Pulpal response to different pulp capping methods after pulp exposure by air abrasion

Lina Maria Cardenas-Duque* / Makoto Yoshida** / George Goto***

Air abrasion is regaining popularity especially in the area of pediatric dentistry due to its ease of use and its advantages. Due to the lost of tactile information, while using this technique, there is an increased risk for pulpal exposure. On the other hand, $Ca(OH)_2$ medicament has been proven to induce dentin bridge formation, but an adequate sealing seems to be even more important that the capping material used. The purpose of this study was two fold: to assess the pulpal response after pulpal exposure by air abrasion and to evaluate the healing potential after using $Ca(OH)_2$ medicament or Liner Bond II™ as a capping agent. Two hundred sixteen teeth from mixed-bred dogs were used in this study. The teeth were divided into three groups, A) pulpal exposure by air-abrasion followed by sealing of the cavity with Liner Bond II™, B) pulpal exposure by air-abrasion and $Ca(OH)_2$ pulp capping and C) pulpal exposure by high-speed followed by air-abrasion and $Ca(OH)_2$ pulp capping as a control group. The animals were sacrificed after 7, 14, 30 and 60 days and a histopathological evaluation was undertaken. After applying Analysis of Variance to compare the groups, it was observed that at earlier observation periods, the inflammatory criteria near the exposure site were different among the groups. As time elapsed, the inflammation was resolved in the pulp tissue, however, the odontoblastic layer and the dentin bridge formation had a highly statistically significant difference ($p<0.001$) among the various groups at all observation periods. In addition, a positive correlation was observed between the organization of the odontoblastic layer and the dentin bridge formation mainly after 30 days. It could be concluded that dentin bridge formation could be achieved with the use of $Ca(OH)_2$ or Liner Bond II™ as capping agent with an adequate sealing. However, the formation is delayed especially when Liner Bond II™ is used as capping agent.

J Clin Pediatr Dent 26(3): 269-274, 2002

INTRODUCTION

The air-abrasion technology is re-gaining popularity, especially among pediatric dentists.[1] This technique utilizes the principle, of kinetic energy to deliver aluminum oxide particles at certain air pressure to cut the tooth structure.[2] The main advantages that have been described with the use of air-abrasion technology, include among others, very little noise and no need for anesthesia for most procedures.[3-5] Due to the distance between the nozzle and the tooth it is easier to make an accidental pulpal exposure when using this method, especially when cavity preparations for restorative materials are being prepared.[6]

When a mechanical pulpal exposure is done accidentally in a healthy pulp, the placement of several capping agents has been advocated. One of the most widely used pulp capping medicaments is calcium hydroxide, which in contact with the pulp tissue seems to produce a necrotic layer that stimulates dentin formation.[7-9] This newly formed dentin, or dentinal bridge, has been considered as one of the signs of pulpal healing. However, pulpal healing is determined by the degree inflammation that is observed in the pulpal tissue. It is very important to consider these two factors (degree of pulpal inflammation and dentin bridge formation) especially when an animal experiment is undertaken, since clinical symptoms do not play a role in this type of studies.[10]

In addition to the calcium hydroxide medicament, some other capping agents have been described. Recent studies have suggested that achieving an excellent sealing of the cavity is even more important for the pulpal healing than the pulp-capping medicament used.[11]

The events that are involve in the formation of dentinal bridge after pulpal exposure by hand piece have been described previously. Yamamura divides the dentin bridge formation into four main stages as follows: a) exudative stage, b) proliferative stage, c) osteodentin stage and d) tubular dentin formative stage.[12]

Department of Pediatric Dentistry, Nagasaki University, School of Dentistry, 1-7-1 Sakamoto-machi, Nagasaki, 852, Japan.

All correspondence should be addressed to Dr. George Goto. Professor and Chairman, Department of Pediatric Dentistry, Nagasaki University, School of Dentistry. 1-7-1 Sakamoto-machi, Nagasaki, 852, Japan.

