BIOLOGIC SEALING OF THE APEX
IN ENDODONTICALLY TREATED HUMAN TEETH

by

John B. Pappin, D.M.D.

ABSTRACT

Controversy continues to exist concerning the sealing abilities of endodontic filling materials and cements, the apical extension of canal preparations and fillings, and the effect that these methods and materials may have on healing in the periapical tissues of treated teeth.

Closure of the apical foramen by cementum (physiologic sealing) has been a goal long sought but seldom achieved. It has been suggested, though never proven in a controlled human investigation, that autogenous dentin chips placed in the canal at the apical constriction may seal the canal and/or potentiate cementum apposition, resulting in a biologic closure of the apical canal.

This study was designed to answer the following questions:

1. Can the apex of a prepared root canal system be sealed with one millimeter of dentin chips?
2. Does the filling material used to obturate the root canal, coronal to the dentin chips, have any additional effect on the apical seal?
3. Will calcified material be deposited in the canal and/or foramen apical to the obturating material?

Sixty single-rooted extracted human teeth were prepared and obturated using a one-millimeter apical plug of dentin chips alone and in conjunction with laterally condensed gutta percha or zinc oxide-eugenol placed with a Lentulo spiral.

Twenty-two single-rooted human teeth with clinically viable pulps were treated *in vivo* with the techniques used in the *in vitro* investigation, except that tetracycline was administered systemically to label sites of active mineralization.

All teeth were sectioned and evaluated for apical leakage following immersion in S^{35} for a period of 48 hours. Teeth from the *in vivo* study were also examined for tetracycline labeling using an ultraviolet microscope.

The results indicate that:

1. The apical one millimeter of the canal was not sealed against penetration by S^{35} using a one-millimeter apical plug of dentin chips.
2. The tested filling materials used to obturate the root canal coronal to the dentin chips had no additional effect on the apical seal.
3. Active calcification occurred in the apical canal adjacent to the dentin chip plug in every case following an *in vivo* test period of 98 days. Calcification was not observed under any other test conditions.

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Dedicated to my children

Kevin, Karin, Katy, Kimberly, and Courtland

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INTRODUCTION

Controversy continues to exist concerning the sealing abilities of endodontic filling materials and cements, the apical extension of canal preparations and fillings, and the effect that these methods and materials may have on healing in the periapical tissues of treated teeth.

It may be suggested that, in spite of clinical studies reporting a success rate of 90% or better, complete healing verified by histology is achieved in only 10-20% of the cases utilizing currently accepted techniques.

Closure of the apical foramen by cementum (physiologic sealing) has been a goal long sought but seldom realized. It has been suggested, though never proven in a controlled human investigation, that autogenous dentin chips placed in the canal at the apical constriction may seal the canal and/or potentiate cementum apposition, resulting in a biologic closure of the apical canal. If biologic closure of the apical foramen could be easily achieved, the remainder of the root canal system might be obturated utilizing less demanding and time-consuming techniques.

This investigation has been designed to evaluate the usefulness of autogenous dentin chips as an apical plug in endodontically treated human teeth.

REVIEW OF THE LITERATURE

It has been reported that the basis of successful endodontic therapy is the thorough debridement and total obturation of the root canal system (Schilder, H., 1975). Recent research has suggested, however, that the removal of all pulpal debris and bacteria from the root canal system may be impossible to achieve at this time (Senia et al., 1971; Coffae and Brilliant, 1975; McComb et al., 1976). It appears, therefore, that successful endodontic therapy may rely primarily on complete obturation and sealing of the root canal system.

In vitro investigations of the sealing abilities of dental materials have been conducted utilizing dyes (Grossman, 1939; Ainley, 1970; Grieve, 1973), bacteria (Fraser, 1929; Kapsimalis, 1965), compressed air (Harper, 1912; Granath and Svenson, 1970), scanning electron microscopy (Moodnik et al., 1975), radioisotopes (Marshall and Massler, 1961; Beal, 1972), passage of electrical current (Jacobsen and Fraunhofer, 1975), and interference with sound waves (Davidson, 1970). The results of these investigations suggest that "hermetic" sealing of the root canal system cannot be achieved using methods and materials currently available. Furthermore, *in vivo* investigations (Going et al., 1968) utilizing a sophisticated technique involving neutron activation analysis for manganese have indicated that some commonly used restorative materials may actually permit more leakage *in vivo* than *in vitro*.

In spite of the acknowledged inadequacies of *in vitro* radioisotope

leakage studies (O'Brien et al.,1968; Roydhouse et al.,1967 and 1968; Going, 1972), it has been suggested (Marshall and Massler, 1961; Beal, 1972) that radioactive isotopes may be useful in comparing *in vitro* sealing capabilities of endodontic sealers. These studies also indicated that the radioactive isotope of sulfur (S^{35}) may be the most sensitive and accurate tracer available for such investigations.

Rogers (1975) has shown that chemography may produce an artifact in any autoradiographic technique. He further indicated that dipping the specimen in a solution of nitrocellulose will coat it with an inert and relatively impermeable layer which is still thin enough to permit penetration by the majority of beta particles. This chemographic variable has neither been considered nor controlled in previous dental leakage studies. Since it is, therefore, impossible to dismiss the possibility that chemographic artifact may have biased previous autoradiographic leakage studies, controls must be established to evaluate this variable.

In addition to poor sealing qualities, all materials currently used in attempts to seal the root canal system are either resorbable, cytotoxic, or both (Langeland, 1974). Langeland stated that: "Strictly speaking, these facts should prevent us from using sealers and pastes at all, but since voids between the canal wall and the filling material are unacceptable, sealers are a necessity". The clinical significance of these voids, however, has never been defined in endodontic research. It is possible that such voids are completely inconsequential if adequate apical and coronal seals can be established and maintained.

The techniques for obturating and sealing root canals are almost as numerous as the clinicians recommending them, and high degrees of clinical success have been reported for most of them. Nevertheless, some

endodontic pathoses fail to heal following treatment. Dow and Ingle (1955) attributed more than 50% of these failures to poorly obturated canals. From the data presented in their report, however, it is impossible to determine whether leakage into the canal through the apical foramen contributed to clinical failure. West (1975) also attempted to implicate leakage of root canal fillings as the critical factor in failure of endodontic therapy. His study, unfortunately, suffered the same lack of controls as did that of Dow and Ingle. Both investigations examined only endodontic failures which showed leakage and completely ignored the possibility that similar leakage may occur in cases which are considered to be clinically successful.

The previously cited investigations have clearly demonstrated that no currently available filling material achieves a complete marginal seal. Jorgensen and Wakumoto (1966) have shown that recurrent caries will occur in a restored tooth only when the marginal defect exceeds fifty microns. Similar criteria do not exist for evaluation of root canal fillings. It must, however, be accepted that (1) the ideals of "thorough debridement and total obturation" of the root canal system have rarely been achieved, and (2) the physiologic retention of endodontically treated teeth does occur in spite of these apparent inadequacies in treatment objectives. It is obvious that resolution of this apparent dichotomy must result from identification and examination of other variables.

Although there are still major disagreements concerning the optimal apical extension of endodontic fillings, it has been demonstrated that carrying these procedures and materials beyond the apical foramen retards, and may even prohibit, complete healing in the periapical tissues

(Blayney, 1927; Strindberg, 1956; Grahnen and Hanssen, 1961; Seltzer et al. 1968).

It has been proposed that, if complete healing is desired in the periapical tissues, all endodontic procedures should terminate at the cemento-dentinal junction (Grove, 1930; Kuttler, 1958). This junction normally corresponds to the apical constriction in the root canal and is located 0.50-0.75 millimeters from the apical foramen (Green, 1956 and 1960; Pineda and Kuttler, 1972). These studies have also shown that the foramen is seldom located precisely at the anatomic or radiographic apex of the tooth. When an instrument is placed into the canal so that radiographically it appears to be flush with the apex it may, in fact, extend beyond the foramen by one millimeter or more in at least 50% of the cases (Palmer et al. 1971). It would appear, therefore, that canal instrumentation should terminate about 1.5 millimeters short of the radiographic (anatomic) apex of the root. However, even if the root canal is flawlessly prepared precisely to the apical constriction, it is virtually impossible to obturate all root canals without extruding some material through the patent apical foramen. Because of these anatomic variables and the demonstrated toxicity of endodontic sealers, a technique for blockading the apical foramen with a biologically acceptable material should, therefore, prove to be very useful.

A recent investigation by Adams (1974) indicated that a plug of dentin chips compacted into the apical one millimeter of the prepared canal formed an apical seal which was impermeable to Ca^{45}. This finding was further documented by Brown et al. (1979). As noted in previous investigations (Marshall and Massler, 1961; Going, 1959), S^{35} is a more sensitive indicator of leakage than Ca^{45}. Therefore, the sealing

potential of dentin chips should be further defined using radioactive sulfur (S^{35}). Adams, furthermore, only investigated the seal of dentin chips incidental to canals obturated with broken files. The possibility that other obturation materials may affect this seal should also be evaluated.

The closure of the apical foramen by the apposition of cementum has been suggested to be the ultimate and perhaps the only physiologic seal (Grove, 1921; Coolidge, 1933; Gottlieb et al, 1950). However, this finding has been reported only rarely in histological investigations of endodontically treated teeth. When cemental closure of the apical foramen has been observed, it has almost always been reported in association with an accidental apical plug of dentin or apical dentin debris. Göllner (1937) was apparently the first to suggest purposely placing a plug of dentin into the apical portion of the root canal. He suggested utilizing such a blockade to prohibit toxic filling materials from coming into contact with the viable apical pulp stump. The concept of an apical plug of dentin chips was further advocated by Mayer (1956). Kuttler (1958) reported from roentgenographic observations that a more rapid resolution of periapical radiolucencies appeared to follow the placing of autogenous dentin chips into the area of the apical constriction. The results of an histological and clinical investigation in human teeth (Ketterl, 1963) suggested that cementum does develop apical to a plug of dentin chips. Ketterl further indicated that complete histologic healing was observed in 68% of the cases so treated, even though complete cemental closure of the foramen was not seen. The results are in sharp contrast with evidence of histologic healing which was reported following more conventional endodontic therapy. Brynolf (1967) observed complete histologic healing in only 7% of 119 root-filled human teeth which were

obtained at autopsy. Although she did not know what materials had been used to obturate the canals, she did observe that, in those cases which demonstrated complete healing, plugs of dentin chips were present in the apical area of the canal. As a result of these observations, Brynolf concluded that "...the prospects of healing are brighter if the root filling does not extend down to the foramen, and if there are fragments of dentin superficially in the foreign material". These observations have been confirmed and documented by other investigators (Waechter and Prinz, 1966; Lambjerg-Hansen, 1974; Herd, 1976 and 1978). Tronstad (1978) investigating tissue reactions apical to a deliberate plug of dentin chips in monkey teeth, reported complete histologic healing as well as total calcified closure of the apical foramen in 22 of 24 teeth so treated. He attributed this high incidence of healing to the utilization of dentin chips and to root filling materials which are known to be only slightly irritating to viable tissues. He further reported complete healing in only 10 of 21 control teeth which were treated conventionally with the same root filling materials. An unexpected finding in this study was that most of the teeth in the control group which demonstrated healing had accidentally been plugged with dentin chips, "although care had been taken that this should not occur".

In addition to the above findings, Gottlieb et al. (1950) reported histologic evidence from dog teeth which indicated calcified bridging of the apex would occur in contact with deliberately placed dentin chips even in teeth with a totally necrotic pulp.

None of the investigators who reported calcification apical to a plug of dentin chips utilized techniques which indicated whether or not the calcification preceded or followed positioning of the dentin plug.

It is difficult, therefore, to eliminate the possibility that their interpretations may have been biased by an incomplete record of physiologic events. More definitive answers might be obtained by utilizing a labeling technique which would positively define the physiologic chronology of the observed calcification.

Tetracyclines have been used *in vivo*, in human research, to label continuing calcification in bone and tooth structure (Milch et al. 1957; Frost and Villanueva, 1960; Epker, 1966; Tagger et al. 1975). These studies have shown that tetracyclines are bound only at sites of active mineralization. The tetracyclines fluoresce a brilliant yellow-gold under ultraviolet illumination, in sharp contrast to the normal blue-gray autofluorescence of unlabeled tissues. Since the tetracyclines are bound only at sites of active mineralization, they appear to be the most sensitive tracer readily available to define the chronology of mineral apposition.

It is obvious from the cited research that a physiologic closure of the apex in endodontically treated teeth is rarely achieved using traditional treatment methods. It is equally obvious that endodontic sealers currently in use do not produce an "hermetic" seal and, in addition, are so cytotoxic that healing may be retarded when they contact periapical tissues.

Some investigators have reported that a plug of dentin chips may produce a seal against isotope penetration which is at least as effective as traditional sealants. Other investigators have observed histologic healing and calcified closure of the apical canal when autogenous dentin chips are placed in contact with apical tissues.

Since there has been no controlled *in vivo* investigation of dentin chips as an apical plug in human teeth, this study has been designed to

answer the following questions:

(1) Can the apex of a prepared root canal system be sealed with one millimeter of autogenous dentin chips?

(2) Does the filling material used to obturate the root canal, coronal to the dentin chips, have any additional effect on the apical seal?

(3) Will calcified material be deposited in the canal and/or foramen apical to the obturating material?

PART I

IN VITRO INVESTIGATIONS

METHODS AND MATERIALS

Sixty extracted single-rooted human teeth selected at random were placed in normal saline and refrigerated at 4 degrees Centigrade until needed for processing. Processing proceeded as follows:

A. Preparation of Canals:

Complete patency of the canals was demonstrated by inserting a number ten root canal file until it was observed at the apical foramen. In pilot studies the canals were prepared with K type endodontic files using the serialized preparation technique previously described by Coffae and Brilliant (1975). It was noted during preparation of these canals that a pigtail of debris and irrigant was extruded through the apical foramen in almost every case (Fig. 1). This same phenomenon has been reported by other investigators (Adams, 1964; Ingle, 1976; Hession, 1977).

It was thought there might be a possibility of creating a bias in the leakage study if debris were impacted into the apical portion of the root canal during the preparation phase. Traditionally root canals are cleaned and enlarged by placing a small instrument to the full working length, as soon as possible, and then enlarging the apex to the desired size. This technique inadvertently packs debris into the apical canal and periodontal tissues. In an attempt to eliminate this inadvertent packing of debris into the apical canal a new preparation technique was developed. Pilot studies indicated that if traditional canal preparation techniques were reversed and the canals were prepared from the crown to the apex instead of from the apex to the crown, this inadvertent extrusion and

packing of debris could be eliminated. Consequently, the test teeth in this investigation were all prepared using the modified preparation technique (Figs. 2-7).

Following normal coronal access opening and removal of the lingual dentin shoulder, a #35 file was placed into the canal without apical pressure until resistance was encountered. The #35 file was then removed from the canal and the canal was flared with Gates-Glidden burs numbers 2, 3, and 4 to the depth which had been penetrated without pressure by the #35 file. Thorough irrigation of the canal was then accomplished using 2 ml of water delivered through a 27 gauge hypodermic needle to the depth of the flare. Next a #30 file was passed into the canal without apical pressure until resistance was encountered. The file was then rotated two complete revolutions and was removed from the canal. This procedure was repeated with consecutively smaller files until the working length was achieved at a point 1.5 mm from the apical foramen. The same filing procedure was repeated again beginning with the #35 file and progressing through the smallest file which would achieve the previously determined working length. This procedure was repeated, beginning each repetition with a file one size larger than previously used, until an apical stop had been prepared to a minimum size forty. Thorough irrigation of the canal using 2 ml of water was accomplished each time the instrumentation procedure was carried to the working length. Ultimately the irrigation needle was passed to the depth of the canal preparation without binding. Upon completion of the canal preparation, excess moisture was removed from the canal using sterile paper points.

B. Establishment of the Apical Seal:

In thirty of the prepared canals dentin chips were produced in the

coronal canal utilizing the Gates-Glidden bur #4. The chips were carried into the apical one millimeter of the canal preparation utilizing a sterile paper point and were then compacted with the final preparation file. This procedure was repeated until one millimeter of compacted dentin chips were present at the apex of the root. In the remaining thirty prepared canals no dentin chips were placed in the apex.

