THE PERMEABILITY OF HUMAN RADICULAR DENTIN AND CEMENTUM

by:

HOWARD M. FOGEL, D.M.D.

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The Permeability of Human Radicular Dentin and Cementum

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Approved: [redacted]

F. James Marshall, Chairman
Department of Endodontology

Approved: [redacted]

R. Quinton-Cox, Chairman
Advanced Education Committee

DEDICATED TO

MY MOTHER
for guiding her children towards intellectual pursuits

MY WIFE
for making everything worthwhile

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CHAPTER 1

INTRODUCTION

1.1 GENERAL

The permeability of dentin has been a subject of interest for some time. Most of the previous research in this area has been on coronal dentin (Anderson et al, 1967; Linden and Brannstrom, 1967; Brannstrom et al, 1967; Reeder et al, 1978; Pashley et al, 1978a; Pashley et al, 1977; Pashley and Livingston, 1978; Merchant et al, 1977; Brown and Wheatcroft, 1966; Brown et al, 1962). Quantitative studies on coronal dentin have been performed by numerous investigators (Reeder et al, 1978; Pashley et al, 1978a; Pashley et al, 1977; Pashley and Livingston, 1978, Merchant et al, 1977). The previous investigations of root dentin permeability have been qualitative in nature (Wantulok and Brown, 1972; Tronstad et al, 1981; Wemes et al; Sorenson and Gatewood, 1966; Martin et al, 1986; Hume and Kenny, 1981). Some authors have studied the qualitative effects of various endodontic medicaments on the permeability of radicular dentin (Anvy et al, 1973;

Marshall et al, 1960; Cohen et al, 1970b; Hampson and Atkinson, 1964). There have been no studies attempting to quantitate the permeability of radicular dentin. This study was therefore undertaken to quantitatively evaluate the permeability of radicular dentin.

Many factors govern fluid movement through dentin. Among the most important factors are the number and diameter of the dentinal tubules and the dentin thickness. No previous studies have attempted to correlate radicular dentin structure with quantitative data on permeability.

A smear layer is formed whenever dentin is cut, scraped, or ground. In endodontics, when canals are enlarged, and in periodontics when roots are planed (scaled), a smear layer is produced. The smear layer masks the underlying dentin matrix and blocks the tubule orifices. The effect of the smear layers produced by endodontic and periodontal instrumentation on the permeability of radicular dentin has never been studied.

It is thought that root structure may act as a "depot" of toxic substances. That is, if the orifices of the dentinal tubules are not sealed, toxic substances from a necrotic pulp may accumulate in the dentinal fluid and later diffuse from the dentinal tubules into the pulp space or in to the periodontium. This may produce a delayed effect after endodontic or periodontal treatment is

complete. Potassium oxalate has been used as a sealing layer to reduce the diffusion of substances across coronal dentin and to reduce the pain from exposed vital dentinal tubules. The effects of a potassium oxalate sealing layer on the pulpal or periodontal surfaces of radicular dentin have not been reported.

The hydrodynamic theory of dentin sensitivity holds that fluid movement in the dentinal tubules results in the mechanical stimulation of sensory nerves in the inner dentin or peripheral pulp to cause pain. The proposed mechanism of action of certain dentin desensitizing agents is the obstruction of the open dentinal tubule orifices in the sensitive area. Pashley (Pashley et al, 1978b; Pashley et al, 1984a; Pashley at al, 1979) has shown that potassium oxalate greatly reduces coronal dentin permeability by forming crystals that block the tubule orifices. Since root dentin is the most common site of dentin hypersensitivity the effects of potassium oxalate must be tested on radicular dentin.

1.2 SPECIFIC AIMS

The purposes of this study were: (1) to measure the effect of distance from the pulp on the permeability (hydraulic conductance) of human radicular dentin; (2) to

determine the effects of intact cementum on root dentin permeability; (3) to determine the influence of dentin thickness on the rates of fluid flow; (4) to correlate dentinal tubule numbers and diameters to root dentin permeability; and (5) to measure the effects of various surface manipulations including scaling, filing, acid etching, and potassium oxalate treatment on the permeability of human radicular dentin.

CHAPTER 2

A REVIEW OF THE LITERATURE

2.1 CORONAL DENTIN PERMEABILITY

Dentin consists of a relatively impermeable mineralized matrix penetrated by numerous cylindrical fluid (Pashley et al, 1978b) and tissue filled tubules. Dentin may be thought of as a semipermeable membrane (Anderson et al, 1967; Linden and Brannstrom, 1967; Anderson and Matthews, 1967). That is, solvent particles may pass through, while solute particles may be reflected. Furthermore, dentin is a diffusion barrier, allowing solute particles to move through it at rates dependent upon many variables.

Several investigators have demonstrated the movement of fluid through coronal dentin (Michelich et al, 1978; Brannstrom et al, 1967, 1968, 1969; Anderson et al, 1958; Brannstrom, 1962, 1966, 1961; Anderson and Matthews, 1967; Brannstrom and Astrom, 1964; Anderson and Ronning, 1962; Horiuchi and Matthews, 1973). Brannstrom's group applied various stimuli to exposed dentin and found that they

caused fluid movement and pain. These stimuli include drilling (Brannstrom et al, 1968), impression taking (Brannstrom et al, 1968), hydrostatic pressure (Brannstrom et al, 1969), a stream of air (Brannstrom et al, 1968, 1967, Brannstrom, 1966), heat (Brannstrom et al, 1968; Brannstrom, 1966), cold (Brannstrom et al, 1967), positive (Brannstrom, 1961), and negative (Brannstrom, 1966) pressure, osmotic pressures (Linden and Brannstrom, 1967; Brannstrom, 1962), and absorbant paper (Brannstrom, 1966; Brannstrom and Astrom, 1964).

Anderson and co-workers (Anderson et al, 1958; Anderson and Matthews, 1967; Anderson and Ronning, 1962) applied various hyperosmotic solutions such as calcium chloride or sucrose, and other substances to exposed dentin. This caused pain *in vivo*. These authors then showed that these solutions produced fluid movement through dentin *in vitro* (Anderson et al, 1967). These studies were qualitative rather than quantitative.

Filtration (fluid movement) is governed by laws expressed by the Poiseuille-Hagen equation (Pashley, 1985):

$$V = \frac{\pi \Delta P r^4}{8 x \eta} \qquad \text{(Formula 1)}$$

where:

V= volume flow, $\mu L\ min^{-1}$

ΔP= hydrostatic pressure difference across dentin

η= viscosity

x= length of tubules

r= radius of tubules

Note that filtration varies with the fourth power of the radius and that the driving force is a pressure gradient. Flow rate varies inversely with the length of the tube and the viscosity of the fluid.

Dentin permeability can be quantified by measuring fluid filtration rate or hydraulic conductance (Lp) (Pashley, 1985):

$$Lp = \frac{Jv}{A(\Delta P)} \qquad \text{(Formula 2)}$$

where:

Jv= fluid flow rate, $\mu L\ min^{-1}$

A= surface area, cm^2

ΔP= hydrostatic pressure gradient across dentin in cm H_2O

The rate of diffusion of a substance across dentin can

be quantified by the Fick equation (Pashley, 1985):

$$J = DA \frac{dc}{dx} \qquad \text{(Formula 3)}$$

where:

J= solute flux

D= diffusion coefficient

A= diffusional surface area

$\frac{dc}{dx}$= change in concentration over a distance, x

Diffusion obeys laws entirely different than does filtration. In diffusion the driving force is a concentration gradient. Note that diffusion is proportional to the diffusional surface area (r^2), whereas filtration varies with the fourth power of the radius. Therefore, changes in tubule diameter would effect filtration more than diffusion.

From these formulae it can be seen that the calculation of hydraulic conductance and diffusion coefficients requires precise knowledge of both surface area and thickness. The earlier studies cited above used cavity preparations in intact teeth to study dentin permeability. This method had several shortcomings. Total dentin surface area was impossible to quantitate and keep

constant because of the variability of enamel thickness and uneven contour of the dentin-enamel junction. Furthermore, the contribution of the tubules in the dentin walls relative to the cavity floor was unknown. Variations in pulp horn and pulp chamber morphology also make dentin thickness highly variable even when the cavity's pulpal floor is made flat. The complexity of the diffusion geometry of even simple occlusal cavities prompted Outhwaite et al (1976) to abandon their use. These authors used dentin disks of known dimensions and placed these in a split chamber device in which dentine surface area and hydrostatic pressure were precisely controlled. They were able to quantitate the effects of changes in surface area, thickness, temperature, and post-extraction time on human coronal dentin permeability *in vitro*. No quantitative studies of radicular dentin have been reported.

As the surface area of exposed coronal dentin increases, so does the filtration rate of fluid passing through it (Outhwaite et al, 1976). This relationship is linear at a constant dentin thickness reflecting constant tubular density (number of tubules/mm^2) and constant tubule dimensions at a constant distance from the pulp. Increasing the available surface area increases diffusion as well (Outhwaite et al, 1976).

The ease with which fluid filters through dentin is

very sensitive to the length of the tubules. As remaining dentin thickness decreases, filtration increases exponentially (Reeder et al, 1978). This is due, in part, to the reduction in the frictional resistance of the shorter tubule walls.

Diffusion is also inversely proportional to dentin thickness (Pashley et al, 1977; Outhwaite et al, 1976). The closer medicaments are placed to the pulp, the greater will be the the diffusion of molecules across the remaining dentin. During diffusion the concentration of substances is dissipated over distance. The concentration of bacterial products on the surface of thick overlying coronal dentin is much greater than that at the pulpal surface (Pashley, 1984).

A ten-fold increase in dentin temperature can almost double its permeability to radioactive iodide (Outhwaite et al, 1976). Post-extraction time had little effect on dentin permeability (Outhwaite et al, 1976).

The resistance to fluid movement through dentin in teeth with vital pulps can be thought of as the summation of a series of three resistances (Pashley et al, 1978a): (1) surface resistance due to the presence of debris partially or totally occluding the peripheral ends of the tubules; (2) intratubular resistance due to mineralized fibrils, nodules, and internal irregularities within the

tubules; and (3) pulpal resistance due to the presence of odontoblastic processes and cell bodies. The odontoblasts may be displaced into or away from the tubules. The adherence of adjacent odontoblasts to one another by junctional complexes may have an additional effect (Pashley and Livingston, 1978).

All of these variables modify the diameter of the spaces within the tubules through which fluid may move. Since the rate of fluid filtration is proportional to the fourth power of the tubule radius, these factors are highly significant (Merchant et al, 1977). Small changes in tubule diameter by blockage or reduction in actual size produce large changes in the rate of fluid flow.

The Poiseuille-Hagen Equation (Formula 1) formalizing the determinants of fluid flow refers to the effective or functional radius of the tubule. This denotes the area within the tubule available for fluid movement, that is, the total tubule diameter minus any intratubular obstructions. Michelich et al (1978) compared the functional versus anatomic tubular radii in coronal dentin. These authors found that, at a constant distance from the pulp, the functional radii varied from 5-40% of the anatomic radii. Expressed another way, this means that the dentinal tubules were 60-95% narrower than they appeared microscopically when partial tubule blockages out of view

were accounted for. They concluded that tubular radii measured microscopically on open tubules only provide a maximum boundary for the functional radii.

The presence or absence of pulp tissue does not significantly affect the permeability of coronal dentin to radioactive isotopes in dogs (Pashley et al, 1981) or in humans (Pashley et al, 1977). This is further supported by studies comparing in vivo versus in vitro dog dentin permeability (Pashley et al, 1981) made on the same teeth. It was observed that rates of isotope permeation were very similar both in vivo and in vitro even after the pulp tissue had been removed by pulpectomy. When the effect of post-extraction time on the permeability of human coronal dentin was studied, (Outhwaite et al, 1976) no change was detected over a 3-4 week period. During this time the odontoblastic processes would have undergone autolysis. Therefore, the presence or absence of odontoblastic processes seems to have little effect on dentin permeability.

The number and diameter of the dentinal tubules have an obvious direct effect on permeability. Tubules are farther apart (less dense) in the peripheral dentin and more dense near the pulp. (Bhaskar, 1976) They are wider near the pulp (3-4 microns) and narrower (1 micron) at the periphery (Garberoglio and Brannstrom, 1976). The ratio of

the number of tubules per unit area at the pulpal and outer surfaces of dentin is about 4:1 in coronal dentin. Thus, superficial dentin is less permeable than deep dentin. This has been confirmed experimentally only in coronal dentin (Outhwaite et al, 1976). Since tubule density decreases corono-apically (Carrigan et al, 1984), radicular dentin should be less permeable than coronal dentin. Apical dentin should be less permeable than cervical root dentin.

In sclerotic dentin, where mineralized deposits obstruct the tubules, permeability could presumably be less. The same may be true of dead tracts, where the tubules are empty, since they are usually sealed at the pulpal interface by reparative dentin. In advance of carious lesions, the tubules may become partially blocked by reprecipitated dentinal minerals known as "caries crystals". Trowbridge (1981) has speculated that caries crystals may reduce dentin permeability.

A substance's ability to diffuse across dentin varies inversely with its molecular size (Pashley et al, 1977; Pashley and Livingston, 1978). Dentin does not discriminate well between isotopes of similar molecular size. Small molecules (less than 3 angstroms diameter) like urea are similar in permeability to water. Molecular weight has a similar effect on diffusion rates (Pashley and

Livingston, 1978; Merchant et al, 1977). Small molecules may pass easily through dentin to the pulp and the systemic circulation. Very large molecules which do not easily permeate can cause water movement (osmosis) out of the dentinal tubules to an extent dependent on the concentration gradient. Thus, the effective osmotic pressure depends on molecular size and weight as well as concentration (Pashley and Livingston, 1978; Pashley et al, 1979; Pashley and Whitford, 1980).

A very critical variable affecting both fluid flow and diffusion is the nature of the dentin surface, that is, whether the surface is coated with a smear layer or not (Reeder et al, 1978). Johnson et al (1973) demonstrated by scanning electron microscopy that the dentinal tubules were open on a fractured dentin surface while cut dentin showed tubules blocked by debris.

Whenever dentin is cut by hand or rotary instruments, the mineralized matrix shatters producing cutting debris analagous to "wet sawdust" (Pashley, 1984). This debris is made up of very small particles of dentin spread over the cut surface and it greatly increases the resistance to fluid movement through dentin. Etching a cut dentin surface with acid removes the smear layer and opens and enlarges the apertures of the tubules. This removes much of the resistance to fluid flow across dentin (Reeder et

al, 1978; Pashley et al, 1978a; Merchant et al, 1977; Pashley et al, 1983b). Clinically this results in increased sensitivity to osmotic, thermal and tactile stimuli (Johnson and Brannstrom, 1974). Removal of the smear layer also removes its effect of steric restriction especially to larger molecules, and greatly increases permeability (Pashley and Livingston, 1978; Merchant et al, 1977; Pashley et al, 1978b, 1979, 1981a, 1983a).