Table 1. Evaluation Criteria for Pulp Healing and Dentin Bridge Formation.

Odontoblastic Layer	Inflammatory Cell Infiltrate S=Superficial D=Deeper	Fibrosis	Hemorrhage	Necrosis	Dentin Bridge formation
0-Regular **1**-Irregular **2**-Absent	**0**-No inflammation **1**-Mild inflammation infiltrate **2**-Heavy inflammation infiltrate **3**-Abscess	**0**-None **1**-Mild **2**-Severe	**0**-None **1**-Slight **2**-Mild **3**-Severe	**0**-No necrotic tissue **1**-Varying amounts of necrotic tissue **2**-Necrotic tissue in incisal or occlusal pulp horn of whole coronal pulp	**0**-None **1**-*IDB* (Initial Dentin Bridge Formation) **2**-*PDB* (Partial Dentin Bridge Formation **3**-*ACDB* (Almost Complete Dentin Bridging **4**-*CDB* (complete Dentin Bridging)

Table 2. Summarized Histological Findings.

Group		Odontoblstic Layer	Inflammatory Cell Infiltration Superficial	Inflammatory Cell Infiltration Deep	Fibrosis	Necrosis	Hemorrhage	Dentin Bridge
7 Days	Group A	1.87, 0.90	1.39, 0.50	0.44, 0.62	0.11, 0.32	0.11, 0.32	1.22, 0.665	0.39, 0.50
	Group B	1.16, 0.77	0.63, 0.60	0.32, 0.58	0.26, 0.45	0.11, 0.32	0.95, 0.91	0.63, 0.76
	Group C	1.44, 0.32	1.11, 0.60	0.33, 0.50	2.00, 0.00	0.00, 0.00	0.11, 0.11	1.89, 0.78
14 Days	Group A	1.13, 0.34	0.74, .069	0.13, 0.34	0.87, 0.29	0.87, 0.29	00.57, 0.66	1.22, 0.42
	Group B	0.85, 0.54	0.39, 0.57	0.04, 0.20	0.39, 0.50	0.04, 0.20	0.66, 0.69	1.97, 0.77
	Group C	1.20, 0.42	0.50, 0.53	0.00, 0.00	0.80, 0.42	0.00, 0.00	0.90, 0.88	3.80, 0.42
30 Days	Group A	1.08, 0.50	0.62, 0.77	0.04, 0.20	0.42, 0.20	0.08, 0.28	0.50, 0.72	1.25, 0.44
	Group B	0.72, 0.58	0.469, 0.62	0.06, 0.25	0.34, 0.48	0.31, 0.69	0.71, 0.81	2.18, 0.69
	Group C	1.00, 0.00	0.60, 0.52	0.00, 0.00	1.30, 0.48	0.00, 0.00	0.20, 0.42	4.00, 0.00
60 Days	Group A	0.47, 0.74	0.40, 0.51	0.00, 0.00	0.13, 0.35	0.07, 0.26	0.33, 0.49	0.59, 0.15
	Group B	0.32, 0.48	0.16, 0.50	0.11, 0.46	0.16, 0.38	0.00, 0.00	0.32, 0.58	3.68, 0.58
	Group C	1.29, 0.76	0.86, 1.46	0.71, 1.50	1.14, 0.38	0.29, 0.76	0.00, 0.00	4.00, 0.00

Group A: Air-abrasion + Liner Bondd IITM; **Group B:** Air abrasion + Ca(OH)2 + temporary filling; **Group C:** High speed + Ca(OH)2 + Temporary filling

However, less attention has been placed on the formation of dentinal bridge after pulpal exposure by air-abrasion. Cardenas and Goto[6], observed that even after pulpal exposure with air-abrasion, pulpal healing and dentinal bridge formation were possible by the use of an adequate capping technique. They also concluded that this was delayed for the air-abrasion group when compared with the high speed group when calcium hydroxide was used as a pulp-capping medicament.