C. Obturation of the Canals:

The teeth used in this study were divided into three groups of twenty teeth each. Each group contained ten teeth with chips and ten teeth without chips. The canals were obturated as follows:

Group I: Twenty canals were obturated with laterally condensed gutta percha points and Roth Root Canal Cement (TM)[1].

Group II: Twenty canals were obturated with a paste of zinc-oxide (USP) and eugenol (USP) utilizing a Lentulo spiral on a slow speed handpiece.

Group III: Twenty canals were left unobturated as controls.

The coronal access openings of all teeth were sealed with 2-4 millimeters of Cavit (TM)[2]. All treated teeth were immersed immediately in normal saline and stored at 4 degrees Centigrade for one week to allow complete setting of the sealer.

D. Evaluation of Seal:

All teeth were removed from storage, were air dried and then were totally immersed in a tracer solution of 0.025% gentian violet and $Na_2(S^{35}O)_4$ containing 100μC of S^{35} per milliliter. Each tooth was then

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[1]Roth Root Canal Cement. Type 811, Elite Grade. Manufactured by Roth Drug Co., Chicago, Illinois.
[2]Non-eugenol zinc-oxide polyvinyl paste distributed by Premier Dental Laboratories, Morristown, Pennsylvania

washed and scrubbed with a tooth brush for five minutes under running tap water. Following washing, dehydration was accomplished by immersion for 36 hours in ethanol (50-70-90%) and an additional six changes of absolute ethanol in the next 48 hours. Dehydration was followed by immersion in two changes of propylene oxide in twelve hours, then propylene oxide plus monomer for 12 hours, and then two changes of monomer alone during 48 hours. The teeth were then embedded in individual blocks of Caroplastic (TM)[3] using a catlyst-monomer ratio of 24 drops:8 ounces.

Following polymerization of the blocks of plastic, serial longitudinal sections 300 microns thick were prepared from each tooth, utilizing a Hamco-Gillings thin sectioning machine equipped with a water-cooled, high-speed diamond saw.

Two autoradiographs were prepared from each section by placing the section in a plastic vise (Figs. 8-9) in contact with the emulsion surface of Kodak ultra-speed dental x-ray film (TM)[4] for a period of 18 hours in a light-proof box. The box and contents were exposed to x-radiation (5Ma, 65 KVP) for 3/10 second to outline the section. When the first autoradiograph was complete the sections were immediately coated by immersion in a 10% solution of nitro-cellulose in ether. The coated sections were air-dried for ten minutes and the second autoradiograph was prepared as above to test for chemographic artifact.

Routine developing and fixing procedures as recommended by the manufacturer were followed throughout.

Each autoradiograph was evaluated for indications of filling and marginal leakage and paired autoradiographs (coated and uncoated) were evaluated for chemographic artifact. Autoradiographs were evaluated and

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[3]Manufactured by Carolina Biological Supply Co., Burlington, North Carolina
[4]Periapical Ultra-Speed Film, DF-48, Eastman Kodak Co., Rochester, New York

measured for leakage at 7X magnification on an Ada enlarger projector (TM)[5].

All sections were microscopically evaluated to determine whether preparation debris had been inadvertently compacted into the apical canal.

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[5]Ada Products, Inc., P.O. Box 17509, Milwaukee, Wisconsin

RESULTS

Mean leakage measurements in millimeters from the apex for three separate evaluations of each autoradiograph are tabulated below. (Measurements were made independently by the investigator and two residents in endodontology).

TABLE I

Controls	D.C. Only	G.P. Only	Z.O.E. Only	G.P. + D.C.	Z.O.E. + D.C.
8.29	5.86	8.00	11.43	3.14	12.00
11.14	4.29	2.29	12.29	6.29	8.29
17.14	6.00	2.86	4.00	2.86	6.57
10.43	9.14	3.43	5.71	3.43	5.71
10.86	4.14	10.00	6.29	9.71	8.00
9.71	4.00	6.57	6.00	6.29	9.43
18.57	3.43	10.26	4.86	3.14	15.14
8.00	6.86	2.86	6.86	7.71	2.57
6.57	4.57	10.00	4.00	4.00	3.43
9.43	6.29		3.43	2.86	2.29
$\bar{X}$=11.01	5.46	6.25	6.49	4.94	7.34
S.D.=3.88	1.73	3.43	3.05	2.41	4.15

D.C. Only: 1 mm apical dentin chips -- no obturating material in canal
G.P. Only: Canal obturated with laterally condensed gutta percha - no dentin chips in apex.
Z.O.E. Only : Canal obturated with zinc oxide-eugenol paste - no dentin chips in apex.
G.P. + D.C.: Canal obturated with laterally condensed gutta percha - dentin chips in apex.
Z.O.E. + D.C. : Canal obturated with zinc oxide-eugenol paste - dentin chips in apex.

An analysis of variance showed significant differences in leakage patterns between obturating techniques. Further analysis with Tukey's Test (least significant difference - 4.2996) indicated significant differences in leakage between the control and all test groups except for zinc oxide-eugenol plus dentin chips (P 99.995, F = 4.546).

Evaluation of paired autoradiographs for chemographic artifact showed no differences in leakage patterns in any case (Figs. 10-12). Microscopic examination showed no dentin debris had been inadvertently compacted into the apical canals.

PART II

IN VIVO INVESTIGATIONS OF DENTIN CHIPS AS AN APICAL PLUG

METHODS AND MATERIALS

Twenty-two single-rooted human vital teeth were utilized in this phase of the study. Patients were selected who had clinically viable pulps in single-rooted teeth which had been designated for extraction by the School of Dentistry Departments of Removable Prosthodontics or Periodontics. Patients were apprised of their "Human Rights" and signed an appropriate form (Appendix A) approved by the Oregon Health Science University Committee on Human Rights. No teeth were utilized in this investigation which could otherwise be retained. Canal preparation was accomplished using the modified canal preparation technique described previously except that all instrumentation was halted 1.5 millimeters short of the radiographic apex.

Two patients having eleven teeth each which met the above criteria were selected for treatment. Immediately following canal preparation to a minimum apical size forty, each patient's teeth were obturated as follows:

a. Controls (No obturation) (one tooth): Following the standard canal preparation a number ten file was passed through the apex to insure apical patency.

b. Dentin chips alone (two teeth): After the standard preparation, a Gates-Glidden drill one size larger than that used for the final preparation was rotated in the canal to create dentin chips. The chips were then moved apically with a sterile paper point and compacted with a file until a one-millimeter apical plug was achieved. The

remainder of the canal was left unobturated.

c. Gutta percha, sealer, and dentin chips (two teeth): After the standard preparation, a one-millimeter plug of dentin chips was created in the apical preparation as described in (b) above. The remainder of the canal was obturated with laterally condensed gutta percha and Roth's root canal cement.

d. Gutta percha and sealer alone (two teeth); After the standard preparation, no attempt was made to plug the apex. The entire canal was obturated with laterally condensed gutta percha and Roth's root canal cement.

e. Zinc oxide-eugenol paste and dentin chips (two teeth): After the standard preparation, a one-millimeter plug of dentin chips was created in the apical preparation as described in (b) above. The remainder of the canal was obturated using a zinc oxide-eugenol paste placed with a Lentulo spiral on a slow speed handpiece.

f. Zinc oxide-eugenol paste alone (two teeth): After the standard preparation, no attempt was made to plug the apex. The entire canal was obturated with zinc oxide-eugenol using a Lentulo spiral on a slow speed handpiece.

All occlusal access openings were sealed with 2-4 millimeters of Cavit.

Two weeks before the root canal filling treatment and one week prior to extraction, oral tetracycline as a calcification marker was administered to the patients for each of three consecutive days, employing the usual clinical dosage schedule for adults (one gram per day). Daily doses were divided into four equal portions. The first patient's teeth were extracted at the 46th post-treatment day and the second patient's teeth were extracted at the 98th post-treatment day.

The extracted teeth were immediately placed in 10% neutral buffered formalin for four days. The teeth were then rinsed thoroughly in water, were dried and the apical five millimeters of each root was submerged in the isotope solution (100μC/ml S^{35}) for 48 hours. The specimens were then embedded in Caroplastic as described for the *in vitro* investigation.

Ten micron serial longitudinal sections were prepared from each tooth using the Jung, Model K microtome fitted with a tungsten carbide knife. To control shattering and separation of the fully calcified tooth structure during sectioning, the surface of the block was covered, as recommended by Ullberg (1977), with double-stick Scotch tape (TM)[6] prior to cutting each section.

Autoradiographs were prepared from a minimum of 10 sections per tooth selected at random intervals throughout the width of the canal. At each interval two autoradiographs were prepared from consecutive serial sections - one coated with 10% nitrocellulose and one left uncoated. These sections were then examined in a Leitz ultra-violet microscope utilizing a UG-1 filter interposed to provide optimum excitation at 3650 Angstroms. A BG-12 filter was also used to enhance contrast. Micro-photographs were prepared using Ektachrome 150 film with an exposure time of 12 seconds.

[6]Minnesota Mining and Manufacturing Co., 3M Center, St. Paul, Minnesota

RESULTS

TABLE II

Location of tooth	Method of treatment	Post-treatment time *in vivo*	Millimeters apical leakage	Evidence of apical Calcification
28	G.P.D.C.	46 days	1.66	-
21	G.P.D.C.		L.I.S.	-
22	G.P. only		L.I.S.	-
11	G.P. only		2.03	-
9	Z.O.E.D.C.		1.72	-
26	Z.O.E.D.C.		0.00	-
8	Z.O.E. only		0.80	-
4	Z.O.E. only		0.56	-
7	D.C. only		1.32	-
23	D.C. only		2.12	-
24	Control	46 days	4.88	-
22	G.P.D.C.	98 days	1.00	+
8	G.P.D.C.		2.12	+
27	G.P. only		0.43	-
9	G.P. only		L.I.S.	-
23	Z.O.E.D.C.		1.12	+
20	Z.O.E.D.C.		1.98	+
10	Z.O.E. only		0.00	-
25	Z.O.E. only		0.83	-
7	D.C. only		1.71	+
26	D.C. only		0.61	+
21	Control	98 days	3.43	-

Control: Canal prepared but left unobturated.
D.C. only: Canal prepared but left unobturated except for 1 mm dentin chips at apex of preparation.
G.P. only: Canal prepared and obturated with gutta percha and sealer. No apical dentin chips.
Z.O.E. only: Canal prepared and obturated with zinc oxide-eugenol. No apical dentin chips.
G.P.D.C.: Canal prepared and obturated with gutta percha and sealer. One mm dentin chips at apex of preparation.
Z.O.E.D.C.: Canal prepared and obturated with zinc oxide-eugenol. One mm dentin chips at apex of preparation.
L.I.S.: Lost in sectioning.

At 46 days no specimen showed evidence of tetracycline labeling in the apical canal whether dentin chips were present or not. Small

areas of labeling were occasionally seen in the cementum along the lateral borders of the root surface (Fig. 15). This was interpreted to indicate normal physiologic remodeling of the root cementum.

At 98 days all teeth plugged with dentin chips showed the tetracycline label in the apical canal (Figs. 17-19). There was no observable difference in the degree of labeling whether the canal was further obturated with gutta percha, or with zinc oxide-eugenol, or with nothing at all. An unexpected finding was the presence of the tetracycline label in the dentin chip plug as well as along the walls of the apical canal. Controls obturated with the same filling materials, but without dentin chips, showed no evidence of labeling in the apical canal (Figs. 20-22). Labeling in the cementum along the lateral borders of the root was again observed in the test as in the control teeth (Figs. 19 and 22).

Average levels of isotope penetration from three separate evaluations of all test sections are presented in Table II, above. Comparison of paired autoradiographs (coated and uncoated) showed no evidence of chemographic artifact.

DISCUSSION

A comprehensive review of the literature indicates there is no dental material tested which forms an isotope-impermeable bond with tooth structure. The results of the present study are therefore not surprising. It is interesting to note, however, that by itself a one-millimeter plug of compacted dentin chips produces a seal against isotope penetration as effective as any apical sealing technique tested.

Previous reports (Adams, 1974; Brown et al.,1979) that dentin chips in the apical millimeter of the canal would seal the root canal were not confirmed by the results of this investigation. It must be pointed out that the Ca^{45} (Mev. 0.254) used in previous investigations had an energy level much greater than the S^{35} (Mev. 0.167) used in this study. Ca^{45}, therefore, affects more silver grains in photographic emulsions at a much greater distance than does S^{35}. This tends to create an "halation" artifact which may mask leakage. Also, in the previous investigations the apices of the test teeth were only submerged in the isotope for a period of two hours. More pronounced leakage may have been observed in the present study because the teeth were exposed to the isotope for a period of 48 hours.

Comparison of serial sections within all *in vitro* test groups showed marked variations in leakage patterns (Fig. 12). Kapsimalis et al.(1965) observed similar inconsistencies in leakage patterns in serial cross sections. They suggested that these variations raised a question concerning the validity of results based on autographs of a single section from one tooth. It is agreed that a single central section taken from a given tooth may show minimal or maximal leakage. It

must be suggested, however, that the variations in leakage patterns demonstrated in serial sections do not detract from the statistical significance of single section observations if the sample size is large enough, and if all other variables are constant, e.g., same filling material, same exposure to isotope, etc. Single central sections taken from a large number of samples would have no greater tendency to pass through an area of maximal than minimal leakage. The mean leakage tables above (Tables I and II) confirm this assumption.

Roydhouse et al. (1967, 1968) observed, when diamond or carborundum wheels are used for sectioning, that steps are created due to variations in hardness of enamel, dentin, and restorative materials. They indicated that these steps might trap debris during the cutting procedure and concluded that "some photographs purporting to show leakage reveal instead an accumulation of dye or radioactive debris at such steps". Scanning electron microphotographs (Pashley, 1977) show that diamond wheel sectioning with the Hamco-Gillings machine creates a smeared layer of debris 10-50 microns thick which covers the cut surface and occludes all voids and crevices including exposed dentinal tubules. Smearing artifacts were visible in the autoradiographs from the present study (Fig. 10, 11). Bubbles and crevices, even those which appeared in the embedding medium in direct juxtaposition to the tooth, accumulated radioactive debris (Fig. 11). Whether or not this mechanical smearing and accumulation of radioactive debris significantly alters observed leakage patterns in autoradiographs has not previously been subjected to controlled evaluation. Therefore additional studies were initiated in an attempt to resolve this question.

Ten micron undecalcified sections from teeth embedded in

Caroplastic were obtained using the Jung microtome. Autoradiographs produced from these sections did not show the artifacts seen on the autoradiographs made from sections prepared by the Hamco-Gillings machine. Further evaluation was attempted by producing alternate serial sections from the same specimen using the Jung microtome for one section and the Hamco-Gillings machine for the next section. Autoradiographs produced from these alternate serial sections showed that the microtome sections produced a more refined autoradiograph with an appearance of enhanced resolution (Fig. 13). Furthermore, the autoradiographs produced from the microtome sections did not exhibit the smearing which was observed on autoradiographs produced from the sectioning machine.

Cavit was used as a coronal sealant in this investigation because it has been shown to be the most effective marginal sealant against bacterial penetration of any material tested (Kapsimalis, 1965). However, all autoradiographs from the present *in vitro* study show extensive penetration of the Cavit mass by the isotope (Figs. 10, 11, 12, 14). Similar observations and virtually identical autoradiographs have previously been published by Weine (1972). Whether or not the apparent penetration of Cavit by isotopes is representative of true leakage patterns or due to the hygroscopic nature of the material or mechanical artifact may be questioned. Microscopic examination of Cavit reveals a sponge-like consistency with large voids occurring throughout the body of the material. Similar voids were seen in zinc oxide-eugenol fillings placed with the Lentulo spiral (Fig. 14). These voids in zinc oxide-eugenol fillings were mechanically filled with radioactive debris during sectioning with the Hamco-Gillings machine, producing artifactual leakage patterns. Positive evidence that the presence of isotope in these voids is due to mechanical smearing is supplied by the absence

of isotope in sections prepared from identical material sectioned with the Jung Model K microtome (Figs. 15, 18, 21). The voids in Cavit produce the same mechanically induced accumulation of isotope. In Figure 14A, it is impossible to differentiate between artifactual isotope accumulation in the Cavit temporary filling and the zinc oxide-eugenol root canal filling. It was impossible to determine from this pilot study whether or not the smearing produced significant artifactual variations in leakage patterns. However, since smearing was not demonstrated on the microtome sections, the decision was made to use the Jung microtome for sectioning teeth from the *in vivo* study.

