

## Glutaraldehyde as a pulp dressing after pulpotomy in primary teeth of baboon monkeys

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### Abstract

*The purpose of this investigation was to assess the pulp healing process after pulpotomy in primary baboon teeth using glutaraldehyde as a pulp dressing with and without induced pulp inflammation. Inflammation was induced by inserting carious dentin into deep Class V cavities for 3 days. Twenty noncarious primary baboon teeth were used. In 10 of these teeth (Group A), inflammation was induced prior to performing a pulpotomy; in the remaining 10 teeth (Group B), pulpotomies were carried out in intact teeth. Two months after treatment, the teeth were extracted and prepared for histologic evaluation. No appreciable difference could be observed between the 2 groups. All teeth were vital and incomplete dentin bridges were seen in 12.5% of Group A and 6.6% of Group B. Most of the teeth presented with a positive tissue response to glutaraldehyde since inflammation, when present, was limited and mild.*

**T**he need for a substitute to formocresol following pulpotomies in primary teeth has been established;<sup>1,2</sup> meanwhile, guidelines restricting its use have been recommended.<sup>3</sup> Diluted formocresol and other materials with less deleterious effects than the conventional Buckley's solution have been utilized; however, an ideal pulp dressing has not been found yet.<sup>4-8</sup>

It has been suggested<sup>9-11</sup> that glutaraldehyde probably could substitute for formocresol for several reasons.

1. It is initially more active chemically.
2. It rapidly forms cross linkages and its penetration is more limited.
3. Glutaraldehyde is not as volatile as formocresol.

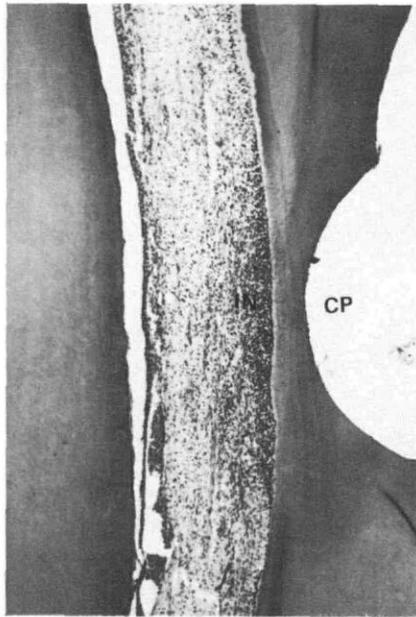
4. There is less apical damage and less necrosis in the glutaraldehyde-treated specimens.
5. There is no evidence of ingrowth of granulation tissue into the apex in the glutaraldehyde-treated specimens.
6. There is less dystrophic calcification in the glutaraldehyde specimens.

Although the positive effect of glutaraldehyde has been demonstrated previously, none of the studies used teeth with experimentally induced inflammation as a model, a condition that may mimic closely the clinical situation. The purpose of this investigation was to study the radicular pulp tissue reaction in primary teeth of baboon monkeys after pulpotomy with and without previously induced inflammation.

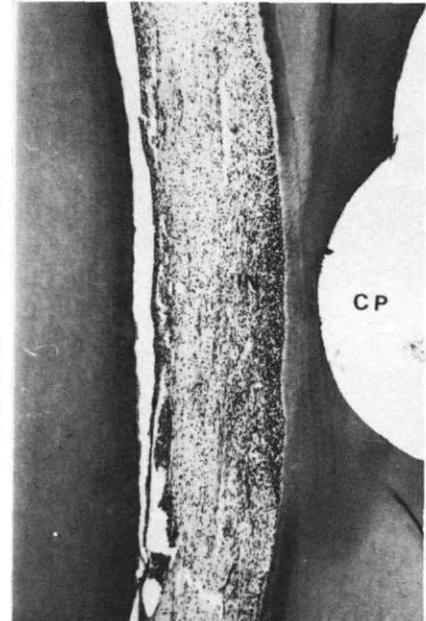
### Methods and Materials

All the teeth of 1 baboon in the primary dentition age were used in the experiment. The 10 maxillary and mandibular primary teeth of 1 side of the mouth formed Group A in which inflammation was induced prior to the glutaraldehyde pulpotomy. In the 10 teeth of the opposite side of the mouth, Group B, the pulpotomies were performed without prior induction of inflammation.

In Group A, inflammation was induced by inserting carious dentin into deep cavity preparations for 3 days as described by Lervik and Mjor.<sup>12</sup> In order to assure the reproducibility of this technique under the present conditions, a previous pilot study was undertaken using 20 primary baboon teeth. All demonstrated moderate to severe inflammation. This inflammation, however, was limited to the area underneath and in close proximity to the cavity preparation (Figs 1a, 1b).



