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We evaluated the histopathological response of the periodontal membrane to intentionally-replanted teeth carrying composite (experimental) and silver amalgam (control) restorations in the middle third of each root. The study revealed that the amalgam produced, in the periodontal tissues, an initial localized inflammation that subsided with the subsequent formation of a fibrous capsule. However, the periodontal membrane adjacent to the composite resin restorations demonstrated chronic inflammation. It was concluded that the composite evoked chronic inflammatory responses of the periodontal tissues in monkeys.

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#### Introduction.

Most of the commercial composite resins used in restorative dentistry today are based on the formulation developed by Bowen in 1962.<sup>1</sup> Composite resins may be described as polymers derived from the aromatic dimethacrylates, principally BIS-GMA (an adduct of bisphenol-A and glycidyl methacrylate). BIS-GMA composites contain fillers coated with a silane coupling agent which bonds the individual filler particles to the resin matrix. Fillers which may be added include fused silica, crystalline quartz, borosilicate glass, and other materials.<sup>1,2</sup>

Composite resin restorations are being utilized extensively in dentistry, because they are dimensionally more stable than either unreinforced methyl methacrylate resins or silicate cements. They offer better color stability and margin integrity, and, therefore, less margin stain and recurrent caries.<sup>3</sup> According to Rupp,<sup>4</sup> composite restorations placed with care will provide long-lasting esthetic restorations, provided they are not placed in areas subjected to load-bearing abrasion. He suggested that composite restorations should be limited to Class III, Class V, and Class IV esthetic restorations.

Based on mechanical and physical properties, the composite resins rank high; however, based on biological acceptance, they rank low. Restorative composites usually cause a degree of pulp inflammation, unless the cavity is shallow or a protective base or liner is used.<sup>5</sup> These pulp responses are similar, whether the composites are self-curing<sup>6</sup> or require ultra-violet radiation for polymerization and hardening.<sup>7</sup> Patient discomfort and histopathologic degenerative changes were observed when a composite resin, employing an activation system, was used in deep cavities.<sup>8</sup> It has been suggested that the pulp response to composites may be due – in certain situations at least – not only to components in the resin itself, but also to microorganism penetration around the restoration.<sup>9</sup> Furthermore, Blank  $et al.^{10}$  demonstrated gingival inflammation associated with well-finished and -contoured composite restorations placed subgingivally.

The purpose of the present study was to evaluate and compare the monkey periodontal membrane response of a new composite formulation with the periodontal response of silver amalgam. The study involved a histopathologic evaluation of the periodontal membrane of intentionally-replanted teeth carrying both composite resin (experimental) and silver amalgam (control) restorations in the middle third of each root, using the same experimental model that was successfully applied to study the effects both of a self-curing acrylic<sup>11</sup> and of silver amalgam restorations on the periodontal tissues of monkeys.<sup>12</sup>

### Materials and methods.

Eight healthy male rhesus monkeys (Macaca mulatta) were used for this study. All the monkeys were adults and were approximately the same size and weight. Each monkey had a full complement of permanent teeth with moderate supra- and subgingival calculus and generalized marginal gingivitis. One wk prior to the experiment, all teeth were scaled and polished. Also at this time, intra-oral radiographs were taken for the purpose of studying the general morphology of the maxillary incisor teeth and surrounding bony structures. A step-by-step sequence of the methods used for tooth replantation was reported previously.13 Briefly, after extractions, the two maxillary central incisors of each monkey were treated endodontically with a root canal sealer<sup>†</sup> and gutta percha points. Subsequently, in these treated teeth, cavities were prepared on the mesial and distal aspects of the roots, and were filled with composite in one cavity and with silver amalgam in the other. The cavities (measuring roughly 3 mm in diameter by 2 mm in depth) were placed approximately half-way between the cemento-enamel junction and the apex, and were prepared with a 588 carbide bur. No further attempt was made to smooth the walls of the cavities. They were packed with composite<sup>‡</sup> and with silver amalgam,<sup>§</sup> with a 1:1 mercuryalloy ratio. During setting, the composite restoration was kept in the cavity with a matrix, and the amalgam (contoured with pluggers and scalers) was left unpolished. The recommendations and instructions of the manufacturers were followed for both the composite and the amalgam restorative materials, especially regarding time intervals relative to mixing and placement procedures. After the restorations had set, the teeth were immediately replanted. Each tooth was, at most, one h out of its socket. Immobilization of the tooth was achieved with an interproximal acid-etch splint.<sup>14</sup> The splints were removed in one wk, at which time all the replanted teeth were firm in their sockets. The monkeys received no medication after

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<sup>&</sup>lt;sup>†</sup>Kerr Pulp Canal Sealer<sup>®</sup>, Kerr, Dental Division of Sybron Corporation, Romulus, MI