Dentinal bridge formation has also been achieved after pulpal exposure by high speed and air-abrasion, when different types of composite resins were used.[13] Moreover, it has been found that even using 4-META composites as a capping agent, when a perfect sealing of the cavity is achieved, dentinal bridge formation is possible in pulpal exposures created by air-abrasion.[14,15]

The purpose of this study was to evaluate the pulpal response and the dentinal bridge formation after pulpal exposure using air-abrasion and high-speed, utilizing calcium hydroxide medicament and 4-META composite resin as a pulp capping material. For this purpose two data sets by Cardenas and Goto, and Yoshida *et al*. were combined and analyzed using the same evaluation criteria.

MATERIALS AND METHODS

A total of 216 teeth obtained from 16 mixed-bred dogs were used in this study. The animals were between 6 to 7 month old. After obtaining general anesthesia by the use of IM Ketamine and IV sodium pentobarbital, the operative procedures were undertaken by two trained operators (C&Y). The teeth were randomly assigned to three experimental groups. For groups A and B, an air-abrasion unit (Whisperjet-American Dental Technologies) was used to perform the pulpal exposures. For group C, or control group, pulpal exposures were performed with high speed as gold standard to study the pulpal effects after accidental pulp exposures.

Figure 1. 60-day specimens. H-E staining. Corresponding to group A (x100). Note that the dentin bridge was covering the whole exposure site. However, the presence of some tunnel defects in the dentin bridge were observed.

Figure 2. 60-day specimens. H-E staining. Corresponding to group B (x100). A1203 particles were observed into the pulp tissue far from the pulp exposure. The newly formed dentin bridge covered the entire exposure site. This dentin bridge presented a more tubular-like appearance and no tunnel defects were observed.

Group A received 4-META (Liner Bond II™), as a pulp capping agent and group B and C received calcium hydroxide medicament (Calvital™) in direct contact with the pulpal tissue.

The animals were sacrificed with an overdose of Pentobarbital at 7, 14, 30 and 60 days respectively. Paraffin blocks (Paraplast Plust™) were obtained and serial sections of 6mm were stained with hematoxilin and eosin. A light microscope (Olympus B12™) was used to observe and grade the samples. A modification of the evaluation criteria by Stanley[16] Fuks[17] Horsted[8] and Kitasako,[18] described by Cardenas and Goto,[6] was utilized to evaluate the pulpal response and the dentin bridge formation. These evaluation criteria includes different parameters as shown in Table 1.

Descriptive Statistics were applied to the obtained data, Analysis of Variance (ANOVA) was used to compare the different groups and Pearson correlation statistics were use to detect the changes over time.

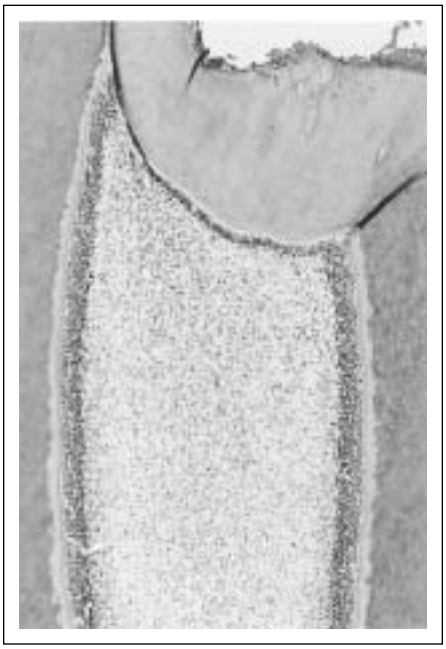

Figure 3. 60-day specimens. H-E staining. Corresponding to group C (x100). Note how a very prominent and well organized dentin bridge was observed in the control group.

RESULTS

The total number of teeth was divided randomly into three groups according to the capping method. In addition four observation periods were considered. Forty-seven samples were observed at seven days post-treatment, while 59 and 66 were observed at 14 and 30 days, respectively. For the sixty-day observation period a total of 44 samples were processed. The control group (or C group) for each observation period consisted of 10 teeth. The summarized results of the histological findings according to the used criteria are shown in Table 2.