**FIG 1a (left).** Photomicrograph of a primary central incisor after induction of inflammation. Notice the inflammatory infiltrate (IN) limited to the area adjacent to the cavity preparation (CP). H&E, 100 $\times$ .



**FIG 1b (right).** Higher magnification of the inflamed area in Fig 1a. Observe the heavy inflammatory infiltrate (IN) and the aspiration of the odontoblast nuclei (N) into the dentinal tubuli (DT). H&E, 440 $\times$ .

All treatments were done with the monkeys anesthetized with sodium pentobarbitone (IV 50 mg/kg).

Preoperative radiographs were taken to assess the state of root development and the absence of pulp pathosis. Root development was complete in all teeth. The treatment was done under sterile conditions; the teeth were isolated with a rubber dam and cleaned with 2% chlorhexidine solution using a cotton swab. Access to the pulp chamber was gained by using a #330 bur mounted in a high-speed turbine handpiece with water coolant. After coronal pulp resection, which was done with a sterile round bur in a low-speed handpiece, the pulp stumps were rinsed with a sterile saline solution and dried with sterile cotton pellets till hemostasis was observed. A cotton pellet moistened with a freshly prepared 2% buffered glutaraldehyde solution<sup>13</sup> was placed over the radicular pulp stumps for 5 min. The pulp stumps were covered by a zinc oxide-eugenol paste and the cavities were filled with IRM<sup>a</sup> filling material. Control radiographs were taken immediately postoperatively and after 1 and 2 months.

The teeth were extracted 2 months postoperatively and the fillings were removed gently in order to facilitate fixation of the radicular pulp. The teeth were fixed in Bovin Holland solution and demineralized in 10% EDTA. After demineralization, the multirouted teeth were divided into individual roots; the roots

then were trimmed, embedded in paraplast, and cut longitudinally to obtain serial 6-micron thin sections. The sections were H&E stained and examined under a light microscope. The results were assessed by "blind testing" of the different preparations and ranged according to a modification of the criteria by Horsted et al.<sup>14</sup> as follows:

1. State of pulp vitality — presence and extent of necrosis
  - a. No necrosis
  - b. Partial necrosis — areas of necrosis at the wound surface or in part of the root pulp
  - c. Total necrosis
2. Presence and extent of inflammation
  - a. Absence of inflammation or a few inflammatory cells limited to the bridge area
  - b. Moderate inflammation evident below the bridge, but limited to the coronal third of the radicular pulp.
  - c. Severe inflammation and circulatory disturbances affecting most of the pulp
3. Presence of a dentin bridge
4. Presence of reparative dentin along the canal, below the dentin bridge area
5. Presence and regularity of an odontoblastic layer
  - a. Regular — present all along the root canal
  - b. Irregular — interrupted or existing in only part of the pulp canal
  - c. Absence of odontoblastic layer
6. Presence of calcifications in the pulp, not related to the bridge.

<sup>a</sup> IRM — LD Caulk Co: Milford, DE.

## Results

### Radiographic Findings

All the teeth presented a normal radiographic appearance with no signs of pathosis; however, dentin bridges could not be observed (Fig 2).

### Histologic Findings

A total of 16 roots were available for histologic evaluation in Group A and 15 in Group B. No appreciable difference could be observed between the 2 groups (Table 1). All teeth were vital and only 2 roots of Group B presented with disintegrating cell nuclei, an evidence of cell necrosis, but limited to the tissue that

had been in contact with the pulp dressing (Figs 3a, 3b). Vertical calcified tissue bands were observed in 1 tooth; these bands could not be interpreted as the expression of a dentin bridge. In addition, signs of osteoclastic activity could be detected as well as fibrosis (Figs 3b, 3c).

Incomplete dentin bridges were present in only 12.5% of the roots in Group A and in 6.6% of Group B (Figs 4a, 4b); these bridges appeared complete in some sections (Fig 4b). Absence of the odontoblastic layer was found in only 1 root of the induced inflammation group (Fig 3a). The reparative dentin was atubular, and in many cases bonelike (Fig 3c). Mild to moderate inflammation was seen in both groups (Figs 3a, 3b).