<sup>&</sup>lt;sup>4</sup>Merdonite – Merdon Super C<sup>®</sup>, AMCO – American Consolidated Manufacturing Co., Philadelphia, PA

<sup>&</sup>lt;sup>8</sup>New True Dentalloy<sup>®</sup>, S.S. White, Dental Products Division of Pennwalt Corporation, Philadelphia, PA

surgery, and they were given Purina Monkey Chow<sup>+</sup> and water ad libitum. They were sacrificed by exsanguination at seven and 15 d, and at one, two, three, four, five, and six mo post-operatively. The maxillary incisors, with the surrounding bone and soft tissues, were excised in block sections. They were fixed in 10% buffered formalin solution and were demineralized in 4% formic acid. After demineralization, the restorations were removed from their beds to avoid tearing the surrounding tissues in the process of sectioning. Each tooth, with attached soft tissues, was cut mid-sagitally into mesial and distal halves, and the restorations were subsequently retrieved from the opened pulpal walls with the aid of a dental explorer (Fig. 1). This operation produced a total of 32 specimens - 16 for evaluation of composite sites and 16 for amalgam sites. These specimens, embedded in paraffin, were sectioned serially, longitudinally, and mesiodistally at 6  $\mu$ , and were stained with Ehrlich's acid hematoxylin and eosin, for histological examination. Twenty-five microscopic sections were evaluated for each of the experimental and control sites.

The inflammatory response of the periodontal membrane which came into contact with the restorative materials used was the primary consideration in the evaluation of the microscopic findings. An arbitrary classification of mild, moderate, and severe was made, based on earlier work.<sup>11,12</sup> The factors considered in the classification were: (1) the type and intensity of inflammatory cell infiltration of the tissues in contact with the restorative materials; (2) the thickness and type of fibroplasia of the capsule, if present, in contact with the materials; and (3) the degree of vascularity of the tissues.

# Results.

The healing process of the periodontal membrane, as a result of tooth replantation, was uneventful and coincided with sequential stages already described in the literature.<sup>15</sup> It was noted that no inflammatory cells were present within the confines of the periodontal membrane in areas coronal and apical to the amalgam and the composite restorations, in 15-day to six-month specimens. Briefly, the marginal tissues reattached to the teeth in seven d. The connective tissue fibers were restored in 15 d, but without displaying their normal arrangement and maturity. Complete maturity was seen only in the four-, five-, and six-month specimens. At these times, new cementum and bone were deposited, covering areas of previous resorption, and no areas of dento-alveolar ankylosis were found.

Histological sections of the seven-day post-operative specimens disclosed, at both the amalgam and the composite sites, a fibrous clot which was undergoing organization. Intense, acute leukocytic infiltration was present. Between this zone and the interdental septum of the alveolar bone, the connective tissue seemed edematous, and the periodontal fibers appeared somewhat disoriented. The original connective tissue outline, however, was preserved without noticeable sloughing.

Healing in the 15-day specimens, at both the amalgam and the composite sites, was considerably advanced over that observed in the seven-day specimens. The collagen fibers of the periodontal membrane showed increased cellularity and a definite tendency toward orientation. There was also evidence of vascular proliferation, since many more blood vessels were seen in the adjacent periodontal tissues. However, lymphocytic and histocytic inflammatory cells were still present to an intense degree along the control and the experimental sites.

A thin, well-defined fibrous capsule of from 20 to 40  $\mu$  in thickness had formed at the amalgam site one mo after surgery. This capsule isolated the amalgam from the adjacent periodontal tissues, and it showed evidence of a low-grade chronic inflammation, with a few plasmocytic cells. Conversely, the periodontal tissues adjacent to the composite restoration showed an intense inflammatory infiltrate.

In two-month specimens, a fibrous capsule of from 40 to 60  $\mu$  thick developed at the amalgam site, filling the periodontal membrane space almost completely, but there was no evidence of inflammation in its connective tissue fibers. The connective tissue adjacent to the composite restoration showed a severe and active chronic inflammation, containing polymorphonuclear leukocytes and mononuclear cells.

In three- to six-month specimens, at the amalgam site, the findings were similar to those of two-month specimens. A well-organized fibrous capsule was the consistent finding, with no inflammatory evidence in its fibrous connective

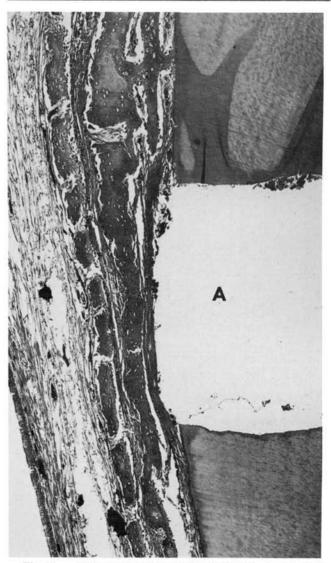


Fig. 1 – Four-months-post-operative specimen. A, the space remaining after the removal of the amalgam restoration. (H&E stain; original magnification 30x).

<sup>&</sup>lt;sup>+</sup>Ralston-Purina Company, St. Louis, MO

tissues (Fig. 2). At the composite resin site, however, an inflammatory cell infiltration was still evident (Fig. 3).

## Discussion.

In the present study, the healing process of the periodontal membrane, as a result of tooth replantation, was uneventful and coincided with sequential stages already described.<sup>15</sup> The periodontal membrane response to the silver amalgam showed a moderate degree of inflammation around the early specimens; however, there was no evidence of inflammation in the fibrous capsule, nor in the adjacent periodontal tissues in the two- to six-month specimens. Conversely, the one- to six-month specimens of the periodontal tissues adjacent to the composite restorations consistently demonstrated severe and active chronic inflammation.

It was noted that the periodontal tissue responses elicited by the composite were consistently more intense than those provoked by cold-curing acrylic<sup>11</sup> and silver amalgam

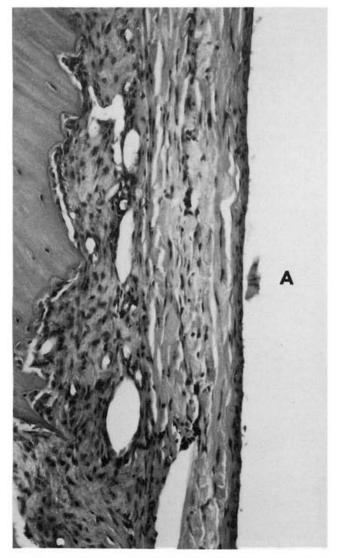


Fig. 2 – Six-months-post-operative specimen. The fibrous connective tissue adjacent to the amalgam (A) restoration showed no inflammation. (H&E stain; original magnification 150x).

restorations.<sup>12</sup> The persistence of a severe chronic inflammatory response to the composite resin may be due to the continued breakdown or release of irritant products from the restoration. It is possible that the composite was undergoing continuing physical-chemical changes resulting in the release of irritant substances. Since we used a cold-cured composite, which is polymerized at room temperature, there could be a possibility for incomplete polymerization, leaving behind monomers which may have caused the observed tissue responses, *e.g.*, remaining unreacted methacrylate groups.<sup>16,17</sup>

The irritating qualities of the composite resins have been well documented in regard to their effects upon the dental pulp.<sup>18-20</sup> Most composites, if not properly lined, cause chronic pulpitis.<sup>2-8</sup> Recent attempts to remove methacrylic acid from the formulations were ineffective in preventing pulpal irritation. Stanley *et al.*<sup>6</sup> found that certain new composites, although free of methacrylic acid and having a neutral pH, were still toxic to the pulp. The intensity of

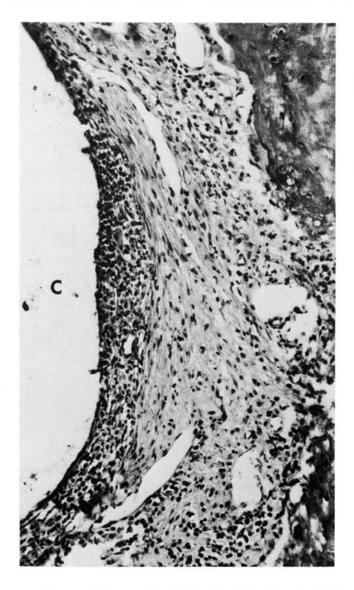


Fig. 3 – Six-months-post-operative specimen. The periodontal tissues adjacent to composite (C) showed a severe chronic cellular inflammatory reaction. (H&E stain; original magnification 150x).

the pulpal response increased after acid pre-treatment procedures.<sup>6</sup> However, when eight basic ingredients of composite resins were individually evaluated for pulp irritation, the study demonstrated, surprisingly enough, that none causes significant inflammation as reflected in its average response. The authors found no abscess formations or lesions predominating in leukocytes.<sup>5</sup>

## Conclusions.

The study showed that the healing process of the periodontal membrane, as a result of tooth replantation, was uneventful. The periodontal membrane response to the amalgam restorations showed that, at two mo and thereafter, there was no evidence of inflammatory infiltrate either in the fibrous capsule or in the adjacent connective tissues. However, in the one- to six-month specimens, the periodontal membrane adjacent to the composite resin restorations demonstrated persistent chronic inflammation. Thus, it was concluded that the composite evoked chronic inflammatory responses of the periodontal tissues in monkeys.

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