A paired comparison of leakage patterns from the *in vivo* and the *in vitro* autoradiographs showed a consistent and statistically significant (Sine Test, P = 0.05) reduction in isotope penetration for the *in vivo* specimens. Since all teeth, both *in vivo* and *in vitro*, were prepared by the same operator using identical techniques, except for the sectioning procedure, this question must be asked: Is the apparent decrease in autoradiographic leakage due to alteration of the sectioning procedure or to biological enhancement of the *in vivo* apical seal? As mentioned earlier, a previous investigation (Going et al. 1968) suggests that some commonly used restorative materials may actually permit more leakage *in vivo* than *in vitro*. It would appear, therefore, that the difference in observed isotope penetration between the *in vitro* and *in vivo* portions of the present study are due to mechanically induced smearing artifact.

Failure to demonstrate chemographic artifact in this study was not inconsistent with previous reports by Rogers (1973). He stated: "Tissue that has been through the process of fixation, dehydration, embedding...sectioning...is less likely to give rise to this type of

artifact than fresh tissue sectioned on a cryostat, for instance. But one of the most striking facts about this particular source of background grains is its unpredictability". And: "This artifact is so serious a pitfall that adequate control measures must be taken in every single experiment". Therefore, in spite of the fact that chemographic artifact could not be demonstrated in this study, it would appear that controls for chemographic variables should be incorporated in all autoradiographic investigations and studies that are not so controlled are suspect.

The results of the present investigation indicate that mechanical artifact may be produced in autoradiographs prepared from sections cut with the Hamco-Gillings saw.

Based on the above observations, all autoradiographic leakage studies in dentistry should be re-evaluated and/or redone since none have reported controls for chemographic artifact and none have reported controls for mechanical smearing artifact.

Regardless of the success of endodontic therapy implied by clinical observation for varying periods of time, if endodontology is to become a truly scientific discipline, the effects of therapy must be subjected to evaluation using all available investigative techniques.

It is generally accepted that optimum results from endodontic therapy are evidenced by (1) calcified closure of the apical foramen, and (2) no evidence of inflammation in the periapical tissues (Kronfeld, 1949; Strindberg, 1956; Herd, 1976). However, it does not appear that these results are routinely achieved using traditional endodontic materials and techniques.

A major difficulty in achieving the desired results has been that all materials which are currently available as sealers are toxic to some

degree (Langeland, 1974; Spangberg, 1969). These reports indicate a decrease in the inflammatory potential of sealers as setting occurs. However, the above investigators reported only those changes which were observed during very short time periods. Studies using longer experimental time intervals (Brown and Friend, 1968; Friend and Brown, 1968) indicate that the initial decrease of inflammation may be followed by a rebound of increased inflammation after a test period of several months. It may be suggested that this chronic inflammation in the periapical tissue might inhibit continuing apposition of cementum and calcified closure of the apical foramen.

Extrusion of a pigtail of debris which was noted during canal preparation (Fig. 1) in the pilot studies for this investigation has previously been illustrated by Ingle (1976). It is possible that this debris may create no problem in teeth which have vital and uninfected tissues within the canal, but such conditions cannot be predicted clinically. Observations reported by Tronstad (1978), Lambjerg-Hansen (1974), and Herd (1976 and 1978) indicated that a calcified bridge may develop as a result of this material contacting the viable apical pulp stump. However, in the tooth with a necrotic pulp, where bacterial invasion may have occurred, apical compaction of debris may not produce such a desirable result. Ingle (1976) has suggested that: "This material could contain billions of bacteria which would act as a nidus for an acute apical abscess". Matsumiya and Kitamura (1956) have demonstrated, however, that residual bacteria in the apical portion of the canal tend to die as healing occurs. Adams (1974) noted in his pilot studies that this same debris was producing the "seal" which occurred in teeth obturated with broken instruments. Recent investigations (Hession, 1978; Tronstad, 1978) indicate that when instrumentation using

traditional preparation techniques extends to a point one millimeter short of the apical foramen,that the apex is plugged accidentally a high percentage (50-70%) of the time regardless of all precautions taken to maintain patency of the apical canal. It is conceivable, particularly when vital tissues remain in the canal, that "successful" endodontic therapy is a result of this inadvertent plugging of the canal with filing debris.

Enhanced healing and potentiation of physiologic calcification appears to result in spite of the fact that the dentin chip plug does not produce an "hermetic" seal. The use of resorbable, cytotoxic materials in attempts to produce an hermetic seal, therefore, appear to be unwarranted,except for the purpose of creating a coronal barrier to bacterial invasion of the canal and subsequent intra-radicular caries.

If contaminated necrotic debris does act as a nidus for post-treatment bacterial infection, as suggested by Ingle (1976), a canal preparation technique which would eliminate impaction of such debris in the apical tissues should be very useful. The results of pilot studies initiated during the present investigation indicate that canal preparation can be accomplished without the inadvertent impaction of debris into the apical portion of the canal and beyond. Preparation of the canal from the crown down to the apex using large instruments first, and all instruments without apical pressure, effectively removes material from the canal coronally, and virtually eliminates apical impaction and extrusion of contaminated debris. Further evaluation of this modified technique is continuing (Morgan and Pappin, 1979) to determine whether it might also eliminate the additional problems of ledging, perforation and apical transposition of curved canals which

has been reported (Weine, 1975) to occur when using other techniques.

Nygaard-Ostby (1953) stated that:"It has not been possible as yet to find any evidence of an obturation of root canals by hard tissues in cases where a total necrosis of the pulp has prevailed". This statement has been widely misinterpreted to indicate that closure of the apical foramen by calcified tissue will not occur in such cases. It has been assumed that once necrosis is initiated in the pulp, the necrosing process will extend throughout the canal to the apical foramen. Recent investigations (Fournier, 1970; Langeland, 1974) indicate, however, that even in teeth with a clinically totally necrotic pulp, viable tissues may remain in the apical two to three millimeters of the canal. It must be accepted, therefore, that vascular defense mechanisms and cells capable of repair can function at this level even in the "totally necrotic" canal. Cleansing of the canal and obturation at the apical constriction should permit phagocytosis of debris and bacteria with subsequent elimination of inflammation. Healing, repair, and return to physiologic function should then occur, provided some neutral barrier can be placed between these viable apical tissues and toxic sealing materials.

Previous investigators (Senia, 1976; McComb, 1976) have shown that the most effective cleansing of the canal is accomplished in the coronal one-half of the canal. Dentin chips obtained deliberately from this portion of the canal as demonstrated in this study are, therefore, unlikely to be contaminated with bacteria and necrotic debris, and may be more readily assumed to form a neutral biological barrier between filling materials and viable tissues in the apical periodontium than the application of uncontrolled total canal debris.

The results of the present investigation indicate that active

calcification is potentiated in the apical one and one-half millimeters of the canal when autogenous dentin chips from the cleansed canal are placed into contact with viable tissues.

Initial deposition of cementum onto dentin during root development occurs when Hertwig's root sheath begins to disintegrate. Apertures appear in this otherwise complete sheath around the dentin of the root, permitting fibroblasts and other cells of the periodontal ligament to "see" dentin. When this occurs, fibroblasts or primitive connective tissue cells differentiate into cementoblasts, secrete a matrix which calcifies around them and become cementocytes. Subsequent layers, formed in the same fashion, result in increased thickness and the anchoring of collagen fibers of the periodontal ligament as Sharpey's fibers. The deposition of cementum which closes the apical foramen of endodontically treated teeth in association with an apical plug of compacted dentin chips probably occurs due to a similar inductive mechanism as is found during the natural developmental, biologic process described above.

It is further interesting to note that calcification as indicated by the tetracycline label occurs within the dentin chip plug. It has been shown that the organic matrix obtained from lyophilized, demineralized dentin will stimulate osteogenesis even in large bony defects which may heal otherwise only by fibrous union (Bang, 1972). It is possible that the organic matrix on the surface of the dentin chips presents a similar matrix for calcification within the apical plug. This possibility was previously suggested by Gottlieb (1950), but has not, to date, been subjected to controlled investigation in the apex of human teeth.

Since it has been established that tissues can remain viable in

the canal near the apex, even when in contact with a totally necrotic pulp, it would appear that elimination of the large pulpal nidus of inflammation and the placing of a calcifiable matrix in contact with a vital periodontium might potentiate apposition of cementum and result in the physiologic closure of the apical canal, regardless of the pre-operative condition of the pulp.

Due to restrictions imposed by time and the limited availability of suitable human volunteers, the experimental population used in this investigation was small. The results do, however, support findings reported by previous investigators. The technique demonstrated and tested (root canal preparation short of the apical foramen and the deliberate placement of an apical plug of coronally obtained dentin chips) resulted in a biologic barrier. The technique did not result in clinical evidence of apical inflammation, i.e., pain and/or swelling, for any tooth so treated.

It must be emphasized that in the present investigation, (1) no medications of any kind were placed in the canal; (2) all irrigation was accomplished with sterile tap water delivered to the depth of the prepared canal using a 27 gauge needle; (3) all treatment on each tooth was completed in a single appointment; and (4) all experimental teeth contained clinically viable pulp tissue. The results may or may not be altered if medications are placed in the canal, if chemical irrigants are used, if treatment requires more than one appointment, or if necrotic material remains in the canal. The techniques developed in this study could well be used in the future evaluation of these variables.

Histologic confirmation of the findings suggested by the tetracycline labeling were not attempted in the present investigation since decalcification would destroy the tetracycline label. Histologic

evaluation of the apical dentin plug in human teeth will be required to truly evaluate the quality of the calcification and the type of hard tissue formed.

Silver amalgam is frequently used for closing the apex when endodontic surgery becomes necessary. It may be suggested, based on the results of this investigation, that dentin chips or lyophilized, demineralized dentin may also be the material of choice for these retrograde fillings.

SUMMARY AND CONCLUSIONS

This investigation studied *in vitro* and *in vivo* techniques in an attempt to answer the following questions:

(1) Can the apical one millimeter of the prepared root canal system be sealed with autogenous dentin chips?

(2) Does the filling material used to obturate the root canal system coronal to the dentin chips have any additional effect on the apical seal?

(3) Will calcified material be deposited in the canal and/or foramen apical to the obturating material?

Sixty single-rooted extracted human teeth were prepared and obturated using a one-millimeter apical plug of dentin chips alone and in conjunction with laterally condensed gutta percha or zinc oxide-eugenol placed with a Lentulo spiral.

Twenty-two single-rooted human teeth with clinically viable pulps were treated *in vivo* with the techniques used in the *in vitro* investigation except that tetracycline was administered systemically to label sites of active mineralization.

All teeth were sectioned and evaluated for apical leakage following immersion in S^{35} for a period of 48 hours. Teeth from the *in vivo* study were also examined for tetracycline labeling using the ultraviolet microscope.

The results indicate:

(1) The apical one millimeter of the canal was not sealed against penetration by S^{35} using a one-millimeter apical plug of dentin chips.

(2) The tested filling materials used to obturate the root canal coronal to the dentin chips had no additional effect on the apical seal.

(3) Active calcification occurred in the apical canal adjacent to the dentin chip plug in every case following an *in vivo* test period of 98 days. Calcification was not observed under any other test conditions.

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FIGURE 1: APICAL PREPARATION DEBRIS

Note the "pig-tail" of debris extruded through the apical foramen when utilizing the traditional (apex to crown) canal preparation technique (lower bicuspid).

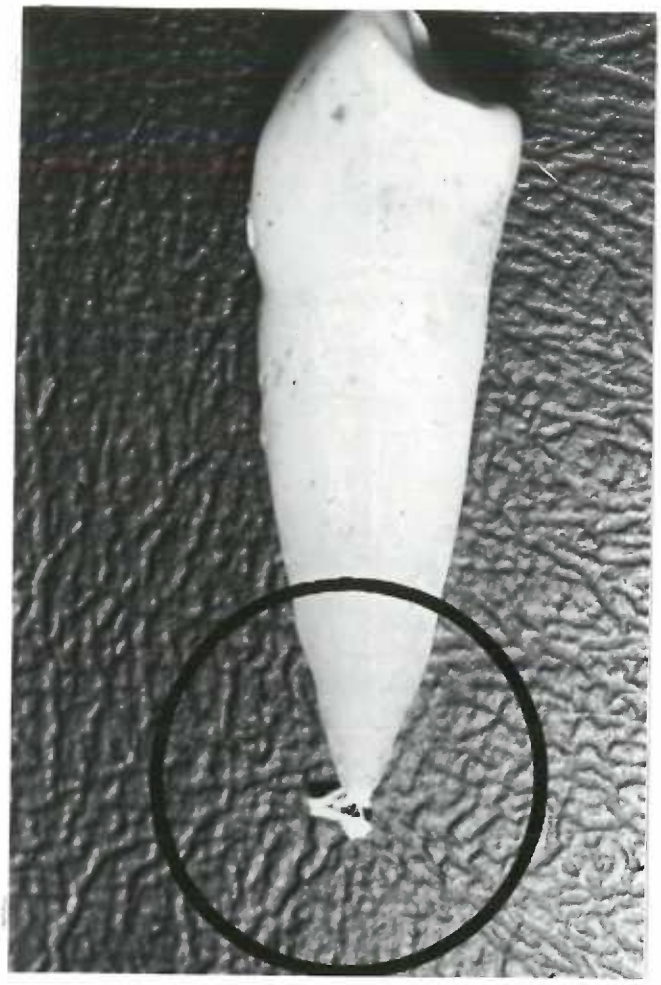


FIGURE 1

FIGURE 2: CANAL PREPARATION TECHNIQUE: CORONAL ACCESS

Traditional coronal access openings were used. Penetration into the pulp chamber was made with the drill directed toward the long axis of the root using a number four round bur. After the bur "dropped" through the roof of the pulp chamber, the pulp chamber was unroofed by cutting on the pull stroke at reduced r.p.m. Using the flange of the bur as an "explorer" and cutting only on the pull stroke, the chamber was unroofed completely and efficiently.

Following unroofing of the pulp chamber, coronal tissue remnants were removed, using thorough irrigation with water and long-shanked endodontic curettes.

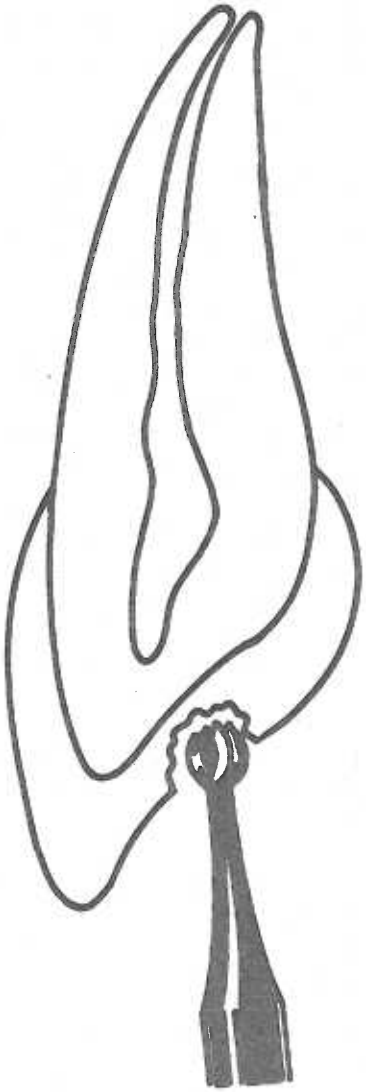


FIGURE 2

FIGURE 3: CANAL PREPARATION TECHNIQUE: RADICULAR ACCESS

The depth of the radicular access preparation was determined by placing a number thirty-five file into the canal without force or rotation to that point where resistance to apical insertion was encountered. This length was then recorded as the depth to which Gates-Glidden drills were used for preparation of the radicular access.

The pulp chamber was flooded with water and a number two Gates-Glidden drill was carried once, in and out, at low r.p.m., to the defined radicular access length. The number two Gates-Glidden drill was followed by the number three Gates-Glidden drill to the same depth. The Gates-Glidden drills should not, at any time, be used forcefully in an attempt to negotiate the canal beyond the length defined by the number thirty-five file.

Upon completion of radicular access with the Gates-Glidden drills, the canals were irrigated thoroughly with a minimum two milliliters of water delivered through a twenty-seven gauge needle to the depth of radicular access.

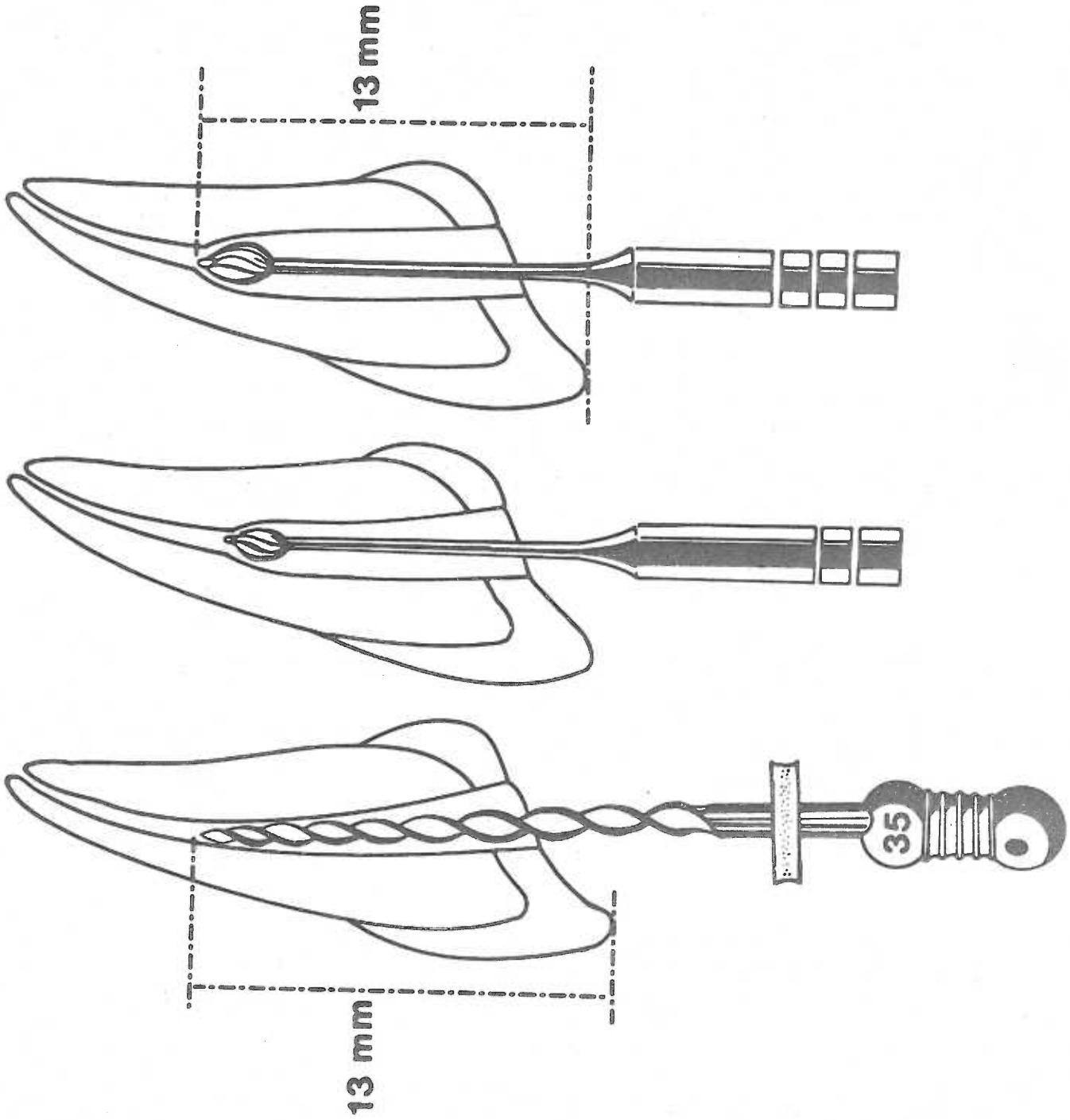


FIGURE 3

FIGURE 4: CANAL PREPARATION TECHNIQUE: APICAL MATRIX

The apical matrix was defined as that portion of the canal preparation that extended from the depth of the radicular access to a point 1.5 millimeters from the radiographic apex of the root.

Preparation of the apical matrix was initiated by placing a number thirty file into the canal just to that point where resistance to apical insertion was encountered. The file was then rotated, clockwise, two complete revolutions and removed from the canal. The same procedure was repeated with consecutively smaller files until a file reached a point three millimeters (Diagnostic Working Length = DXL) from the radiographic apex as measured from the pre-operative radiograph.

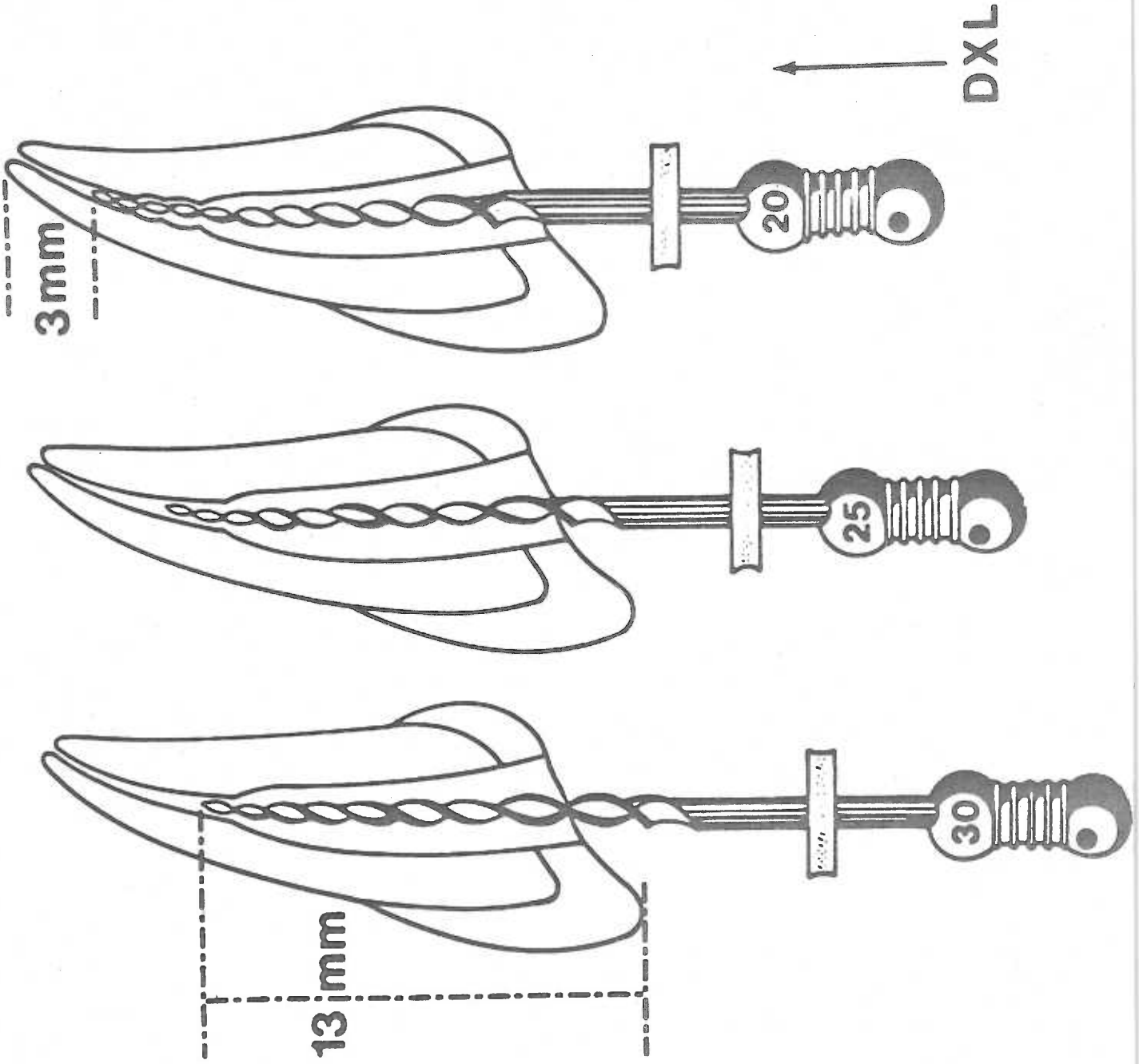


FIGURE 4

FIGURE 5: CANAL PREPARATION TECHNIQUE: TRUE WORKING LENGTH

The first file that achieved the diagnostic working length (DXL) was left in place in the canal and a radiograph was exposed to verify the true length of the root. Following verification of true working length, the file was rotated two complete revolutions and removed from the canal. Consecutively smaller files were then placed into the canal without apical pressure and rotated two complete revolutions and removed until the true working length (TWL) was achieved at a point 1.5 millimeters from the radiographic apex.

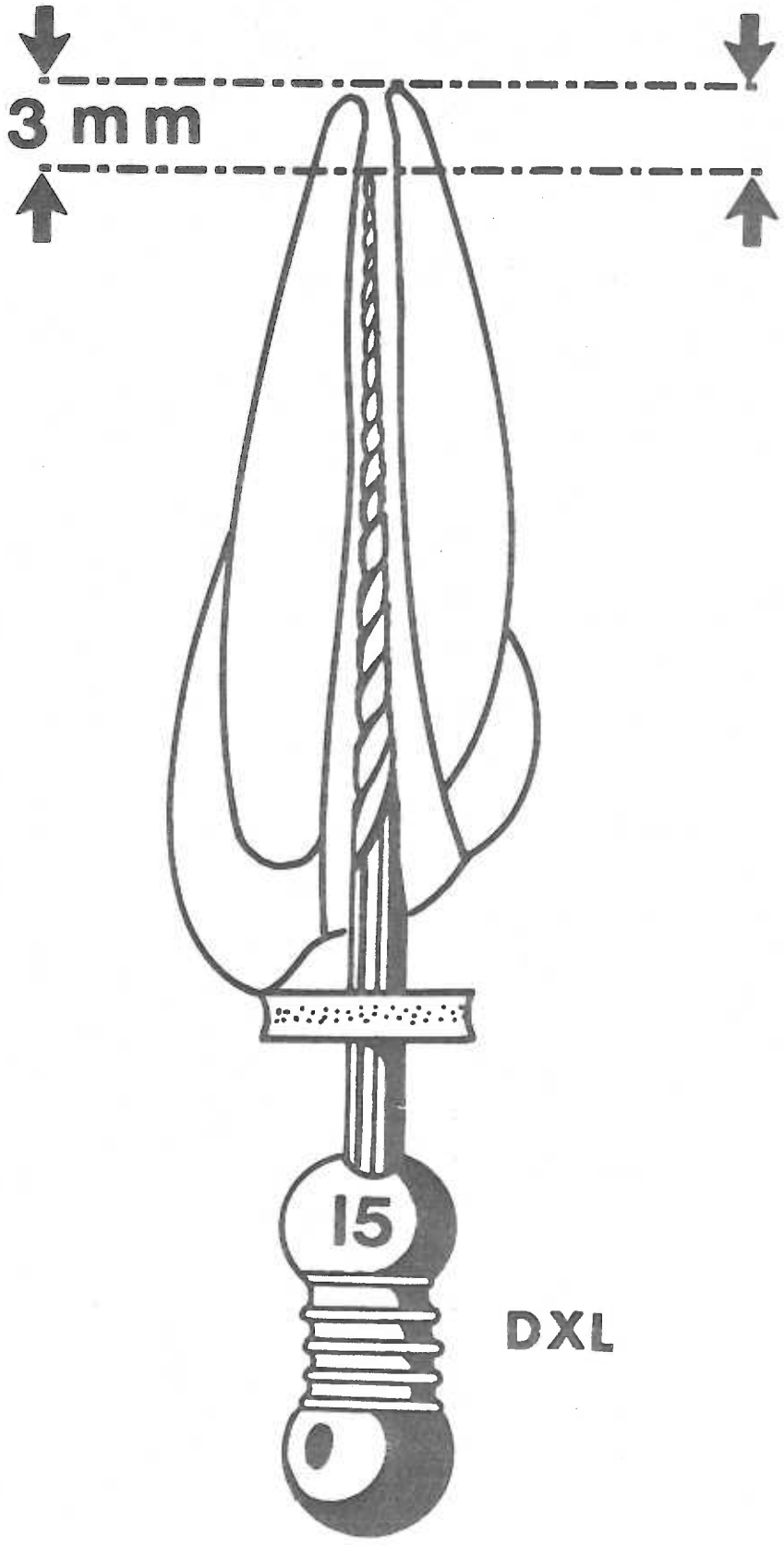


FIGURE 5

FIGURE 6: CANAL PREPARATION TECHNIQUE: APICAL STOP

Following definition of the true working length, an apical stop was completed at that depth, utilizing the inverse reaming procedure illustrated.

A number thirty-five file was placed into the apical matrix until resistance was encountered. The file was rotated two complete revolutions and removed from the canal. This procedure was repeated with consecutively smaller files until the true working length was achieved.

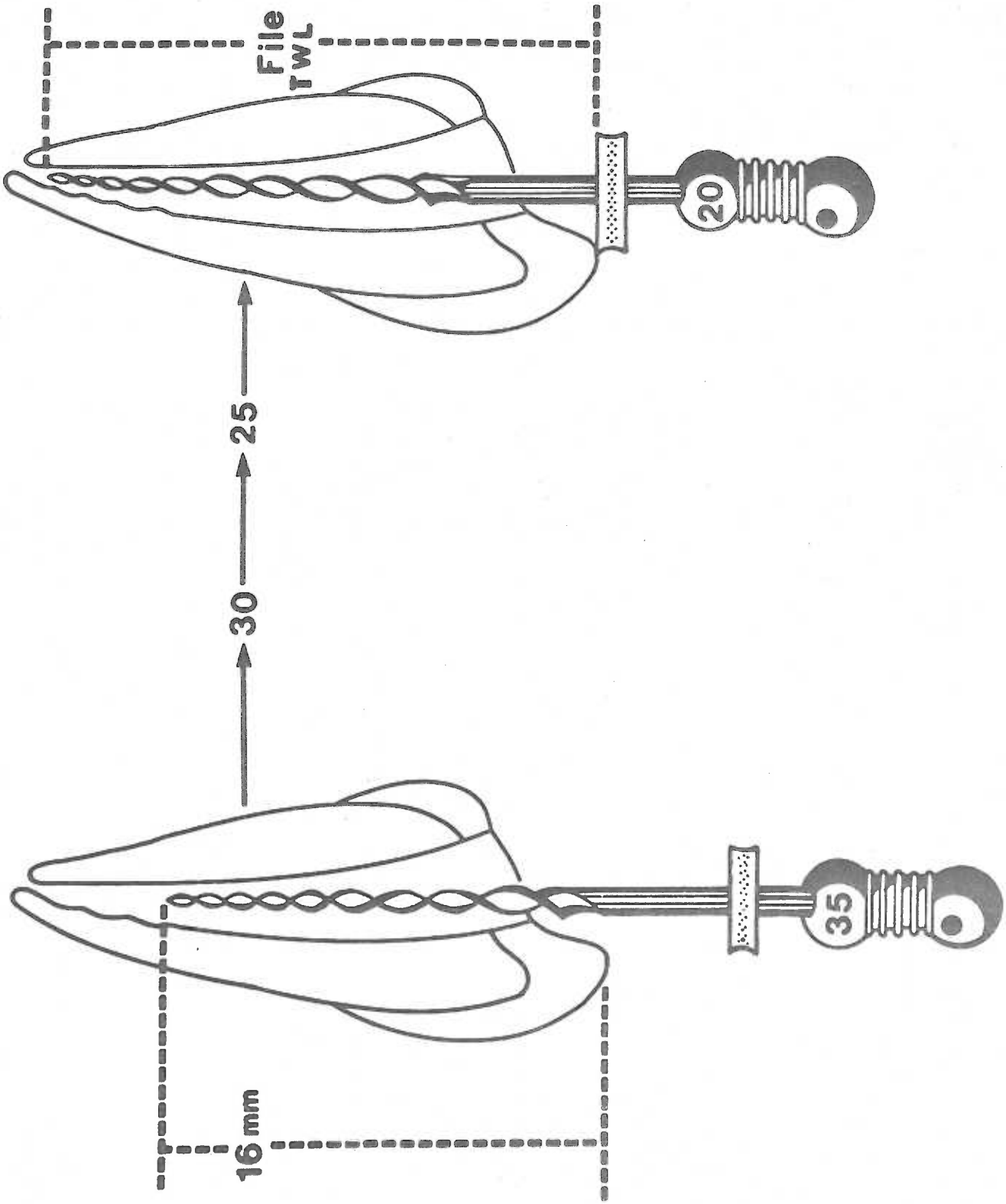


FIGURE 6

FIGURE 7: CANAL PREPARATION TECHNIQUE: APICAL STOP

The apical stop was completed by repeating the inverse reaming procedure using a file one size larger at the initiation of each apical progression through the apical matrix.

The final size of the apical stop was determined by canal morphology and the judgment of the operator.

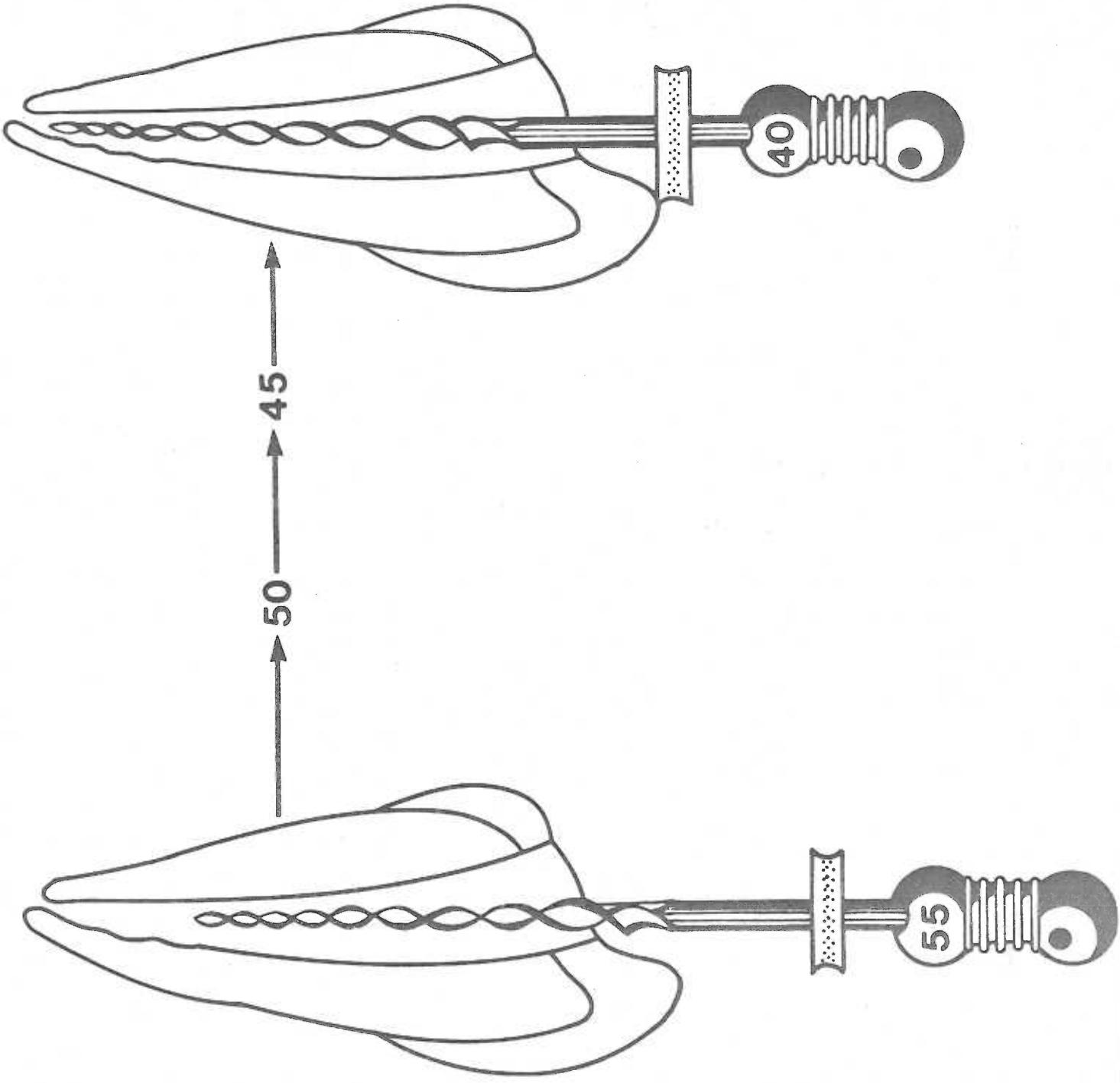


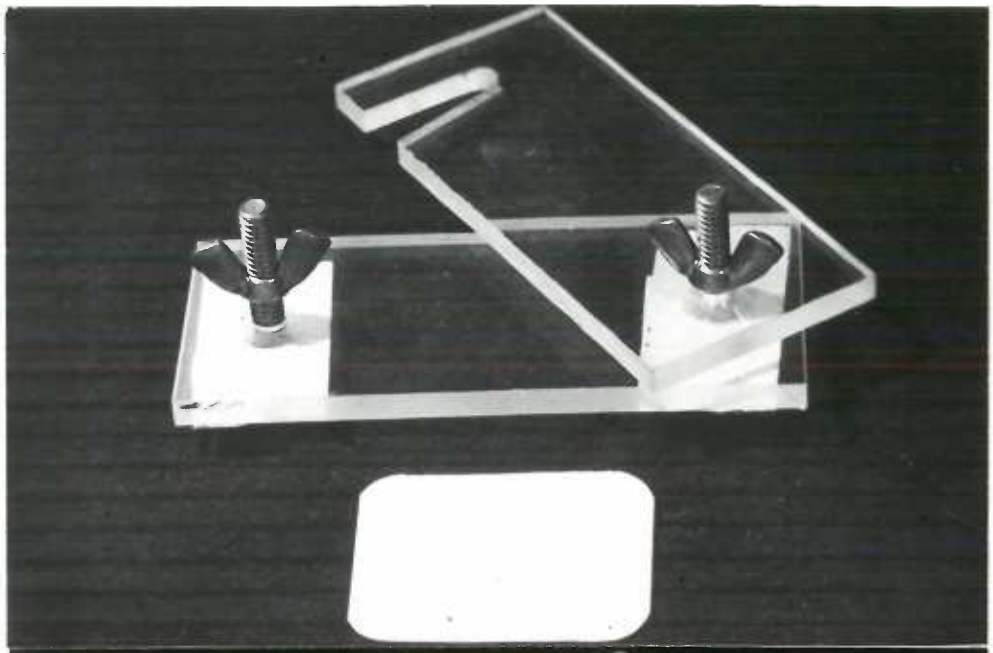
FIGURE 7

FIGURE 8: AUTORADIOGRAPHIC EXPOSURE TECHNIQUE

Figure 8A shows double-clamping plastic vise and dental x-ray film used to prepare autoradiographs.

Figure 8B shows tooth section placed on emulsion surface of film in place between jaws of plastic vise.

A



B

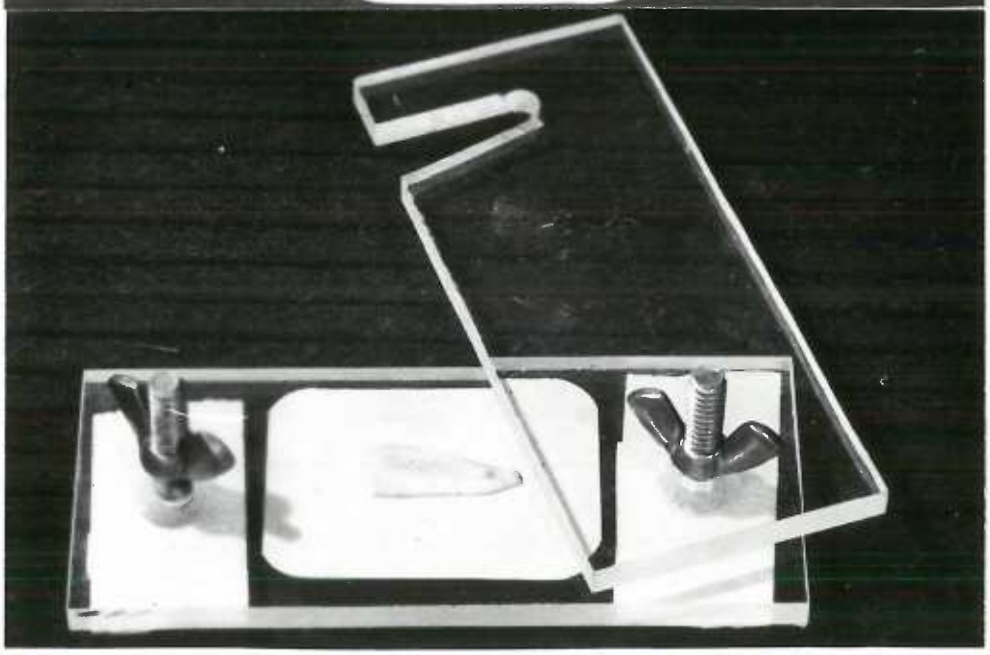


FIGURE 8

FIGURE 9: AUTORADIOGRAPHIC EXPOSURE TECHNIQUE

Figure 9A shows section of tooth in place on emulsion side of film with jaws of plastic vise secured in place.

Figure 9B shows vise and contents placed in light-tight box. Box is closed during exposure time required for preparation of autoradiograph and during the soft x-ray exposure.

A

B

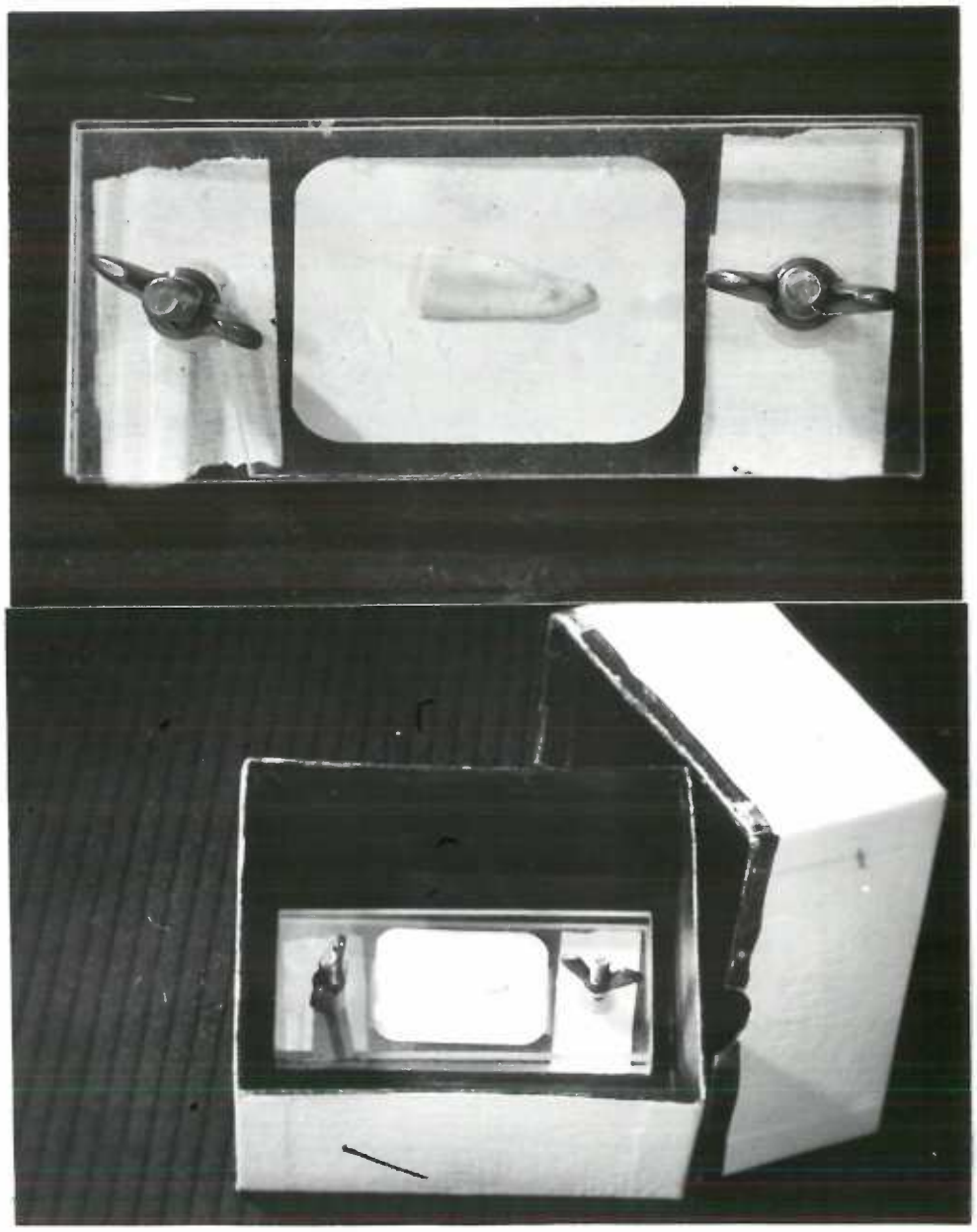


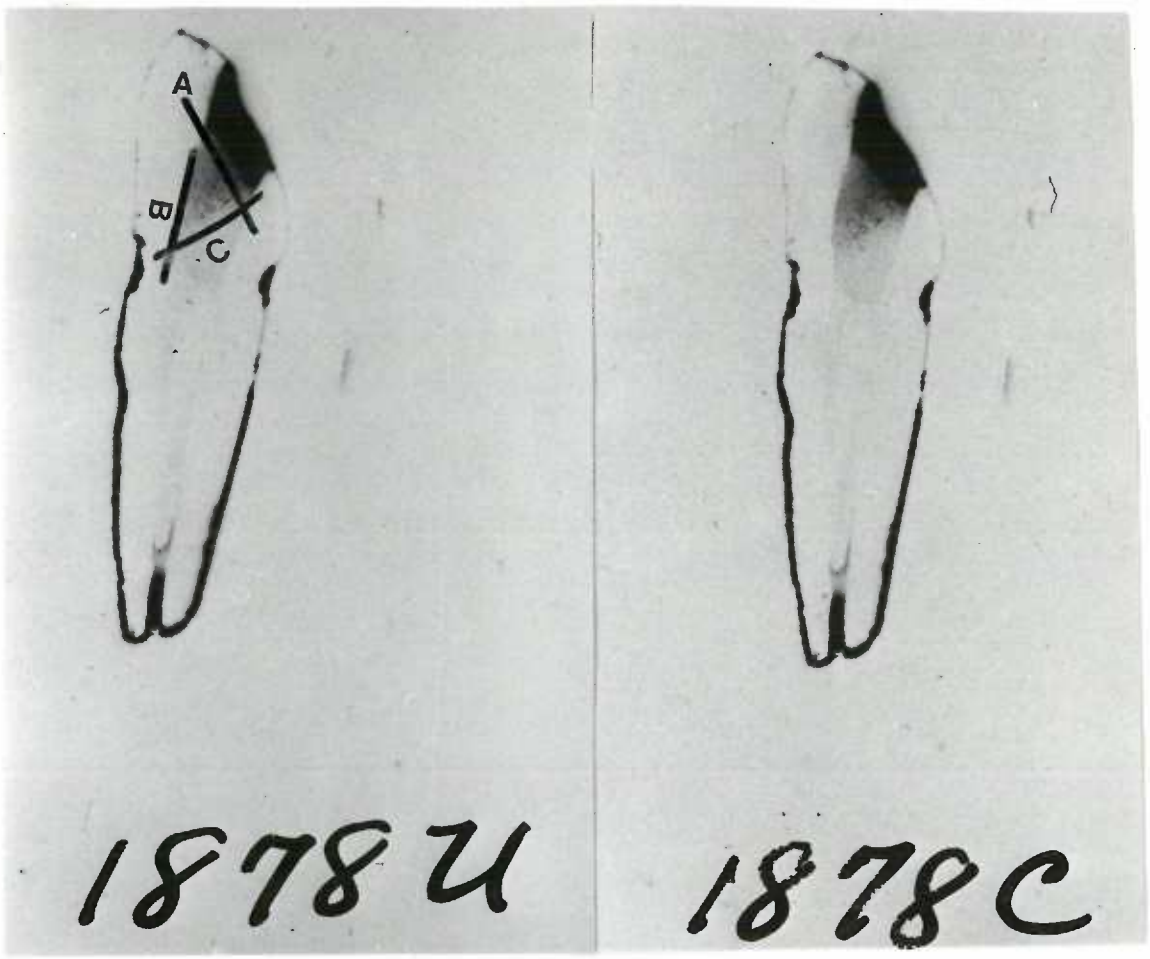
FIGURE 9

FIGURE 10: CHEMOGRAPHIC CONTROLS

Autoradiographs were prepared from sections three hundred microns thick, cut with a Hamco-Gillings saw. Following preparation of autoradiograph 10A, the section was coated with 10% nitrocellulose in ether: absolute alcohol. A second autoradiograph, 10B, was then prepared as a control for chemographic artifact. No evidence of chemographic artifact could be identified in any case.

Note penetration of the isotope into the Cavit temporary filling material to the depth of line drawn at A. There is further accumulation of isotope in the triangle demarcated by line B. The isotope in this triangle is interpreted as artifactual smearing created as the saw blade passed through the temporary filling (direction of saw blade is indicated by curved line C. The black spots in triangle B correspond to voids which are characteristically present in Cavit as well as in zinc oxide-eugenol fillings which are placed with a Lentulo spiral. The dark spots are definitely due to smearing and accumulation of isotope in these voids. Compare these dark spots in saw-prepared sections to the absence of such spots in microtome-prepared sections (Figs. 15, 18, 21).

The canal in this specimen was obturated with inadequately compacted gutta percha and sealer. In this specimen there is evidence of the isotope throughout the canal wall-filling interface. There is no way to determine, however, whether this represents isotope penetration or artifactual accumulation.



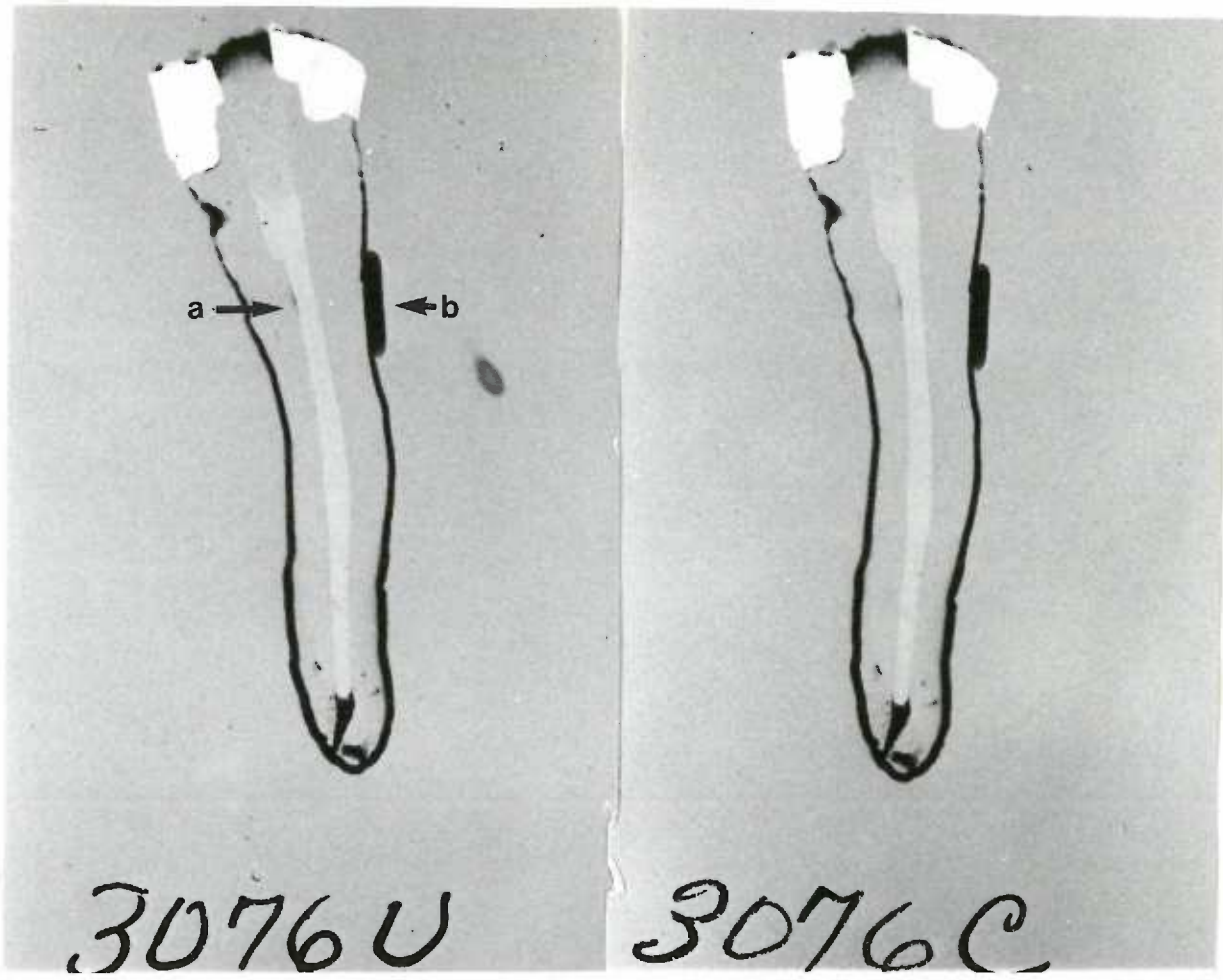
A B

FIGURE 10

FIGURE 11: CHEMOGRAPHIC CONTROLS

Autoradiographs prepared from a section 300μ thick, cut with Hamco-Gillings saw. Following preparation of autoradiograph A, the section was coated with 10% nitrocellulose. A second autoradiograph, B, was then prepared as a control for chemographic artifact. No evidence of chemographic artifact could be identified.

The canal was obturated with laterally condensed gutta percha and sealer. Note that there is minimal isotope penetration along the canal wall-filling interface. There is, however, artifactual accumulation of the isotope in a void (arrow a) at the interface in the coronal one-third of the canal. There is also an artifactual accumulation of isotope in an embedding bubble (arrow b) on the lateral border of the root.

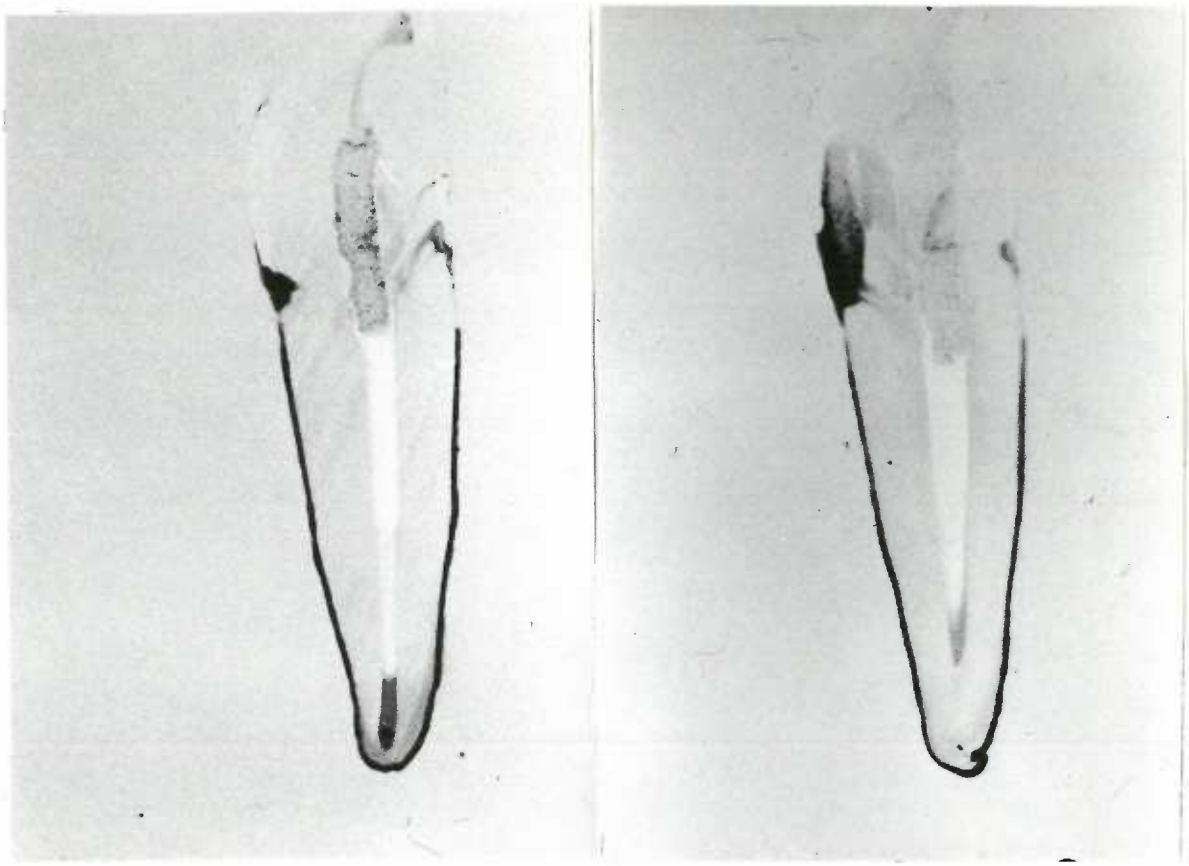


A B

FIGURE 11

FIGURE 12: SMEARING CONTROLS

Autoradiographs prepared from alternate sections, 300μ thick, from the same tooth. Sections were prepared with the Hamco-Gillings saw. Note that Figure 12A shows minimal penetration of the isotope at the apex. Figure 12B, however, shows accumulation of the isotope throughout the canal wall-filling interface. Note, also, that Cavit temporary filling material shows artifactual accumulation of isotope in voids similar to autoradiographs produced from zinc oxide-eugenol sections cut with Hamco-Gillings saw (Fig. 14).

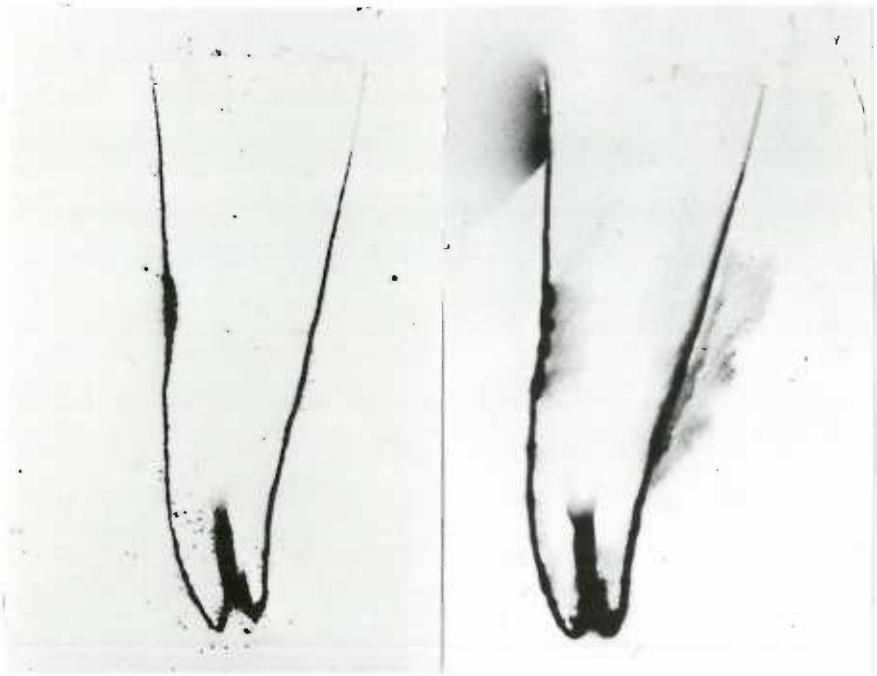


A B

FIGURE 12

FIGURE 13: SMEARING CONTROLS

Serial sections from a specimen obturated with laterally condensed gutta percha and sealer. Figure 13A shows autoradiograph prepared from 10μ section cut with the Jung microtome. Figure 13B shows autoradiograph prepared from 300μ section cut with the Hamco-Gillings saw. Note (1) absence of smearing artifact and (2) refinement of autoradiographic tracing in 13A as compared to 13B.

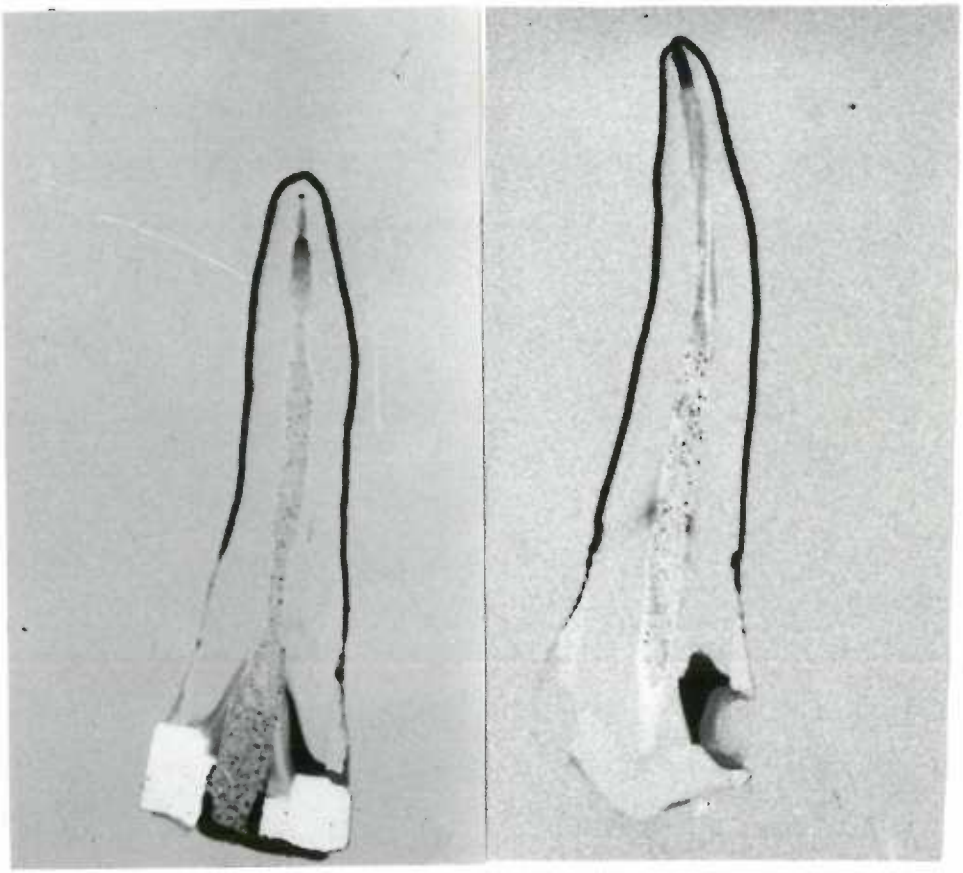


A B

FIGURE 13

FIGURE 14: SMEARING CONTROLS

Canals in two specimens which were obturated with zinc oxide-eugenol paste inserted with Lentulo spiral on slow-speed handpiece. These 300μ sections were prepared with the Hamco-Gillings saw. Note artifactual accumulation of isotope in voids throughout entire body of filling material. Compare this to total absence of isotope in 10μ sections cut with the Jung microtome (Figs. 15, 18, 21).

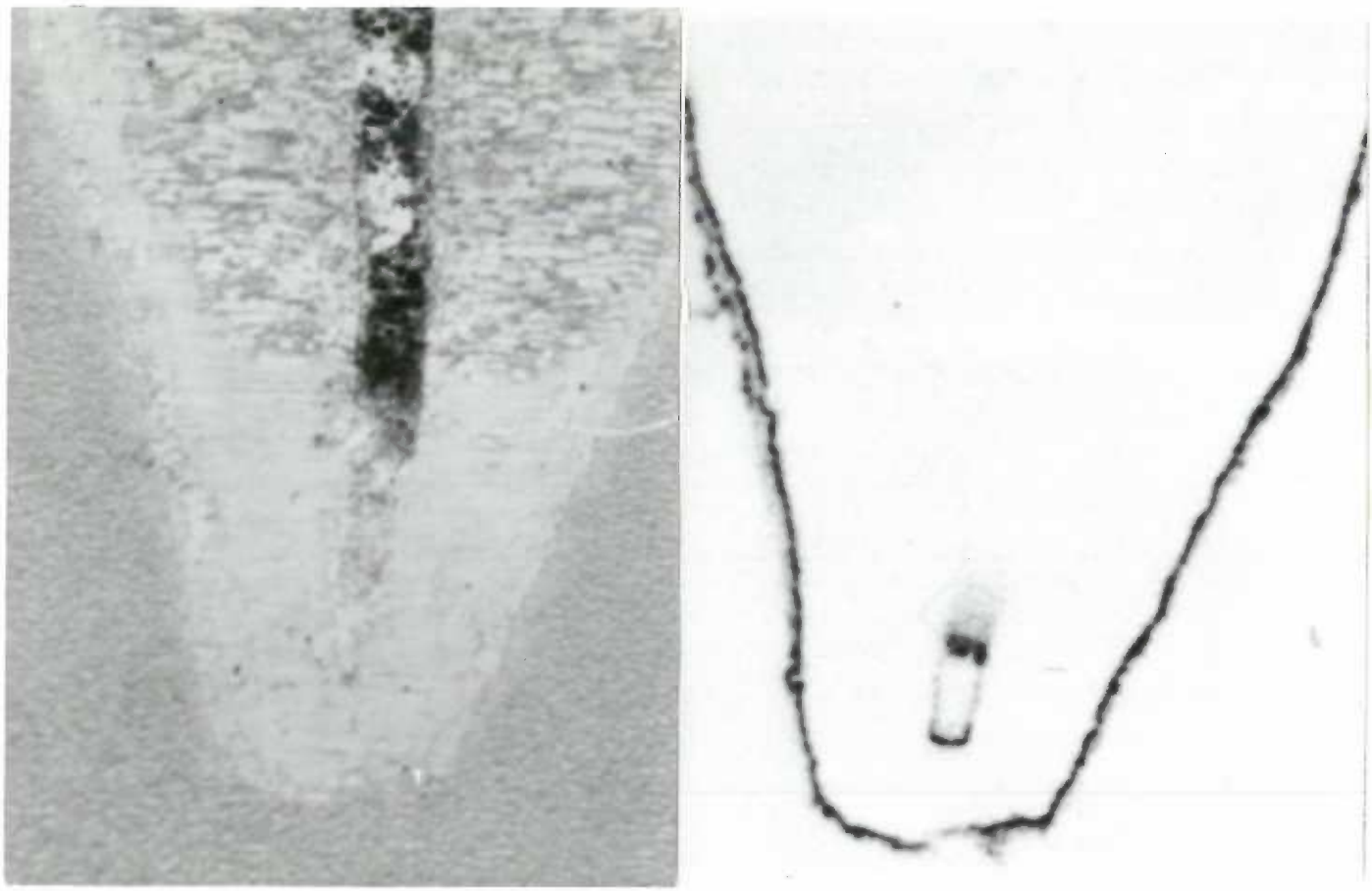


A B

FIGURE 14

FIGURE 15: SMEARING CONTROLS

Photomicrograph (15A) and autoradiograph (15B) prepared from 10μ section cut with the Jung microtome. Canal was obturated with zinc oxide-eugenol paste inserted with a Lentulo spiral on slow speed handpiece. Note that, even at 20X, there is no autoradiographic evidence of isotope in voids present in filling materials.



A B

FIGURE 15

FIGURE 16: TETRACYCLINE LABELING SERIES

Control specimen extracted at 46th post-treatment day. Canal was prepared and number 10 file was extruded through apical foramen to insure patency. Canal was left unobturated and coronal access was closed with 4 millimeters of Cavit. Note traces of tetracycline labeling (arrows) along lateral borders of root. This labeling, also seen on other specimens, is interpreted to be evidence of physiologic remodeling of cementum.

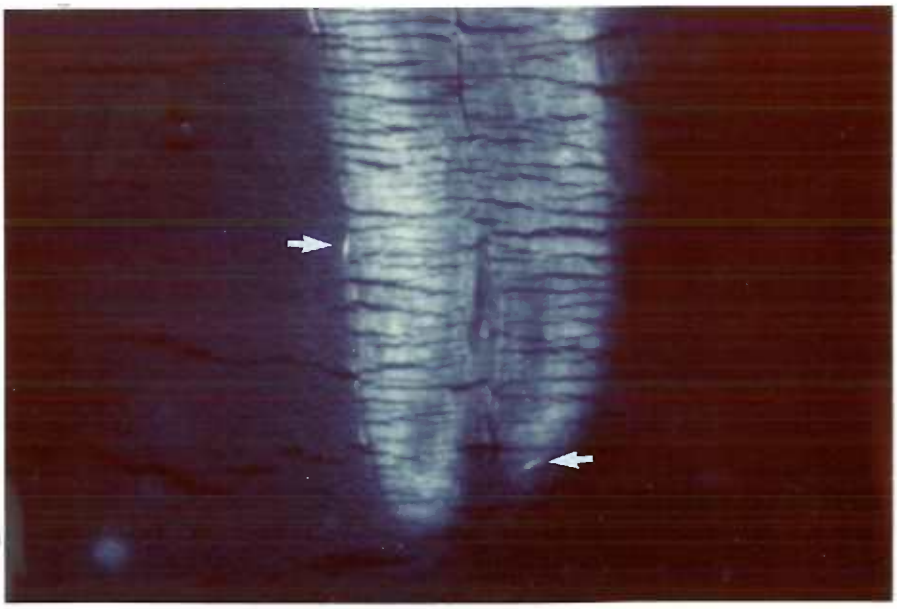


FIGURE 16

FIGURE 17: TETRACYCLINE LABELING SERIES

Photomicrograph, 17A, of a specimen extracted at the 98th post-treatment day. The canal was obturated with laterally condensed gutta percha and a sealer. A one-millimeter plug of autogenous dentin chips was placed into the apical portion of the canal. The section is slightly tangential to the canal. The dark area in the canal between the apical tetracycline label (ATL) and the labeled dentin chip plug (DCP) is the outer margin of the canal wall. The apical tetracycline label represents active calcification on the lateral borders of the canal wall. Labeling in the dentin chip plug represents a passive up-take of the label, possibly on the surface of exposed dentin matrix in the plug.

Autoradiograph, 17B, shows penetration of isotope in small area of dentin chip plug apically (DCPA), but no evidence of isotope in dentin chips more coronally placed.

A

B

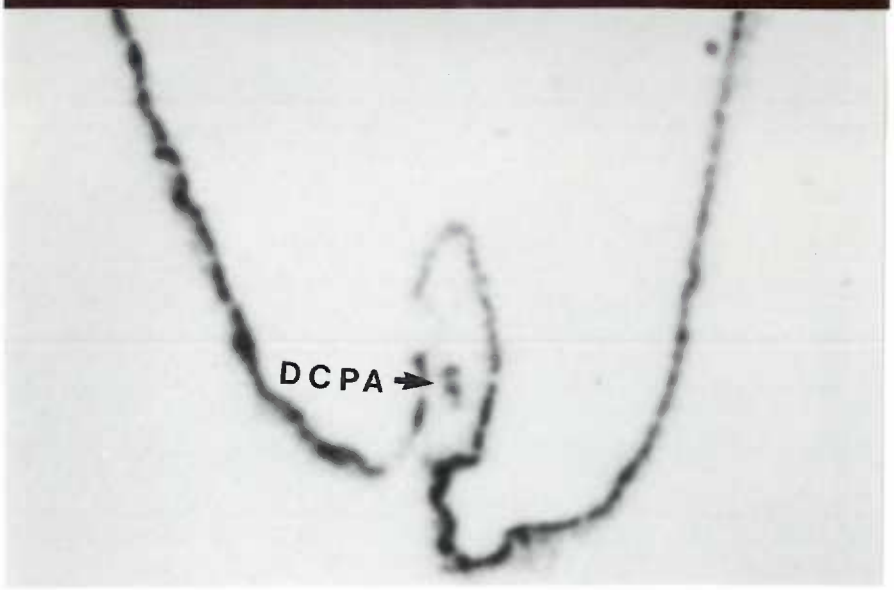


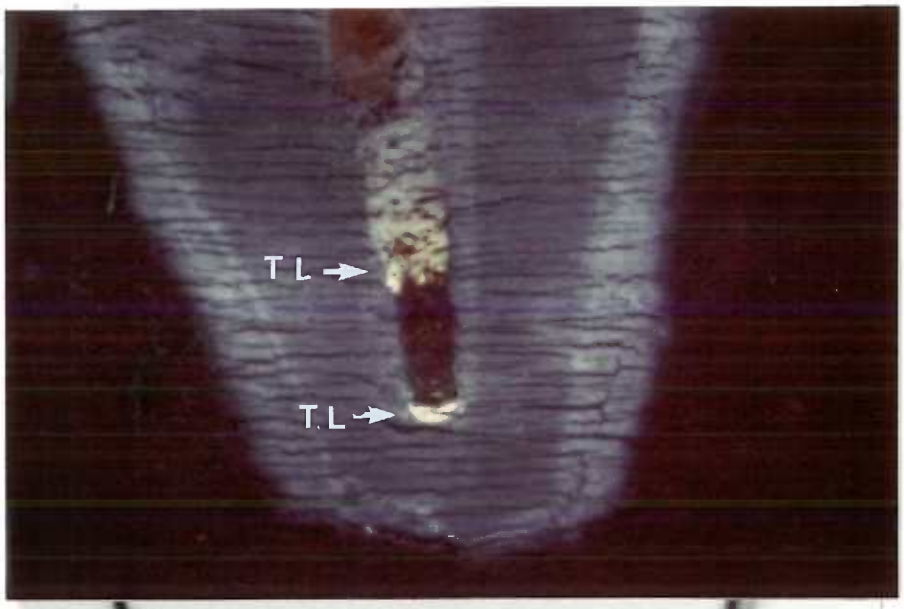
FIGURE 17

FIGURE 18: TETRACYCLINE LABELING SERIES

Photomicrograph, 18A, of a specimen extracted at the 98th post-treatment day. The canal was obturated with zinc oxide-eugenol paste placed with a Lentulo spiral on slow speed handpiece. A one-millimeter plug of autogenous dentin chips was placed in the apical portion of the canal. Note evidence of tetracycline label (TL) in dentin chip plug. Dark area between labeled areas is where a portion of the dentin chips fragmented away during sectioning.

Autoradiograph, 18B, of same 10μ section shows penetration of isotope into, but not completely through, the dentin chip plug (DCP).

A



B

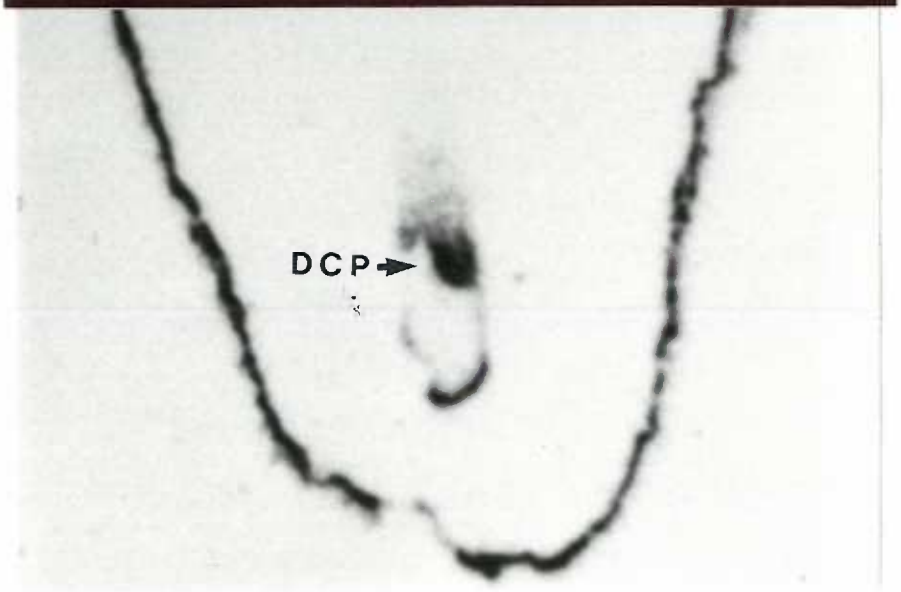


FIGURE 18

FIGURE 19: TETRACYCLINE LABELING SERIES

Photomicrograph, 19A, shows a specimen extracted at the 98th post-treatment day. The canal was prepared but left unobturated except for a one-millimeter apical plug of autogenous dentin chips. Section, lateral to central canal, was selected to show tetracycline label (TL) along lateral borders of the canal apical to the dentin chip plug. Note, also, the tetracycline labeling along the lateral border of the root.

A

B

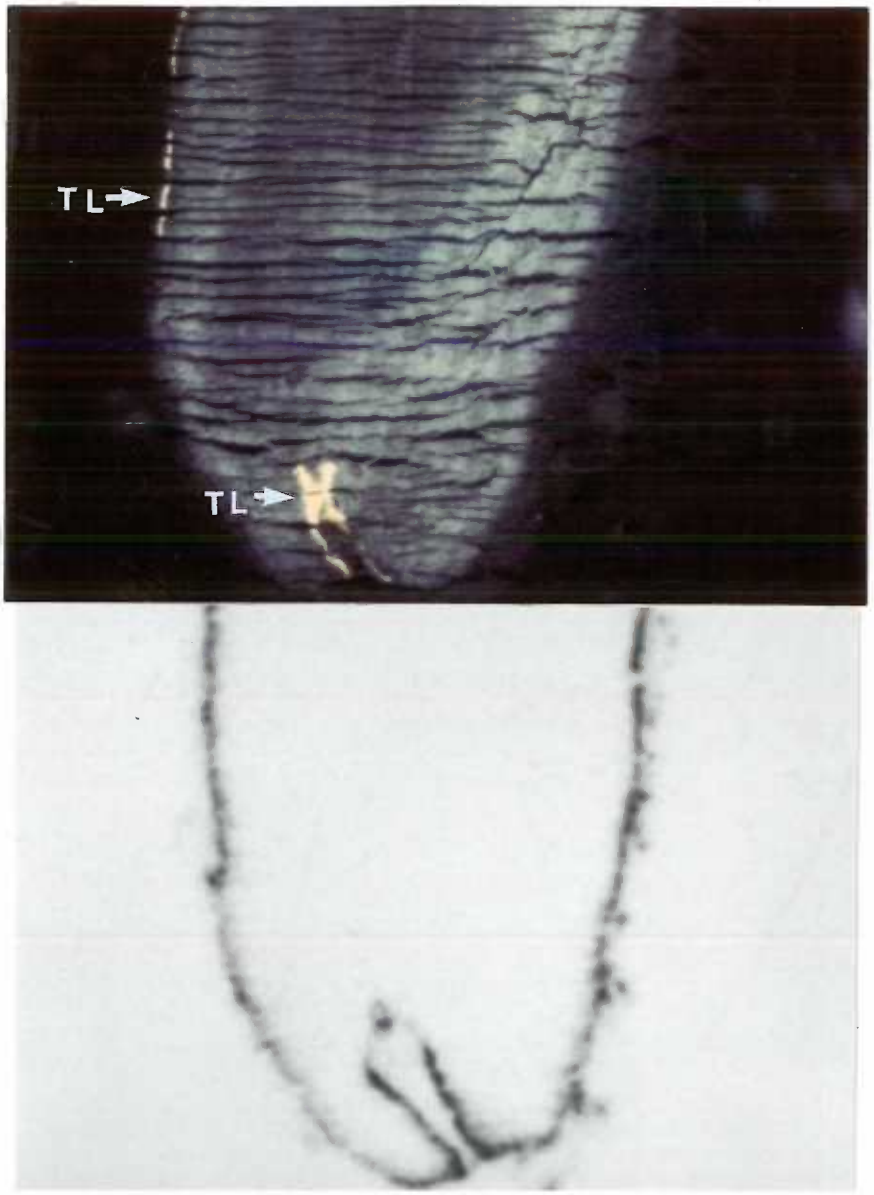


FIGURE 19

FIGURE 20: TETRACYCLINE LABELING SERIES

Photomicrograph, 19A, shows a specimen extracted at the 98th post-treatment day. The canal was obturated with laterally condensed gutta percha and a sealer but contained no apical dentin chips. No evidence of tetracycline labeling is visible. Note clear differentiation of autofluorescence between cementum (cem) and dentin (den).

Autoradiograph, 20B, prepared from same 10μ section. Autoradiograph shows no penetration of the isotope coronal to gutta percha filling material.

A

B

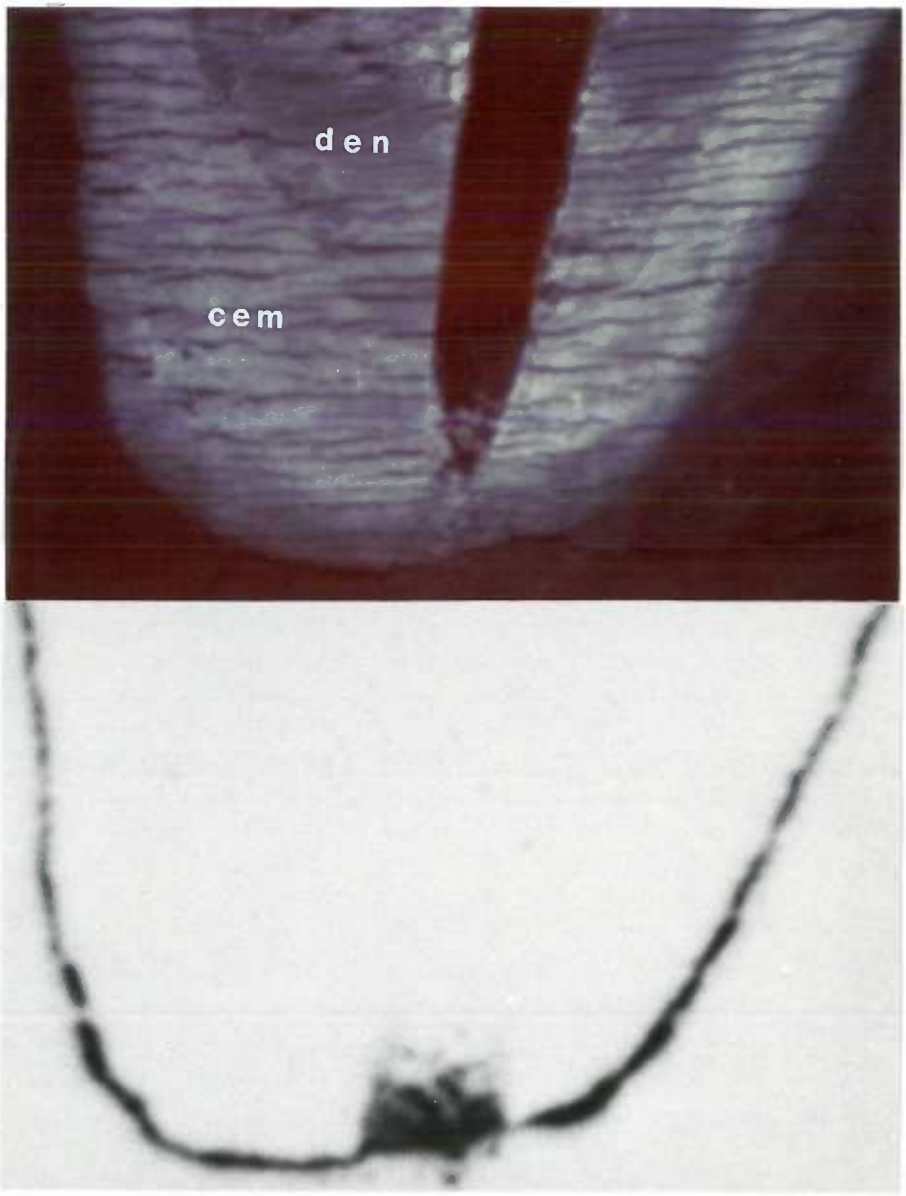


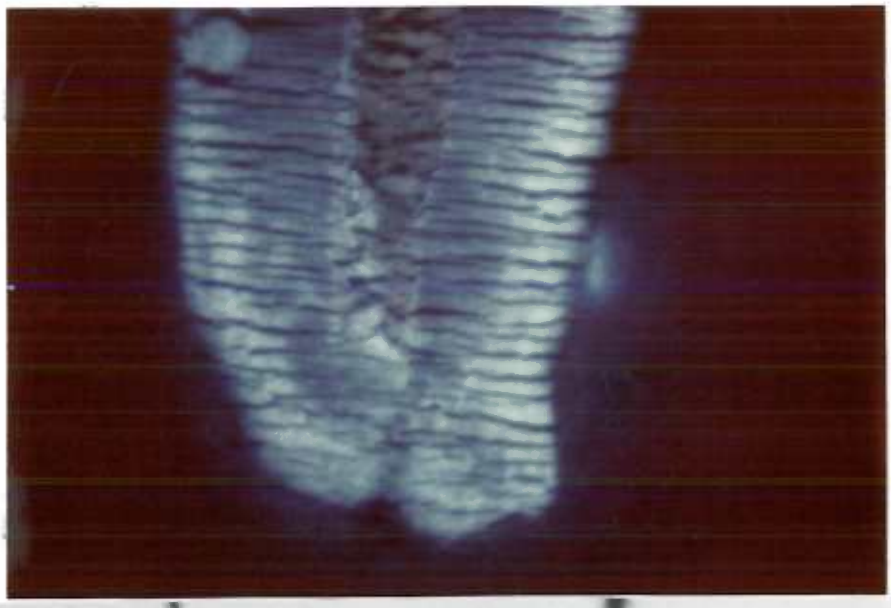
FIGURE 20

FIGURE 21: TETRACYCLINE LABELING SERIES

Photomicrograph, 21A, shows a specimen extracted at the 98th post-treatment day. The canal was obturated with zinc oxide-eugenol placed with a Lentulo spiral on slow speed handpiece. No dentin chips were placed in the apical portion of the canal. No evidence of tetracycline labeling is seen.

Autoradiograph, 21B, prepared from the same 10μ section. Autoradiograph shows only minimal penetration of the isotope into the body of the zinc oxide-eugenol filling material. Note, again, that there is no artifactual accumulation of the isotope in voids which are present throughout the body of the filling material.

A



B

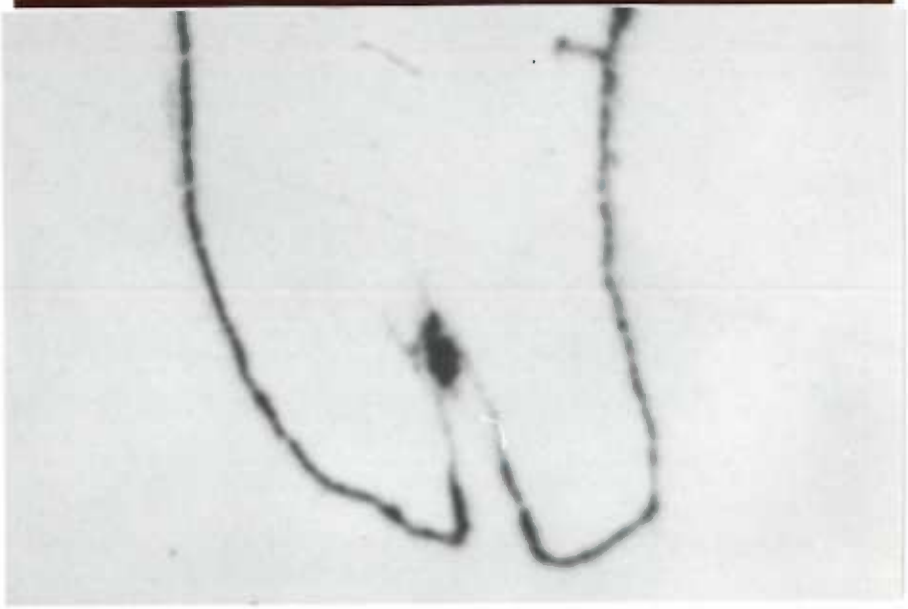


FIGURE 21

FIGURE 22: TETRACYCLINE LABELING SERIES

Photomicrograph, 22A, shows a control specimen extracted at the 98th post-treatment day. The canal was prepared and a number 10 file was extruded through the apical foramen to insure patency. The canal was left unobturated and the coronal access was closed with 4 millimeters of Cavit. This section, although lateral to the central canal, was selected to show the cementum with a tetracycline label (TL) near the root apex but lateral to the apical foramen.

A

B

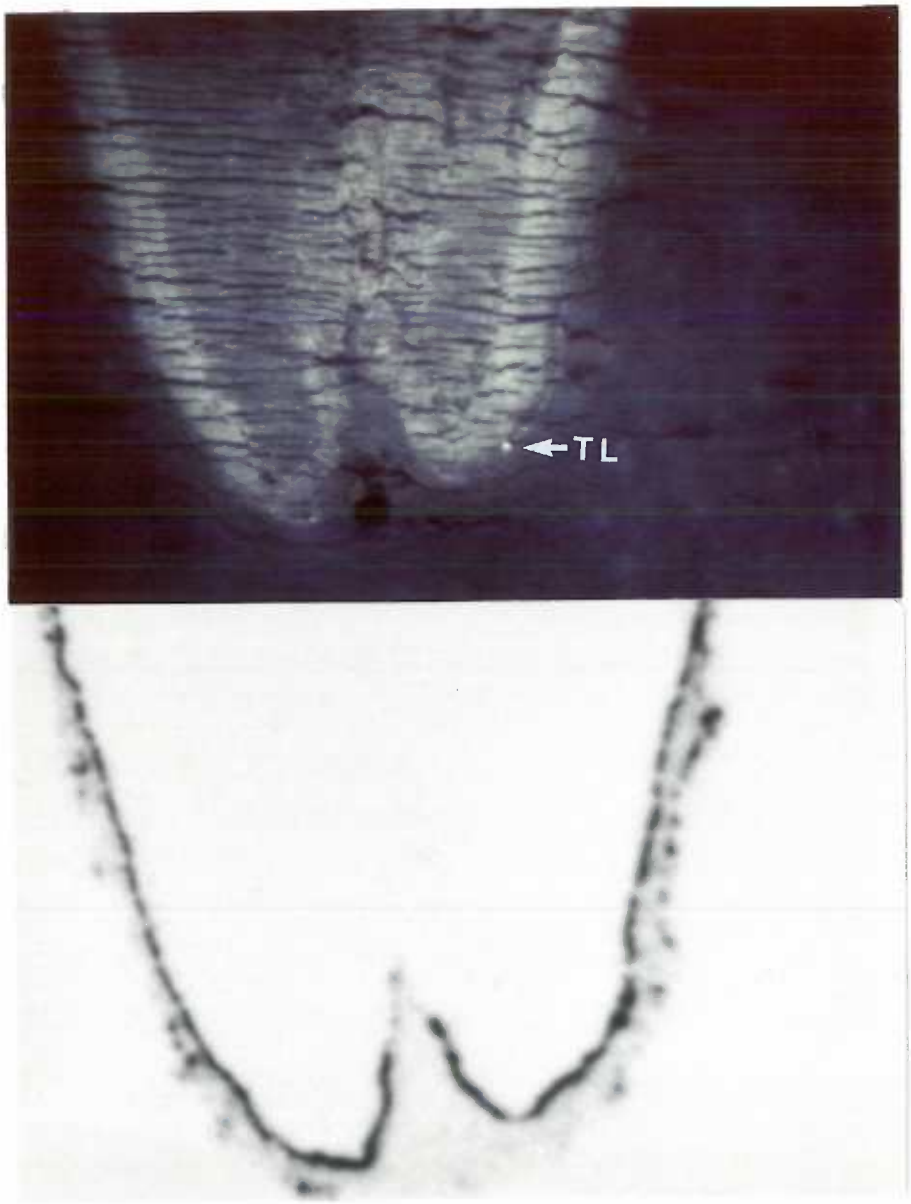


FIGURE 22

APPENDIX A

INFORMED CONSENT - NARRATIVE

Biologic Sealing of the Apex in Endodontically Treated Teeth

You are being asked to take part in a study of treatments that save abscessed teeth from extraction. The research data that you will help to provide may eventually lead to reduction in the number of teeth lost due to dental decay and infection.

This research study is designed to evaluate the effectiveness of sealing the root canal with dentin filings from the patient's own tooth. It is well established that tissues for medical treatments which are taken from a person's own body are far less likely to create toxic reactions than are foreign tissues or synthetic materials. Further, complete healing in the tissues surrounding the tooth has been seen more frequently when dentin filings were accidentally placed in the apical area of the root canal.

The study will require these commitments from you:

1. Completion of a general medical questionnaire routinely used at the University of Oregon Health Sciences Center School of Dentistry.
2. Participation during one or more appointments while endodontic therapy is being performed on selected teeth in your mouth which have been determined to be damaged beyond repair.
3. Two separate three-day courses of oral tetracycline administration at intervals seven weeks apart.
4. Extraction of the project teeth at the end of the study period.

The risks to your general and oral health are considered minimal. The discomfort you will experience will be the normal bleeding and mild pain which would normally occur when your teeth are extracted. Participation in this project will be of no direct benefit to you. However, you will be helping in the generation of important information.

If you have any further questions pertaining to this project or your participation therein, please contact: Dr. John B. Pappin, Principal Investigator.

I, _____, agree to participate in this project entitled "Biologic Sealing of the Apex in Endodontically Treated Teeth", under the direction of Dr. John B. Pappin. I understand that by this agreement I will permit endodontic therapy to be performed on teeth in my mouth which have been diagnosed as damaged beyond repair. I also understand that I will be given usual clinical dosages of the oral antibiotic, tetracycline, at two intervals seven weeks apart, and that the treated teeth will be extracted six weeks following the initial treatment. I have received a copy of the project narrative for my information and have been given a verbal description of my role in the project.

I understand I am free not to participate or to withdraw from participation at any time and it will in no way affect my relationship with the University of Oregon Health Sciences Center.

I have read the foregoing statements.

Witness my signature this

_____day of _____, 19__.

_____

Signature of Participant

Witness:

_____