Clinically, hot and cold stimuli applied to teeth produce pain. Temperature has an effect on fluid movement within dentin via several mechanisms (Pashley et al, 1983b). Temperature changes cause larger changes in the volume of dentinal fluid than in the volume of the dentinal tubules themselves due to disparate coefficients of thermal expansion. This leads to pressure changes and the movement of fluid in the tubules to balance the pressure (Van Hassel and Brown, 1969). Because the viscosity of dentinal fluid varies inversely with temperature, tubule fluid flows more easily as it is heated. Flow also increases when the temperature of dentin is raised because it expands and the tubule diameters increase slightly. As noted earlier, a small change in tubule diameter produces large changes in fluid flow. If a smear layer is present it provides a noteable resistance to fluid movement that is not thermally sensitive due to the low coefficient of thermal expansion

of the debris (Pashley et al, 1983b).

When dentin is dessicated there is decreased fluid movement compared to rehydrated dentin (Polhagen and Brannstrom, 1971). Evaporation of water from the tubules may lead to increased intratubular concentrations of solutes. The formation of insoluble salts and organic substances such as albumin (Pashley et al, 1984b) partially blocks the tubules, decreases the functional radii, and thereby decreases fluid flow.

The contact of saliva or plasma with dentin greatly reduces its hydraulic conductance (Pashley et al, 1982; Pashley, 1984). Saliva contains glycoproteins which adsorb to the peritubular dentin and reduce fluid flow. Likewise, plasma proteins are capable of the same effect. Plasma also contains platelets which, following pulpal trauma, may reach open dentinal tubules, clog them, and reduce fluid flow. Whole saliva contains bacteria which can also enter the tubules of exposed dentin (Olgart et al, 1974; Brannstrom and Nyborg, 1973; Michelich et al, 1980) and reduce fluid flow. Clinical experience has shown that due to unknown causes, hypersensitivity in exposed dentin sometimes disappears spontaneously. The processes discussed above may be responsible, in part, for this phenomenon (Pashley et al, 1982).

The mechanism for dentin sensitivity is not fully

understood; nor is the mechanism of the effect of desensitizing agents. Pashley and co-workers (Pashley et al, 1983b, 1984a) studied several desensitizing agents in use clinically to evaluate their ability to reduce the rate of fluid flow through dentin in vitro. Flouride, barium sulfate, silver nitrate and potassium oxalate were effective. These substances, especially oxalate, form crystals that block the dentinal tubules. These authors suggest that tubule blocking is one of the mechanisms of action of some desensitizing agents. Further support for this suggestion comes from Pashley et al (1987) who tested the effects of burnishing dentin with various desensitizing pastes on dentin permeability. They found that dentin permeability was decreased 50-80% by burnishing alone and that the paste constituents were insignificant. This may lend some support to the hydrodynamic theory of the mechanism of dentin sensitivity. These studies, however, where performed on disks of coronal dentin. These data using coronal dentin may not pertain to root dentin where most cases of dentin hypersensitivity actually occur.

In addition to desensitizing agents, other structures may occupy and partially obstruct dentinal tubules. Several factors may induce the displacement of odontoblast organelles (Brannstrom, 1961, 1966; Johnson et al, 1973) due to outward fluid movement in the tubules. In

pulpal inflammation polymorphonuclear leukocytes and red blood cells have been observed within the dentinal tubules. Red blood cells are easily deformed by pressures within the physiologic range. Their presence in dentinal tubules results in a large reduction in dentin hydraulic conductance (Pashley et al, 1981b).

On average, normal intrapulpal pressure is about 25 mm Hg (Van Hassel, 1971). Outward fluid movement under physiologic pulpal pressure acting on open dentinal tubules is capable of causing a reduction in the odontoblast layer due to aspiration of the cells into the tubules (Johnson et al, 1973). The subsequent autolysis of these cells and pulpal contamination by cell breakdown products diffusing back out of the tubules may cause inflammation.

These products must contact the pulp in sufficient concentrations if tissue damage is to occur. The concentration of substances diffusing through dentin depends both on the rate of permeation and on the rate of removal by the microcirculation of the pulp (Pashley, 1979). Pulps with existing inflammation or compromised blood flow due to occlusal trauma, excessive orthodontic forces, restorative manipulations, or vasoconstrictors, may be more susceptible to damage by toxic agents diffusing through dentin. Van Hassel (1971) reported regional differences in intrapulpal pressure such that inflamed

areas had increased pressure while non-inflamed areas remained normal. Thus, total pulpal blood flow need not fall in order to allow the local accumulation of inflammatory mediators. Path and Meyer (1980) have reported that dog pulpal blood flow varies with location in the pulp. The flow is higher in the crown than the root. Therefore radicular pulp may be more susceptible to inflammation caused by substances diffusing across dentin and there are fewer odontoblast replacement cells.

Dentin's permeability to bacteria and bacterial products is of particular interest. Several investigators have shown that bacteria may penetrate dentinal tubules (Chirnside, 1961; Shovelton, 1964; Michelich et al, 1980; Vojinovic et al, 1973; Olgart et al, 1974; Akpata and Blechman, 1982). Olgart et al (1974) reported that the outward flow of dentinal fluid due to physiologic intrapulpal pressure may hinder the ingress of bacteria. However, the presence of a smear layer was found to be a more important obstruction to bacterial penetration (Michelich et al, 1980; Olgart et al, 1974). Removal of the smear layer by acid etching (Vojinovic et al, 1973; Olgart et al, 1974) or allowing it to be washed away by saliva (Michelich et al, 1980) resulted in the ingrowth of bacteria. However, dentin with open tubule apertures is still a significant barrier to bacteria and only a fraction

of organisms on the dentin surface actually penetrate all the way to the pulp (Michelich et al, 1980). Dentin acts as a "Millipore filter" and 99.8% of the bacteria are filtered out by the irregular constrictions in the tubules.

Since diffusion and filtration obey different laws (formulae 1 and 3), substances may diffuse pulpally against an outward hydrostatic pressure gradient (Pashley, 1979). The pulp reacts to caries long before bacteria penetrate the enamel and dentin into the pulp chamber (Vojinovic et al, 1973, Bergenholtz, 1977). It is bacterial products diffusing through the enamel and dentin which initiate the pulpal inflammation.

It should be noted that while the presence of a smear layer prevents bacterial penetration, it does permit fluid filtration and the penetration of bacterial products at reduced rates (Pashley, 1984). Bergenholtz and co-workers (Bergenholtz, 1977, 1981; Bergenholtz and Lindhe, 1975) placed bacterial products in contact with cut dentin surfaces and found pulpal inflammation in response. Bergenholtz et al (1977) demonstrated a pulpal immune response to bovine serum albumin placed on cut dentin. Treatment of exposed dentin with calcium hydroxide (Pashley et al, 1986) or potassium oxalate (Pashley et al, 1984a) may reduce the permeability of dentin to bacterial products and prevent the associated pulpal inflammation. Cavity

varnishes and certain bases are also effective at reducing dentin permeability (Pashley et al, 1985). Studies of the effects of these substances have been restricted to coronal dentin.

2.2 SMEAR LAYER

Throughout the previous discussion of dentin permeability, smear layer has been referred to several times. It is worthwhile to look at this subject more closely since it occupies such a unique place in clinical dental practice. A smear layer is formed whenever dentin is cut by a bur or chisel or ground with a stone. In endodontics, when canals are enlarged, and in periodontics when roots are planed, a smear layer is likewise produced. The smear layer masks the underlying dentin matrix and decreases permeability by blocking the tubule openings (Dippel et al, 1984). Dental materials that are capable of flowing into open tubules (eg. resins, root canal sealers, cements) are prevented from doing so. The smear layer also interferes with materials capable of adhering to the dentin matrix (eg. glass ionomers, polycarboxylate cements). This results in decreased retention and increased marginal leakage (Hoppenbrouwers et al, 1974; Goldman et al, 1984; Causton and Johnson, 1982, Jodaikin and Austin, 1981;

Goldman et al, 1984)

The smear layer may be beneficial in that it provides an obstruction to bacterial penetration of dentin and decreases dentin's permeabiltiy. However, Akpata and Blechman (1982) found that a smear layer created by endodontic instrumentation was permeable to streptococci. In addition, the smear layer may slowly dissolve if exposed to subsequent leakage or plaque acids. This would leave a void between the dentin and the adjacent dental material which saliva could fill. Bacterial colonization of the now unprotected dentin surface might then occur (Vojinovic et al, 1973). The smear layer may also harbour bacteria or bacterial products and so provide a resevoir of potential irritants beneath a restoration or in a treated root canal (Pashley, 1984). For these reasons many investigators have looked at ways of removing the smear layer (Goldman et al, 1984; Causton and Johnson, 1982; Jodaikin and Austin, 1981; Goldman et al, 1982; Goldman et al, 1981; Brannstrom et al, 1980; Bowen, 1978). When the smear layer is removed its sealing benefits are then lost. Others have tried to fix the smear layer to improve its bonding to the underlying dentin (Hoppenbrouwers et al, 1974; Powis et al, 1982). Still others have tried to remove the smear layer and replace it with an uncontaminated chemical sealing layer (Hoppenbrouwers et

al, 1974; Bowen and Cobb, 1983; Greenhill and Pashley, 1981). Such a sealing layer may be less permeable than the original smear layer. Pashley et al (1978b, 1979) and Merchant et al (1977) have shown that potassium oxalate greatly reduces coronal dentin permeability by forming crystals that block the tubule orifices. This is the proposed mechanism of action of oxalate as a desensitizing agent in dentin hypersensitivity, although it has only been tested on coronal dentin. It may not be necessary to remove the smear layer in order to effectively reduce dentin permeability with potassium oxalate (Pashley and Galloway, 1985), however it may be desirable if the smear layer is contaminated with bacteria and bacterial products.

The effects of the smear layers produced by endodontic and periodontal instrumentation on the permeability of radicular dentin has never been studied. Likewise, the effects of a potassium oxalate sealing layer on the pulpal or periodontal surfaces of radicular dentin have yet to be reported upon.

2.3 RADICULAR DENTIN PERMEABILITY

The general principles that govern the movement of fluids though minute tubules apply equally well in radicular dentin as in coronal dentin. Likewise, so do the

laws governing the diffusion of molecules across a semipermeable membrane. However, there are certain differences in the structure of radicular dentin which affect its permeability. These effects have yet to be quantified.

Several authors have examined, in a qualitative fashion, the permeability of root dentin to endodontic medicaments and materials. Radicular dentin is permeable to penicillin (Curson and Jackson, 1960; Bennett and Miles, 1955; Wach et al, 1955) and to formalin (Sorenson and Gatewood, 1966). Marshall and Massler (1961) used various radioactive tracers to evaluate the sealing of root canals and found that isotopes diffused outwards from the canal into the dentinal tubules and concentrated at the cementodentinal junction. Marshall et al (1960) found that cervical and midroot dentin were very permeable to all isotopes tested. However, the apical dentin was highly impermeable. Radicular dentin is also permeable to methylene blue dye (Antoniazzi et al, 1968) from the cemental surface inwards. Martin et al (1968) found that apical root dentin was permeable to penicillin, formocresol and CMCP (camphorated monochlorophenol) from the pulp outwards. More recently, Taylor et al (1976) compared aqueous PCP (parachlorophenol) with CPCP (camphorated parachlorophenol). They confirmed the findings of Anvy et

al (1973) that the aqueous PCP penetrated the entire thickness of the root dentin from the pulp outwards. The CPCP penetrated less than 1/5 the tubule length. In a study of the effects of calcium hydroxide root fillings on the pH of dental tissues Tronstad et al (1981) found that hydroxyl ions diffused outwards from the canal space into the radicular dentin. Formocresol is also capable of penetrating root dentin (Wemes et al, 1982). In monkeys, replanted teeth root filled with gutta-percha and Roth's sealer showed reduced periodontal healing and enhanced root resorption (Andreasen, 1981). These findings suggest that root dentin is permeable to some component of these materials. Haapasalo and Orstavik (1987) found that bacteria (E. fecalis) could penetrate the tubules of root dentin from the canal outwards. These authors tested the disinfection ability of CMCP and a calcium hydroxide compound (Calasept) as intracanal medicaments. Liquid CMCP rapidly and completely disinfected the tubules whereas gaseous CMCP and Calasept did not.

The effect of endodontic medicaments on the permeability of the dentin with which they come into contact has also been investigated. Marshall et al (1960) used radioactive isotopes to test the effect of various treatments on radicular dentin permeability. Sulfuric acid decreased dentin permeability markedly. Hydrogen peroxide

and sodium hypochlorite solutions used alternately increased permeability significantly. The CDJ acted as a barrier to the passage of isotopes from inside the canal outward and from outside the canal inward. Mechanical instrumentation had no effect on permeability. Silver nitrate, 5% chloramine, EDTA, Cettimide, and chlorhexidene increased permeability while tricresol decreased it. Hampson and Atkinson (1964) found that apical dentin was impermeable while middle and cervical third root dentin permeability was variable to radioisotopes. Cohen and co-workers (1970a) tested the effects of various drugs and irrigating solutions on human radicular dentin. These authors found that a combination of R-C Prep (an EDTA-containing paste) and sodium hypochlorite increased apical third permeability. So did urea peroxide and sodium hypochlorite. Sulfuric acid decreased dentin permeability to methylene blue dye. Conflicting conclusions were reached by Fraser and Laws (1976). They found that EDTA, R-C Prep, Decal and Largal Ultra (chelating agents) all decreased dentin permeability to phloxine dye. In this study, however, the chelating agents were not used in conjuction with canal enlargement procedures. Normal canal instrumentation creates a smear layer which may decrease permeability. Chelating agents may remove this smear layer to increase permeability over instrumentation alone.

There are several indications that root dentin is permeable to some bacteria and bacterial products (Rowe and Binnie, 1977). Shovelton (1964) stated that bacteria were capable of invading root dentin to depths that varied considerably. Canal enlargement removed the infected dentin. These results were confirmed by Bence et al (1973) who found bacteria penetrated predentin and circumpulpal dentin in pulpless teeth. Bergenholtz (1974) studied micro-organisms from the necrotic pulps of infected teeth. He found a correlation between the presence of bacteria in the canal and external root resorption. This implies that bacterial products are capable of diffusing outwards through root structures to initiate an inflammatory reaction in the periodontium. The outward diffusion of bacterial toxins are also capable of causing pathologic changes in the overlying cementum (Armitage et al, 1983).

Sorensen and Gatewood (1966) found radicular dentin from human anterior teeth permeable to radioactive formalin used for tissue fixation. Johnson et al (1985) found no pulpal response to the topical application of citric acid to root-planed surfaces in cats. This suggests that cat radicular dentin is not very permeable to citric acid.