During the seven-day observation period there was a statistically significant difference on the inflammatory cell infiltration in group A and B ($p<0.001$); and between

group A and C ($p<0.0001$). Hemorrhage was also statistically significant different between group A and C ($p<0.001$). The dentinal bridge formation was statistically significant different between groups A and C ($p=0.0001$), but no difference was detected between groups A and B ($p>0.05$). As time elapsed, most inflammation parameters decreased as can be observed in Table 2.

In the 14-day samples, there was no difference in the odontoblastic layer between group A and B, but a statistically significant difference between group A and C was noted ($p<0.05$). In this observation period, the dentin bridge formation was statistically different among all groups; between A and B the p value was < 0.05 and between groups A and C and B and C. The p value was<0.001.

The most important findings in the 30-day observation period were the statistically significant difference in the odontoblastic layer between groups A and B ($p<0.05$), A and C ($p<0.0001$) and B and C ($p<0.05$). In addition the dentinal bridge formation was also different among the groups. A and B showed a highly statistically significant difference ($p<0.0001$), as well as A and C ($p<0.0001$) and finally, B and C ($p<0.0001$).

In the 60 day observation time, the dentinal bridge formation was different among the groups. Between group A and B, this difference was statistically significant ($p<0.05$) as well as between groups B and C.

When correlation statistics were applied to the data set, it was found that there was a positive correlation between superficial inflammatory cell infiltration and necrosis in group C at 60 days ($r=0.80$). In addition, a strong correlation was also found between the odontoblastic layer and the dentin bridge formation for group B at 60 day observation period ($r=0.91$). Figures 1 to 3.

DISCUSSION

The use of air-abrasion technology has many advantages related to ease of use and to patient comfort. However, since there is certain distance between the tooth surface and the nozzle, tactile information is lost and the risks of creating an accidental pulp exposure are highly increased.[6] The present study examined the pulpal effects after pulp exposure by air-abrasion and concentrated on the dentin bridge formation as one of the signs of favorable pulpal response to capping agents. The results of the study are also confirmed by previous studies.[6,19]

The accepted gold standard for pulp capping agents is $Ca(OH)_2$ which under ideal conditions such as healthy pulp, hemorrhage and subsequent clot formation control, has been proven to induce dentinal bridge formation by stimulating odontoblastic differentiation. When comparing this gold standard (control group or group C) with pulpal exposures by the use of air-abrasion, the dentin bridge formation was always achieved. However, the main difference was the speed of dentin bridge formation, which was significantly delayed for group A or air-abrasion/Liner Bond II™. Similar findings have been reported in monkeys when pulp exposures by high-speed had been followed by pulp capping with resin materials.[16]

Regarding the pulpal inflammation, represented in the studied criteria, in most observation periods no significant difference was observed among the three groups. However, the odontoblastic layer, in many instances, presented a statistically significant difference. Moreover, the odontoblastic layer and the dentinal bridge formation presented a positive correlation, which also explains the importance of the stimulation of odontoblastic differentiation in order to obtain dentinal bridging, which is a phenomenon that has been described previously. In addition, though it was not a common finding in this study, some chronic inflammation was observed at later observation periods justifying the fact that dentin bridge is a very important marker of pulp healing, but not the only one. The pulpal tissue is more complex than that and an observation of the degree of inflammation using a universal criteria is imperative in order to obtain compelling data, at is has been described in the pulpal biology literature.[16]

Although a dentinal bridge was obtained in all groups studied, the delay in both air abrasion groups and especially when Liner Bond II™ was used suggests that longer observation periods are needed when evaluating this type of material.[19]

The results of this study are encouraging, however, further investigation is needed both with animal studies and by human clinical trials, as ethically feasible.

