Vital Pulp Therapy for Complicated Crown Fracture of Permanent Canine Teeth in Dogs: A Three-Year Retrospective Study

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Summary:

A 36-month retrospective study compared the results of vital pulp therapy based on the duration of pulp exposure for complicated crown fracture of 97 permanent canine teeth in 76 dogs. Postoperative oral and radiographic examinations were performed at 3, 12, and 36-months following treatment. Based on the 36-month postoperative examinations, 88.2 %, 41.4 %, and 23.5 % of teeth were vital when treated within 48-hours, 1-week, and 3-weeks of pulp exposure, respectively. There was a significant difference in the incidence of tooth vitality between groups. The duration of pulp exposure following complicated crown fracture influenced the success of vital pulp therapy. Vital pulp therapy should be performed as soon as possible following traumatic pulp exposure. ***J Vet Dent 18 (3); 117-121, 2001***

Introduction

The most common endodontic condition in dogs is crown fracture usually involving the canine, maxillary fourth premolar, or mandibular first molar teeth.[1] The severity of tooth damage reflects the type and amount of force applied to the tooth during injury. Uncomplicated crown fractures involve only the enamel, or the enamel and dentin. Complicated crown fractures involve the enamel and dentin resulting in pulp exposure.[2] When pulp tissue is compromised and its vitality threatened or destroyed, endodontic therapy is recommended to maintain teeth that might otherwise be exfoliated or extracted.[3] Treatment options for complicated crown fracture include standard root canal therapy, vital pulp therapy (partial coronal pulpectomy and direct pulp capping), or extraction. Extraction may be the treatment of choice when the owner has financial restrictions or the practitioner does not have the equipment, materials, skills, or referral sources available for endodontic therapy. Treatment by standard root canal therapy has the advantages of removing irreversibly inflamed or necrotic pulp, sealing the apex and coronal pulp chamber from future infection, and, in human studies, providing an excellent prognosis for long-term success.[3,4] Standard root canal therapy may be contraindicated in young dogs with thin-walled, immature teeth that have open apices. The root canal procedure cannot seal the open apex adequately and removing the pulp tissue prevents continued growth of dentin, which may further weaken the tooth.[5] Vital pulp therapy may allow for maintenance of pulp vitality, continued root development, and healing of the coronal pulp wound.[5] Prolonged pulp exposure leading to progressive pulpitis and pulp necrosis inhibits root development and dentin production, and increases the incidence of periapical osteomyelitis and systemic manifestations of dental disease.[6]

The degree of crown trauma, as well as the extent of bacterial contamination may have a direct effect on pulp vitality. The dental pulp is encased in a rigid environment making it particularly sensitive to fluctuations in vascular pressure, affecting tissue perfusion and pulp viability.[7] Changes in pulp microcirculation and hemodynamics are closely related to the severity of the inflammatory response that increases with the duration of pulp exposure.[8-12] Therefore, it is quite possible that the success of vital pulp therapy could be related to the degree of pulp trauma and the duration of pulp exposure.

Human studies have reported excellent results for vital pulp therapy in cases of deliberate pulp exposure and complicated crown fractures treated acutely.[13-17] Whereas in dogs, vital pulp therapy for complicated crown fracture with pulp exposure of extended duration (> 7-days) was associated with a poor prognosis for maintenance of tooth vitality.[18] An excellent prognosis has been reported for maintenance of vitality in teeth undergoing crown reduction and vital pulp therapy in dogs and cats.[18] However, a recent study in primates reported a high failure rate for crown reduction and vital pulp therapy performed under controlled, aseptic conditions.[19] The purpose of the study reported here was to determine the success of vital pulp therapy in teeth with complicated crown fracture in relation to the duration of pulp exposure.

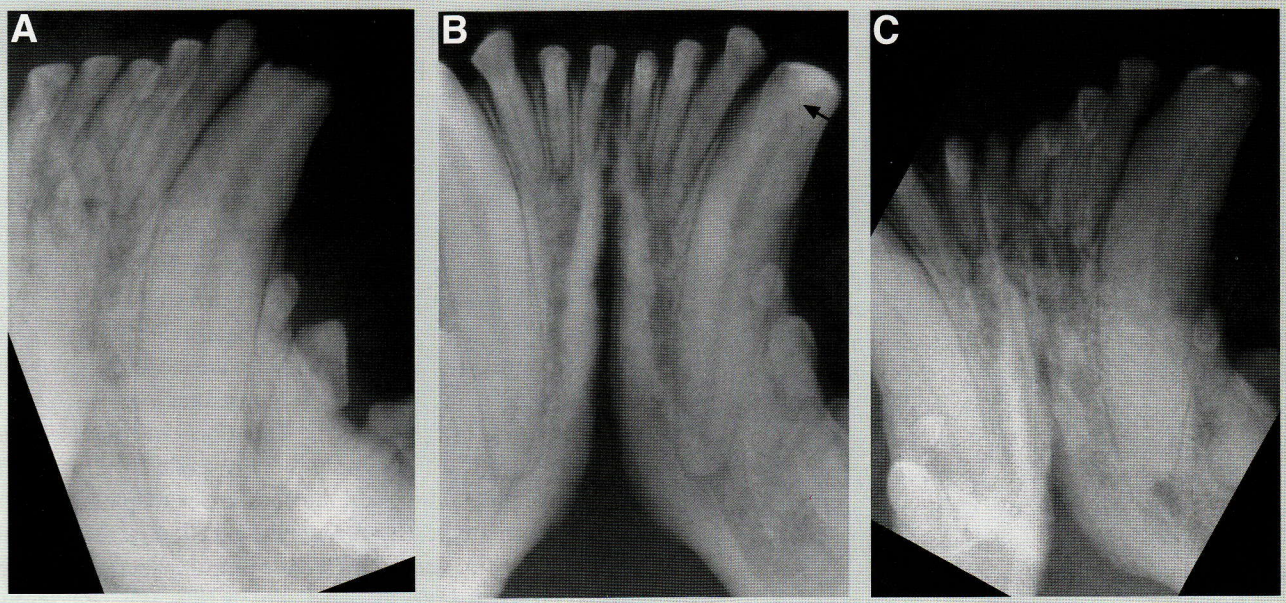
Materials And Methods

Ninety-seven complicated crown fractures of the permanent canine tooth were evaluated in 76 dogs. All complicated crown fractures were supra-gingival. Clinical and radiographic signs of periodontal disease were absent in all teeth. Additional criteria for patient inclusion in this study were: 1) owner knowledge of the time of pulp exposure; 2) vital pulp therapy consisting of partial coronal pulpectomy and direct pulp capping performed within 3-weeks of complicated crown fracture; and, 3) the owner's desire to maintain the vitality of the tooth. Failure to meet these criteria resulted in standard root canal therapy or extraction of the injured tooth. Only vital pulp therapy was evaluated in this study.

Factors that resulted in patients being excluded from the study included: a diagnosis of necrotic pulp; presence of systemic disease (diabetes mellitus, renal insufficiency, etc.); administration of non-steroidal, anti-inflammatory drugs (NSAIDS), corticosteroids, or systemic antimicrobials within

Figure 1

Intraoral radiographs of a mandibular right canine tooth (404) with complicated crown fracture in a two-year-old Boxer dog. Vital pulp therapy was performed within 24-hours of injury. Preoperative (A) and postoperative radiographs taken at 3 (B) and 36 (C) months show an intact composite restoration, dentin bridge formation (arrow), progressive narrowing of the pulp canal, and no radiographic signs of periodontal or periapical pathology.



6-months of tooth injury; and, failure to achieve hemostasis within 5-minutes of partial coronal pulpectomy.

Teeth were categorized into three groups based on pulp exposure duration of < 48-hours (n = 34) [48-hour group], 2 to 7-days (n = 29) [1-week group], or 1 to 3-weeks (n = 34) [3-week group]. Patient age, sex, or breed were not considered during group assignment. The mean age (+/- SD) for dogs in the 48-hour, 1-week, and 3-week groups was 28 (4), 24 (2), and 31 (5) months, respectively. Males were represented more than females by a ratio of 2:1.

Postoperative assessment including oral and radiographic examinations was performed at 3, 12, and 36-months. Radiographic signs considered consistent with tooth vitality included formation of a dentin bridge, continued dentin formation with narrowing of the pulp canal, and absence of periapical and periodontal pathologic changes (Fig. 1). All teeth considered non-vital were either extracted or received standard root canal therapy after consultation with the owner.

Preanesthetic examination and laboratory data were within normal limits for each dog. Subcutaneous preanesthetic medication (atropine sulfate[a] 0.05 mg/kg, acepromazine maleate[b] 0.05 mg/kg, buprenorphine[c] 0.0075 mg/kg) was followed 30 minutes later by anesthetic induction (ketamine[d] 5.0 mg/kg, diazepam[e] 0.25 mg/kg IV). Each dog was intubated and general anesthesia was maintained with isoflurane[f] 1-2 %, oxygen (10 ml/kg), and N_2O (20 ml/kg). Monitoring was performed using apAlert[g] (respiration rate), pulse oximeter[h] (heart rate and spO_2) and Doppler[i] (blood pressure). A preoperative intraoral radiograph was taken of each tooth to rule-out subgingival/root fractures, periapical pathology, and periodontal disease that would indicate tooth extraction using occlusal, E-speed intra-oral film[j] and a bisecting angle technique. The oral cavity and fractured tooth were irrigated with a 0.12 % chlorhexidine digluconate[k] solution. Using a #1 pear-shaped diamond bur in a high-speed handpiece with water-cooling, approximately 5-mm of coronal pulp was excised. Hemostasis was obtained with saline-soaked sterile cotton pellets. Following removal of the pellet, a 1-mm layer of fresh calcium hydroxide USP powder[l] was placed over the remaining pulp. A 1-mm layer of hard-setting calcium hydroxide paste[m] was placed over the powder followed by a final composite restoration[n]. The manufacturers' recommendations for acid-etching and bonding agents were followed to place the composite. (**Editor's Note**: Please see the *Step-by-Step* feature on page 154 for a pictorial guide describing partial coronal pulpectomy and direct pulp capping). All dogs were prescribed (amoxycillin and clavulanic acid[o] (20 mg/kg Q8h X 10-days) and ketoprofen[p] (1mg/kg Q24h X 5-days).

Differences in tooth vitality between groups were analyzed using a non-parametric Wilcoxon's Rank Sum test with values of $P < 0.05$ considered significant.

Results

The results of tooth vitality assessment following vital pulp therapy are summarized in Table 1. At the 3-month postoperative examination, all treated teeth appeared vital based on oral examination, normal periodontal probing depths, intact composite restorations, and no obvious change in tooth

Table 1

Results of tooth vitality assessment in 97 permanent canine teeth receiving vital pulp therapy for complicated crown fracture in dogs.

			Postoperative Examinations					
			3-months		12-months		36-months	
Duration of Pulp Exposure	# of Dogs Treated	# of Teeth Treated	# Vital Teeth	% Vital Teeth	# Vital Teeth	% Vital Teeth	# Vital Teeth	% Vital Teeth
< 48 - Hours	33	34	34	100	32	94.1	30	88.2
2 to 7 - Days	25	29	29	100	14	48.3	12	41.4
1 to 3 - Weeks	18	34	34	100	9	26.5	8	23.5

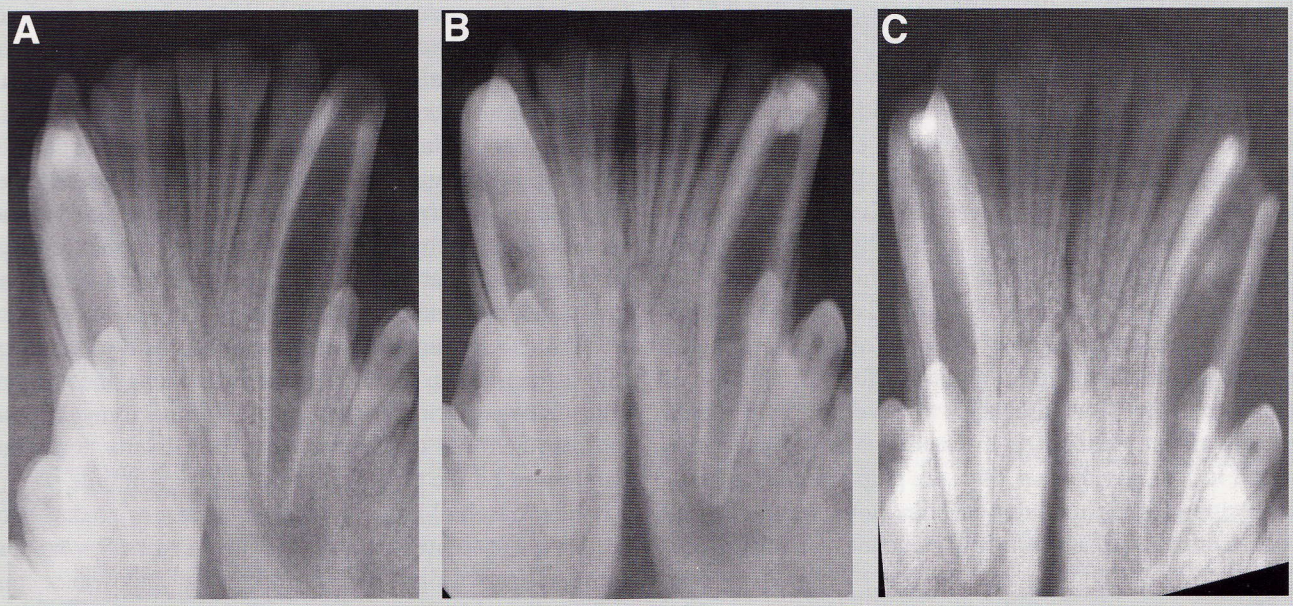
color. There were no radiographic signs of periapical or periodontal pathology. The width of the pulp canal appeared narrower in all cases indicative of tooth vitality, with dentin bridge formation in 75.0 %, 82.0 %, and 65.0 % of teeth treated within 48-hours, 1-week, and 3-weeks of pulp exposure, respectively.

At the 12-month postoperative examination, all treated teeth appeared vital based on oral examination, normal periodontal probing depths, intact composite restorations, and no obvious change in tooth color except for 1 tooth in the 48-hour group. This tooth lost its composite restoration and had exposed, necrotic pulp tissue (Fig. 2). An additional tooth in this group had radiographic signs of pulp necrosis. The width of the pulp canal appeared narrower indicative of tooth vitality in 94.1 %, 48.3 %, and 26.5 % of teeth treated within 48-hours, 1-week, and 3-weeks of pulp exposure, respectively. There was a significant difference in the incidence of tooth vitality between groups.

At the 36-month postoperative examination, the visual appearance of the treated teeth was clinically normal in 88.2 %, 70.0 %, and 53.0 % of the teeth from the 48-hour, 1-week, and 3-week groups, respectively. The number of teeth with

Figure 2

Intraoral radiographs of the mandibular left (304) and right (404) canine teeth with complicated crown fractures in an eleven-month-old Bull Terrier dog. Vital pulp therapy was performed within 24-hours of injury. Preoperative (A) and postoperative radiographs of 404 show narrowing of the pulp canal similar to 304 by 3-months (B) following treatment. At 10-months (C), the composite restoration of 404 has been lost and the pulp canal is wider compared with 304 indicative of pulp necrosis and failure of vital pulp therapy. (Note: the apparent periapical lucency of 404 is an artifact based on positioning of the mandible.)



a normal clinical appearance was significantly different between groups. Lost composite restorations and necrotic pulp were evident in 1, 2, and 5 teeth from the 48-hour, 1-week, and 3-week groups, respectively. Teeth that lost the composite restoration had no radiographic signs of a dentin bridge. The width of the pulp canal appeared narrower indicative of tooth vitality in 88.2 %, 41.4 %, and 23.5 % of teeth treated within 48-hours, 1-week, and 3-weeks of pulp exposure, respectively. For teeth considered vital, there was no clinical or radiographic evidence of periapical or periodontal pathology, however dentin bridge formation was not necessarily apparent radiographically. There was a significant difference in the incidence of tooth vitality between groups. There was no significant relationship between maintenance of tooth vitality and patient age, sex, or breed.