Linden (1968) qualitatively demonstrated fluid movement through radicular dentin. In a recent abstract Frydenlund and Krell (1986) reported the effects of root

planing with sharp and dull curettes on fluid flow across root dentin. Sharp curettes were found to cause an increase in filtration rates while dull scalers decreased fluid flow. In a second abstract Krell and Frydenlund (1986) reported that the smear layer produced by root planing was not removed by etching with 50% citric acid for 1 min. The complete data behind these research findings has not been published to date.

2.4 DENTIN STRUCTURE

When considering dentin structure in relation to its permeability, it may be thought of as a relatively impermeable mineralized matrix penetrated by numerous cylindrical fluid filled tubules (Pashley et al, 1978b). The dentinal tubules are, perhaps, the most characteristic feature of dentin. Dentin's permeability is a direct consequence of the presence of these tubules.

The number and diameter of the dentinal tubules have a critical influence on the rates at which fluid flows through dentin. Since filtration varies with the fourth power of the tubular radius (Formula 1) small changes in tubule diameter would have a large effect on fluid flow. Since dentinal tubules converge from the dentino-enamel junction or cemento-dentinal junction towards the pulp,

they are less dense in peripheral dentin and more dense near the pulp (Bhaskar, 1976; Garberoglio and Brannstrom, 1976). Garberoglio and Brannstrom (1976) studied fractured surfaces of human coronal dentin under the SEM. Near the pulp the tubule density was 45,000 tubules/mm^2, with a mean diameter of 2.5 microns. In the middle of the dentin, there were 29,500 tubules/mm^2, and the diameter was 1.2 microns. Peripherally there were 20,000 tubules/mm^2 and these had an average diameter of 0.9 microns. These authors found no great difference between old and young intact teeth. These values were similar to those found in other species (Forssell-Ahlberg et al, 1975). One can calculate total tubular surface areas from Garberoglio and Brannstrom's data to be about 1% at the DEJ, 10% halfway through dentin, to about as high as 45% at the pulp chamber (Pashley, 1983).

The figures of Garberoglio and Brannstrom (1976) on the tubule diameters of undecalcified dentin differed from other reports such as that of Bradford (1955) on decalcified dentin. There are indications that the peritubular dentin is dissolved to a large extent during decalcification in acid (Isokawa et al, 1970). In Bradford's study, the tubule diameters were measured on decalcified sections. This gives an excessively high value (Bradford, 1955) and makes reports based on such samples

not valid (Garberoglio and Brannstrom, 1976).

Whittaker and Kneale (1979) studied the dentin-predentin junction under SEM in both coronal and radicular dentin. Their findings of tubule density agreed closely with those of Garberoglio and Brannstrom (1976) in coronal dentin. In root dentin they found that tubule numbers decreased in a corono-apical direction until there were only approximately 8,000 tubules/mm^2 in the apical regions.

Carrigan et al (1984) examined the dentin of 30 extracted human maxillary central incisors under SEM. They used undecalcified samples and looked at the pulpal dentin surface in the crown and in three areas of the root: cervical, middle, and apical. The mean tubule densities found in this study were 44,243 tubules/mm^2 in coronal dentin, 42,360 tubules/mm^2 in crevical root dentin, 39,010 tubules/mm^2 in mid-root dentin, and 8,190 tubules/mm^2 in apical root dentin. These results agree with those of Garberoglio and Brannstrom (1976) on coronal dentin. However, Carrigan et al did find an age related difference. They concluded that tubule numbers decreased significantly with age so that, for example, there were no tubules in the apical root dentin of the 80 years and above age group. A possible explanation for the difference between these two studies as to the significance of age is offered by the studies of Nalbandian et al (1960) and of Bang and Ramm.

(1970). Nalbandian et al found that dentinal sclerosis was an age related change that started at the root apex and progressed coronally. Bang and Ramm quantitated the extent of transparent (sclerotic) dentin in one thousand teeth in an attempt to create a data base for age determinations of unknown bodies for forensic dental work. The root dentin usually becomes transparent at the tip of the root and advances coronally with age. This alteration is believed to be caused by a reduction in the diameter of the dentinal tubules caused by increasing intratubular calcification. They found that sclerosis begins around the age of 30 years at the apices. From their data it can be seen that very few teeth would have sclerotic dentin in the crowns prior to age 60. Since Garberoglio and Brannstrom studied only coronal dentin, and since their oldest age group was 40-60 years, it is reasonable that they reported no age-related differences between old and young teeth. It is necessary to study radicular dentin (also see Nalbandian et al, 1960) and to have a large sample ranging in age from 20 to 80 years to detect age-related differences in dentinal sclerosis.

Whittaker and Kneale (1979) measured tubule diameters in the apical areas where age changes would be expected to be most marked. They found mean diameters ranged from 0.1-1.5 microns. No correlation between age

and diameter was apparent. The authors were surprised at these findings and speculated that some tubules may remain unaffected by the formation of transulcent dentin or that the tubules are not all obliterated at the pulpal ends.

Furseth (1974b) studied the structure of peripheral root dentin in young human premolars using light and electron microscopy. Although actual tubule numbers were not reported, both methods showed that cervical, midroot, and apical areas of peripheral root dentin had very few dentinal tubules. Many tubules also ended short of the cemento-dentinal junction. The distance from the CDJ was not specified. This disagreed with Ten Cate's (1972) suggestion that Tomes' granular layer is caused by a random looped configuration of the terminations of the dentinal tubules. Furseth found unmineralized areas in the peripheral dentin which had the shape of small interglobular areas.

The extent of the odontoblast process is a question that is both interesting and controversial. It is disputed whether or not one can identify the odontoblast process by light or SEM techniques. Brannstrom and Garberoglio (1972) in an SEM study, reported that the processes were limited to the inner third of the dentin. Similar observations were made by Thomas and Payne (1983), by Thomas and Carella (1984), by White et al (1986), and by Webber and Zaki

(1986). These findings have been challenged by Maniatopoulous and Smith (1983) and by Sigel et al (1985) who report that the processes go all the way to the DEJ.

LaFleche et al (1985) offer a compromise position which states that the processes reach to the DEJ in vivo but retract to the inner third upon extraction. Only Weber, Thomas and LaFleche have examined this problem using TEM. Thomas and Weber both agree that a tubular structure in a dentinal tubule may not be called an odontoblast process unless one can identify its trilamminar plasma membrane by TEM.

From a functional point of view, one should ask if the presence of odontoblast processes modify fluid movement (or pain transmission) across dentin. According to the widely accepted hydrodynamic theory, fluid movement in the dentinal tubules results in mechanical distortion of nociceptive nerve endings in the pulp and innermost dentin. Brannstrom and Astrom (1964) left cavities unrestored in a group of patients. In a short time, the odontoblasts were destroyed and there was pulpal inflammation yet the dentin remained sensitive. In a more recent study by Hirvonen and Narhi (1986), the intradental nerves of dogs were isolated and the activity recorded in response to air blasts. After obtaining baseline responses, they subjected the teeth to prolonged (2-5 hours) air blasts which disrupted the

odontoblasts and displaced their nuclei into the tubules. There was no observable change in the sensitivity of these teeth. They concluded that pulpal nerve function and morphological changes of the pulp are not clearly correlated. They had thought that displacement of odontoblastic nuclei into the tubules would have modified the resistance to fluid movement which might then modify nerve activity, but they found no such change.

In unpublished studies, Pashley (1987) measured hydraulic conductances in dog dentin in vivo before and after attempts to osmotically alter odontoblast dimensions within the tubules by filtering pure water or 3M NaCl across dentin. There were no significant changes in Lp (hydraulic conductance). They also exposed other teeth to a prolonged air blast similar to Hirvonen and Narhi, but could not detect any difference in Lp even though histologically, most of the tubules exhibited displaced odontoblast nuclei.

As cited earlier (Outhwaite et al, 1976) autolysis of the odontoblast processes after tooth extraction seems to have no effect on dentin permeability.

From these types of observations, it may be concluded that odontoblast processes do not contribute significantly to the resistance to fluid movement across dentin and are not necessary for pain transmission or

perception.

2.5 CEMENTUM PERMEABILITY

Several authors have qualitatively studied the permeability of cementum. Conflicting results have been reported concerning the permeability of cementum to penicillin (Martin et al, 1968; Curson and Jackson, 1960; Bennett and Miles, 1955; Wach et al, 1955). Stewart-Ross (1933) and Antoniazzi et al (1968) reported that cementum was permeable to methylene blue. Wainwright (1953) found that cementum was permeable to some isotopes. Sorenson and Gatewood (1966) noted that radioactive formalin penetrated cementum. Formocresol was found to penetrate cementum (Wemes et al, 1982) while glutaraldehyde did not.

Marshall et al (1960) studied the effects of various endodontic procedures and drugs on radioisotope penetration of raducular dentin. Isotopes (iodine, sodium, sulphur and phosphorus) failed to pass beyond the cemento-dentinal junction from inside the canal outwards and from outside the canal inwards. This was confirmed by Marshall and Massler (1961). These observations suggest that the cemento-dentinal junction is not permeable to the isotopes tested. This contradicted earlier studies by Wainright (1953) and by Wasserman et al (1941) which had indicated

that cementum was permeable to radioactive iodine and radioactive phosphorus.

Tronstad et al (1981) studied pH changes in dental tissues after root canal filling with calcium hydroxide. They found that calcium hydroxide did not penetrate the cementum. Taylor et al (1976) stated that CPCP and aqueous PCP did not penetrate cementum from the canal outwards.

Linden (1968) demonstrated the flow of water completely through cementum and identified the pathways for fluid flow through cementum to be the cementocyte lacunae and canaliculi as well as Sharpey's fibres.

Selvig (1977) identified a hypermineralized layer on cemental surfaces exposed to the oral cavity. This may decrease the permeability of diseased cementum.

"Pathologic granules" were found in the cementum of teeth with heavily infected root canals (Armitage et al, 1983), suggesting that bacterial toxins diffuse outwards from the pulp space into the cementum.

Periodontally involved cementum was found to be toxic to cell cultures (Johnson et al, 1973), suggesting that it is penetrable by cytotoxic substances. On the other hand, Nakib et al (1982) found that endotoxin did not penetrate cementum.

Lantelme et al (1976) found narrowing of pulp canal

diameters associated with periodontally involved teeth. This implies the penetration of pulpal irritants inwards through cementum and radicular dentin. The same conclusion was reached by Bergenholtz and Lindhe (1978). In this investigation the effects of experimentally induced marginal periodontitis and periodontal scaling on the pulp were examined. Secondary dentin formation and/or a mild inflammatory reaction in the pulp were seen opposite root planed areas. In another paper Bergenholtz and Nyman (1984) speculated that due to root dentin permeability to bacterial products, periodontal disease is related to pulpal disease.

Frydenlund and Krell (1986) found that scaling with sharp instruments increased root permeability while permeability was decreased by scaling with dull instruments. They speculated that dull instruments may have a burnishing effect that partially occludes dentinal tubules (also see Pashley et al, 1987). Krell and Freydenlund (1986) found that the smear layer created by root planing was not removed by etching with citric acid.

No results have been published quantitating the permeability of human radicular dentin with attached cementum.

2.6 CEMENTUM STRUCTURE

The pathways for fluid flow through cementum have been identified as the cementocyte lacunae and canaliculi, and Sharpey's fibres (Linden, 1968). The cementum in the cervical and middle part of roots of young human premolars is acellular (Furseth, 1974a). It is 4-5 microns thick and consisted mainly of Sharpey's fibres (also see Selvig, 1965). In the cervical areas of some teeth there is no cementum. Mayhall (1984) looked at the relationship of the cementum to the enamel at the cementoenamel junction and found three different relationships. In 51% of teeth cementum overlapped enamel. In 31% there was an end-to-end relationship. Gaps existed between the cementum and enamel in 18% of teeth. Substances may pass more easily across the root structure in such areas.

There is a great deal of evidence that, in periodontal disease, cementum undergoes pathologic changes. Many authors have reported that cementum exposed to the oral cavity develops a hypermineralized layer (Selvig and Hals, 1977; Eide et al, 1983, 1984; Wirthlin et al, 1979). Such areas may be less permeable than normal cementum.

Carious human cementum also develops a hypermineralized surface layer under which is found a subsurface demineralization. This is similar to the

pattern found in carious enamel and dentin. The effects of the processes of demineralization and remineralization on cementum permeability are not known.

The effect of periodontal instrumentation on root surfaces is of interest in a discussion of root permeability. Van Volkinburg et al (1976) studied various forms of instrumentation and found that the cementum was almost always completely removed by use of a curette alone or together with a Cavitron or Alpha-Sonics instrument. In contrast, some cementum was usually left behind when the Cavitron or Alpha-Sonic intruments were used without curettage. Woodruff et al (1975) also found that the ultrasonic instruments tested did not expose the dentin. If cementum acts as a diffusion barrier, and seals the peripheral ends of the dentinal tubules, then root planing with curettes would be expected to increase root permeability.

2.7 THE EFFECT OF EXTERNAL ROOT RESORPTION

Andreasen (1985) speculated that the cementum acts as an insulating layer that protects the periodontal ligament against the penetration of bacterial products from the pulp space and dentinal tubules. External root resorption may cause an increase in root permeability by denuding

areas of cementum and superficial dentin and exposing the underlying dentinal tubules. This would be expected to allow more rapid diffusion of substances across the remaining dentin from the periodontal tissues to the pulp or vice versa. There are many causes of external root resorption.

2.7a Physiologic Resorption

The roots of the primary teeth gradually resorb as the permanent teeth erupt. The roots of primary teeth with no permanent successors resorb at various rates or not at all. This process of physiologic resorption is not well understood.

Some degree of cemental resorption appears to be normal in the permanent dentition as well. Henry and Weinmann (1951) found that 90.5% of the teeth in their study had varying numbers of resorptive areas. The average number of resorptive areas per tooth was 3.5. The average size of these areas was not reported. Resorption was more commmon in molar teeth. Older patients had more resorption than younger ones. The apical third of the root was the most common site and the authors related this to the presence of cellular cementum. Resorptive areas were more numerous on mesial root surfaces. The authors speculated that this was due to the pressure of physiologic tooth

movement (mesial drift) and that, in general, trauma was the most important etiologic factor.

2.7b Trauma

Trauma can lead to three forms of external root resorption: surface, inflammatory and replacement resorption. The incidence and type of resorption is related to the extent of the injury (Andreasen, 1970). Thus, resorption is less common and less severe in luxation injuries than in avulsion. The interval from the time of injury until the reduction of the displacement plays a role in the etiology of resorption. Surface resorption starts as early as 1 week following tooth replantation (Andreasen, 1980) and is generally repaired by cementum deposition.