CONCLUSIONS

The following conclusions from this study can be drawn:

1. For most of the inflammation criteria studied, pulpal response was not statistically different after pulpal exposure by air-abrasion or high speed.
2. The main difference in pulpal healing after pulpal exposure by the two studied techniques, was the dentinal bridge formation, which was statistically significantly delayed for both air-abrasion groups.
3. Sealing of the pulpal exposure plays a very important role in pulpal healing, probably even more important that the capping agent used. However, when Liner bond II™ was used as a capping agent, the formation of dentinal bridge was statistically significantly delayed.
4. $Ca(OH)_2$ medicament proved to still be the gold standard when an accidental pulp exposure occurs.

REFERENCES

1. Goldstein RE, Parkins FM. Using air-abrasive technology to diagnose and restore pit and fissure caries. JADA 126: 761-766, 1995.
2. Goldstein RE, Parkins FM. Air-abrasive technology: its new role in restorative dentistry. JADA 125: 551-557, 1994.
3. Goldberg MA. Airbrasive: patient reactions. J Dent Res August, abstract 118, 1952.

4. Goto G, Zhang Y. Kinetic cavity preparation: protection of the cavosurface enamel. J Clin Pediatr Dent 21 : 61-65, 1996.
5. Kotlow LA. Air Abrasion and the KCP-1000. JSSPD 3: 32-33, 1993.
6. Cardenas-Duque LM, Goto G. Histological effects of the pulp exposure by air-abrasion on the dentin bridge formation. Ped Dent J 10: 49-59, 2000.
7. Hendry JA, Jeansonne BG, Dummett CO, Burrell W. Comparison of calcium hydroxide and zinc oxide and eugenol pulpectomies in primary teeth of dogs. Oral Surg 54: 445-451, 1982.
8. Horsted P, Al Attar K, Langeland K. Capping of monkey pulps with Dycal and a Ca-eugenol cement. Oral Surg 52: 531-553, 1981.
9. Jerrel R, Court FJ, Stanley HR. A comparison of two calcium hydroxide agents in direct pulp capping of primary teeth. J Dent Child 34-38, 1984.
10. Mochizuki K, Fuji H, Machida Y. Dentin bridge formation following direct pulp capping in dogs. Bull Tokyo Dent Coll 39: 31-39, 1998.
11. Shovelton DS, Friend LA, Kirk EEJ, Rowe AHR. The efficacy of pulp capping materials. Brit Dent J 130: 385-91, 1971.
12. Yamamura T. Differentiation of pulpal cells and inductive influences of various matrices with reference to pulpal wound healing. J Dent Res 64, Special Issue, 530-540, 1985.
13. Matsuo T, Nakanishi T, Shimizu H, Ebisu S. A Clinical Study of Direct pulp capping applied to carious-exposed pulps. J Endodont 22: 551-55, 1996.
14. Tsuneda Y, Hayakawa T, Yamamoto H, Ikemi T, Nemoto K. A histopathological study of direct pulp capping with adhesive resins. Op Dent 20: 223-29, 1995.
15. Kitasako Y, Inokoshi S, Fujitani M, Otsuki M, Tagami J. Short-term reaction of exposed monkey pulp beneath adhesive resins. Op Dent 23: 308-17, 1998.
16. Stanley H. Methods and criteria in evaluation of dentin and pulp response. Int Dent J 20: 507-527, 1970.
17. Fadavi S, Anderson AW. A comparison of the pulpal response to freeze-dried bone, calcium hydroxide, and zinc oxide-eugenol in primary teeth in two cynamalgus monkeys. Ped Dent 18: 52-56, 1996.
18. Kitasako Y. Sealing ability and effect of adhesive resins as direct pulp capping materials on wound healing. A'ihonshika hozon-gaku zashi 40: 414-444, 1997. (article in Japanese)
19. Yoshida M, Hosoya M, Kimura A, Goto G. Histo-pathological study on influence of direct pulp capping with the adhesive resin on the exposed pulp by air-abrasion. Shonishikagaku zashi 38:1061-1074, 2000.