found that intrapulpal temperatures rose from 7.2 ^{O}C to 12.3 ^{O}C while using polymerizing resins to form provisional restorations on crown-prepped teeth. The

exothermic reactions from the polymerization of these materials after a period of 2-3 minutes of contact was able to generate enough heat to push pulpal temperature over the 5.5 ^{O}C threshold set by Zach and Cohen(49). Cooling techniques have since been suggested, including repeatedly removing the provisional crown and using air or water coolant as polymerization is taking place(50).

The threat of overheating the pulp is increased as dentin thickness decreases. Dentin is an efficient insulator, and will dissipate heat if an adequate amount of thickness remains. Therefore, trauma to the pulp tissue via heat is dependent on the proximity of the heat source to the pulp(31). Ettinger and Qian have postulated that leaving 2mm of dentinal thickness is sufficient to provide the pulp with protection from most restorative procedures(51).

Desiccation is often associated with heat, and may also be a contributing factor to pulpal inflammation during crown preparations. As little as 30 seconds of drying dentinal tubules in teeth with uninflammed pulps has been shown to cause significant aspiration of odontoblastic nuclei and pulp inflammation. Pulp necrosis was seen associated with the tubules that had been desiccated(32). Other studies have implicated desiccation as the cause of pulpal trauma inflicted by high temperatures, not heat itself(15).

Even in the absence of heat and desiccation, however, there is a greater risk for pulpal inflammation anytime a dentinal tubule is exposed. Crown preparations, specifically, expose more tubules than cavity preparation, expose tubules for a longer period of time over multiple appointments, and rely on imperfect provisional crown margins to prevent microleakage. Opening a tubule to the outside environment will always

bring the risk of potential contamination, as bacterial cells are 0.5 to 1µm in diameter and dentin tubules range from 0.6 µm near the DEJ to 3 µm near the pulp chamber(6, 18).

As crown preparations get closer to the pulp, the chance of bacterial contamination of the pulp chamber increases. In teeth that are vital, this risk is lowered due to a constant outward flow of dentinal fluid. This flow helps impede the access of noxious agents from the oral environment into dentinal tubules(52). Despite this mechanism, greater pulpal inflammation is still seen in crown-prepped teeth that had bacterially contaminated cavities, indicating that bacterial infiltration through dentin still occurs in vial pulps(53). In necrotic teeth, even if the necrosis is not bacterial in nature, exposing a dentinal tubule for a crown preparation leaves unimpeded access to the pulp chamber for oral microbes. These teeth inevitably become infected and ultimately develop to apical periodontitis in the future.

Resin-based materials such as self-adhesive resin cements used to lute crowns have been shown to have cytotoxic effects on specific cell types, such as fibroblasts. These cements release uncured, free monomers that can diffuse through dentinal tubules to the pulp (54). These uncured resin components have been shown to inflict localized pulpal inflammation when in close proximity to pulp tissues(55). Resin- based cements are most often used to lute all-ceramic crowns, but can used for other full-coverage restorations as well.

Microleakage has been defined as the movement of fluids carrying bacteria and other molecules at the boundary between a restoration and a tooth(56). When a crown is cemented, precise marginal fit and adequate cementation is essential to prevent microleakage and subsequent inflammation of the pulp. Temporary cements are used to

lute provisional crowns in place for the days to weeks before a definitive restoration is ready. Microleakage associated with temporary cements has been attributed to their weak mechanical properties and get worse over time and exposure to oral fluids(57). Possible mechanisms of failure in temporary cements include dissolution, lack of bond between cement and tooth structure, mechanical failure or shrinkage of cement upon setting(58). Baldissara found that all six temporary cements studied, including zinc phosphate, zinc oxide eugenol (ZOE), and non-eugenol cements, exhibited microleakage to some degree during the one week protocol(59). Arora corroborated these findings, showing polycarboxyate, ZOE, and non-eugenol cements all exhibited microleakage when cementing both acrylic and resin-based temporary crowns. Cements containing eugenol were found to have significantly more microleakage than non-eugenol cements(58). Farah found that glass ionomer and resin-based temporary cements also display microleakage after 24 hours(60). It is important to note that the chemical properties of non-resin cements do not seem to be irritating in nature. Polycarboxylate, zinc phosphate and ZOE cements have all been found to elicit little to no inflammation to nearby pulp tissues(61, 62).

Cementation of a definitive crown can also affect microleakage potential. Many practioners use temporary cements to lute definitive restorations in order to achieve adequate retreivability of restorations if they need to be removed. These cements yield the same poor mechanical properties used in conjunction with definitive crowns as previously mentioned with provisional crowns. Newer permanent cements such as glass ionomer, resin-modified glass ionomer (RGMI) and resin cements have stronger mechanical properties than their predecessors, and are more resistant to dissolution in oral fluids(63).

These cements display adhesive properties, improving the mechanical retention of the restoration and decreasing the risk of microleakage(64-66).

It has been largely accepted that the performance of cements and risk of microleakage under definitive restorations is affected by the marginal integrity of the restoration. Bergenholtz found that poorer marginal fit of a crown was directly proportional to the amount of inflammation in the associated pulp tissues(67). Poor crown margins can lead to an accumulation of plaque, increasing the risk for recurrent decay under a restoration(68). As anytime a dentinal tubule is exposed there is risk for pulpal contamination, inadequate margins can encourage the ingress of bacteria to otherwise healthy pulp tissue(6).

Marginal integrity will also affect cement stability. When less marginal adaptation is seen, more cement is exposed to oral fluids. The amount of cement exposed has been related to the amount of dissolution, which in turn will promote microleakage(69). Mondelli confirmed this using radioactive sodium iodide, finding the amount of microleakage under full-coverage crowns is directly influenced by marginal adaptation of the restoration and film thickness of the cement(70).

It is unclear whether marginal fit is affected by type of marginal preparation. Goldman found that there was no different in microleakage extent or pattern between crowns placed with chamfer, shoulder, or shoulder with bevel margin preparations(71). Shillingburg found that shoulder preparations produce less distortion and have better marginal integrity than chamfer finish lines(72). Gavelis, however, found that featheredge and parallel bevel preparations demonstrated superior marginal seal over shoulder preparations(73).

The linear coefficient of thermal expansion of crown material has been identified as an important factor contributing to microleakage. As materials are subjected to extremes in temperatures, such as crowns are when submerged in an iced drink and returning to the temperature of the oral cavity seconds later, they expand and contract accordingly. Overtime, these expansions and contractions can contribute to microfractures at the restoration-cement interface(74). A meta-analysis reviewing 16 thermocycling experiments on human teeth with cast base metal, cast gold, and ceramic crowns reported no significant difference between materials with respect to microleakage. However, statistical analysis was hindered by lack of standardization between experimental protocols(75).

Marginal integrity at the time of cementation is more dependent on the technique used to create crowns, rather than the material of the crown margin itself. Base metal alloys created with the lost wax technique have been shown to have superior internal fit and marginal adaptation when compared to milled all-ceramic crowns in SEM investigations. However, once porcelain veneering is preformed, this improved internal adaptation is lost(76). Laser sintering of alloys is a new technique that enables better marginal fit of cast metal copings when compared to milled, milled wax, or lost wax techniques(77).

Currently, all-ceramic crowns can be impressed and milled chairside, bypassing the use of any dental laboratory to produce crowns. Studies are inconclusive as to whether this new technology yields similar marginal fits to those crowns synthesized in a dental laboratory(78-82). While chairside impression and milling systems do fall in a clinically

acceptable range for marginal integrity, accuracy is most likely dependent on the practitioner and preparation smoothness(81-83).

Incidence of Endodontic Therapy After Crown Placement

There are many factors that contribute to the development of pulpitis or apical periodontitis in a tooth needing a coronal restoration. Insults to the pulp, whether it be from caries, restorative treatment, or trauma, are cumulative. Each time an insult occurs, the pulp has a lowered capacity to recover and remain vital. Removing dentin for a crown preparation, curing materials needed for provisionalization, and microleakage under a temporary crown can all contribute to possible preexisting pulpitis(84).

The current literature available about incidence and probability of root canals after full-coverage crown placement are sparse. Many studies have been performed in dental schools or similar teaching institutions where practitioners and oversight from faculty is inconsistent. Other prospective studies fall victim to poor follow-up, diminishing the power needed to analyze risk factors.

In a case-control study at the University of North Carolina School of Dentistry, 3265 patients with single crowns placed within a four-year period were followed to see if root canal therapy (RCT) was preformed 2-6 years after crown delivery. 92 patients returned for RCT on the teeth which had crowns placed. Multivariable logistic regression analysis was performed to identify predictive factors of the case status. Younger age and greater extent of destruction of coronal and root structure were found to be significant predictors of RCT after single-crown placement(85).

In a similar prospective study of PFM crowns or bridges delivered over an 8-year period in a hospital in Hong Kong, Cheung followed 284 single-unit PFM crowns. Patients were recalled, where a clinical exam, endodontic testing, and radiographs of the tooth in question were performed. They found that 15.9% of single crowns had endodontic involvement at the time of recall. The range of time between restoration placement and root canal ranged from 6 months to 14 years after crown delivery. Survival probabilities for single-unit PFM crowns were estimated to be 84.4% after 10 years and 81.2% after 15 years(1).

In another study by the same author and in the same hospital, 34 all-ceramic, 32 cast gold metal and 48 PFM crowns were followed up after delivery. The average time from delivery to follow-up was 40.1 months, 31.4 months, and 33.4 months for ceramic, metal, and PFM crowns, respectively. Only one tooth in each the all-ceramic and cast metal groups, and no teeth in the PFM group became endodontically involved(86).

Valderhaug examined radiographic changes in over 291 vital single-crown or bridge abutment teeth over a time period of 25 years. Radiographs were taken post-operatively and after every 5th year during a follow-up examination. After 25 years, 28% of the patients remained in the study, and 13 vital teeth had developed apical periodontitis. Estimated percentage of crowned vital teeth that would remain free of periapical pathology were 92% after 10 years, 87% after 20 years and 83% after 25 years. Crown material was not reported in this study(2).

In a study of 603 teeth recalled after delivery of single crown or fixed partial denture delivery, Jackson found that of the 437 crowns that were vital before restoration placement, 25 (5.7%) had or needed RCT after cementation of the prosthesis. There was

no significant difference found between single crowns and fixed prosthesis abutments, or between anterior and posterior teeth(87).

In a large systematic review analyzing numerous clinical complications associated with fixed prosthodontic procedures, Goodacre found five studies reporting incidence of endodontic treatment on teeth with single crowns. The mean incidence for single crowns excluding all-ceramic crowns was 3%, while a 1% incidence was found between twelve studies evaluating outcomes of exclusively all-ceramic crowns(3).

MATERIALS AND METHODS

The data for this study was obtained from the electronic insurance enrollment and claims database for Delta Dental of Wisconsin. The database contained claims data representing 13,329,249 patient encounters between January 1, 2008 and December 31, 2017. From the dataset, 88,409 patients who underwent full-coverage crown deliveries were identified based on the Code on Dental Procedures and Nomenclature (CDT) codes for full-coverage crown delivery. CDT codes are used to properly and uniformly document dental treatment procedures in patients' health records , and to process insurance claims.

Type of Crown	*CDT Code*
All-ceramic	D2740
Porcelain-fused-to-metal (PFM)	D2750 D2751 D2752
Cast metal	D2790 D2791 D2792

TABLE 1: INITIATING EVENT CDT CODES

All-ceramic crown (D2740), porcelain fused to metal crown (PFM) (D2750, D2751, and D2752), and cast metal crown (D2790, D2791, and D2792) codes were identified as initiating events (Table 1). Teeth that had a root canal treatment (RCT)

performed before crown placement were excluded from the analysis. Teeth that received crowns within the first 12 months of the time period studied were also excluded to eliminate crown deliveries performed as they may have been a consequence of a previous RCT. Untoward events were defined as having initial root canal therapy (D3310, D3320, D3330), extraction (D7140, D7210), endodontic retreatment (D3346, D3347, D3348), or apicoectomy (D3410, D3421, D3425) as defined by CDT codes. Treatments were determined successful until an untoward event or a lapse in the patient's enrollment status occurred.

Untoward Event	*CDT Code*
Root canal therapy (RCT)	D3310 D3320 D3330
Extraction	D7140 D7210
Endodontic Retreatment	D3346 D3347 D3348
Apicoectomy	D3410 D3421 D3425

TABLE 2: UNTOWARD EVENT CDT CODES

For each encounter, information was collected regarding crown material, age of patient, location of tooth, and type of provider placing crown. Crown material was divided

into groups of all-ceramic crowns, PFM crowns and cast metal crowns as determined by CDT code. Patients were divided into 6 groups based on their age: Under 30 years, 31 to 40 years, 41 to 50 years, 51 to 60 years, 61 to 70 years, and 71 and above years. Location of tooth consisted of groups of anterior teeth, premolar teeth, and molar teeth. Provider type included groups of general dentist, prosthodontists, and "other" providers as defined by the Delta Dental database. The category of "other" consisted of all the providers not categorized as prosthodontists or general dentists.

Data was analyzed using SAS 9.4 software (SAS Institute Inc., Cary, NC). Survival time was taken from time of crown placement to the time of an untoward event. The effect of predictors on tooth survival was analyzed. Hazard ratios were calculated using a univariate Cox proportional hazards regression model. The variable of tooth location did not satisfy the proportional hazard assumption in the model, and therefore, analysis was stratified on tooth location. Kaplan Meier curves were plotted for each variable, and log-rank tests were performed to identify differences of Kaplan Meier curves in each variable group. A significance level (alpha) of $p < 0.05$ was used throughout all analyses.

RESULTS

After the exclusion criteria were applied to the dataset, 88,409 teeth with crowns placed were identified. The vast majority of crowns placed were all-ceramic (41.50%) or PFM (49.64%). All-metal crowns only represented 8.86% of all crowns placed. General dentists placed almost all of the crowns in the data set, with prosthodontists and "other" providers constituting the remaining 1.23% of crown providers. Over half of patients receiving crowns were between the ages of 51 and 70 years of age. Molar teeth represented 75.39% of the teeth being crowned, followed by premolars (20.52%) and anterior teeth (4.09%)(Table 3).

	All (n=88409)	
Variable	*N*	*%*
Crown Material		
All-metal	7834	8.86
PFM	36692	41.5
All-ceramic	43883	49.64
Provider Type		
General Dentist	87318	98.77
Prosthodontist	745	0.84
Other	346	0.39
Age Group		
Under 30 years	2472	2.8
31 to 40 years	7269	8.22
41 to 50 years	15862	17.94
51 to 60 years	30317	34.29
61 to 70 years	27377	30.97
71 years and above	5108	5.78
Tooth Location		
Anterior	3617	4.09
Premolar	18144	20.52
Molar	66648	75.39

TABLE 3: DESCRIPTIVE SUMMARY OF INITIATING EVENTS BASED ON VARIABLE

Of the 88,409 teeth that were crowned, 4.82% of teeth underwent an untoward event during the time period studied. Out of these 4,259 teeth that underwent an untoward event, 72.41% of events were primary root canal therapy. Apicoectomies and non-surgical root canal retreatments only constituted 5.40% of all untoward events, and extractions accounted for the remaining 22.19% of events. The complete summary of untoward events with respect to crown material and patient age can be seen in Table 4.

	Outcome			
Variables	*Total N=4259*	*Extraction N=945*	*Root Canal N=3084*	*Retreatment N=230*
Crown Material				
All Metal	326 (100.0)	104 (32.7)	212 (66.7)	10 (3.1)
All porcelain	1505 (100.0)	267 (18.4)	1166 (80.3)	72 (4.8)
Metal & Porcelain	2428 (100.0)	574 (24.7)	1706 (73.5)	148 (6.1)
Age Group				
Under 30 yrs	112 (100.0)	15 (15.0)	82 (82.0)	15 (13.4)
31 to 40 yrs	427 (100.0)	67 (16.6)	326 (80.7)	34 (8.0)
41 to 50 yrs	941 (100.0)	155 (17.4)	721 (80.7)	65 (6.9)
51 to 60 yrs	1442 (100.0)	301 (21.7)	1070 (77.1)	71 (4.9)
61 to 70 yrs	1102 (100.0)	310 (28.9)	755 (70.3)	37 (3.4)
71 yrs or above	235 (100.0)	97 (41.8)	130 (56.0)	8 (3.4)

TABLE 4: DESCRIPTIVE SUMMARY OF UNTOWARD EVENTS BASED ON CROWN MATERIAL AND AGE OF PATIENT

The Cox Regression model results compared survival times of teeth based on the variables studied. Larger hazard ratios equate to a greater likelihood of an untoward event in one variable compared to another variable (Table 4). Tooth location did not satisfy the

proportional hazard assumption in the regression model and therefore, analysis was stratified with respect to tooth location.

Metal crowns have a hazard radio of 0.73 when compared to PFM crowns, indicating that they 27% lower hazard rate ($p<0.0001$). All-ceramic crowns have a hazard ratio of 1.09 when compared with PFM crowns ($p<0.01$). While there was no significant difference between the survival rates of teeth treated by prosthodontists compared to those treated by general dentists, there was a significant difference between all other providers and general dentists ($p<0.05$). When analyzing patient age, the interval from 51 to 60 years of age was used as a reference group. Groups 30 years and under, 31 to 40 years, and 41 to 50 years of age all had significantly higher hazard rates than the group from ages 51-61. The group consisting of patients aged 61 to 70 had significantly lower hazard rate than the group aged 51 to 60 years. There was no significant difference in hazard rates when comparing the age groups 71 and above years to 51 to 60 years (Table 5).

Hazard Ratio and 95% Confidence Interval				
Variable	*Hazard Ratio*	*95% Confidence Interval*		*P-value*
Crown				
All-Metal vs PFM	0.73	0.65	0.82	<0.0001
All-Ceramic vs PFM	1.09	1.03	1.17	0.0081
Provider				
Prosthodontist vs General Dentist	1.10	0.80	1.52	0.5525
Other vs General Dentist	1.55	1.07	2.23	0.0198
Age Group (in years)				
30 and younger vs 51-60	1.38	1.14	1.67	0.0014
31-40 vs 51-60	1.45	1.30	1.61	<0.0001
41-50 vs 51-60	1.30	1.19	1.41	<0.0001
61-70 vs 51-60	0.89	0.83	0.97	0.0049
71 and above vs 50-60	0.94	0.82	1.08	0.3251

TABLE 5: HAZARD RATIOS AND CONFIDENCE INTERVALS FOR VARIABLES

The estimated survival of all crowns drops from 97.3% after one year, to 90.4% after 10 years. The overall probability of survival for all teeth with crowns can be seen in Table 6.

Time (years)	*Number at risk*	*Observed Events*	*Survival Probability*
1	62989	17	0.9727
2	48300	5	0.9595
3	36587	0	0.9492
5	20379	1.00	0.9322
9	4087.00	0.00	0.9041

Table 6: Overall estimates for survival of teeth with crowns

The following plots demonstrate the cumulative incidence of the first untoward event occurring over time following placement of a single-unit crown (Figure 1). Plots were created to display survival rates between variables, including crown material, tooth location, provider type, and age of patient (Figures 2, 3, 4, 5). Significant differences were seen in survival rates with respect to age groups, tooth location, and crown material ($p<0.0001$).

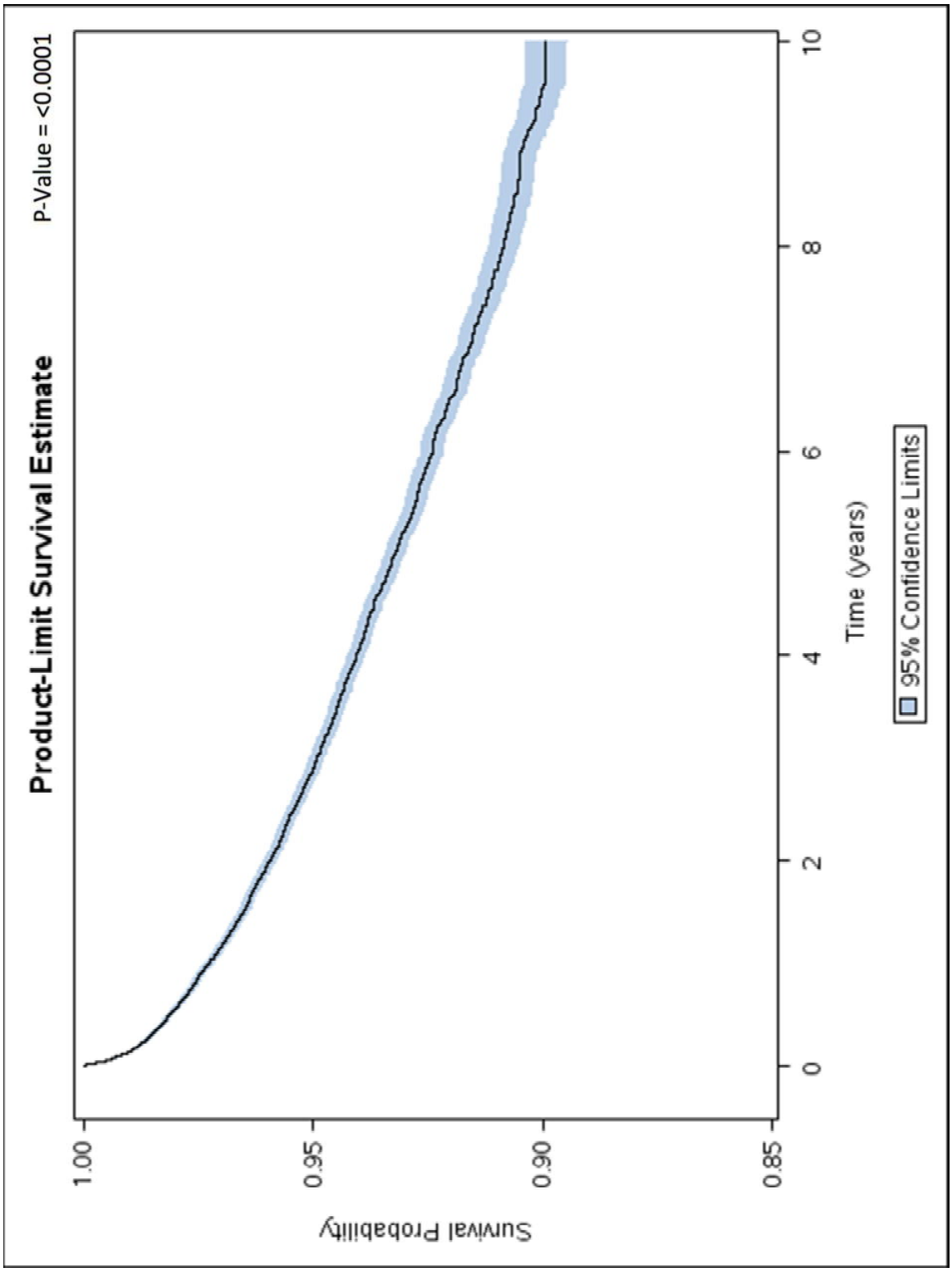

Figure 1: Survival estimates of all teeth after crown placement

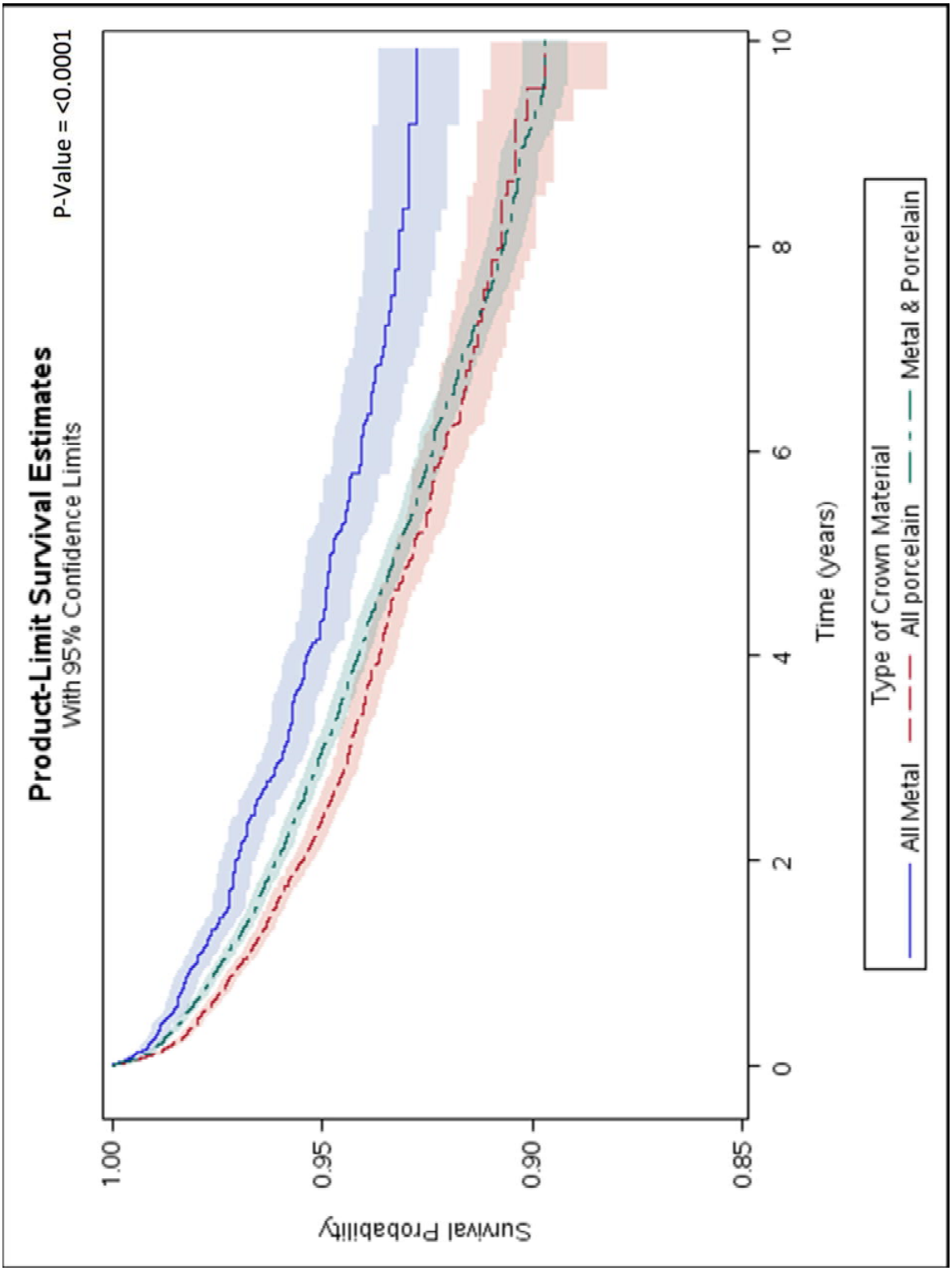

Figure 2: Survival estimates of all teeth after crown placement based on crown material

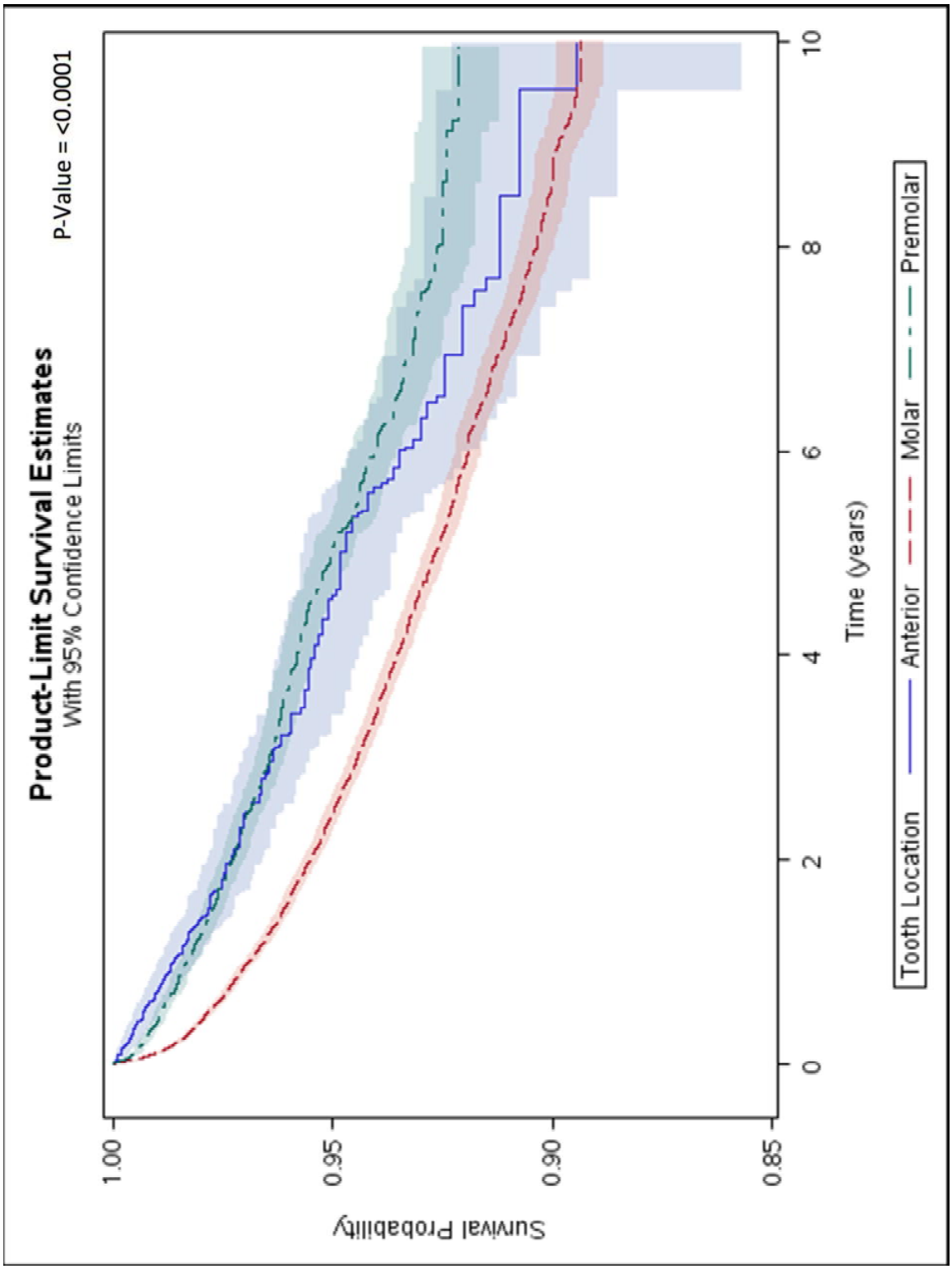

Figure 3: Survival estimates of all teeth after crown placement based on tooth location

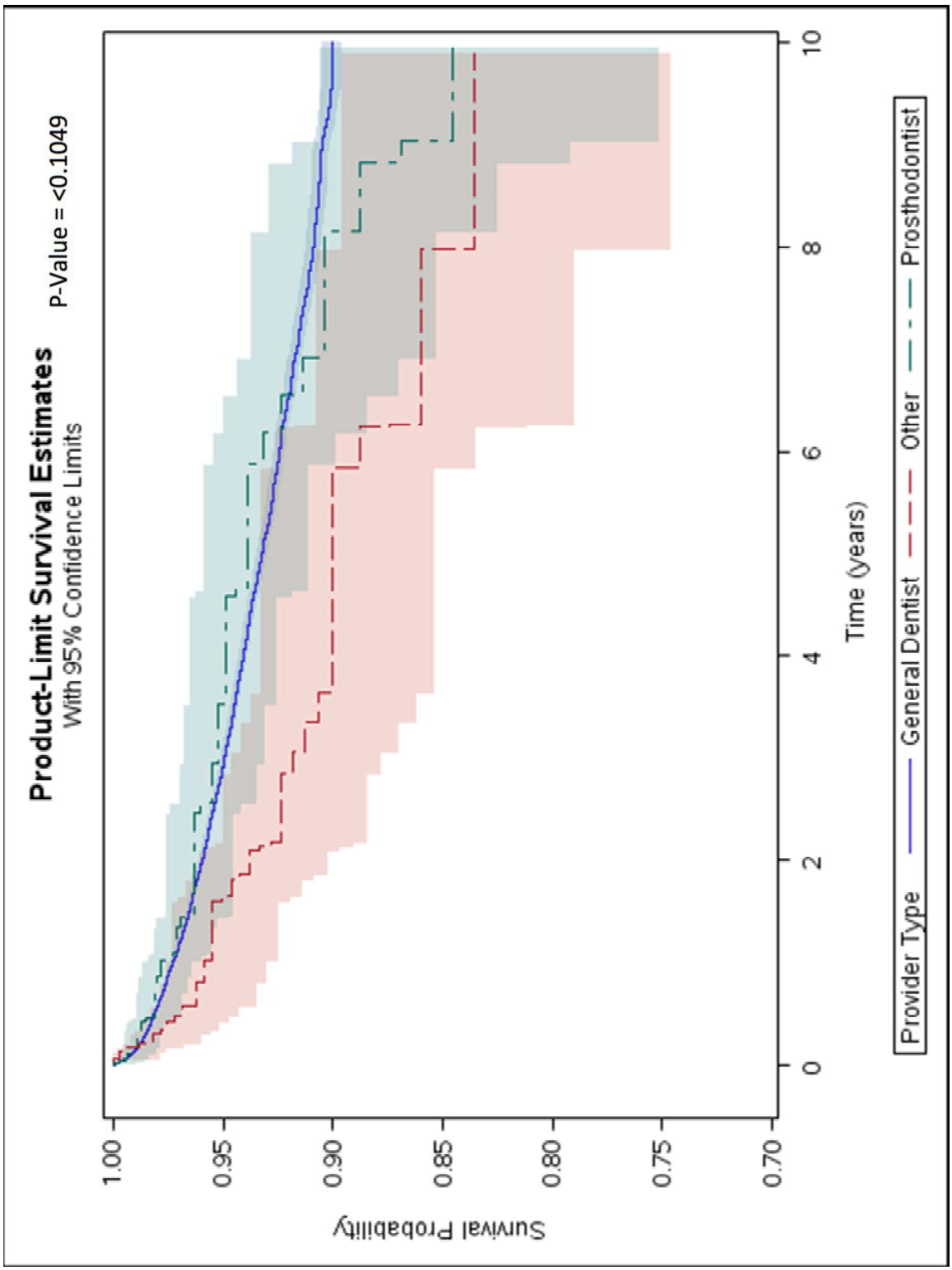

Figure 4: Survival estimates of all teeth after crown placement based on provider type

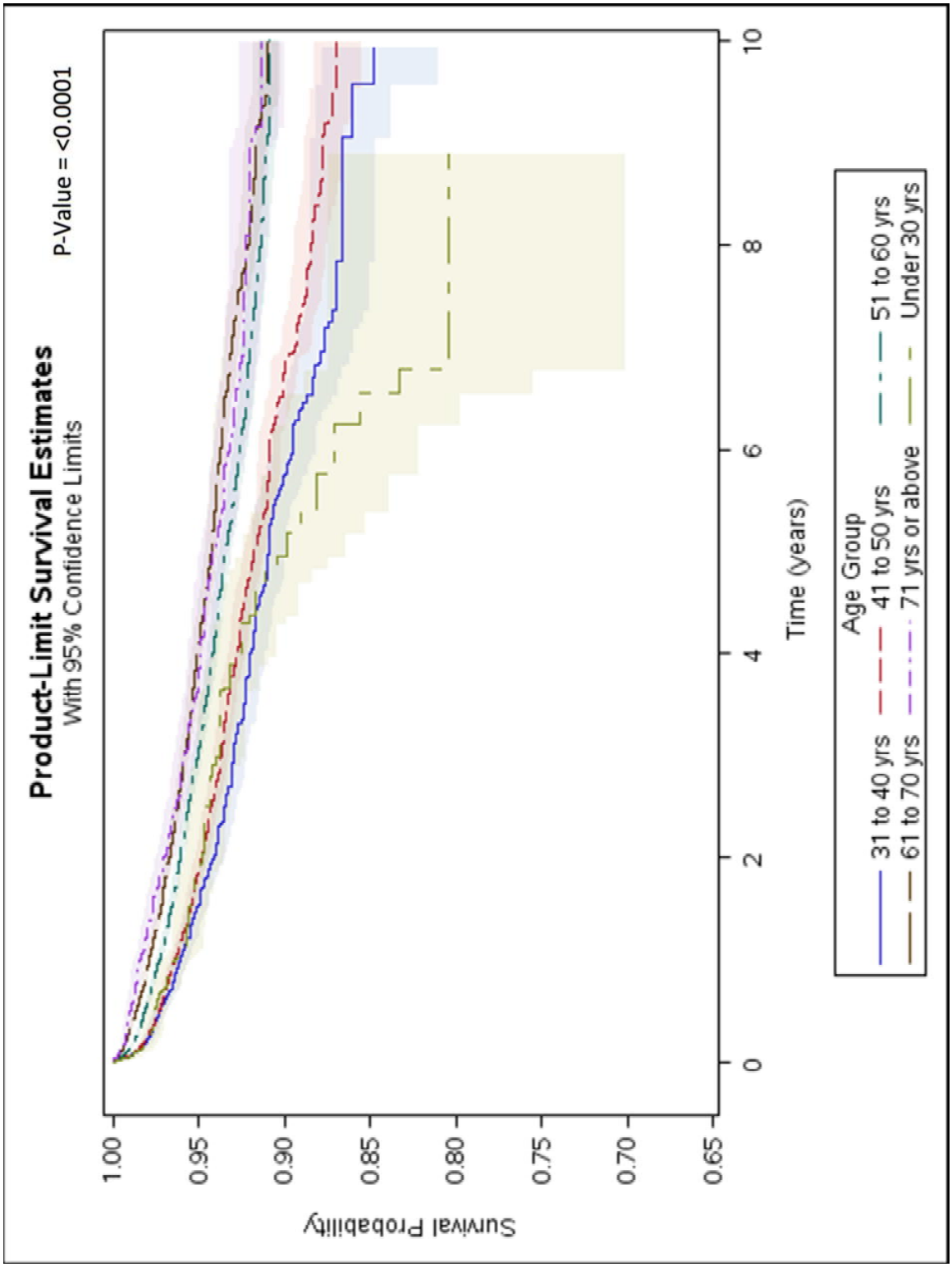

Figure 5: Survival estimates of all teeth after crown placement based on patient age

DISCUSSION

The primary objective of this study was to identify variables affecting the likelihood of endodontic intervention after placement of a full-coverage, single-unit restoration. By utilizing the Delta Dental of Wisconsin insurance database, a substantial number of records were available for analysis, contributing power and meaning to the results.

In order to more closely approximate a population of teeth that had intact pulpal tissues before the placement of a crown, two exclusion criteria were applied to our data. The first was to eliminate any tooth in which endodontic therapy had been performed before placement of the crown. This was only effective for teeth that had the procedure completed during the ten-year span of our insurance coverage. Teeth with crowns placed within the first year of the coverage period were also excluded. This aimed to eliminate teeth that had recently underwent endodontic therapy, and had crowns placed as a result.

Even with these exclusion criteria, some endodontically treated teeth were inevitably included in the study. This can be confirmed by the few teeth that had untoward events consisting of endodontic retreatments and surgeries (Table 3). In time-to-event analyses, such as a survival analysis, decisions cannot be made based on future events, and therefore, these data points must remain in the data set in order for statistical analysis to be accurate. Removing these events would result in a biased population of teeth undergoing untoward events. However, retaining these data points allows calculation and examination of their impact on the overall population. Fortunately, only 4.50% of all untoward events consisted of treatments that would suggest root canal therapy before crown placement,

indicating that the inclusion of this group is unlikely to have a major impact on the overall results.

Similarly, extractions were included as untoward events due to this same principle, as well as the unavailability of clinical information that may provide insight as to the prognosis of a tooth. Unfortunately, there are many justifications for tooth extraction, and there is no way to distinguish teeth that are extracted due to pulpal pathology from teeth that are extracted for other reasons with the given insurance information. Standardization of providers and understanding rationale for treatment is also impossible with a retrospective insurance-based study. While one provider may perform NS-RCT on a tooth with pulpal pathology, another may extract the same tooth in favor of an implant. Even with the inclusion of extractions and endodontic retreatments as untoward events, the overwhelming majority (72.41%) of all untoward events consisted of initial NS-RCT.

There are certain limitations of using insurance data-based studies. Patient diversity is limited, as only patients living in Wisconsin, who have Delta Dental of Wisconsin insurance were included. Individuals with private dental insurance may potentially present with contrasting outcomes compared to uninsured individuals due to differences in access to care and patient expectation. Correspondingly, providers of crowns were limited to only those who are contracted under the Delta Dental network.

This study also depends on the accuracy of coded procedures, as erroneous codes would lead to a misrepresentation in data and inaccurate results. Other factors that may impact pulpal health were not available for analysis, such as remaining dentin thickness, previous restorations, type of handpiece used, amount of water spray utilized, marginal finish line design, and pulpal status before crown preparation. Thus, it is impossible to

control for these variables. Coding for NS-RCT is the only way we are able to confirm the presence of pulpal disease within our dataset. This creates another limitation, as there is likely a small population of teeth that exhibited pulpal pathology that did not undergo NS-RCT, as well as teeth that underwent NS-RCT for restorative reasons instead of biologic reasons.

Regardless of these limitations, this is the first large-scale study designed to identify factors contributing to the likelihood of NS-RCT after crown placement. Of the 88,409 teeth that were crowned, 4.82% teeth underwent an untoward event. The 10-year prediction of survival reaches 90.41%. This value is comparable to past studies despite varying follow up times and inclusion criteria (1-3, 84-87). In all, 87,318 of these crowns were placed by general dentists, which equated to 98.77% of all treatments. The remaining providers included categories of prosthodontists and "other", which accounted for just 0.84% and 0.39% of total treatments, respectively. Results from the logrank test indicate that there are no significant differences between the survival rates of teeth between these three groups (Figure 4).

The majority of teeth receiving crowns were molars with constituted 75.39% of teeth, followed by premolars which constituted 20.52% of teeth, and anterior teeth, which constituted 4.09% of teeth. This variable did not satisfy the proportional hazard assumption for the Cox regression analysis, which states that the ratio of the hazards for two individuals is constant over time. This suggests that the variable of tooth location does not make a linear contribution to the survival model. Therefore, no results comparing the survival rates of teeth with respect to tooth location could be determined. Further analysis was stratified based on tooth location.

The analysis of crown material was divided into three categories – all-metal, PFM, and all-ceramic. This was a natural division, as the CDT codes for crowns fall into these three classifications. Furthermore, these three classes of full-coverage restorations display unique properties that are exclusive/ specific to each group, such as depth of crown preparation and production technique. Stainless steel crowns were not included in this study as there are few indications for use of prefabricated stainless-steel crowns as definitive restorations on permanent teeth.

All-metal crowns represented just 8.86% of all crowns placed. PFM crowns represented 41.5% of crowns, and all-ceramic crowns representing the remaining 49.64% of crowns. This is likely due to increasing demands for esthetic dentistry. Cox regression analysis showed a significant difference in the hazard rates of these materials. PFM crowns have approximately a 27% higher hazard rate than all-metal crowns, and all-ceramic crowns have approximately a 9% higher hazard rate than PFM crowns. Interestingly, these hazard ratios increase with the amount of tooth structure removal indicated for each crown material. This is in contrast with a previous systematic review, which reported lower rates of failure associated with all-ceramic crowns (3).

The age of the patient at the time of crown delivery was also analyzed. Over 65% of patients had crowns delivered while they were between the ages of 51 and 70 years old, while very few patients had crowns placed who were under the age of 30 and over the age of 71. A higher percentage of patients aged 71 and above who had crowns placed experienced extraction as an untoward event. This is likely due to increased likelihood of fracture, increased number of restorations, and higher caries rate in elderly populations, leading to a increased possibility of non-restorable teeth(88).

While patients aged 30 years old and younger were least likely to have a crown placed, they also had the highest rates of failure after crown placement than any other age group. In general, the Cox regression results showed that younger individuals were at a increased risk for an untoward event after crown placement than older individuals. The only groups not exhibiting a statistically significant difference in survival rates were the age groups of 51 to 60 years and 71 years and above (Table 4). There could be multiple explanations for this pattern. Younger individuals generally have larger pulp spaces due to less deposition of secondary dentin, making pulpal tissues closer to the heat and desiccation at the cavosurface margin during crown preparation(5). It may also indicate a correlation between crowns at a young age and pulpal pathology; crowns placed in individuals of younger ages may be necessary due to poor oral hygiene, high caries risk, or unfavorable oral conditions that may also cause a predilection for pulpal pathology.

Kirakozova previously found that younger age and greater extent of tooth destruction were significant predictors of root canal treatment after delivery of full-coverage restorations(85). The results of the current study confirmed these findings, however, without access to radiographs in the current study, tooth destruction was unable to be measured. However, assuming proper preparations were produced for each crown type, the present results indicate that crown materials that require more structure reduction are significantly more likely to undergo an untoward event.

The primary focus of this study was to identify variables that impact pulpal health after crown placement. Since hazard rates increase with lower age and with crown materials that necessitate deeper preparations, these results, in whole, suggest that remaining dentin thickness likely plays a large factor in the development of pulpal

pathology after crown procedures. Additional research is needed to further understand the relationship between the variables discussed in this study, as well as variables that are unable to be followed in an insurance database. Specifically, a large-scale, prospective study which accounts for pulpal diagnosis before crown placement, remaining tooth structure, status of current restorations would allow a more thorough and accurate investigation of the factors contributing to this pathologic process.

CONCLUSION

This is the first insurance-based study aimed to identify and analyze the factors that contribute to the incidence of NS-RCT after the delivery of single-unit full-coverage restorations. Within the constraints and limitations of this study, the rate of untoward events after crown placement was low, with a 90.41% predicted survival rate after 9 years. Survival rate decreases as age at the time of crown placement decreases, and with the placement of PFM and all-ceramic crowns. These findings suggest remaining dentin thickness at the time of crown prep has a significant impact on the likelihood of untoward events after the crown has been delivered. Future research in this area should be focused on prospective studies to standardize procedure protocol, and obtain results based on pre-operative and post-operative pulpal diagnosis.

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