Several factors influence the development of inflammatory resorption. All are related to the extent of the injury to the cementoblasts and periodontium. Transient apical breakdown is a mild, self-limiting type of inflammatory resorption seen after luxation injuries. Inflammatory resorption is seen 1 week after reimplantation and progresses rapidly unless endodontic therapy is instituted (Andreasen and Hjorting-Hansen, 1966) In replantation the pulp status and the degree of cell damage to the root surface play a role (Andreasen, 1981). If surface resorption exposes dentinal tubules in a tooth with

a necrotic pulp inflammatory resorption ensues. In a vital tooth, repair by cementum occurs (Andreasen, 1981). In fact, the presence of external inflammatory root resorption can be used to confirm the diagnosis of pulpal necrosis for intruded and reimplanted teeth (Jacobsen,1980). These observations suggest that radicular dentin and cementum are permeable to bacterial products. Root topography enters into the equation as resorption occurs more frequently on the convex buccal and lingual surfaces (Andreasen, 1981). Rigid splinting may increase resorption in reimplanted teeth that had a short extraoral period (Andreasen, 1975). Extraalveolar storage and cleansing procedures can affect the extent of inflammatory resorption (Weinstein et al, 1981). Wet storage is better (Andreasen, 1980). Air drying and removal of the PDL increase the severity of inflammatory resorption (Andreasen and Kristerson, 1981). In general, the more physiologic the extraoral conditions are, and the less time the teeth are out of the socket, the less severe the cell damage and consequent resorption.

Replacement resorption (ankylosis) begins at 2 weeks and decreases with time following replantation (Andreasen, 1980). Ankylosis is increased when the periodontal ligament is removed (Andreasen, 1981b; Andreasen, 1981a) or dried (Andreasen and Kristerson, 1981) prior to reimplantation. In rats apicoectomy procedures on molar

teeth resulted in a transient ankylosis at 7 days that was resolved by cementum repair by 28 days (Andreasen, 1976).

Pulp status plays a role in external root resorption that is independent from trauma. Winter and Krammer (1972) experimentally induced pulpal necrosis in the deciduous teeth of monkeys and related this to root resorption. Walton and Garnick (1986) induced pulpal necrosis in the permanent teeth of monkeys. Resorption adjacent to inflamed areas of the periapical tissues was a consistent finding. The presence of bacteria in the necrotic pulps of traumatized teeth corellates well with external root resorption (Bergenholtz, 1974). Simon et al (1981) compared the cellular cementum of normal and periapically diseased teeth of humans by SEM. The diseased cellular cementum contained more projections, more cementum lacunae, fewer fibers, and an amorphous layer. Increased cemental resorption was also a consistent observation.

2.7c Endodontic Treatment

Endodontic treatment has been linked to resorption in both non-traumatized and reimplanted teeth. Andreasen (1981) found that pulp extirpation did not affect resorption on reimplanted teeth. Endodontic therapy prior to reimplantation, however, was found to enhance root resorption. Teeth with gutta-percha/Kerr sealer root

fillings consistently showed increased frequency of surface and replacement resorption, and may suppress inflammatory resorption. Intracanal calcium hydroxide during the first week after reimplantation increases inflammatory resorption (Andreasen and Kristerson, 1981). For these reasons, endodontic therapy should be postponed 1-2 weeks until the periodontal inflammation has subsided.

Conservative endodontic therapy has been reported to cause root resorption (Strindberg, 1956; Brynolf, 1967; Seltzer et al, 1967; Kerekes et al, 1980). Simon et al (1983) studied the effects of endodontic instrumentation on root resorption in non-traumatized monkey teeth. They concluded that intrumentation alone caused resorption that was not exacerbated by disinfection of the root canal with agents containing formaldehyde. Several authors have related surgical endodontic treatment to root resorption (Andreasen and Rud, 1972b,a; Rud et al, 1972; Andreasen, 1973) Harrington and Natkin (1979) associated external inflammatory resorption with the internal bleaching of discolored pulpless teeth. They speculated that hydrogen peroxide diffused through the dentinal tubules into the cervical periodontal ligament.

2.7d Orthodontic Treatment

Orthodontic treatment has been reported to increase

the incidence and degree of external root resorption (Newman, 1975). This was not related to systemic, genetic, or occlusal factors. The magnitude of the orthodontic force seems to be a determining factor in the direction of resorptive activity (Lilja et al, 1983). Lilja et al (1983) found a lack of enzyme activity in the central parts of the pressure zones within the PDL. This indicated the presence of necrotic tissue in these areas. Necrotic tissue is chemotactic for macrophages and stimulates resorption. Mattison et al (1984) found no difference in external root resorption between endodontically treated and vital teeth that were subjected to orthodontic forces.

2.7e Other

Other causes of root resorption have been reported and include periodontal disease (Harvey and Zander, 1959), periodontal re-attachment procedures (Gottlow et al, 1984; Klinge et al, 1981; Magnusson et al, 1984), and idiopathic.

2.7f Treatment

The treatment for inflammatory external resorption due to trauma or apical external resorption of pulpal etiology often involves cleaning, shaping and filling the canal with calcium hydroxide (Cvek, 1973; Heithersay,

1975; Biesterfeld and Taintor, 1980; Stewart, 1975; Frank, 1979). Therapy relies on the diffusion of calcium hydroxide outwards through the dentinal tubules. Tronstad et al (1981) has shown that calcium hydroxide is not capable of effecting pH changes in cementum. However, in resorption areas, an alkaline pH was reported at the exposed dentinal surface. This treatment is often effective in arresting external root resorption.

CHAPTER 3

METHODS AND MATERIALS

3.1 Experimental Design

The experiment was divided into six parts. Each is outlined below:

a) Effect of distance from the pulp on radicular dentin permeability (Inner vs. Outer root slabs)

i) Outer root slabs (5 specimens)

ii) Inner root slabs (5 specimens)

b) SEM correlation of tubule density and diameter to radicular dentin permeability

i) counts of tubule numbers on radicular dentin slabs (8 inner & 8 outer slabs)

ii) measurements of tubule diameters on fractured root slabs (8 specimens)

c) Comparison of radicular and coronal dentin permeability (6 specimens)

i) Coronal dentin disks (6 specimens)

ii) Outer root slabs (6 specimens)

iii) Inner root slabs (6 specimens)

iv) Effects of Potassium Oxalate (6 specimens)

d) Effects of Reduced Thickness on Rate of Fluid Flow

i) Outer root slabs (5 specimens)

ii) Inner root slabs (5 specimens)

Smear layers were removed by acid etching and hydraulic conductance (Lp) was measured before and after each reduction in thickness.

e) Effects of Surface Manipulations

i) outer root surfaces

-sharp vs. dull currettes

-of acid-etching

-potassium oxalate

ii) inner root surfaces

-endodontic files

-acid-etching

-NaOCl

Smear layers were removed by acid etching and hydraulic conductance (Lp) was measured before and after each manipulation.

3.2 TOOTH PREPARATION

Extracted unerupted human third molars were placed in isotonic saline containing 0.2% sodium azide to inhibit microbial growth, and stored at room temperature.

Specimens were not stored longer than 3 weeks (Outhwaite et al, 1976). Only teeth with a relatively broad, flat root surface were selected (Fig. 1). The crowns were removed using a high-speed, water-cooled diamond saw and the roots were attached to mounting stubs with epoxy resin. Inner and outer root slabs were prepared utilizing a low-speed diamond saw (Buehler LTD, Isomet, low speed saw) with water coolant. Cuts were made parallel to the long axis of the root. The cut surfaces were treated with 50% citric acid for 2 min to remove the smear layer created by the saw which obstructed the tubule orifices (Figs. 2, 3). The specimens were handled by their edges to prevent alterations of the surfaces to be studied.

3.3 EXPERIMENTAL METHOD

After acid-etching, the coronal disks or root slabs were placed in a plastic split chamber device and connected to the apparatus described by Pashley and Galloway (1985)(Fig. 4). Briefly, a nitrogen tank supplied the hydrostatic pressure gradient which moved isotonic saline from the reservoir, through a micropipette, into the lower compartment of the split chamber and across the root slabs to the open upper compartment. The movement of an air bubble in the micropipette was measured in mm/min and,

knowing the volume of the micropipette, this was converted to μL/min. Each measurement was repeated 4 times. A microsyringe was used to control the position of the air bubble in the system. The dentin surface area studied was limited by identical rubber "O"-rings on each side of the slab. Experiments were conducted at room temperature (Outhwaite et al, 1976). Reductions in slab thickness were accomplished using 320 grit silicon carbide sand paper (3M Co. Ltd.) from the cementum surface inward. Each sanding procedure reduced the thickness about 0.08 mm. A machinist's micrometer was used to measure slab thicknesses to the nearest 0.01 mm. Ten teeth were used (5 in each experiment) yielding a total of 10 inner dentin slabs and 10 outer slabs consisting of peripheral dentin and cementum. Measurements of hydraulic conductance (L_p) were confined to cervical root dentin.

To measure tubule diameters, eight dentin slabs prepared as described above were fractured in liquid nitrogen, stored in a solution of 5% paraformaldehyde and 5% glutaraldehyde, air dried, sputter coated with a thin layer of gold (Hummer X, Anatech Ltd, Alexandria, VA), examined under SEM (AMRay 1000, Advanced Metal Research Corp. Burlington, MA) and photographed at 5000X. Scanning electron micrographs were made of the cervical area on the inner surfaces of the inner slabs and on the outer surfaces

of the outer slabs. Pilot trials had shown that there were no significant differences in mean tubule diameters when comparing these surfaces to the inner surfaces of the outer slabs or the outer surfaces of the inner slabs. Tubule diameters were measured on SEM prints to the nearest 0.01 mm using an electronic digital caliper.

Tubule densities were studied in the cervical area on the cut surfaces of eight inner and eight outer root dentin slabs that had been acid etched to remove the smear layer and prepared for SEM as described above. Tubule densities were recorded by marking tubules on acetate templates of known area on the SEM monitor as they were counted.

3.4 CALCULATIONS

Hydraulic conductance (L_p) was calculated using this formula:

$$L_p = \frac{Jv}{A\Delta Pt} \qquad \text{(Formula 3)}$$

where:

Jv= fluid flow in μL

A = dentin surface area in cm^2

ΔP = hydrostatic pressure gradient in cm H_2O

t = time in min

L_p= hydraulic conductance

in $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$

Fluid movement, or Jv was calculated from mm of bubble movement per min. Hydrostatic pressure was controlled by the regulator on the nitrogen tank. To account for distortion of the "O"-rings when they were compressed during assembly of the chamber, the surface area described by the "O"-rings was determined by running methylene blue dye through the system with a slab in place. The slab was then placed on a computer graphics tablet (Bit Pad One, Summagraphics, Fairfield, CT) which was connected to a computer (PPDII/03-L, Digital Equipment Corp.; Maynard, MA) and a monitor (Digital VT-100). The outline of the dyed area was traced with a crosshairs on a movable cursor, and, by command, the area was calculated immediately and displayed on the monitor.

Due to variations in permeability between different teeth, results were expressed as percent changes from the hydraulic conductances of the acid etched root slabs prior to any manipulation. Thus, each slab acted as its own control.

Mean tubule diameters were determined by dividing the measurements in mm by the magnification and expressed in micrometers. Tubule densities were determined by dividing the tubule counts/template by the area of the template.

Mean tubule densities were expressed in number of tubules per square mm.

To account for the effect of differences in tubule density and radius between inner and outer root dentin on the measured hydraulic conductances, the following ratio was derived:

$$\frac{r_i \; X_i L_{po}}{r_o \; X_o L_{pi}} \qquad \text{(Formula 4)}$$

where:

r_i = mean tubule radius of inner slabs in μm

r_o = mean tubule radius of outer slabs in μm

X_i = mean tubule density of inner slabs in number/mm²

X_o = mean tubule density of outer slabs in number/mm²

L_{pi} = mean hydraulic conductance of inner slabs in $\mu L \; cm^{-2} \; min^{-1} \; cm \; H_2O^{-1}$

L_{po} = mean hydraulic conductance of outer slabs in $\mu L \; cm^{-2} \; min^{-1} \; cm \; H_2O^{-1}$

3.5 DATA ANALYSIS

The tubule densities and diameters, and the hydraulic conductances calculated using Formula 2 were entered into a microcomputer statistics program (Stat Pack, The Basic

Business Software Co., Inc.) The Student's t test was used to determine whether significant differences existed between the mean Lp's of the coronal, inner and outer dentin slabs, between the mean Lp's before and after surface manipulations, between the mean tubule densities and diameters of inner and outer dentin slabs. Least squares regression lines were calculated for inner and outer dentin hydraulic conductances. All tests for statistical differences were conducted with alpha = 0.05.

CHAPTER 4

RESULTS

4.1 Calculations

Pilot trials had shown that in most cases, for radicular dentin slabs, the use of 25 microliter pipettes (65 mm long) and a hydrostatic pressure gradient of 15 psi (1050 cmH_2O) resulted in rates of bubble movement that were convenient for recording. The surface area studied was 8.76×10^{-2} cm^2. This was the largest area possible due to the topography of the samples. For coronal dentin disks 50 microliter pipettes (100 mm long) and a pressure gradient of 10 psi (700 cmH_2O) were convenient. The surface area studied here was larger: 2.22×10^{-1} cm^2. The largest possible surface area was used here to obtain representative hydraulic conductances (Lp's) since there are regional differences in the permeability of coronal dentin disks (Andringa et al, 1986) To minimize experimental error, pressures were kept constant throughout each experiment.

4.2 Effect of Distance from the Pulp

The mean L_p for inner root dentin was 2.69×10^{-2} and for outer root dentin was 2.76×10^{-3}. The difference between these means was statistically significant ($p<0.01$). Table 1 shows the hydraulic conductances of the inner and outer root slabs individually, and the difference betweeen the inner and outer L_p's expressed as a percentage. The inner root slabs accounted for approximately 90% of the fluid movement (Fig. 6) while the outer slabs consisting of peripheral dentin and cementum accounted for only approximately 10%.

4.3 Tubule Density and Diameter

The mean tubule density for the inner surface of the inner slabs was 40,691 +/- 7107 tubules/mm^2 ($\bar{X}$ +/- SD) while for the outer surface of the outer slabs it was 20,895 +/- 2817 ($\bar{X}$ +/- SD) tubules/mm^2 (Table 2; Figs. 7, 8). Eight inner and eight outer slabs were studied. Tubules were counted on 3 SEM fields per root slab.

The mean tubule diameter for the inner slabs was 1.56 +/- 0.29 µm ($\bar{X}$ +/- SD) and for the outer slabs it was 1.07 +/- 0.18 µm ($\bar{X}$ +/- SD) (Table 3; Figs. 7, 8). Tubules were

measured on 1 SEM print each from 4 inner and 4 outer slabs. A total of 68 inner tubules and 26 outer tubules were measured.

Correcting for differences in tubule numbers and diameters yields the ratio of 0.90 (Formula 3) for inner and outer radicular dentin. That is, 90% of the differences in the hydraulic conductances of inner and outer dentin could be accounted for by the differences in tubule densities and radii.

4.4 Coronal vs Radicular Dentin Permeability

The mean coronal Lp was 1.32×10^{-1}. All coronal dentin disks were made 1.00+/-0.01mm thick by sanding (Reeder et al, 1978). The mean Lp for the inner radicular slabs was 1.12×10^{-2}. The mean Lp for the outer radiucular slabs was 3.50×10^{-3} (Table 4, Fig. 9). Expressed another way, the inner root dentin Lp was 8.48% that of the coronal dentin, while the outer root dentin Lp was only 2.65% that of the coronal dentin.

The mean percent reduction in Lp after potassium oxalate application to coronal dentin was 99.2% (Table 15). The mean percent reduction in Lp for inner radicular dentin was 88.73% (Table 13). For outer root dentin potassium oxalate reduced Lp by 61.28% (Table 14, Fig. 19). However,

there were no significant differences in the mean Lp's after potassium oxalate was applied to coronal dentin, inner radicular dentin, or outer radicular dentin.

4.5 Effects of Thickness

Reductions in slab thickness were from the cementum surface inward. Each sanding procedure reduced thickness about 0.08 mm. There was a linear inverse relationship between radicular dentin thickness and permeability for the inner dentin slabs (Fig. 10). As the thickness was reduced, L_p increased. For the outer slabs, there was an exponential inverse relationship (Fig. 11). The initial permeability values for each slab were with the cementum attached. (Table 6). The thickness value was the actual thickness of the slab, not the distance from the pulp. The permeability did not increase after removal of the cementum. An additional 0.2 mm of dentin had to be removed before Lp increased (Fig. 11).

4.6 Surface Manipulations

a) Outer Root Slabs

The use of both sharp and dull curettes created smear layers which significantly reduced the permeability

of the outer root slabs (Tables 9, 10; Figs. 12, 13, 14). When sharp curettes were used to root plane the outer (cemental) surface of the outer root slabs a mean reduction in Lp of 29.42% resulted. The use of dull curettes caused a mean reduction in Lp of 35.45%. There was no significant difference between the means of these two groups. The smear layer created by both sharp and dull curettes could be removed by acid etching (50% citric acid for 2 min) which raised the Lp to the original value or higher. Potassium oxalate application to this acid etched surface resulted in a mean reduction in Lp of 61.28% (Table 14; Figs. 18, 19).

The use of endodontic files on the inner surface of the outer root slabs resulted in a mean reduction in Lp of 25.24% (Table 12; Fig. 15, 16). This smear layer could be removed by acid etching and the Lp returned to its original value or higher.

b) Inner Root Slabs

When sharp curettes were used to root plane the outer (dentin) surface of the inner root slabs, the mean reduction in Lp was 69.46% (Table 7, Figs. 12, 14). The use of dull curettes caused a mean reduction in Lp of 67.08% (Table 8, Fig. 13, 14). Again, there was no significant difference in the mean reduction in Lp between

sharp and dull curettes. This curette-created smear layer could be removed by acid etching and this resulted in a return of Lp to the original value or higher.

The use of endodontic files on the inner surface of the inner root slabs resulted in a mean reduction in Lp of 48.56% (Table 11; Figs. 15, 16). This file-created smear layer could be removed by acid etching and this resulted in a return of Lp to the original values or higher. NaOCl application did not affect the Lp of the smear layer (Table 16). Application of potassium oxalate to this acid etched surface caused a mean reduction in Lp of 88.73% (Table 13; Figs. 17, 19). There was a significant difference between the mean reductions in Lp caused by file-created smear layers and by potassium oxalate.

CHAPTER 5

DISCUSSION

Root slabs prepared and stored in the manner described in the methods and materials section of this paper had empty dentinal tubules (Fig 5). Saline storage allowed the odontoblastic processes to undergo autolysis. Although the presence or absence of odontoblastic processes does not appear to have much effect on fluid flow across dentin (see above), the hydraulic conductances measured in this *in vitro* study should be regarded as maximum limits of the *in vivo* values.

5.1 Effect of Distance From the Pulp

The inner dentin slabs were more permeable than the outer slabs due, in part, to the higher tubule density but mostly due to the larger diameter tubules in the inner slabs. The permeability was greater because fluid flow varies with the fourth power of the radius of the tubule (Formula 1). The differences in inner and outer Lp were 9.75-fold (Table 1). The differences in tubule density accounted for 1.95 of the 9.75-fold difference. The tubule

radii raised to the fourth power accounted for 4.52 of the 9.75-fold difference. The two together accounted for 8.8 of the 9.75-fold difference or 90%. This is shown by the ratio of 0.90 to 1 (Formula 4) when tubule density and radius raised to the fourth power are considered. The fact that the differences in Lp do not sum directly may be due to differences in functional versus anatomic tubular radii (Michelich et al, 1978). Functional radius refers to the area of the tubule actually available for fluid movement after partial tubule blockages are accounted for. Anatomic radii, as measured microscopically in this study, only provide a maximum boundary for the functional radii. However, if one assumes that the functional radii represent a similar amount of intratubular debris in both inner and outer radicular dentin, the results agree well with theoretical calculations.

5.2 Tubule Density and Diameter

Garberoglio and Brannstrom (1976) studied fractured surfaces of human coronal dentin under SEM. Close to the pulp they found approximately 45,000 tubules per square mm with a diameter of 2.5 µm. The findings of this study for radicular dentin near the pulp were 40,961 tubules per

square mm with a diameter of only 1.56 μm (Tables 2, 3; Figs. 7, 8). Their findings of greater tubule density and larger diameter tubules would explain the greater permeability of coronal dentin. The ratio of the number of tubules per unit area at the pulpal and outer root surface in this study is only about 2:1, rather than the 4:1 reported in coronal dentin by Garberoglio and Brannstrom.

The results of Carrigan et al (1984) agreed closely with those of Garberoglio and Brannstrom (1976). Furthermore, they found that the number of tubules decreased in a corono-apical direction. Observations of radicular dentin disclosed tubule densities that decreased from approximately 42,000 tubules per square mm in cervical dentin to approximately 8,000 tubules per square mm in apical root dentin. The findings of the present investigation are similar to those of Carrigan et al (1984) with respect to tubule density of cervical root dentin.

The mean tubule diameter of inner radicular dentin is greater than that of outer radicular dentin (Table 3, Figs. 7, 8). This is supported by the findings of Garberoglio and Brannstrom (1976) who reported that tubule diameters decreased progressively as they coursed from the pulp to the enamel in coronal dentin.

5.3 Coronal vs Radicular Dentin

Radicular dentin is much less permeable than coronal dentin. When the coronal dentin hydraulic conductance figures from this study are compared with those of previous studies (Table 5) there is close agreement. The mean coronal Lp found in this study was 1.32×10^{-1} $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$ (Table 4). The mean coronal Lp from the pooled results of the previous studies were 1.29×10^{-1} $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$ (Table 5). These data demonstrate that the permeability of inner radicular dentin is only 8.48% that of coronal dentin. The L_p of outer radicular dentin is only 2.65% that of coronal dentin. In spite of using only unerupted third molar teeth, biologic variations played a part. Biologic variations in permeability between different teeth make precise application of these percentages to individual teeth difficult.

5.4 Effect of Thickness

Fluid movement was found to be inversely proportional to dentin thickness (Tables 6; Figs. 10, 11). This agrees with the results reported by others for coronal dentin (Pashley et al, 1977; Outhwaite et al, 1976) and is due to a reduction in the frictional resistance to flow of the

tubule walls as thickness is reduced.

It is interesting to note that, the permeability of the outer dentin slabs did not increase markedly when the cementum was removed (Fig. 11). The teeth were not examined microscopically for remaining cementum, but the thickness was reduced enough to ensure than none remained. Cementum normally forms a layer averaging approximately 50-100 μm in thickness. It is thickest in the apical regions and may be only 4-5 μm thick in cervical areas (Furseth, 1974a). Several tenths of a mm (i.e. several hundred micrometers) of the adjacent peripheral dentin had to be removed before increases in L_p became apparent. The reason for this low permeability of peripheral dentin may be due to fewer dentinal tubules (Furseth, 1974b) with relatively narrow lumens. The comparatively low permeability of peripheral root dentin may help to explain why periodontally treated teeth, in which the cementum has been removed by root planing, do not routinely show more pulpal inflammation. This relatively impermeable layer may prevent the diffusion of bacterial products from the surface plaque reaching the pulp. Furthermore, this relatively impermeable peripheral root dentin may also restrict the diffusion of toxic products from a necrotic pulp to the periodontium. Toxic products must accummulate in sufficient concentrations if tissue damage is to occur. The accumulation of substances

diffusing through dentin in either direction depends both upon the rate of permeation and the rate of removal by the local microcirculation (Pashley, 1979). Pulps with existing inflammation or compromised blood flow due to caries, restorative procedures, occlusal trauma, excessive orthodontic forces, or vasoconstrictors may be more susceptible to damage by toxic agents diffusing through dentin.

Dentin thicknesses used in this study were similar to the root dentin thickness that remain after endodontic instrumentation. These thicknesses (0.15-1.07 mm, $\overline{X}$ = 0.53 mm) are similar to the dentin thicknesses remaining remaining after post-space preparation in molars (Abou-Rass et al, 1982). They also agree with the thicknesses found by Montgomery (1985) after biomechanical preparation of mandibular molars using conventional circumferential filing or engine-driven reamers. Endodontic instrumentation would result in increased permeability of the remaining root structure by decreasing remaining dentin thickness. It should be noted, however, that endodontic instrumentation removes the deepest, most permeable dentin. Outhwaite et al (1976) found that reductions in thickness made from the pulpal side resulted in a slower rate of increase in permeability than reductions made from the enamel side. Thinning of the root structure from the inside-out, ie. in

the opposite direction from that used in this study, would result in less of an increase in permeability than an equal reduction in thickness from the outside-in. A graph of such a procedure would be less steep in slope than that of Figures 10 and 11. This is because thinning from the inside-out leaves the outer dentin and cementum intact and these form the most effective barrier to fluid flow across the root.

Thickness was found to have an inverse linear relationship to hydraulic conductance in inner radicular dentin. The relationship was exponential in outer radicular dentin. The reason for this difference may be explained on the basis of the geometry of the tubule surface area available for fluid flow. As the cementum and outer dentin are removed, the most effective permeability barrier is removed. The deeper dentin that is exposed has more tubules per unit area and wider tubules. Thus the cross-sectional area of the tubules available for fluid flow increases more dramatically than in inner dentin where no such barrier is removed by thinning.

In discussing the diffusion of molecules across dentin Outhwaite et al (1976) point out that the Fick equation (Formula 3) relates diffusion to membrane surface area and thickness. It assumes that the membrane geometry is constant; i.e. the number and dimensions of the pores

per unit volume is constant. This is not the case for dentin. Although the concentration gradient driving diffusion (dc/dx) becomes steeper as thickness is reduced from either direction, the change in the number and diameter of tubules available for diffusion (actual diffusional surface area) is entirely dependent upon the direction in which the dentin thickness is reduced, even though the apparent diffusional surface area (within the "O"-rings) is kept constant. In other words, as dentin thickness is reduced from the pulpal side of the root, although the concentration gradient would increase, the actual diffusional surface area would decrease due to the increase in calcified intertubular matrix per unit surface area. As dentin thickness is reduced from the cementum side the same increase in concentration gradient is achieved, but the diffusional surface area increases due to a relative reduction in the amount of intertubular dentin.

An examination of the variables determining hydraulic conductance (Formula 2) reveals that the variable of dentin thickness is not included, yet the present results and those of Reeder et al (1978) on coronal dentin demonstrate clearly that filtration rate is very sensitive to changes in dentin thickness. Reeder et al (1978) tried to correct for this by multiplying their data on hydraulic conductance by thickness. This did not improve their

ability to predict filtration rates under specific conditions of thickness. They presumed that this was due to the fact that as dentin thickness is reduced from the enamel side, the number of tubules per unit surface area increases.

5.5 Effect of Endodontic Instrumentation

Endodontic preparation techniques remove the innermost layers of predentin and dentin. The precise effect of such procedures on increasing the permeability of the remaining root structure *in vivo* are difficult to predict. Many additional factors come into play. The effects of thickness alone are complex as discussed above. Canal enlargement increases the canal wall surface area as well as changing the thickness. Both diffusion (Outhwaite et al, 1976) and filtration (Reeder et al, 1978) are related to available surface area. Since canal anatomy is complex, endodontic instruments do not reach all canal walls (Walton, 1976). The final shape of the prepared canal is also geometrically complex and this makes estimates of canal wall surface area rough at best.

Unlike unerupted teeth, erupted teeth generally have a layer of secondary dentin covering the primary dentin. Nalbandian (1960) has shown that this is an age-

dependent phenomenon. Secondary dentin has fewer tubules than primary dentin, however, these are wider in diameter. The net effect of secondary dentin removal is a matter of speculation, however, the root permeability would probably be increased. In teeth exposed to trauma, caries, or any other pulpal irritant, tertiary or reparative dentin is also layed down. Tertiary dentin has fewer tubules and more irregular tubules than primary or secondary dentin (Holland et al, 1979). This would make it less permeable. The extent to which canal enlargement removes this protective layer would tend to increase the permeability of the remaining dentin. In areas of the canal with no tertiary dentin, the innermost, most permeable dentin would contain the highest concentrations of bacteria and bacterial products. Its removal would be in accord with the therapeutic aims of endodontic treatment.

5.6 Effect of Endodontic Irrigation

Sodium hypochlorite is one of the most commonly used endodontic irrigants. It has been reported to have antimicrobial effects (Shih et al, 1970), and to dissolve pulp tissue (Senia et al, 1971), necrotic tissue (Hand et al, 1978), and predentin (Rosenfeld et al, 1978). The depth that this irrigant penetrates into the dentinal

tubules has not been investigated. In a study of the efficacy of several endodontic irrigating solutions Goldman et al (1981) found that sodium hypochlorite alone did not remove the smear layer. This is supported by the finding of the present investigation since sodium hypochlorite was found to have no effect on the hydraulic conductance of radicular dentin slabs with endodontic smear layers (Fig. 26). In a subsequent paper however, Goldman et al (1982) found that when sodium hypochlorite (NaOCl) was used after EDTA as a final flush in an instrumented canal, the smear layer was more effectively removed than when EDTA was used after NaOCl as a final flush. For this reason, they speculated that the smear layer may contain an organic component. It seems likely that this is true since endodontic instruments in contact with healthy and necrotic pulp tissue, bacteria, and odontoblast processes must "smear" some tissue on to the canal walls. The ability of sodium hypochlorite to dissolve the organic component of the smear layer does not seem to influence the permeability of the remaining dentin (Table 16).

Since sodium hypochlorite is irritating to vital tissue (Rutberg et al, 1977; Pashley et al, 1985), and since some studies have stated that there is no difference in debris removal between NaOCl and saline (Senia et al, 1971; Baker et al, 1975), saline is also used for

irrigation. Saline has no effect on the permeability of instrumented or acid-etched dentin. This is demonstrated by the repeatability of the Lp measuremments in all of the present experiments when saline was used as the fluid. The inability of saline to remove the smear layer may be a disadvantage to debridement and disinfection of necrotic canals when the smear layer and dentinal tubules contain bacteria or their products.

Citric acid is also used as an endodontic irrigant. A 50% solution is reported to remove the smear layer and open the tubule orifices to allow the penetration of resin to seal the canal (Tidmarsh, 1978). A 10% solution alternated with NaOCl again produced clean canal walls and patent tubules (Wayman et al, 1979). In this study, 50% citric acid effectively removed the smear layers created by K-files and returned the hydraulic conductance to control values or slightly higher depending on the amount of thickness reduction (Tables 11, 12; Figs. 15, 16).

5.7 Effect of Root Planing

The use of both sharp and dull curettes created smear layers which similarly reduced the hydraulic conductance of outer root slabs (Tables 9, 10; Figs. 12-14). These findings are in contrast to those of Frydenlund

and Krell (1986) who found that sharp curettes increased Lp. This would suggest that sharp curettes were capable of cutting dentin "cleanly" without leaving a smear layer. This seems unlikely and is further disputed by the present SEM evidence that sharp scalers did create smear layers (Fig. 12).

Krell and Frydenlund (1986) also found that 1 min applications of 50% citric acid failed to effect the hydraulic conductances of root-planed dentin. They concluded that the smear layer found on root surfaces was not easily etched way, and may have a higher organic component than smear layers found elsewhere on cut dentinal surfaces. The findings of the present study show that the smear layers created by both sharp and dull curettes on the cemental surface of radicular dentin slabs can be removed by etching with 50% citrate for 2 min (Tables 9, 10). This process returns the Lp to control values or higher, depending on the amount of thickness reduction. The differences between the findings of this study and that of Krell and Freydenlund (1986) may be related to differences in pressure used while scaling. This was not controlled in either study. Perhaps scaling with greater pressure creates more of a burnishing effect that is not easily etched away.

It is possible that the original reduction in Lp

that occurred in this study could also be reversed by the dissolution of the smear layer by saliva or plaque acids *in vivo*. If the root-planing was overly vigorous and removed enough cementum and adjacent dentin to cause an increased permeability, dentinal hypersensitivity could result. In addition, such an increase in dentin permeability could allow the inward diffusion of bacterial products from plaque and dietary products. In teeth with compromised pulpal blood flow, the accumulation of such pulpal irritants could result in pulpal inflammation if their concentration excedes the pulp's threshold.

5.8 Potassium Oxalate

Potassium oxalate was shown to significantly reduce the permeability of dentin regardless of where it was applied. The hydraulic conductances of coronal dentin, and inner and outer radicular dentin were reduced to similar very low values by a 2 min application of 3% monopotassium oxalate (Tables 13-15, Fig. 19). The percent reductions in permeability achieved in coronal dentin agree closely with those of other investigators (Greenhill and Pashley, 1981; Pashley et al, 1984a). There were no significant differences between the mean Lp's achieved after potassium oxalate treatment in coronal or radicular

dentin. Potassium oxalate reacts with the calcium in the dentinal fluid to form calcium oxalate crystals. These crystals obstruct the tubule orifices (Figs. 17, 18), reduce the functional radii of the tubules, and this causes a profound reduction in the ease with which fluid flows across the dentin (Greenhill and Pashley, 1981).

The fact that potassium oxalate was found to work equally well on radicular dentin supports its use as a desensitizing agent in cases of cervical hypersensitivity.

Potassium oxalate may also be of use in endodontics. The smear layer created by endodontic instrumentation may harbour bacteria or bacterial products (Pashley and Livingston, 1978). It may also reduce the ability of irrigants and medicaments to penetrate into dentinal tubules dissolve tissue and kill bacteria. For these reasons many investigators have looked at ways of removing the smear layer (Bowen, 1978; Goldman et al, 1981, 1981, 1984; Brannstrom et al, 1980; Causton and Johnson, 1982; Jodaikin and Austin, 1981). However, the benefits of reduced dentin permeability are then lost. The creation of a potassium oxalate sealed layer after chemomechanical canal preparation has been completed may be an advantage. Since potassium oxalate reduces dentin permeability, any bacteria remaining in the tubules would be "entombed" between the sealed layer on the pulpal side and the

relatively impermeable dentin/cementum layer found peripherally. The diffusion of toxic substances from necrotic tissue in such tubules would also be greatly reduced (Michelich et al, 1978). However, potassium oxalate would not seal large canal ramifications that could potentially harbor greater amounts of irritants than the dentinal tubules could. The actual clinical effect of potassium oxalate used in this way is unknown.

5.9 External Root Resorption

External root resorption occurs as a consequence of damage to the root surface. This may be due to orthodontic treatment (Newman, 1975; Lilja and Hammarstrom, 1983). Periodontal reattachment procedures that include citric acid-etching can cause resorption (Gottlow et al, 1984; Klinge et al, 1981; Magnusson et al, 1984). Internal bleaching of pulpless discolored teeth with hydrogen peroxide has been associated with cervical external root resorption (Harrington and Natkin, 1979; Cvek and Lindvall, 1985). Radiographic and histologic evidence of external root resorption has been reported following conservative endodontic therapy (Strindberg, 1956; Brynolf, 1967; Kerekes et al, 1980; Seltzer et al, 1967) as well as following surgical endodontics (Andreasen and Rud, 1972b,

a; Andreasen, 1973; Rud et al, 1972). The presence of bacteria in the root canal has been related to root resorption (Bergenholtz, 1974). In deciduous teeth, replacement resorption is common (Kurol, 1981, 1984) but its etiology is unknown. It usually occurs in furcation areas and may have a familial tendency (Kurol, 1984).

Andreasen (1985) speculated that the cementum acts as an insulating layer that protects the periodontal ligament against the penetration of bacterial products from the pulp space and dentinal tubules. He thought that external root resorption may cause an increase in root permeability by denuding areas of cementum and exposing the underlying dentinal tubules. Experimental removal of cementum caused inflammatory resorption in immature human teeth, whereas in mature teeth repair with cementum took place (Andreasen, 1973). The immature teeth had wider dentinal tubules and this allowed the penetration of bacteria into the periodontal ligament. In this experiment, however, the amount of peripheral dentin removed with the cementum was not recorded.

The results of the present investigation indicate that cementum is not a complete barrier, since fluid may move across it, albeit at low rates. In addition, cementum removal does not affect the permeability of radicular dentin. More than 0.2 mm of peripheral root dentin must

also be removed by clastic activity before the permeability of the remaining dentin is increased. This changed permeability would be expected to allow more rapid diffusion of substances across the remaining dentin from the periodontal ligament to the pulp or vice versa. In traumatized or reimplanted teeth with necrotic pulps, this could lead to inflammatory resorption (Andreasen, 1981; Jacobsen, 1980). If endodontic medicaments or materials are placed prematurely, or the necrotic pulp allowed to remain too long, resorption could be exacerbated. Whether or not the loss of cementum and peripheral dentin will have significant sequelae depends on the area of dentin exposed, the number and diameter of the tubules in this area, the remaining dentin thickness, the initial pulp status, and the concentration and toxicity of the substances available for diffusion.

5.10 Other Areas Yet to be Investigated

The effects of exposed surface area on radicular dentin permeability can be assumed from previous studies of coronal dentin, however it would be interesting to repeat these experiments on radicular dentin. In particular, it would be of interest to study the effects of canal enlargement procedures on root permeability.

The effect of other endodontic irrigants on radicular dentin permeability is another area of interest. EDTA has been found to remove the smear layer and open tubule orifices (Goldberg, 1977; Goldman et al, 1981). It is antibacterial (Bystrom and Sundqvist, 1985; Patterson, 1963) and increased root dentin permeability to methylene blue dye (Cohen et al, 1970b). Alternating hydrogen peroxide with sodium hypochlorite has been advocated (Svec and Harrison, 1977) for better debris removal. Irrigating with sodium hypochlorite and EDTA alternately has been claimed to increase smear layer removal (Goldman et al, 1982) and to increase the antibacterial effect (Bystrom and Sundqvist, 1985) more than the use of hypochlorite alone. The precise effects of these irrigants on root permeability should be quantitated.

Carrigan et al (1984) found than the number of dentinal tubules decreased corono-apically. Bang and Ramm (1970) found than apical sclerosis increased with age. The permeablity of radicular dentin should also be correlated with its corono-apical location and with the age of the patient.

The present study showed that more than 0.2 mm of peripheral root dentin had to be removed before permeability increased. Exact amount of peripheral dentin removal needed to increase Lp significantly is not known.

The effect of diseased cementum on radicular dentin permeability is a significant question in the treatment of periodontal disease when cementum is exposed to the oral cavity, and in endodontics when cementum is involved in periapical disease.

Correlation of the amount of fluid movement through the dentinal tubules with stimulation of pulpal nerve endings and pain perception in humans is of great interest in understanding pulpo-dentin physiology and in treatment of hypersensitive dentin.

The permeability of radicular dentin with respect to endotoxin is of particular importance to the understanding of the pathogenesis of various disease processes. Endotoxin has been implicated in endodontic disease (Meryon et al, 1986; Pitts et al, 1982; Griffee et al, 1982). It has been demonstrated in root canals and in periapical lesions (Schein and Schilder, 1975; Schonfeld et al, 1982; Dahlen and Bergenholtz, 1980). Endotoxin has been implicated in periodontal disease as well (Aleo et al, 1974; Pitaru et al, 1984; Selvig et al, 1971; Johnson et al, 1976; Ito et al, 1985) Limulus lysate positive substances, presumably endotoxin, have been isolated from periodontally involved cementum (Aleo et al, 1974). The permeability of radicular dentin and cementum to endotoxin has not been studied.

CHAPTER 6

CONCLUSIONS

6.1 Distance from the Pulp

The inner dentin slabs were more permeable than the outer slabs due, in part, to a higher tubule denstiy but more importantly, to larger tubules in the inner slabs. The hydraulic conductance of inner radicular dentin is approximately ten-fold greater than that of outer radicular dentin.

6.2 Tubule Density and Diameter

The mean tubule density of inner radicular dentin is greater than that of outer radicular dentin. The mean tubule density of the inner root slabs was 40,691 tubules/mm^2, while for the outer slabs it was 20,895 tubules/mm^2.

The mean tubule diameter of inner radicular dentin is greater than that of outer radicular dentin. The mean

tubule diameter for the inner slabs was 1.56 μm and for the outer slabs it was 1.07 μm.

These differences in tubule density and diameter accounted for the greater permeability of inner radicular dentin. The latter difference predominated due to the fact that fluid flow varies with the fourth power of the radius.

6.3 Coronal vs Radicular

Radicular dentin is much less permeable than coronal dentin. Coronal dentin had a mean hydraulic conducatance (Lp) of 1.32×10^{-1} μL cm^{-2} min^{-1} cm H_2O^{-1}. Inner root dentin had a mean Lp of 1.12×10^{-2} μL cm^{-2} min^{-1} cm H_2O^{-1}. Outer root dentin had a mean Lp of 3.50×10^{-3} μL cm^{-2} min^{-1} cm H_2O^{-1}. Thus inner radicular dentin is only 8.48% as permeable as coronal dentin, while outer radicular dentin is only 2.65% as permeable.

6.4 Effect of Thickness

Fluid movement is inversely proportional to dentin thickness. This relationship was linear for inner radicular dentin and exponential for outer radicular dentin. The permeability did not increase after removal of all of the cementum; rather, more than 0.2 mm of dentin

had to be removed before Lp increased. This root dentin adjacent to cementum acts as a significant barrier to permeability. Dentin slabs with intact cementum did allow fluid movement, albeit at very low rates. Cementum is not an absolute barrier to fluid movement.

6.5 Surface Manipulations

The use of both sharp and dull curettes created smear layers which reduced the permeability of outer and inner root slabs. There were no significant differences between the reductions in Lp for sharp or dull instruments. The smear layers created by both sharp and dull instruments could be removed by acid-etching (2 min 50% citric acid) to restore the Lp to control values.

The use of endodontic files created smear layers which reduced the permeability of inner and outer root slabs. Sodium hypochlorite application did not effect the hydraulic conductance of such smear layers. These smear layers could be removed by acid-etching to restore the Lp to control values.

Application of potassium oxalate to radicular dentin resulted in dramatic reductions in hydraulic conductance. The mean Lp of inner root slabs was reduced by 88.7%, while the mean reduction in Lp for the outer root slabs was 61.3%

after potassium oxalate application.

6.6 General

The *in vitro* rates of fluid flow through cervical radicular denitn have been determined. Some of the factors governing the rates of fluid flow such as tubule density and radius, dentin thickness, and the effect of various types of smear layers and surface treatments have been quantified. The low permeability of radicular dentin has important implications in periodontics and endodontics. Much more work is required to complete the characterization of the permeability properties of this very important type of dentin.

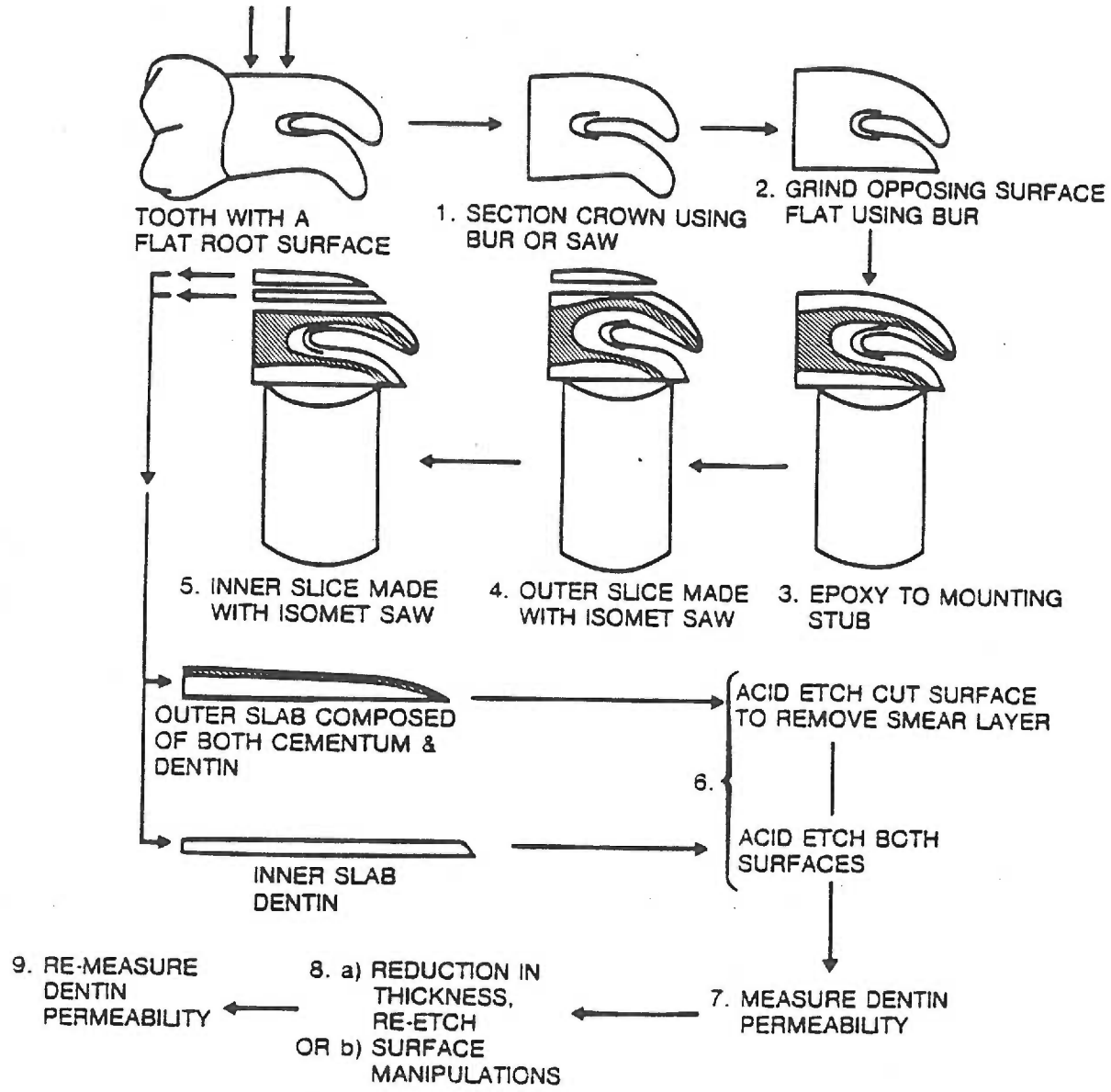

Figure 1. Preparation of root dentin slabs

Figure 2. SEM of radicular dentin covered by a smear layer made by diamond saw cut of radicular dentin slab. (magnification = 1000x)

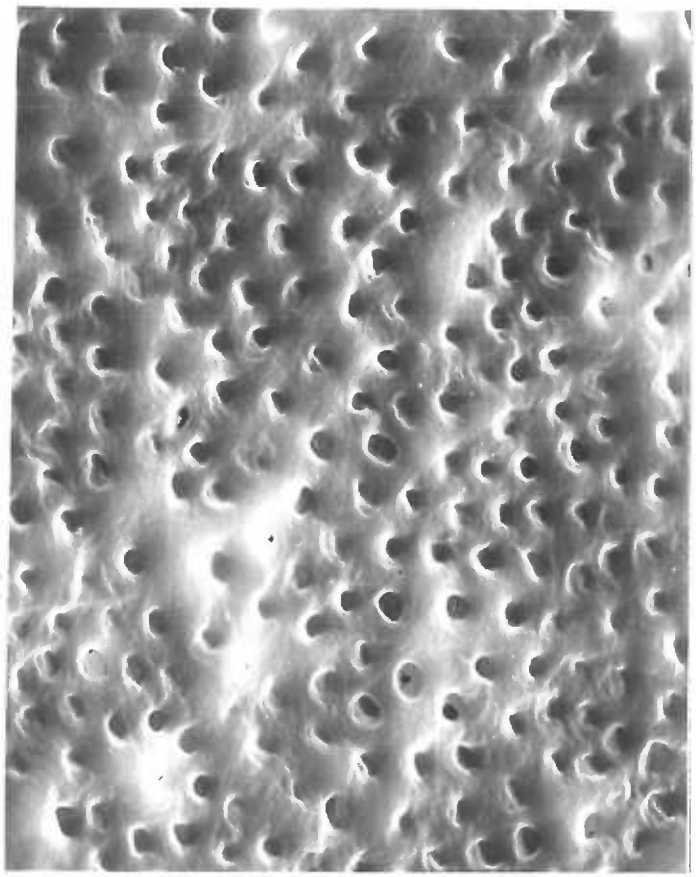

Figure 3. SEM of radicular dentin surface acid etched (2 min 50% citric acid) after saw cut. (magnification = 2000x)

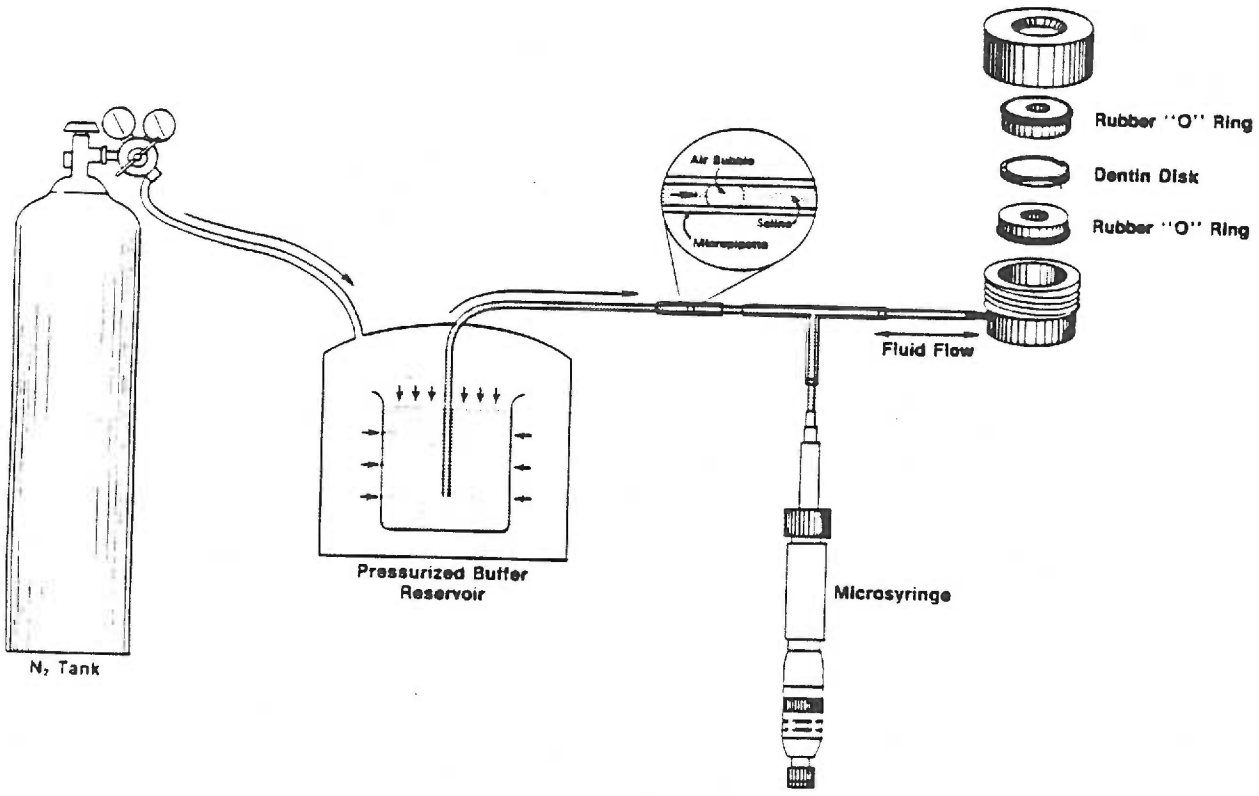

Figure 4. Scheme of the apparatus and split-chamber device used to measure hydraulic conductance (Lp). The movement of the air bubble toward the chamber was used to determine the rate of fluid filtering across the dentin.

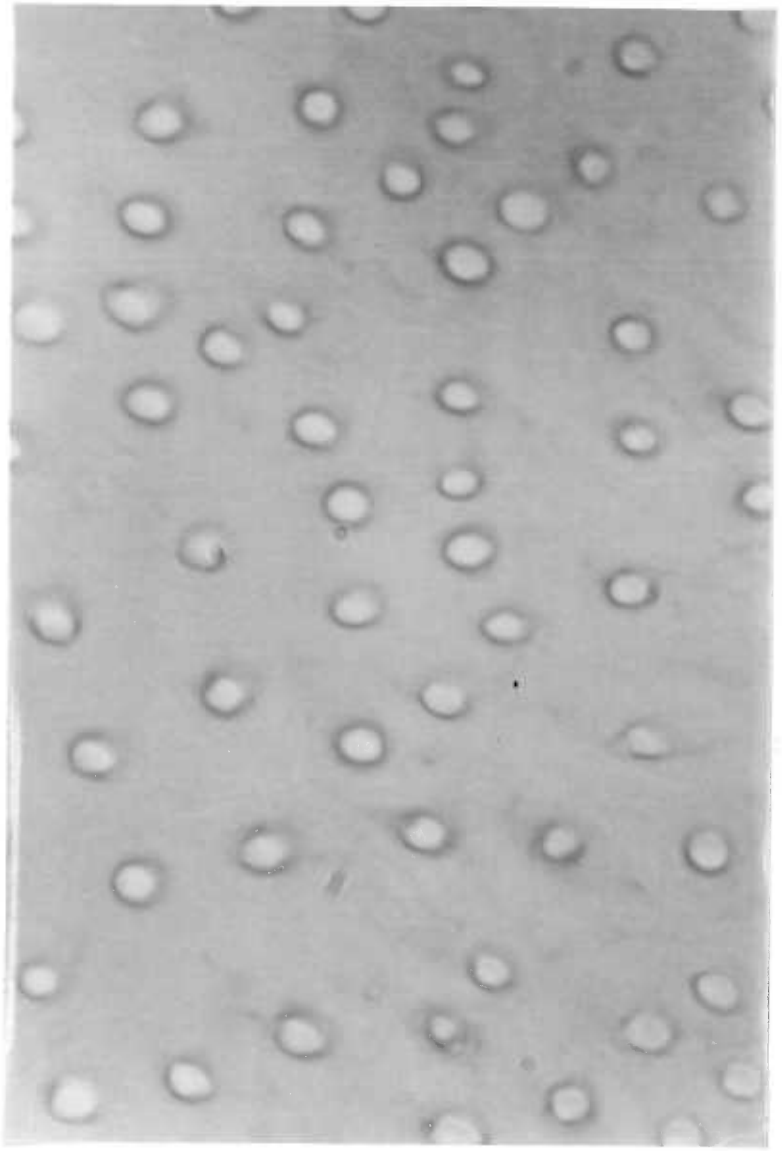

Figure 5. Decalcified radicular dentin, stored in saline, H.&E., 5 um thick, 512x. Taken with Zeiss Photoscope using IX Daylight filter and Kodacolor A.S.A.100 film. Dentinal tubules appear empty.

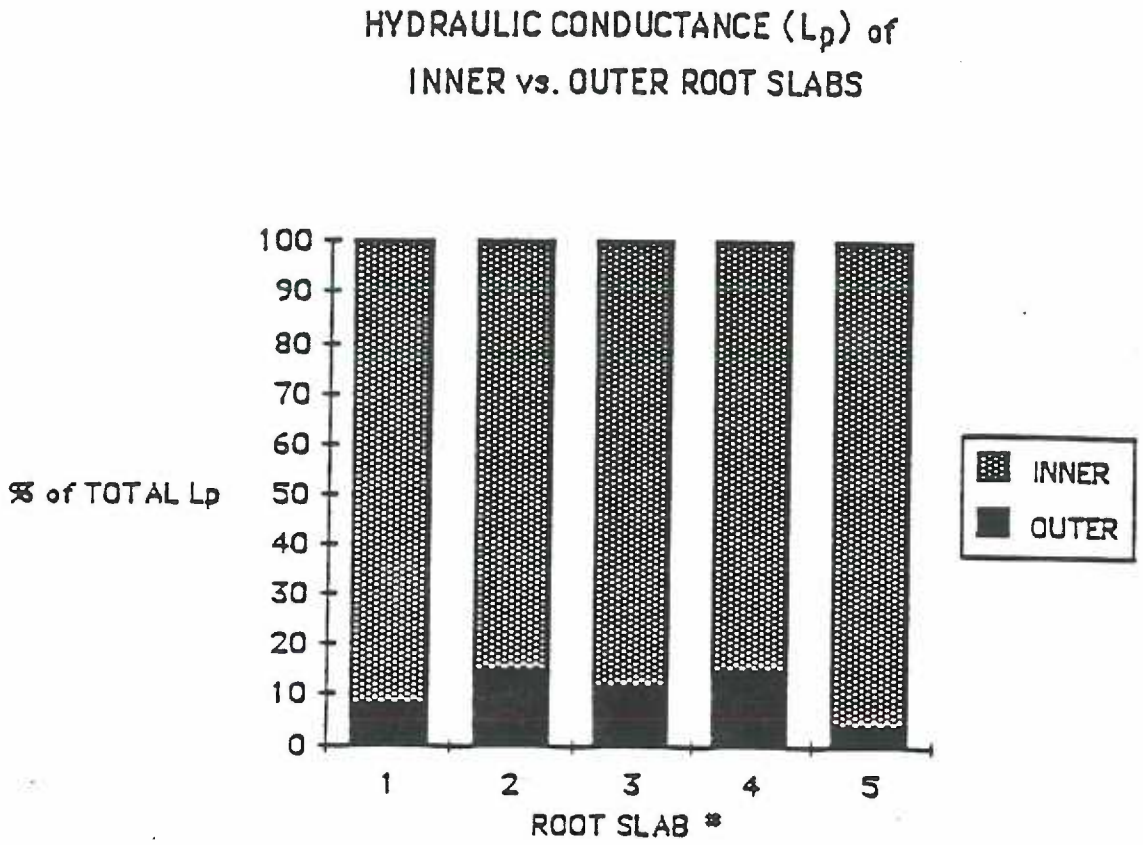

Figure 6. Effect of distance from the pulp on hydraulic conductance (Lp) of radicular dentin for 5 teeth. Lp's of inner and outer root slabs are expressed as a percentage of the total Lp for each sample.

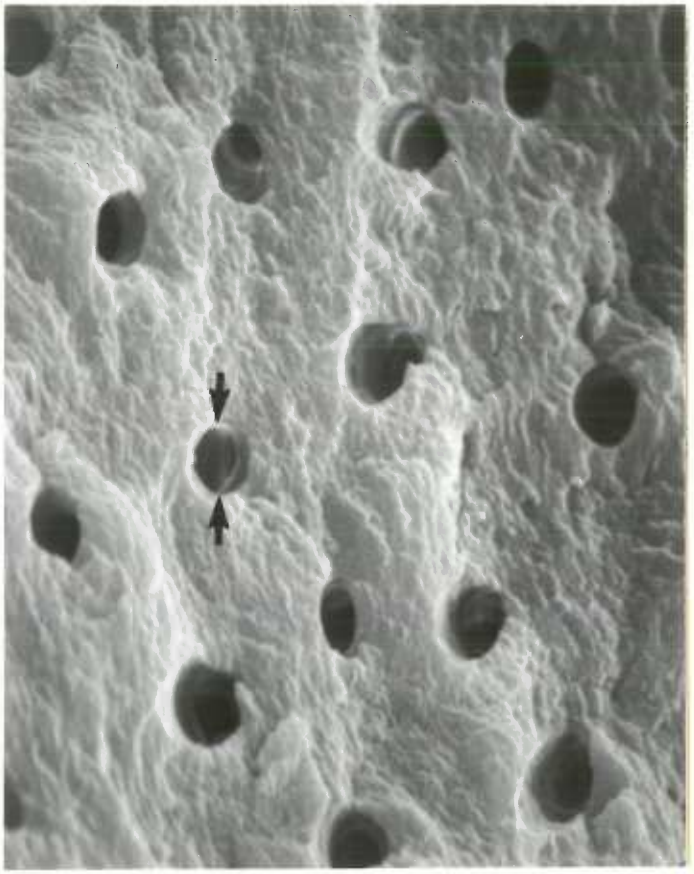

Figure 7. SEM of inner radicular dentin; cervical region; fractured surface; 5000x; showing tubule diameter (arrows). Note large size of tubules and large number of tubules per unit surface area. Arrows show how tubules were measured.

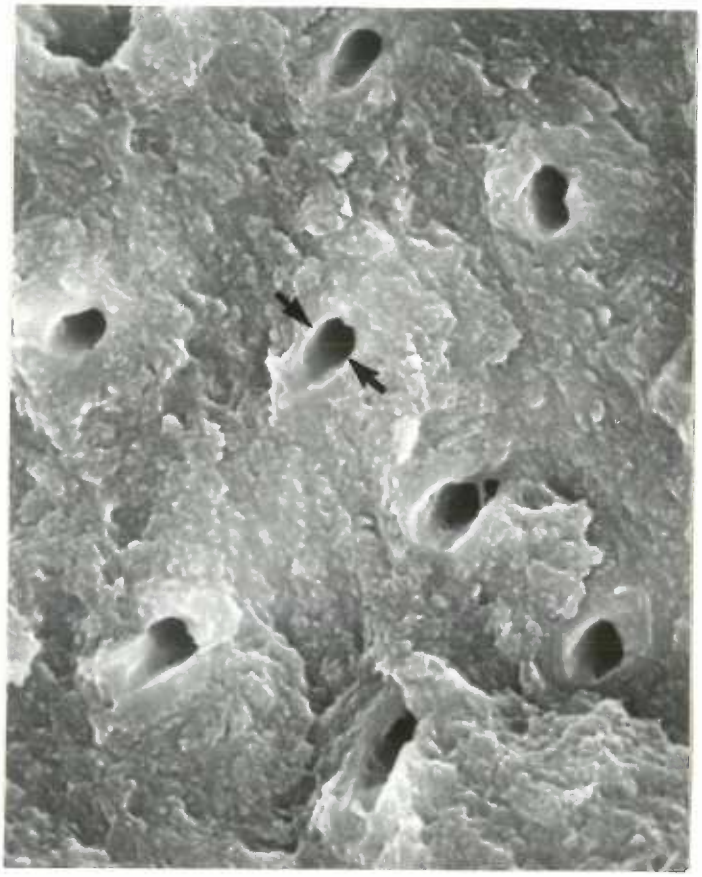

Figure 8. SEM of outer radicular dentin; cervical region; fractured surface; 5000x; obliquely fractured tubules were measured across their smallest diameter (arrows). Note small size of tubules and few tubules per unit surface area.

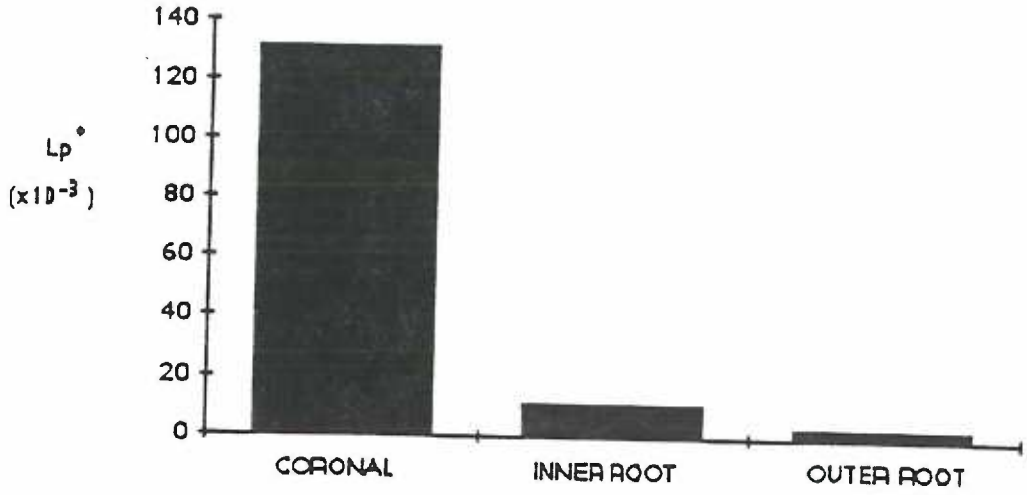

Figure 9. Hydraulic conductance (Lp) of coronal vs radicular dentin for 5 teeth. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

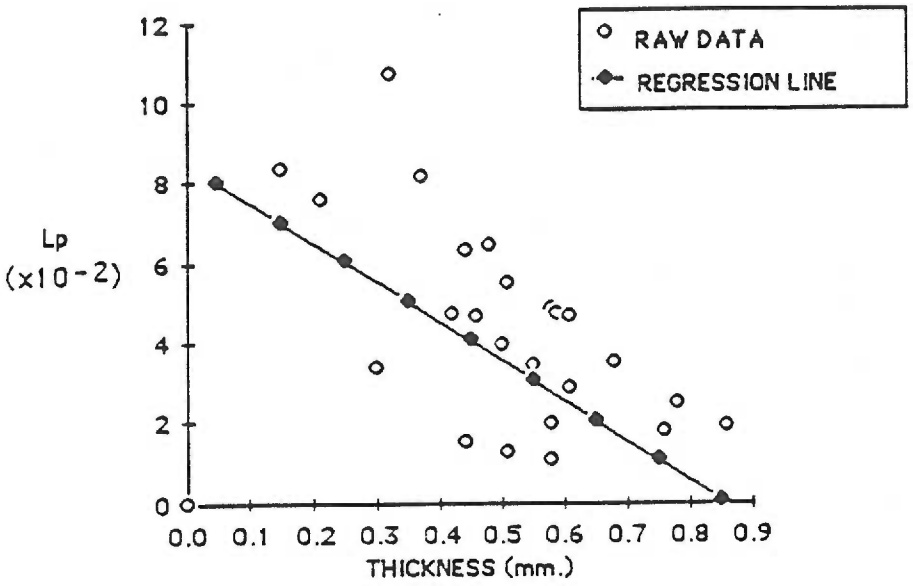

Figure 10. Effect of dentin thickness on hydraulic conductance (Lp) of five inner root slabs. Thickness was reduced from the outer surface of each slab. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$. The relationship between Lp (y) as a function of dentin thickness (x) is described by: $y = 9.94x10^{-2}(x)+0.09$; $r=-0.67$; $p<0.001$

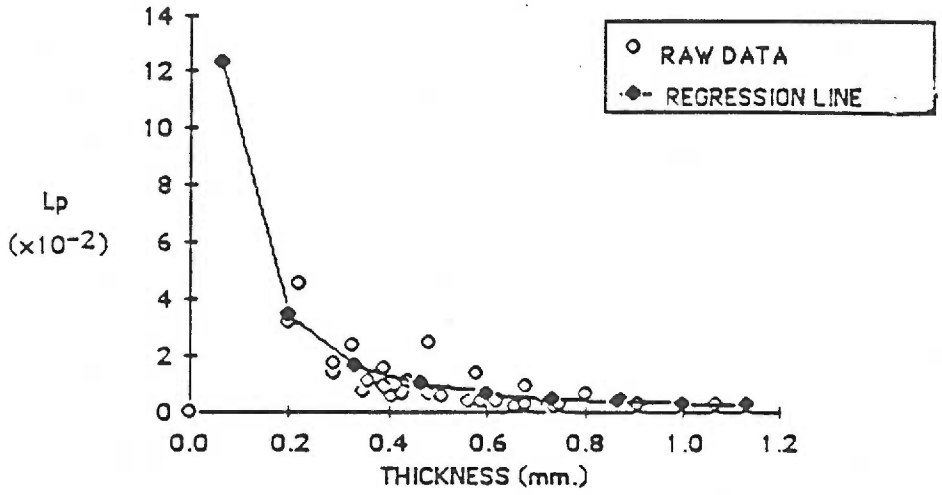

Figure 11. Effect of dentin thickness on hydraulic conductance (Lp) of five outer root slabs. Reductions in thickness were made from the outer surface. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$. The relationship between Lp (y) as a function of dentin thickness (x) is described by: $y = 1.78 \times 10^{-3} (x^{-1.84})$; $r=-0.81$; $p<0.001$

Figure 12. SEM of dentin covered by a smear layer created when a sharp curette was used on outer radicular dentin; 1000x. Note that the dentinal tubule orifices were completely obliterated.

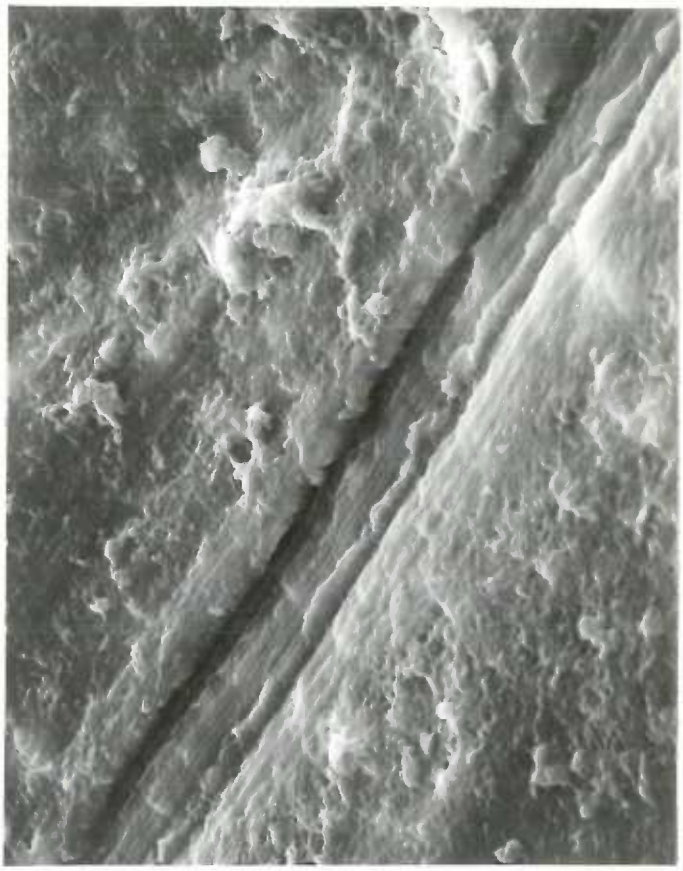

Figure 15. SEM of dentin covered by a smear layer created when an endodontic file was used on inner radicular dentin; 1000x. Note that the debris appears to be less densely packed than the smear layers created by curettes (Figs. 12, 13).

Figure 17. SEM of inner radicular dentin that was acid etched (2 min, 50% citric acid) and treated with 3% monopotassium oxalate for 2 min; 1000x. No tubule orifices are visible.

Figure 18. SEM of outer radicular dentin that was acid etched (2 min, 50% citric acid) and then treated with 3% monopotassium oxalate for 2 min; 1000x. Only an occasional dentinal tubule orifice is visible.

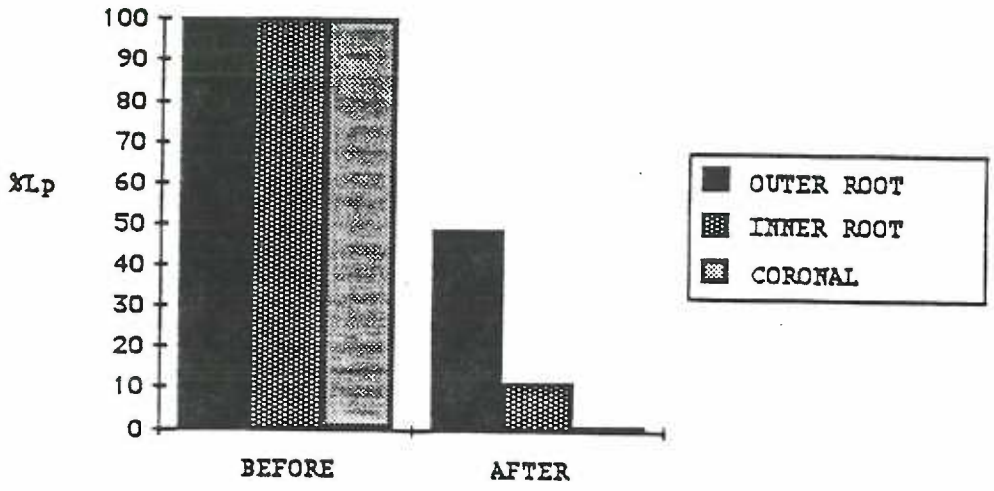

Figure 19. Effect of 3% monopotassium oxalate application on the hydraulic conductance (Lp) of coronal and radicular dentin. Results are expressed as percent reductions from the original (control) values.

SAMPLE	INNER ROOT Lp	OUTER ROOT Lp	% DIFFERENCE
1	$3.24X10^{-2}$	$2.81X10^{-3}$	91.35
2	$1.06X10^{-2}$	$1.76X10^{-3}$	83.01
3	$3.76X10^{-2}$	$4.89X10^{-3}$	86.96
4	$1.78X10^{-2}$	$2.97X10^{-3}$	83.14
5	$3.61X10^{-2}$	$1.38X10^{-3}$	96.12
mean	$2.69x10^{-2}$	$2.76x10^{-3}$	88.12

Table 1. Effect of distance from the pulp on hydraulic conductance (Lp) of radicular dentin. Student's t test of difference between inner and outer Lp's: differences were significant at $p<0.01$; $t=4.72$; $df=4$. Lp in $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	DENTINAL TUBULE DENSITY # of TUBULES PER TEMPLATE INNER SLAB	OUTER SLAB
1	149	109
	151	118
	163	114
2	209	96
	225	102
	241	100
3	220	-
	208	-
	191	-
4	237	85
	203	-
	206	-
5	209	136
	226	125
	260	140
6	269	110
	280	99
	272	119
7	199	97
	180	89
	176	99
8	184	112
	175	105
	227	102

Table 2. Number of tubules in human radicular dentin counted in the area of a standardized template (area of template = 5.184×10^{-3} mm^2). Tubules/template was multiplied by 1.93×10^2 to convert to tubules/mm^2. