Titanium endodontic implants: a scanning electron microscope, electron microprobe, and histologic investigation

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Titanium cones were examined before and after implantation in the root canals and bones of two mongrel dogs. A thin, adherent film was detected on the implants after they were removed. Severe, inflammatory lesions developed around the implants in both dogs after six months. A hypothesis for this occurrence is offered.

In a previous study[1] we found that Vitallium endodontic implants in the teeth of dogs corroded, and the corrosion products were cytotoxic to the periapical tissues. Metal particles from the Vitallium were found dispersed in the alveolar bones at considerable distances from the implant sites. Several other negative features of those implants were detected. Among these were the production of moderate to severe periapical inflammatory responses and induction of ankylosis in several teeth. In addition, the mandibular nerve and artery were severed, and the maxillary sinuses were penetrated by several of the implants.

Titanium would appear to be a desirable, biocompatible material for use as an endodontic implant, based on reported corrosion and tissue toxicity studies.[2-4] All metals corrode in the human tissue fluids. However, the corrosion products of titanium, which have been reported to be oxides, such as $Ti_2O_3 \cdot 3.5TiO_2$,[5] passivate the titanium surface and protect it from the attack of electrolytes.[6] Thus, the corrosion rate of titanium appears to be lower than that of most other metals such as stainless steel (type 316L) when tested in physiologic saline[6] and Ringer's[7] solutions.

Favorable results have been reported when titanium has been implanted in the bones of rabbits, sheep, dogs, and humans. The findings indicated that the implants were well tolerated by the bones.[4,8,9] Also, when the titanium was fabricated to have open pores or was threaded, the bone grew into the pores and irregularities of the metal.[10-15]

The present investigation was instituted to determine whether titanium endodontic implants were tolerated by the periapical tissues of dogs when implanted for about six months. It was also of interest to investigate the reactions of inflamed periapical tissues to the titanium implants. To avoid the possible induced inflammation of cements or sealers, the implants were inserted without these materials. The investigation used the modalities of scanning electron microscopy, electron probe microanalysis, tissue culture, radiography, and histopathology. Thus, the effect of the periapical tissues on the metal and the effect of the metal on the periapical tissues could be studied.

MATERIALS AND METHODS

Thirty-four titanium cones were fabricated at the Franklin Institute Research Laboratories in Philadelphia. After polishing, the cones were treated with a modified Kroll's etching solution (2% by volume of hydrofluoric acid in water with the addition of 3% by volume of concentrated nitric acid).[16] The cones then were cleaned ultrasonically in ethyl alcohol and autoclaved before use.

The cones were examined in the scanning electron microscope (SEM) and photographed at magnifications ranging from $\times 60$ to $\times 6{,}000$. Electron microprobe analyses of four of the cones also were made.

The teeth of two dogs were used. In the first dog, a 2-year-old mongrel weighing about 50 lb, the teeth were radiographed. The pulps were removed from ten mandibular premolars and molars under the aseptic conditions usually used for root canal therapy. After pulp extirpations, the root canals were instrumented from 4 to 10 mm beyond the apices with hand instruments (stainless steel reamers and Hedströem files). The canals then were irrigated with physiologic saline solution and dried with sterile paper points.

Twenty-two such root canal preparations were made in 12 teeth. Nine titanium cones then were inserted to the depth of the preparations without sealers. The access cavities were closed with Cavit* and amalgam. Postoperative radiographs then were taken. Forty-two days later, nine other implants were inserted, sealed, and radiographed as before. The root canals of two teeth remained unfilled except for closure of the access openings, and these teeth served as controls for 112 days. The animal was killed by perfusion with fixatives 140 days later.

In another 2-year-old mongrel dog weighing about 60 lb, the pulps of ten mandibular and four maxillary premolars and molars were exposed with high-speed instruments. The pulp tissues in the root canals then were macerated with barbed broaches and root canal files. The root canals were left open for three months to create periapical inflammatory lesions. The presence of such lesions was confirmed by radiographs.

As in the first dog, the root canals were instrumented with hand instruments, irrigated with physiologic saline solution, and dried with paper points. Eight implants were placed, without sealers, at one operating session. The access openings then were sealed with Cavit and amalgam. Forty-two days later, eight additional implants were placed in the other root canals, except the canals of four teeth that acted as controls. Access openings again were sealed with Cavit and amalgam. The animal was killed 139 days later by perfusion with fixatives.

The mandibles of both animals were radiographed, and block sections, each containing about three implants, were made with a bone saw.

After decalcification, the implants were removed for examination with the SEM and for microprobe analysis. The tissue blocks then were processed in the usual manner for histologic examination.

Six implants from each dog as well as particles of titanium dioxide were tested on a tissue culture monolayer of mouse fibroblasts, strain L-929. The agar-diffusion method of Guess and associates[17] was used.

RESULTS

Immediately before killing the dogs, clinical examinations showed that numerous sinus tracts had developed in the mandibles of both animals.

SEM Examinations

All of the titanium cones were examined before insertion. The surfaces of the titanium cones, having been etched chemically, showed clearly outlined grain boundaries. Grain boundaries are regions of mismatch between blocks of crystals or grains of metal. Such grains result from the crystalline array of atoms that occurs when a metal solidifies during casting. Grain boundaries are high-energy areas that are more active chemically, hence are usually attacked more rapidly than the grain faces when the metal is exposed to a corrosive.[18] Within the grains there were microscopic pits that probably resulted from acid treatment at the time the cones were manufactured (Fig 1). Coronal, middle, and apical portions of the cones all looked alike. Microprobe analyses showed that the cones were composed of pure titanium (Fig 2).

When the cones were removed six months later, some seemed to be deformed (Fig 3), and it appeared as if tissue debris or coagulated blood was present on their surfaces (Fig 4). Microprobe analyses of the cones showed the absence of other elements in some cones (Fig 5). In others, the presence of sodium and chlorine in addition to titanium was detected. In a few cones, potassium, sulfur, and iron also were detected.

After ultrasonic cleaning, the surface debris was apparently removed, showing a topography similar to that of the unused cones (Fig 6).

Tissue Culture Studies

Before use, the cones were tested in tissue culture. After removal from the dogs and ultrasonic cleaning and autoclaving, the cones again were tested. None of the cones proved to be toxic to mouse fibroblasts. No zones of inhibition were detectable. Similar negative findings were obtained with particles of titanium dioxide.

Radiographic Studies

Radiographic and histologic examinations of the teeth of the first dog (vital pulp extirpations) showed that five root canals had been perforated, and the implants had been inserted through the perforations (Fig 7). In the second dog (periapical areas), two root canals were perforated and the implants were inserted through the perforations into the periodontal ligament and alveolar bone (Fig 8).

All of the other implants appeared to pass through the root canals into the periapical tissues.

Examination of the radiographs of the teeth of both dogs showed large areas of rarefaction around every tooth that received the implants, as well as the control teeth (Fig 9). The rarefactions were present periapically and, in many instances, laterally to the roots. The size of the areas appeared to be similar in the teeth of both dogs (Figures 6 through 8).