## Discussion

Formocresol pulpotomy has been utilized as the treatment of choice for vital pulpally exposed primary teeth, since conventional endodontic treatment is many times difficult to perform.<sup>15,16</sup> The remaining radicular pulp should be maintained healthy and free of inflammation, a condition that cannot be attained with the pulp dressing materials currently available.<sup>2,15,16-19</sup>

Glutaraldehyde has been proposed as a substitute for formocresol and its favorable results have been demonstrated in several in vitro and in vivo studies.<sup>9,10,13,20,21</sup> The findings of the present study confirm the results of previous reports where inflammation, when present, is limited to the area located immediately under the pulpotomy.<sup>9,20,21</sup>

The similar histologic picture of the radicular pulp in both groups indicates that the presence of induced inflammation in the coronal pulp did not affect the final outcome of the treatment, when glutaraldehyde

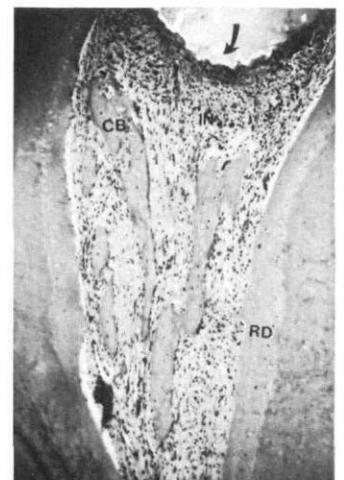


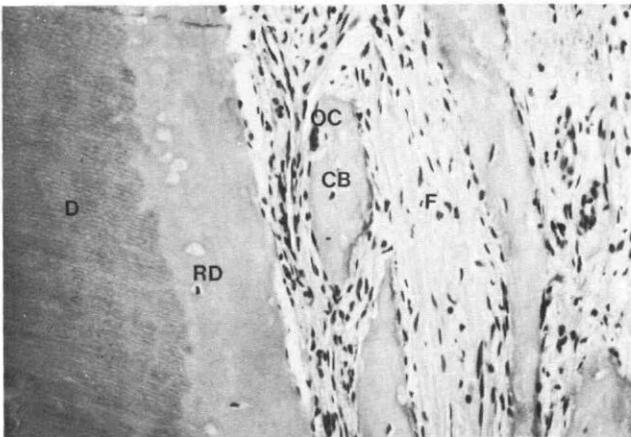
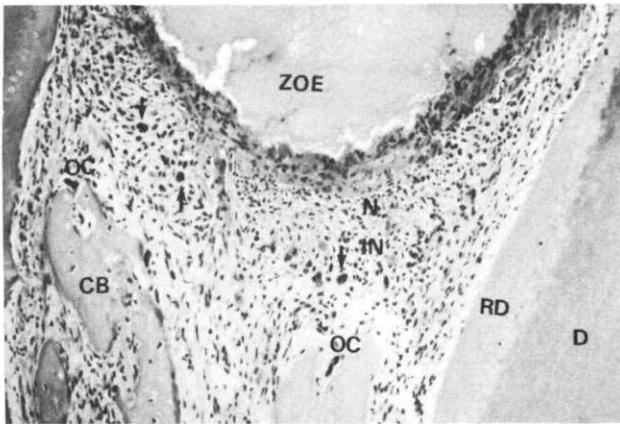
FIG 2. Periapical radiograph of the pulpotomized lower incisors. The pulp and periapical tissues appear normal; no dentin bridges are evident.

TABLE 1. Histologic findings.

		Group A (Inflamed)	Group B (Intact)
Number of roots examined		16	15
Pulp Vitality	No necrosis	16 (100%)	13 (86.7%)
	Partial necrosis	— (0%)	2 (13.3%)
	Total necrosis	— (0%)	0 (0%)
Inflammation	Absent or mild	10 (62.5%)	7 (46.6%)
	Moderate	6 (37.5%)	8 (53.4%)
	Severe	0 (0%)	0 (0%)
Presence of dentin bridge		2 (12.5%)	1 (6.6%)
Reparative dentin		15 (93.7%)	15 (100%)
Odontoblastic layer	Regular	7 (43.8%)	7 (46.6%)
	Irregular	8 (50.0%)	8 (53.4%)
	Absent	1 (6.2%)	0 (0%)

FIG 3a. Palatal root of a pulpotomized maxillary second molar of Group A showing an area of fixation (arrow); below it, an area of limited inflammation (IN) and necrotic cells, vertical calcified tissue bands (CB), and reparative dentin (RD). The odontoblastic layer has been destroyed and the predentin is missing. H&E, 100 $\times$ .





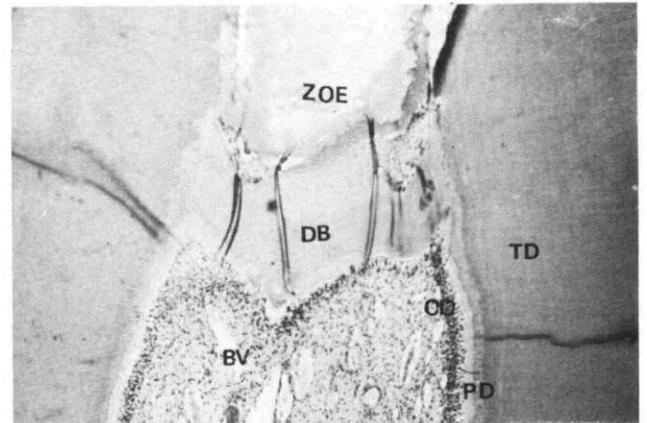
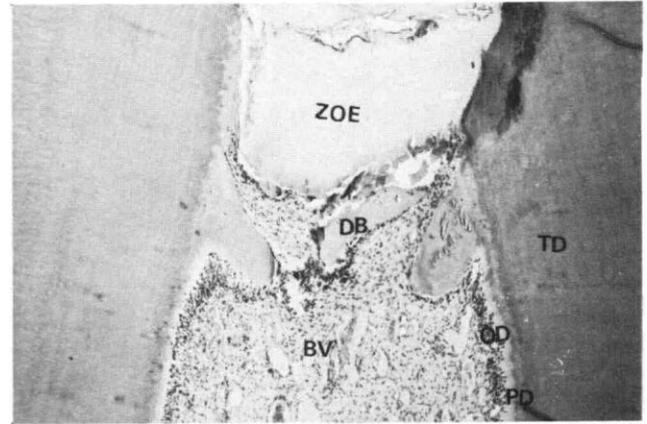
**FIG 3b (top).** Higher magnification of Fig 3a. Note remnants of the pulp dressing (ZOE) in contact with the fixed tissue (F); localized inflammation (IN) and disintegrating cell nuclei (N); giant cells (arrows); osteoclasts (OC); dentin (D); reparative dentin (RD); calcified tissue bands (CB). H&E, 440 $\times$ .

**FIG 3c (bottom).** Higher magnification of Fig 3a, showing fibrosis (F) and bone-like calcified tissue bands (CB) undergoing osteoclastic activity (OC); dentin (D); reparative dentin with cell inclusions (RD). H&E, 440 $\times$ .

is utilized as a pulp dressing. This fact may lead to the assumption that either the radicular pulp was normal in both groups or that eventual mild, localized inflammation in Group B was reduced after treatment.

The presence of reparative dentin in almost all of the treated teeth confirms that the fixative effect of glutaraldehyde is limited to the pulpotomy site. This has been attributed to the cross-linking properties of glutaraldehyde.<sup>9,20</sup>

Dentin bridges have been considered a sign of pulp healing after pulpotomy.<sup>22</sup> However, dentin bridge formation per se is not a sign of healing, since bridges have been found in monkey teeth which manifested chronic inflammation after formocresol pulpotomies.<sup>17</sup> The formation of dentin bridges has been reported to be induced by calcium hydroxide. Induction of hard tissue formation results from mild irritation



**FIG 4a & b.** Distal root of a pulpotomized mandibular second molar of Group B in process of dentin bridge formation: a. (top) Incomplete dentin bridge (DB) is evident below the pulp dressing (ZOE). b. (bottom) Adjacent section showing a complete dentin bridge (DB); normal odontoblastic layer (OD); predentin (PD); tubular dentin (TD); increased number of blood vessels (BV).

of coagulation necrosis caused by the calcium hydroxide. The coagulated tissue calcifies and dentin subsequently is formed by newly differentiated odontoblasts.<sup>23</sup> A more biologic mechanism of bridge formation was observed when an enriched native collagen solution was utilized; bridges resulted from cellular activity starting at the interface between the sound pulp odontoblasts and the collagenous dressing.<sup>8</sup>

The pulp response to glutaraldehyde may lie somewhere between these 2 response mechanisms. The fixative, nonbiologic properties of glutaraldehyde do not promote cell proliferation. However, its deleterious effects are limited to tissue fixation without causing coagulation necrosis, that ultimately mineralizes and forms part of the bridge, as in the calcium hydroxide pulpotomy procedure. The lack of necrosis may be the expression of a tissue response to a mild, self-limited irritant, without the formation of a dentin bridge. This might be the reason for the lack of bridge formation in most of the teeth treated in the present

study. A longer follow-up time could allow for development of these bridges in additional teeth.

The overall appearance of the material examined demonstrated a positive tissue response to glutaraldehyde, since all the teeth were vital and the inflammation, when present, was mild and limited in both groups.

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