Discussion

The structural elements of the pulp consisting of cells, fibers, and ground substance are similar to loose, fibrous connective tissue found elsewhere in the body. The pulp also has a neurovascular supply. Histological examination of the pulp from its periphery towards the center shows the odontoblast layer, cell-free zone of Weil, cell-rich zone, and pulp core. The principle cells in the pulp are the odontoblasts, fibroblasts, undifferentiated mesenchymal cells, and host-defense cells. Odontoblasts are highly differentiated mesenchymal cells with a primary function to produce dentin. They contain a cellular body, located in the odontoblastic layer, and an odontoblastic process which extends into the dentin through an open tubule. As the tooth ages, odontoblasts produce secondary dentin, which can be demonstrated radiographically as a thickening of the dentin layer with a narrowing of the pulp canal. Located in the cell-rich zone, fibroblasts are the most common cell type in the pulp, producing collagen and ground substance. Undifferentiated mesenchymal cells have the capacity to differentiate into other types of cells such as odontoblasts and fibroblasts. Thus, undifferentiated mesenchymal cells may play a critical role in the healing of damaged pulp in response to direct pulp capping with calcium hydroxide. Placement of a thin layer of calcium hydroxide over an exposed vital pulp results in superficial pulp necrosis. Host-defense cells including macrophages and occasionally lymphocytes, polymorphonuclear leukocytes, plasma cells, and mast cells from the healthy pulp respond to pulp inflammation.[20] An appropriate cellular response to the inflammation and necrosis coupled with the transformation of undifferentiated mesenchymal cells into odontoblasts allows production of reparative dentin (dentin bridge) to cover the pulp tissue. The pulp of teeth reported in this study would be expected to respond in a similar manner since the pulp dressing was calcium hydroxide and partial coronal pulpectomy was performed to excise inflamed tissue following guidelines established previously.[5]

The success of vital pulp therapy may be impacted by multiple variables that should be controlled if possible. The technical skills of the operator may influence the outcome of any dental procedure. In this study, the same operator performed all vital pulp treatments. Although technical variation may have occurred between individual cases within this study (e.g. amount of calcium hydroxide for the pulp dressing, exact depth of partial coronal pulpectomy), the overall results should be comparable between groups based on the sample size in each group.

Another variable influencing treatment success may be the severity of bacterial pulpitis at the time of vital pulp therapy. There are numerous species of oral bacteria, with varying degrees of virulence, that contaminate and invade the exposed pulp tissue. Maintenance of tooth vitality may be related to the type and number of bacteria, as well as the depth of bacterial invasion.[12] In this study, only 5-mm of pulp tissue was excised during partial coronal pulpectomy. Bacteria may have colonized pulp > 5-mm apically especially in cases of prolonged pulp exposure. The author is not aware of any studies reported in the veterinary literature that have analyzed the type or depth of bacterial pulpal colonization following complicated crown fracture. A study in the human dental literature reported that exposed, untreated pulp tissue in germ-free animals consistently healed, whereas the exposed pulp in conventionally treated animals became necrotic.[21] The pathologic effect of bacteria in pulpitis and pulp necrosis may be further supported by the fact that, in this study, teeth which lost their composite restorations before dentin bridge formation had pulp re-exposure and subsequent necrosis. Unfortunately, bacterial cultures were not performed on the exposed, necrotic pulps in this study.

A bacteria-tight seal protecting the healing pulp may be the most critical factor influencing successful vital pulp threrapy.[12] Microleakage of the restoration occurs when bacteria and/or oral fluids traverse the space between the tooth wall preparation and restoration. Microleakage could have been a potential problem in this study since some restorations may have been better than others even though all restorations were performed in the same manner, by the same operator. Additionally, restorations were subjected to different conditions (e.g. food type, chew objects, masticatory forces) that may have disrupted the mechanical integrity of the restoration causing microleakage. A glass ionomer may have been a more appropriate intermediate base layer compared with hard-setting calcium hydroxide since it adheres to dentin and is less soluble.[22]

Pulp necrosis may have occurred secondary to irreversible inflammation from the traumatic event or iatrogenic pulp damage during partial coronal pulpectomy. It could be theorized that the degree of pulp inflammation would vary based on the type of trauma (e.g. automobile trauma, a cricket/baseball bat, chewing on a bone or stick). Many dogs were presented with complicated crown fracture presumably from a fight with a sibling, but the owners could not confirm this type of trauma because the dog had been

playing with hard objects in the yard. Since owners were often unable to accurately identify the type of trauma that caused the complicated crown fracture, a relationship between the relative degree of trauma and pulp necrosis could not be determined. It is generally recommended that all inflamed pulp be excised during partial coronal pulpectomy. However, pulp inflammation may not be confined to the coronal aspect of the tooth. Therefore, assessment of pulp inflammation remains indirect based on persistent hemorrhage following pulp excision.[5] Teeth were excluded from this study when pulp hemorrhage persisted > 5-minutes following partial coronal pulpectomy indicative of irreversible pulp inflammation.

Iatrogenic pulp damage leading to excessive inflammation was unlikely in this study since all teeth were treated by the same individual, in the same manner, including use of sterile hand instruments and new burs and materials. Additionally, fresh calcium hydroxide was used for the pulp dressing since it has been reported that calcium hydroxide may react with atmospheric moisture and carbon dioxide to form ineffective calcium carbonate.[20]

In this report, there was continued apexogenesis following vital pulp therapy that was unrelated to the duration of pulp exposure. This observation is consistent with another study that reported 3 of 4 teeth continued to produce dentin sufficient to perform standard root canal therapy even though pulp necrosis had occurred eventually.[23] Apexogenesis allows continued dentin growth, narrowing of the pulp canal, and root apex closure.[5] Vital pulp therapy may be a viable treatment option for immature teeth with irreversible pulpitis since even minimal dentin production and apical closure may allow a standard root canal procedure to be performed. All teeth in this study showed continued pulp canal narrowing indicative of tooth vitality despite several treated teeth having pulp necrosis at the 12 or 36-month reexaminations.

In summary, results of this study support other work that shows subjective assessment parameters are not reliable indicators of tooth vitality following vital pulp therapy.[19,23] Further, professional postoperative assessment of vital pulp therapy, including oral and radiographic examinations, should be performed for an extended period of time, as reported here, in order to accurately determine outcome. Finally, vital pulp therapy for complicated crown fracture has a relatively high success rate (88.2 %) if the duration of pulp exposure is < 48-hours. Therefore, vital pulp therapy should be performed as soon as possible following complicated crown fracture.

[a] Atropine injection, Apex Laboratories Pty Ltd, Australia
[b] Promex 2, Apex Laboratories Pty Ltd, Australia
[c] Temgesic injection, Reckitt & Colman, Australia
[d] Ketamine injection, Parnell Laboratories Pty Ltd, Australia
[e] Pamlin injection, Parnell Laboratories Pty Ltd, Australia
[f] Forthane, Abbott Australia Pty Ltd, Australia
[g] apAlert Respiratory Monitor, Model RM5D, MBM Enterprises, Australia
[h] Vet/Ox, SDI, Australia
[i] 811-B, Ultrasonic slow detector, Parks Medical Enterprises, Australia
[j] Eastman Kodak Co, Rochester, NY
[k] Hibiclens, Coopers Animal Health, Wilks-barre, PA
[l] Amend Drug & Chemical Co, Australia
[m] Life, Kerr Laboratory Products Div, Emeryville, CA
[n] Z100, 3M Dental Products, St. Paul, MN
[o] Clavulox, Pfizer Animal Health, Exton, PA
[p] Ketofen, Merial, Iselin, NJ

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