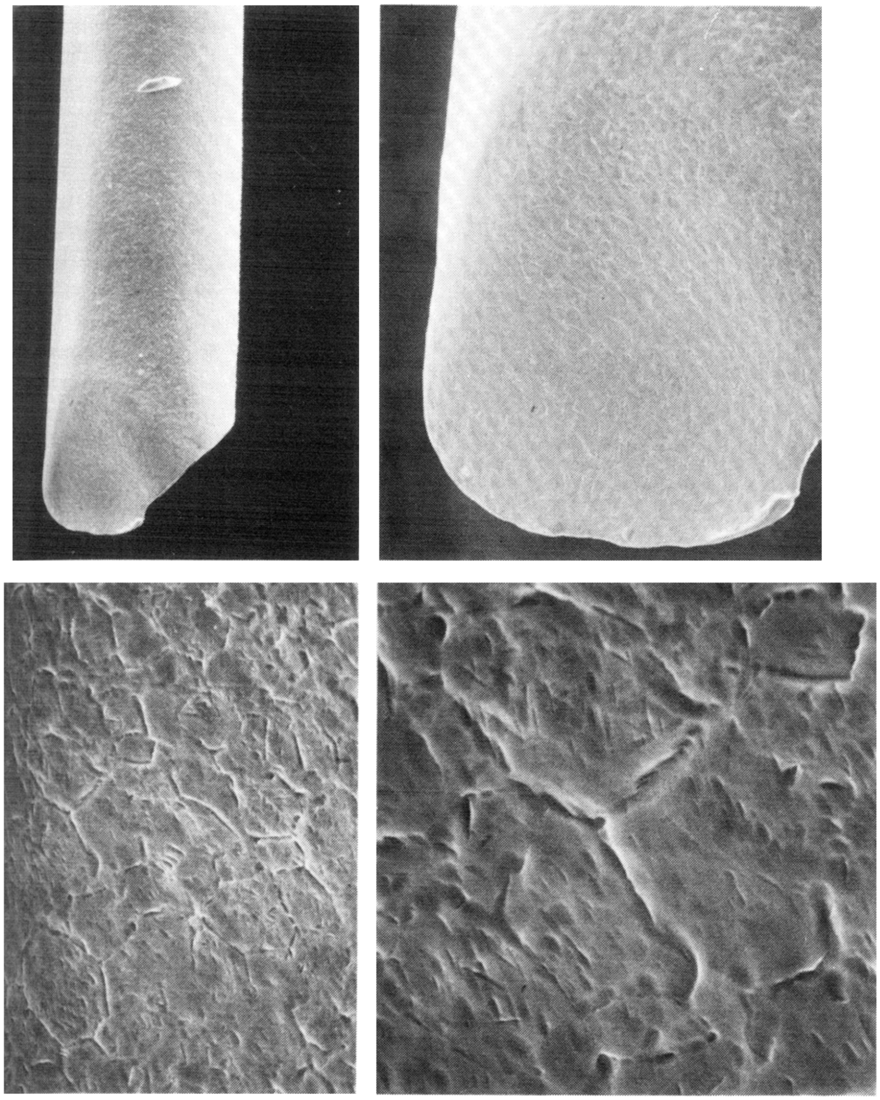


Fig 1—Titanium cone before use: top left (orig mag ×60); top right (orig mag ×180); bottom left (orig mag ×600); bottom right (orig mag ×1,800).

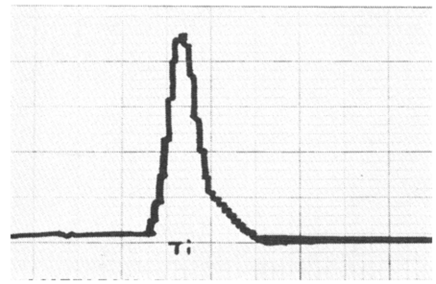


Fig 2—Microprobe analysis of titanium cone. Full-scale for period of 400 seconds (orig mag ×600).

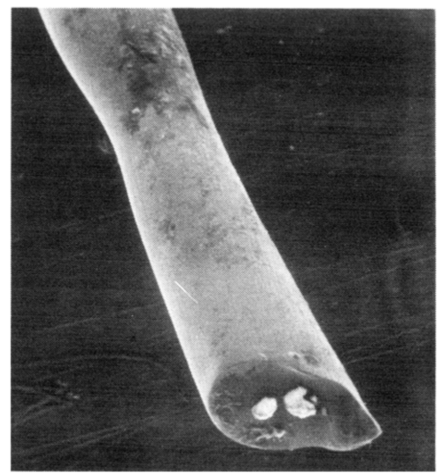


Fig 3—Deformed titanium cone removed from dog after six months (orig mag ×60).

Histologic Findings

In both dogs, severe periapical inflammatory responses were found around all of the implants as they emerged from the roots of the teeth. In a few cases, epithelium lined the implants for short distances (Fig 10). Deeper into the cancellous bone, the implants were surrounded by dense, fibrous capsules. Inflammation was absent in these deeper areas.

The severe inflammation found periapically in the first dog (vital pulp extirpations) was similar both for the control teeth and the teeth with implants.

In the second dog, the periapical inflammatory responses of the teeth with implants appeared to be greater than the responses of the control teeth.

In all of the teeth with perforated roots, severe inflammation was found around the implants. In two cases, massive bone destruction was accompanied by epithelial proliferation, resulting in the formation of deep periodontal pockets (Fig 11).

In the first dog, the mandibular nerve was found to have been severed by the implant procedure (Fig 12).

In the second dog, two teeth were found to be ankylosed (Fig 13).

DISCUSSION

According to McQuillan and McQuillan,[16] the tissue film, found on the surface of the titanium cones, apparently could not be caused by cor-

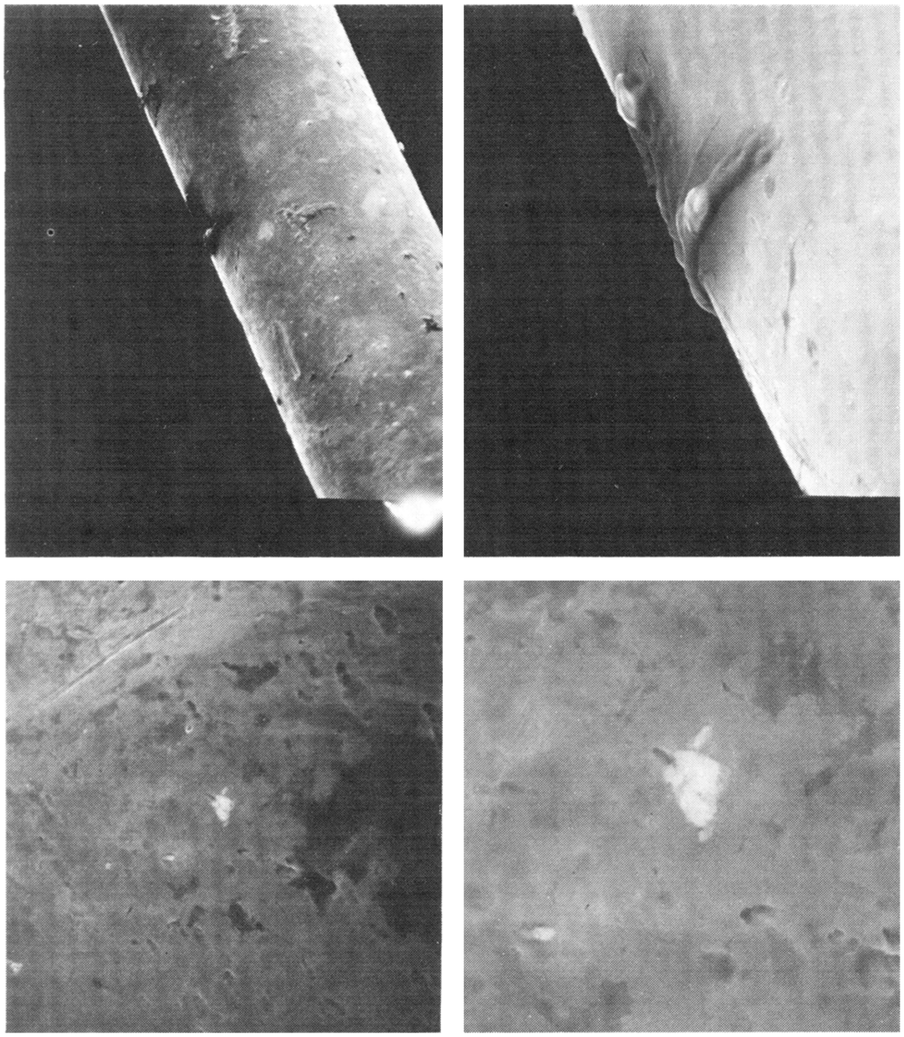


Fig 4—Titanium cone six months after implantation: top left (orig max ×60); top right (orig mag ×180); bottom left (orig mag ×600); bottom right (orig mag ×1,800).

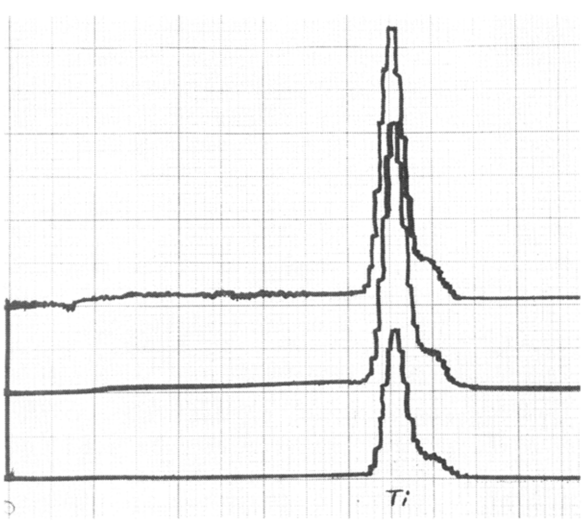


Fig 5—Microprobe analysis of titanium cone six months after implantation. Analyses made in three different areas; all show only presence of titanium. Full-scale for period of 400 seconds (orig mag ×600).

rosion of the cones by formic acid. Thus, Formalin fixation probably was not responsible for the surface changes noticed on the cones after they were removed.

The nature of the thin film found absorbed on the surface of each titanium implant could not be ascertained by electron microprobe analysis. Sodium, chlorine, potassium, sulfur, and iron were found on some cones. The film may have been proteinaceous in character. This film was removed with difficulty by ultrasonic cleaning, showing an apparently unchanged implant surface. Apparently, even clean, smooth metal surfaces become coated when inserted in the body. For example, DePalma and associates[19] found that five different metals implanted in the canine inferior vena cava became coated with a thin, labile, ill-defined proteinaceous deposit. The surface of the metals had been cleaned until totally organic-free by a special glow discharge treatment. In such treatment, low-energy ions, produced at reduced gas pressures, gently bombarded the specimens, thereby scrubbing them free of all organic materials.

The titanium cones removed from the dogs did not prove to be toxic to mouse fibroblasts. Similarly, powdered titanium dioxide produced no inhibition of growth of mouse fibroblasts in tissue culture. However, the tissue culture method used may not have been sensitive enough to show discrete toxic reactions to insoluble metals or oxides. Possibly, another method for measuring toxicity, such as the one used by Spangberg,[20] might have been more sensitive for the detection of cell toxicity.

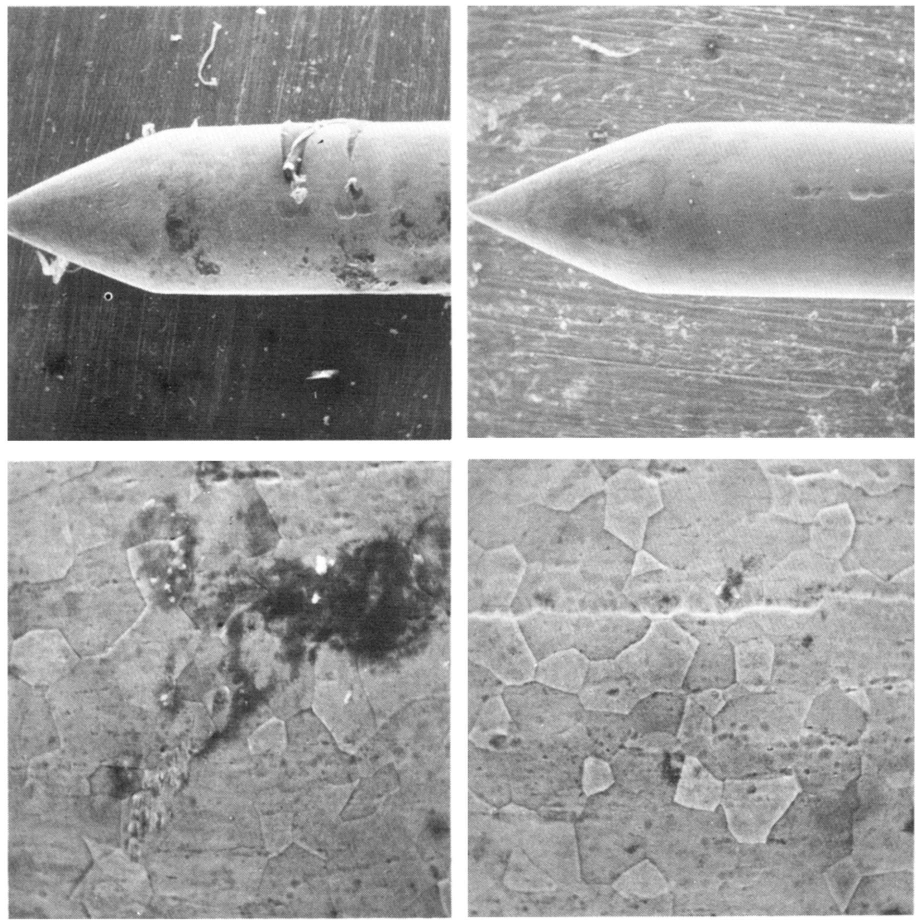
Corrosion of metallic implants is capable of causing inflammatory responses leading to infection or rejection.[21,22]

Whether or not the periapical inflammatory lesions were caused by irritation from corrosion products could not be ascertained. An analysis of pre- and postoperative cones in both animals showed no visible evidence of corrosion. However, titanium oxide film cannot be seen with the naked eye or with a microscope. The film is very thin, forms on the surface, and acts as a protective layer preventing further corrosion from taking place. When the oxide is removed from the surface of the metal, the metal can become highly corrosive until a new film of oxide is formed. Thus, the metal could corrode from stress when the cone is inserted into the tooth and removed, temporarily, before final insertion.

An electrochemical attack would be analogous to that which occurs to a piece of iron that is partially immersed in saline solution. In the beginning, numerous anodic and cathodic areas develop over the surface. However, because the oxygen needed for the main cathodic reaction is replenished readily only near the water line, the cathodes on the lower part of the specimen stop functioning and become wholly anodic. Inasmuch as the total cathodic current must equal the total anodic current, the part near the water line becomes predominately cathodic, and the corrosion products precipitate at the level where the cathodic and anodic zones meet.[23] In the case of the titanium implants, a similar phenomenon may have occurred with the cathodic and anodic titanium zones joining at the areas where the implants emerged from the root.

The inflammatory response directly around the apices of the teeth would be expected to be greater because of differences in the consistency of the periodontal ligament compared to that of bone. Because of the interchange of electrolytes in this area, a greater inflammatory response should occur directly opposite the apices of the teeth. Some cones appeared to be deformed, possibly by shearing forces, and looked different-

Fig 6—Titanium cone immediately after removal from dog: top left (orig mag ×60); bottom left (orig mag ×600). Same cone after ultrasonic cleaning: top right (orig mag ×60); bottom right (orig mag ×600).



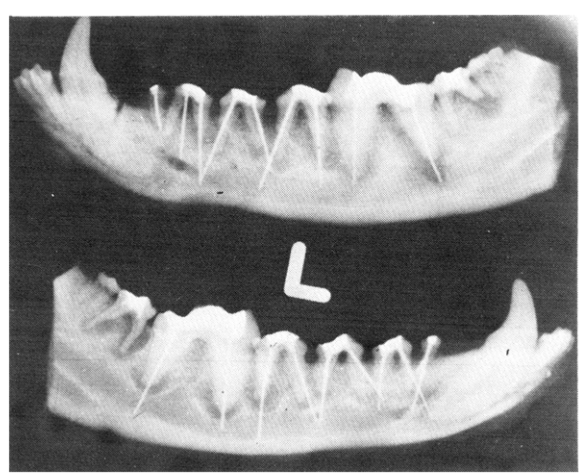
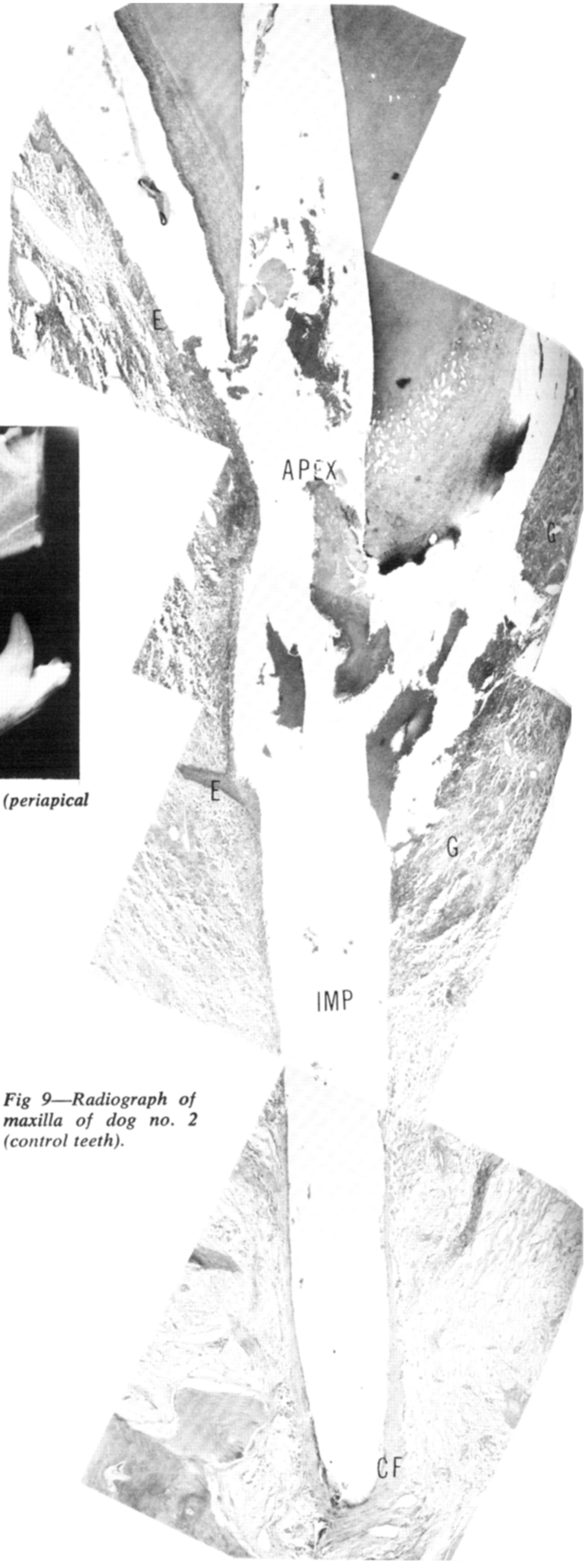


Fig 7—Radiograph of mandible of dog no. 1 (vital pulp extirpation).

Fig 10—**Composite photomicrograph** shows granulomatous tissue **(G)** present directly around apex of tooth (APEX). Deeper in bone, portion of implant **(IMP)** is covered by epithelium (E). Deepest portion of **implant** is surrounded by dense collagen fibers (CF) (H & E orig mag ×50).



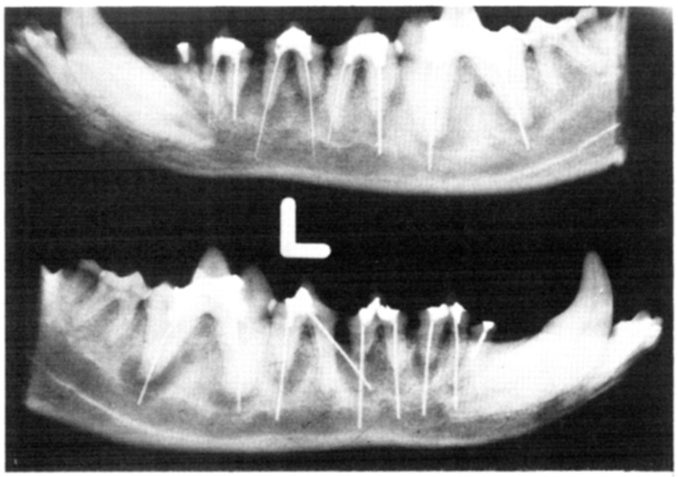


Fig 8—Radiograph of mandible of dog no. 2 **(periapical** areas).

