

**ENDODONTICS***Editor: Richard E. Walton***Use of mineral trioxide aggregate for repair of furcal perforations**

Thomas R. Pitt Ford, BDS, PhD,<sup>a</sup> Mahmoud Torabinejad, DMD, MSD,<sup>b</sup>  
 Douglas J. McKendry, DDS,<sup>c</sup> Chan-Ui Hong, DDS, MS,<sup>d</sup> and Stalin P. Kariyawasam,<sup>e</sup>  
 London, England, Loma Linda, Calif., and Seoul, South Korea.  
 UNITED MEDICAL AND DENTAL SCHOOLS, LONDON, LOMA LINDA UNIVERSITY SCHOOL OF  
 DENTISTRY, AND DANKOOK UNIVERSITY SCHOOL OF DENTISTRY

The histologic response to intentional perforation in the furcations of 28 mandibular premolars in seven dogs was investigated. In half the teeth, the perforations were repaired immediately with either amalgam or mineral trioxide aggregate; in the rest the perforations were left open to salivary contamination before repair. All repaired perforations were left for 4 months before histologic examination of vertical sections through the site. In the immediately repaired group, all the amalgam specimens were associated with inflammation, whereas only one of six with mineral trioxide aggregate was; further, the five noninflamed mineral trioxide aggregate specimens had some cementum over the repair material. In the delayed group, all the amalgam specimens were associated with inflammation; in contrast only four of seven filled with the aggregate were inflamed. On the basis of these results, it appears that mineral trioxide aggregate is a far more suitable material than amalgam for perforation repair, particularly when used immediately after perforation. (*ORAL SURG ORAL MED ORAL PATHOL ORAL RADIOL ENDOD* 1995;79:756-62)

In endodontic practice, procedural accidents such as furcal perforation may occur and affect the prognosis of root canal treatment. In an analytic study of endodontic failures, Ingle<sup>1</sup> reported that perforations were the second greatest cause of endodontic failure and account for 9.6% of all unsuccessful cases.

Perforations, especially in the furcation during endodontic treatment, have a notably detrimental effect on prognosis. Several studies<sup>2-4</sup> have shown that furcal perforation predisposes a tooth to periradicular breakdown and eventual loss of periodontal attachment, which in most instances is irreparable and frequently leads to loss of the tooth.

Periodontal tissue reactions to experimentally induced perforations have been studied in dogs<sup>3, 5-11</sup> and

monkeys<sup>2, 12, 13</sup>; some clinical investigations of root perforations have also been done in human beings.<sup>4, 14</sup> In general, investigators have agreed that the prognosis for root perforations in the apical and middle third of the root was much better than those in the cervical third of the root or in the floor of the pulp chamber. Immediate repair of these perforations has also been recommended.<sup>14</sup>

Materials such as Cavit (Espe, Seefeld, Germany),<sup>6, 7</sup> zinc oxide-eugenol,<sup>2, 11</sup> calcium hydroxide,<sup>7, 9, 11</sup> amalgam,<sup>7, 10, 13</sup> gutta-percha,<sup>5, 10</sup> tricalcium phosphate,<sup>9, 13</sup> and hydroxyapatite<sup>13</sup> have been used to repair furcation perforations. Despite its shortcomings, amalgam has been the standard material for repairing furcal perforations.<sup>7, 13</sup>

Clinical reports indicate that furcal perforations have usually been treated conservatively.<sup>4, 14</sup> However, histologic studies have often demonstrated unfavorable tissue responses at the treatment sites.<sup>3, 5, 7, 8</sup> The aim of treatment of furcal perforations is to maintain healthy tissues against the perforation without continuing inflammation or loss of periodontal attachment. In the case of already existing lesions, the aim is to re-establish tissue attachment, which has proved to be extremely difficult to achieve with available materials.

<sup>a</sup>Senior Lecturer, Department of Conservative Dental Surgery, UMDS, London.

<sup>b</sup>Professor, Department of Endodontics, Loma Linda University.

<sup>c</sup>Associate Professor, Department of Endodontics, Loma Linda University.

<sup>d</sup>Associate Professor, Dankook University School of Dentistry, Seoul.

<sup>e</sup>Senior Chief Laboratory Scientific Officer, Division of Anatomy and Cell Biology, UMDS, London.