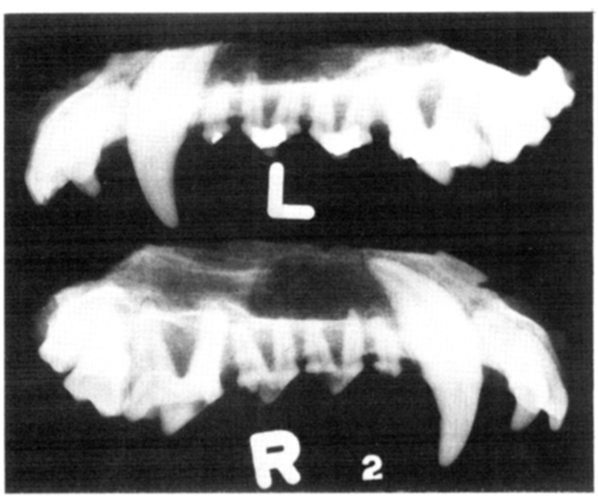
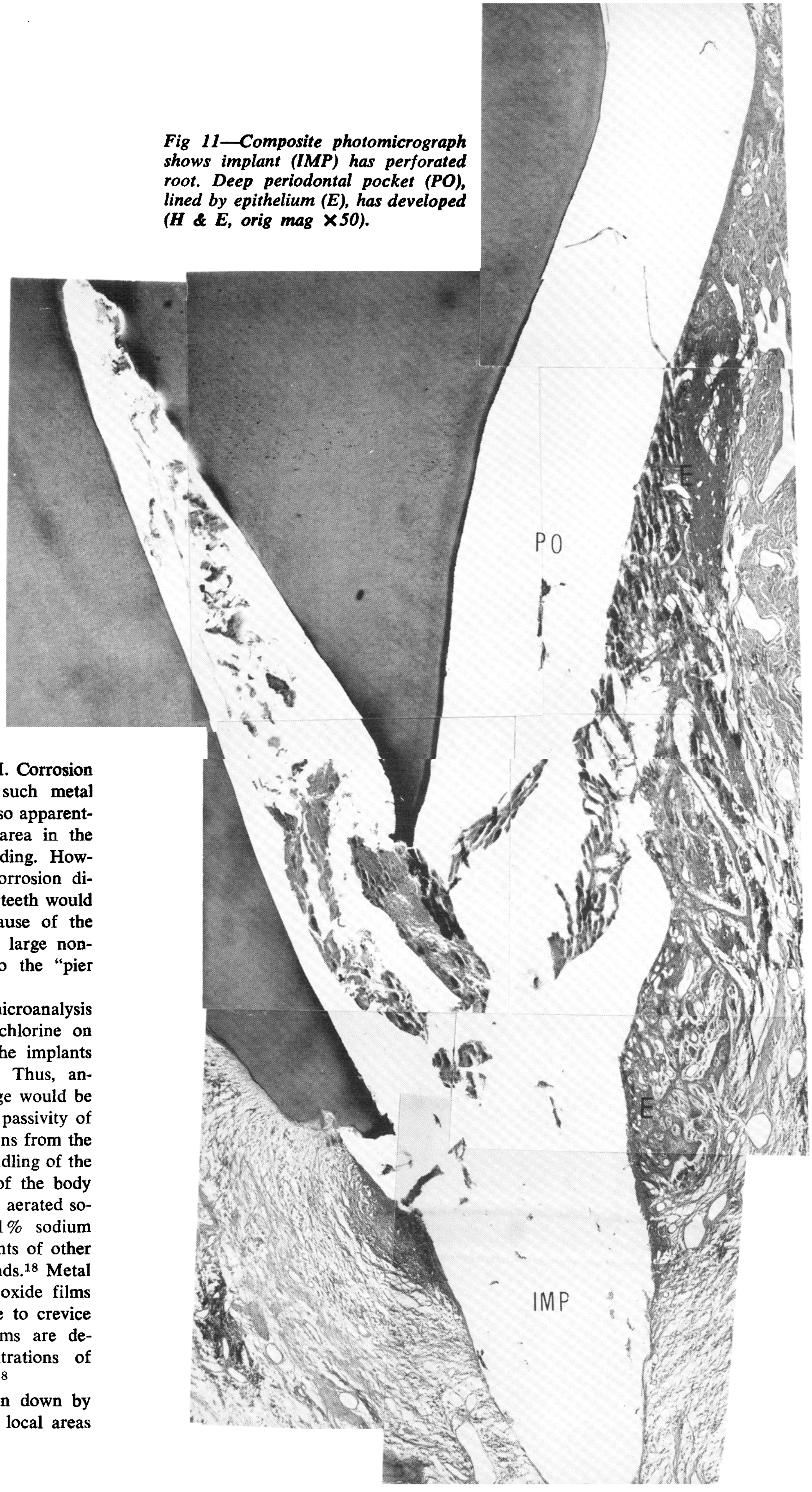


Fig 9—Radiograph of maxilla of dog no. 2 (control teeth).

Fig 11—Composite photomicrograph shows implant (IMP) has perforated root. Deep periodontal pocket (PO), lined by epithelium (E), has developed (H & E, orig mag ×50).



ly from others in the SEM. Corrosion would be enhanced by such metal stressing. Corrosion was also apparently enhanced by a large area in the bone that was not corroding. However, small amounts of corrosion directly at the apices of the teeth would be grossly enhanced because of the small area relative to the large noncorroding area, similar to the "pier effect" aforementioned.

Electron probe microanalysis showed the presence of chlorine on the surface of some of the implants after they were removed. Thus, another possibility for damage would be the breaking down of the passivity of the titanium by chloride ions from the hands through careless handling of the implants or by chlorides of the body fluids. The body fluids are aerated solutions containing about 1% sodium chloride plus minor amounts of other salts and organic compounds.[18] Metal or alloys that depend on oxide films are particularly susceptible to crevice corrosion because the films are destroyed by high concentrations of chloride or hydrogen ions.[18]

Passivity may be broken down by chloride ions at points or local areas

12—Composite photomicrograph vs implant (IMP) has penetrated t of tooth (APEX), where it is ounded by granulomatous tissue Deeper in bone, mandibular nerve has been severed by implant. Col-n fibers (CF) surround implant ɔ in bone (H & E, orig mag ×50).

on metal, resulting in pitting corrosion. The locally contaminating areas become anodic and the larger areas of passive metal are cathodic. Corrosion then takes place at the anodes.[24,25]

Sealers were not used to avoid the possibility that inflammatory changes would be induced by the chemical irritation of the ingredients of the cements. As Frank and Abrams[26] and Langeland and Spangberg[27] have pointed out, the cements are complicating irritants in endodontic implants. Cement particles induce intense inflammatory reactions; a nonirritating cement is not currently available.

The presence of a fibrous capsule and the absence of inflammation deep in the bones indicated that possibly more oxygen was available to the metal deep in the bone, resulting in an oxide film and greater passivity of the metal. Passivity refers to the loss of chemical reactivity of certain metals under particular environmental conditions.[18,23,24] On the other hand, at the titanium-root interface, lack of a protective oxide film may have resulted in increased and continued corrosion.

The findings of thick, fibrous capsules around the titanium cones deep in the bones conflict with those of Rhinelander and associates.[28] They found that titanium showed the least, if any, fibrous lining around disks of nonporous titanium, Vitallium, and stainless steel implanted in dog femurs for periods up to six months. However, others have found thick, fibrous

Fig 13—Composite photomicrograph shows implant (IMP) surrounded by granulomatous tissue (G). Within granuloma, there are a number of abscesses (ABS). Resorption (R) of teeth and bone has resulted in ankylosis (ANK) (H & E, orig mag ×50).

capsules around various metal implants in animals.[26,29-33]

In the teeth with perforated roots, the reactions of the periodontal ligament and bone to the implants was of academic interest because lateral endodontic stabilizers have been advocated for treatment of periodontally involved teeth.[34-35] The severe tissue destruction that occurred in those teeth raises questions concerning the wisdom of using lateral endodontic stabilizers for treatment of such periodontally involved teeth.

If titanium is used for endodontic implants, it might be desirable to alloy the metal with platinum, as suggested by Fontana and Greene.[18] During exposure to a corrosive environment the titanium could be preferentially dissolved because platinum is virtually inert in electrolyte solutions. The corrosion would allow accumulation of sufficient platinum on the surface to cause spontaneous passivation. Should the platinum be removed from the surface by abrasion, the process of titanium removal would be repeated and additional platinum enrichment would occur. Thus, the corrosion resistance of titanium would noticeably improve.

SUMMARY

After vital pulp extirpations, titanium endodontic implants were inserted into the prepared root canals of ten mandibular teeth of a mongrel dog for 182 days. In a second mongrel dog, 16 implants were inserted into the root canals of teeth with induced periapical lesions for 181 days. SEM studies showed the presence of a thin film that was adsorbed on the surfaces of the implants. Electron microprobe analyses showed the presence of sodium, potassium, chlorine, sulfur, and iron on the surfaces of some of the implants. Tissue culture studies failed to show toxicity of the cones.

Radiographic and histologic studies showed severe periapical and lateral inflammatory lesions in both dogs. Where the roots were perforated, deep periodontal lesions developed. However, deep in the bone, the implants were surrounded by thick collagen fiber capsules.

In one dog the mandibular nerve was severed. In the other dog two teeth were found to be ankylosed.

It was speculated that corrosion of the implants caused the inflammatory responses directly at the apices of the teeth.

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