Copyright © 1995 by Mosby-Year Book, Inc.  
 1079-2104/95/\$3.00 + 0 7/15/63817

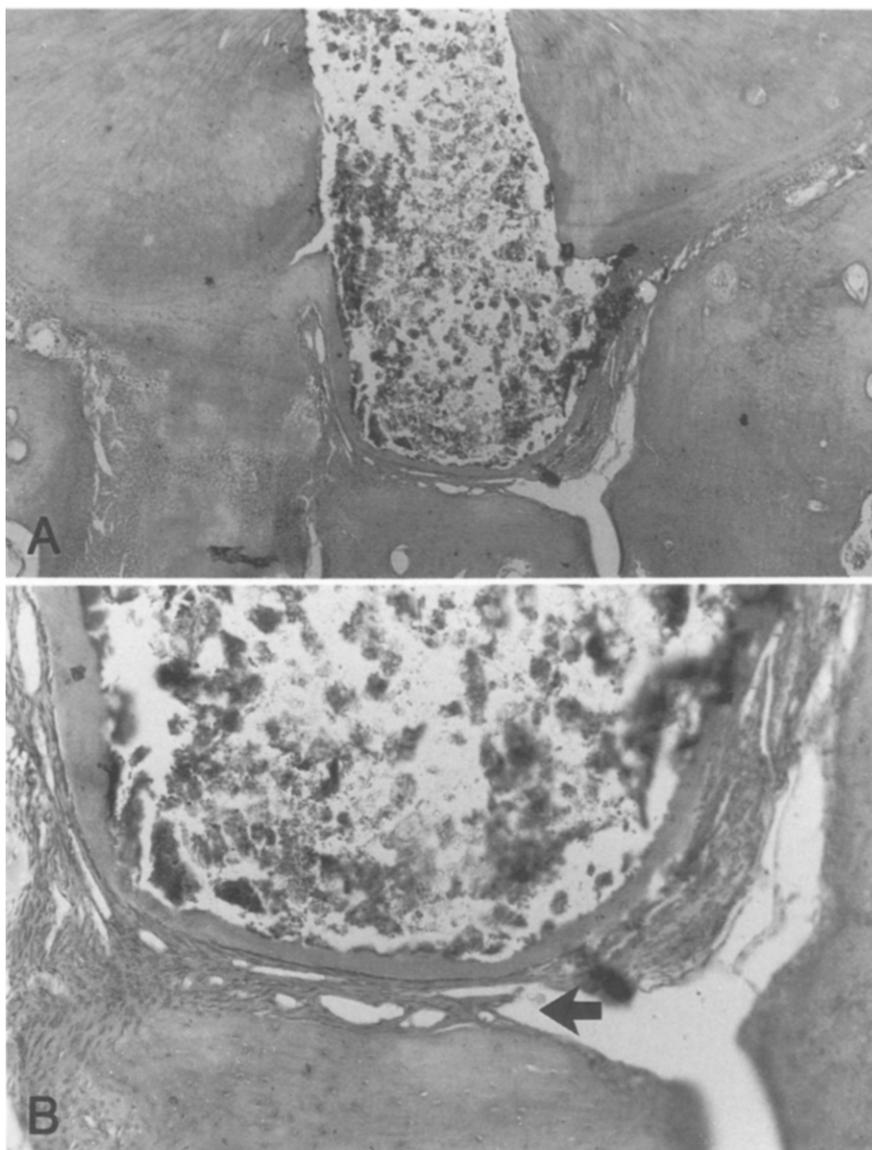


Fig. 1. A, Perforation filled immediately with MTA shows a layer of continuous new cementum over the excess material. (Original magnification  $\times 20$ .) B, Higher magnification ( $\times 50$ ) shows narrow periodontal ligament space (arrow) between cementum and bone. (Hematoxylin and eosin stain.)

**Table I.** Number of specimens that show inflammation, cementum over the repair material, epithelial proliferation, and detectable bacteria for the two materials under immediate and delayed conditions

Material	Conditions	Number of specimens	Inflammation	Cementum over material	Epithelial proliferation	Bacteria detected
Amalgam	Immediate	7	7	0	1	0
MTA	Immediate	6	1	5	0	0
Amalgam	Delayed	8	8	0	3	3
MTA	Delayed	7	4	2	2	0

Recently, a new material, mineral trioxide aggregate (MTA), has been developed at Loma Linda University, to fill and seal root end cavities during endodontic surgery. Encouraging results have been found for the

sealing ability in extracted teeth<sup>15,16</sup>; furthermore, histologic findings of preparations carried out in dogs have confirmed laboratory observations that this material has great potential to facilitate tissue healing.<sup>17</sup>

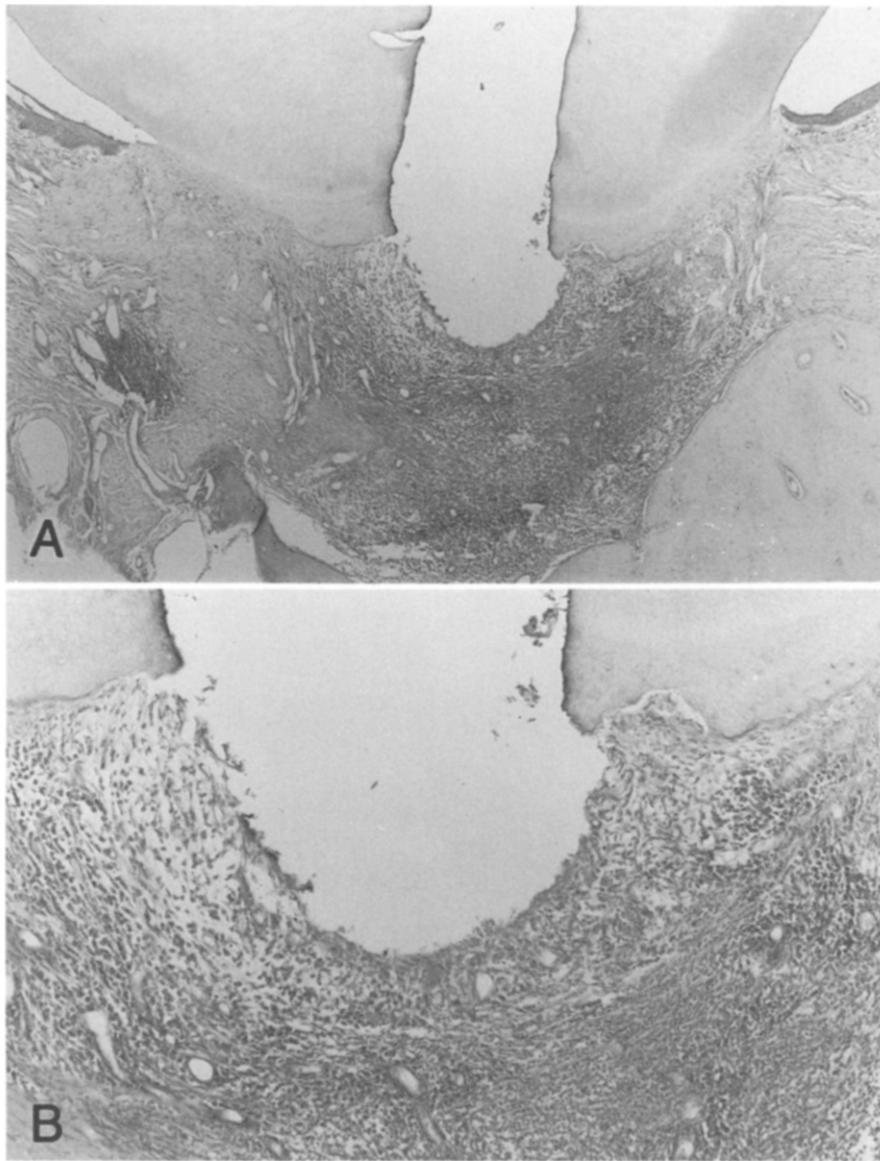


Fig. 2. A, Perforation filled immediately with amalgam shows severe inflammation extending  $<0.5$  mm. (Original magnification  $\times 20$ .) B, Higher magnification ( $\times 50$ ) shows absence of cementum around repair material. (Hematoxylin and eosin stain.)

**Table II.** Severity and extent of inflammation (when found) for the two materials under immediate and delayed conditions

Material	Conditions	Inflammation							
		Severity				Extent (mm)			
		None	Few	Moderate	Severe	0	$<0.2$	$<0.5$	$>0.5$
Amalgam	Immediate	0	1	2	4	0	1	6	0
MTA	Immediate	5	0	1	0	0	1	0	0
Amalgam	Delayed	0	0	2	6	0	3	3	2
MTA	Delayed	3	0	2	2	3	3	1	0

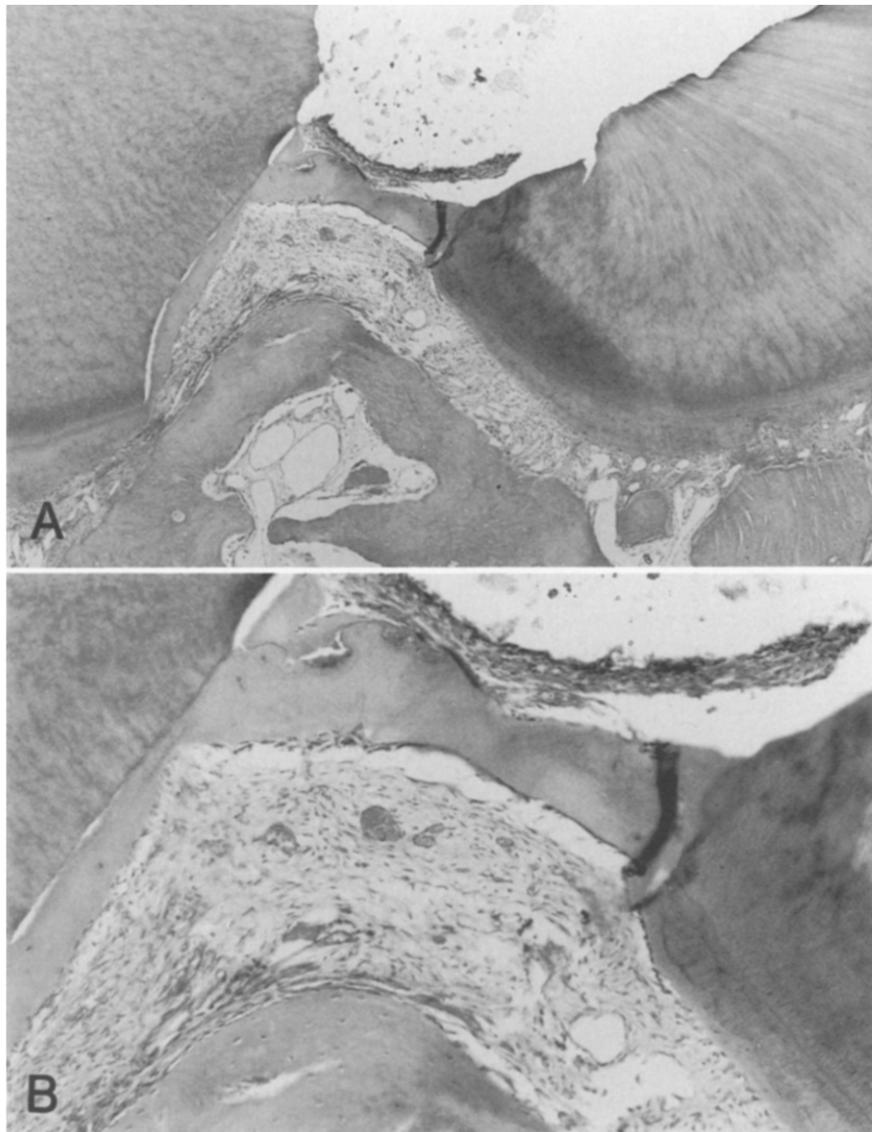


Fig. 3. **A**, Perforation filled after 6 weeks with MTA shows layer of new cementum over material. (Original magnification  $\times 20$ .) **B**, Higher magnification ( $\times 50$ ) shows a bridge of cementum with cells beyond and adjacent to the material; no inflammation is seen. (Hematoxylin and eosin stain.)

The purpose of this study was to examine histologically the tissue response to experimentally induced furcal perforations, repaired with amalgam or MTA either immediately or after salivary contamination.

#### MATERIAL AND METHODS

A total of 30 furcal perforations were made in mandibular premolars in seven adult beagle dogs. With the dog under general anaesthesia with sodium pentobarbital (30 mg/kg body weight), the root canals in mandibular premolars were cleaned, shaped, and obturated with gutta-percha and sealer. A trephine bur (ISO size 014) was used at low speed to

make a perforation through the floor of the pulp chamber into the furcation. In half the teeth, the perforation was filled immediately with amalgam (Sybraloy, Kerr, Romulus, Mich.) or MTA (Loma Linda University, Calif.) after control of hemorrhage by rinsing with saline solution and use of cotton pellets. In the rest of the teeth, the perforations were left open to salivary contamination for 6 weeks to allow for bacterial contamination and the formation of inflammatory lesions in the furcation. The presence of a lesion was confirmed by radiographic evidence of bone loss. These perforations were then cleaned by copious irrigation with sodium hypochlorite, dried, and filled

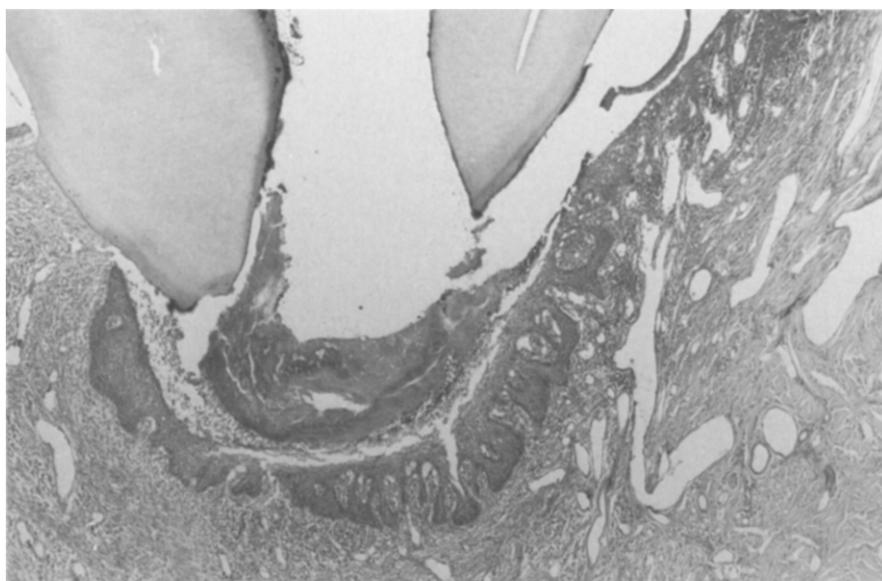


Fig. 4. Perforation filled after 6 weeks with amalgam shows epithelial proliferation from the gingival sulcus (right); inflammation is present below; there is infected necrotic tissue between epithelium and perforation. (Hematoxylin and eosin stain; original magnification  $\times 20$ .)

with amalgam or MTA. The access cavities were filled with MTA. The animals were killed 4 months later, and the jaws perfused with 10% buffered formalin; the respective sections of mandible were removed and placed in 10% buffered formalin before demineralization in formic acid buffered with sodium citrate. Tissue blocks were then dehydrated and embedded in paraffin. Longitudinal step-serial sections were prepared buccolingually at  $8 \mu\text{m}$  through the area of furcal perforation. Slides were stained with hematoxylin and eosin, Masson's trichrome, and by the Brown and Brenn method. Sections were examined by two observers independently; in cases of disagreement, specimens were reexamined jointly. They were assessed for the presence of inflammation, its maximum severity, and extent at the site of perforation from all the sections examined according to previously used criteria.<sup>18</sup> The presence of cementum covering the repair was also noted together with the presence of epithelium and detection of bacteria at the site.

The number and distribution of teeth available for histologic examination is given in Table I; two specimens were lost during preparation.

## RESULTS

The number of teeth with inflammation at the furcation after 4 months is shown in Table I. The extent and severity of inflammation is reported in Table II. In teeth repaired immediately with MTA, the re-

sponse was characterized by a lack of inflammation and formation of cementum in five of six teeth (Fig. 1), whereas amalgam specimens were always associated with inflammation that was often moderate or severe (Fig. 2). In the specimens where repair was delayed, three of seven filled with MTA were free from inflammation (Fig. 3), but four were inflamed. In contrast, teeth repaired with amalgam were always associated with inflammation (Fig. 4) that was frequently severe and more extensive. Table I gives details on the presence of cementum over the material, epithelial proliferation, and detection of bacteria at the site of perforation.

## DISCUSSION

Experimental studies of the treatment of furcal perforations have invariably produced disappointing results,<sup>7, 10, 13</sup> so it was with some surprise that the favorable response to MTA in the immediately filled group was noted; five of six specimens were free of inflammation. Furthermore, these specimens all demonstrated some evidence of cementum formation that was continuous with the cementum covering the root surface. As with the hard tissue bridges formed in pulp capping,<sup>19</sup> the hard tissue layer in some specimens was incomplete in serial sections. It is possible that it would have been complete had the follow-up period been longer. Where the MTA had been extruded into the bony defect, the cementum had formed around the excess material (Fig. 1). An

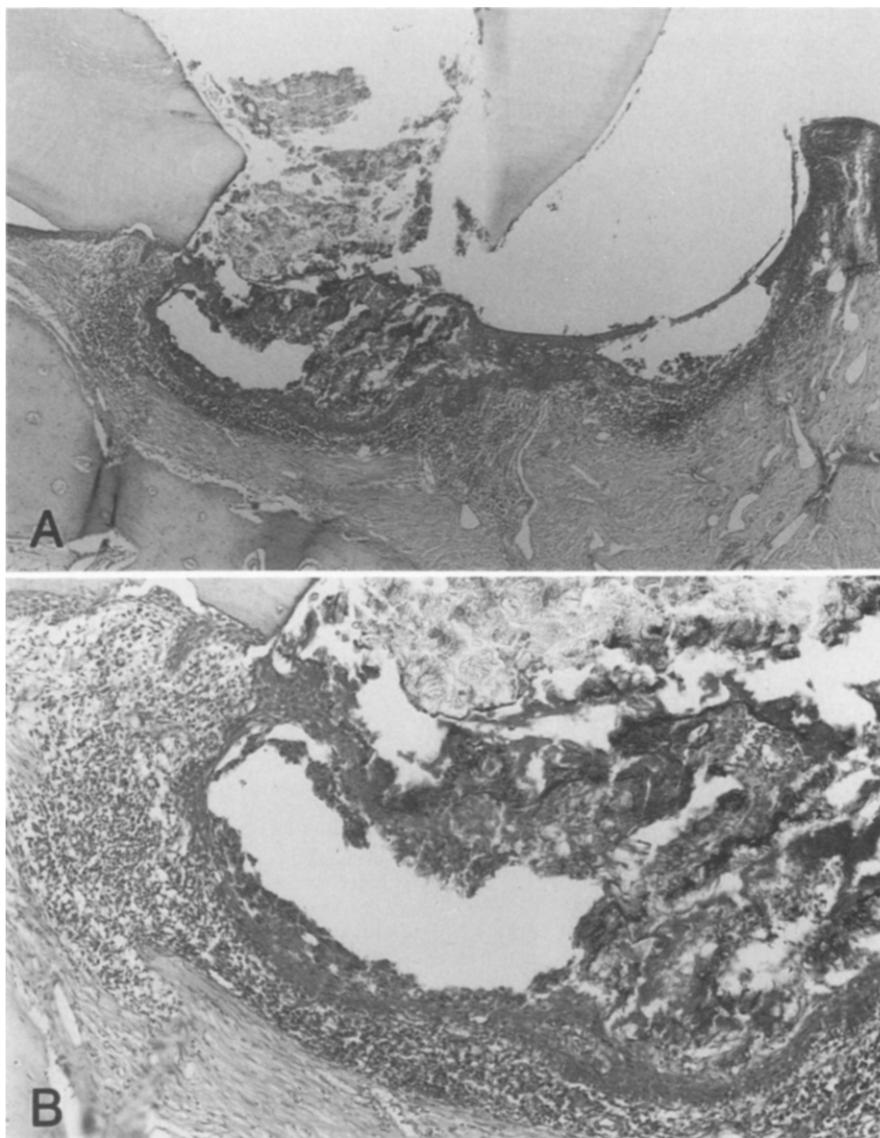


Fig. 5. A, Perforation filled after 6 weeks with MTA shows discharging sinus tract (right) and abscess cavity under perforation. (Original magnification  $\times 20$ .) B, Higher magnification ( $\times 50$ ) shows repair material, abscess cavity, epithelium, and surrounding inflammation. (Hematoxylin and eosin stain.)

apparent periodontal ligament separated this cementum from bone; the appearance was very similar to that observed at root ends filled with MTA.<sup>17</sup>

Only one<sup>11</sup> of a large number of experimental studies into furcal perforations has reported the presence of new cementum formed against the repair material, a mixture of calcium hydroxide and iodoform. The use of calcium hydroxide has been investigated in two studies<sup>7,9</sup> in which worse results were found than with other materials investigated; this could possibly have been due to the nonsetting nature of the material, difficulty of packing, and its known initial caus-

tic effect on vital tissue.<sup>20</sup> Unlike calcium hydroxide, MTA sets hard within 4 hours, therefore it provides a solid barrier against which tissue can organize, and like calcium hydroxide it also has a high pH.<sup>21</sup>

Clinicians refer to the size of perforations that affect prognosis, but as in pulp-capping studies there is no substantive evidence from the literature to support it. The size of perforations in this investigation was standardized at 1.4 mm in common with previous studies.<sup>7,9,10,13</sup>

The poor prognosis of furcal perforations is without doubt because of the crucial role of infection; this has

been particularly marked in unfilled controls in previous studies.<sup>2,7</sup> The majority of experimental studies have not specifically sought to eliminate bacterial leakage around the restoration in the access cavity, and therefore it is possible that as a consequence of nonsealing materials used for repair of the perforation, the tissue response has been poor.<sup>7,10,13</sup> Two groups of workers<sup>12,13</sup> stained their histologic sections for bacterial contamination and found that a proportion of specimens were contaminated. In this study, bacteria could only be detected by staining in 3 of the 20 teeth that demonstrated inflammation. Bacterial stains are known not to be completely reliable particularly adjacent to amalgam,<sup>18</sup> but their ability to demonstrate bacteria was lower than in an earlier investigation<sup>22</sup> of root apices when the same laboratory method was used.

In the group in which repair was delayed, epithelial proliferation was observed in 5 of 12 inflamed specimens. The presence of epithelial proliferation has been reported in previous studies.<sup>7-10,13</sup> In Fig. 2, a furcation of a dog mandibular premolar shows clearly in the buccolingual section just how little area of periodontal fiber attachment is present. The dog is a severe experimental model because of anatomic relations. If a treatment procedure or material can succeed in this model, it should succeed in man where the furcation is usually deeper into the alveolus. In inflamed furcations it was not surprising to see epithelial proliferation from the gingival sulcus (Fig. 4); in the more advanced lesions epithelium almost joined both buccal and lingual gingivae. Fig. 5 shows a sinus tract from the perforation site to the gingival sulcus in a specimen after delayed filling with MTA. It had been decided to prepare histologic sections buccolingually in an attempt to observe any sinus tracts; in contrast most previous investigators prepared sections mesiodistally.<sup>2,7,10,13</sup>

When perforations are allowed to remain open to saliva, the prognosis is much poorer,<sup>4,14</sup> and that was mirrored in this study. Nicholls<sup>14</sup> recommended that contaminated perforations were washed out with hypochlorite or hydrogen peroxide, although few, if any, investigators seem to have followed his recommendations. The good response in three of the specimens in which repair with MTA was delayed could probably be attributed to the effective use of hypochlorite irrigation. With hindsight in the delayed group the gingival sulci should have been probed before repair to observe if pockets had already formed. If they had, then any conservative treatment would have been compromised. It is interesting to speculate if such teeth could be successfully treated by obturation of

the repair with MTA and combined surgical curettage of the pocket.

## CONCLUSIONS

Histologic evidence has shown that MTA has potential as a material for immediate repair of furcal perforations. In particular, five of six teeth had some cemental repair over the material. When repair of furcations was delayed, more specimens were associated with inflammation that appeared to be linked to infection. Overall, MTA allowed a considerably more favorable response than amalgam.

## REFERENCES

1. Ingle JJ. A standardized endodontic technique utilizing newly designed instruments and filling materials. *ORAL SURG ORAL MED ORAL PATHOL* 1961;14:83-91.
2. Seltzer S, Sinai I, August D. Periodontal effects of root perforations before and during endodontic procedures. *J Dent Res* 1970;49:332-9.
3. Bhaskar SN, Rappaport HM. Histologic evaluation of endodontic procedures in dogs. *ORAL SURG ORAL MED ORAL PATHOL* 1971;31:526-35.
4. Meister F, Lommel TJ, Gerstein H, Davies EE. Endodontic perforations which resulted in alveolar bone loss. *ORAL SURG ORAL MED ORAL PATHOL* 1979;47:463-70.
5. Lanz B, Persson PA. Periodontal tissue reactions after root perforations in dogs' teeth: a histologic study. *Odontol Tidskr* 1967;75:209-20.
6. Jew RCK, Weine FS, Keene JJ, Smulson MH. A histologic evaluation of periodontal tissues adjacent to root perforations filled with Cavit. *ORAL SURG ORAL MED ORAL PATHOL* 1982;54:124-35.
7. ElDeeb ME, ElDeeb M, Tabibi A, Jensen JR. An evaluation of the use of amalgam, Cavit, and calcium hydroxide in the repair of furcation perforations. *J Endodon* 1982;8:459-66.
8. Petersson K, Hasselgren G, Tronstad L. Endodontic treatment of experimental root perforations in dog teeth. *Endod Dent Traumatol* 1985;1:22-8.
9. Himel VT, Brady J, Weir J. Evaluation of repair of mechanical perforations of the pulp chamber floor using biodegradable tricalcium phosphate or calcium hydroxide. *J Endodon* 1985;11:161-5.
10. Aguirre R, ElDeeb ME, ElDeeb M. Evaluation of the repair of mechanical furcation perforations using amalgam, gutta-percha, or indium foil. *J Endodon* 1986;12:249-56.
11. Bramante CM, Berbert A. Root perforations dressed with calcium hydroxide or zinc oxide and eugenol. *J Endodon* 1987;13:392-5.
12. Beavers RA, Bergenholtz G, Cox CF. Periodontal wound healing following intentional root perforations in permanent teeth of *Macaca mulatta*. *Int Endod J* 1986;19:36-44.
13. Balla R, LoMonaco CJ, Skribner J, Lim LM. Histological study of furcation perforations treated with tricalcium phosphate, hydroxyapatite, amalgam, and Life. *J Endodon* 1991;17:234-8.
14. Nicholls E. Treatment of traumatic perforations of the pulp cavity. *ORAL SURG ORAL MED ORAL PATHOL* 1962;15:603-12.
15. Torabinejad M, Watson TF, Pitt Ford TR. Sealing ability of a mineral trioxide aggregate when used as a root end filling material. *J Endodon* 1993;19:591-5.
16. Torabinejad M, Higa RK, Pitt Ford TR. Dye leakage of four root end filling materials: effect of blood contamination. *J Endodon* 1994;20:159-63.
17. Pitt Ford TR, Torabinejad M, Hong CU, Kariyawasam SP.

- Assessment of mineral trioxide aggregate as a retrograde root filling. *J Dent Res* 1994;73:804.
18. Pitt Ford TR, Andreasen JO, Dorn SO, Kariyawasam SP. Effect of IRM root end fillings on healing after replantation. *J Endodon* 1994;20:381-5.
  19. Cox CF, Bergenholtz G, Heys DR, Syed SA, Fitzgerald M, Heys RJ. Pulp capping of the dental pulp mechanically exposed to the oral microflora: a 1-2 year observation of wound healing in the monkey. *J Oral Pathol* 1985;14:156-68.
  20. Schröder U, Granath LE. Early reaction of intact human teeth to calcium hydroxide following experimental pulpotomy and its significance to the development of hard tissue barrier. *Odontol Revy* 1971;22:379-96.
  21. Torabinejad M, Hong CU, McDonald F, Pitt Ford TR. Physical and chemical properties of a new root end filling material. *J Endodon* (in press).
  22. Pitt Ford TR. The effects on the periapical tissues of bacterial contamination of the filled root canal. *Int Endod J* 1982;15:16-22.

*Reprint requests:*

T. R. Pitt Ford, BDS, PhD  
Department of Conservative Dental Surgery  
UMDS, Guy's Hospital  
London SE1 9RT  
England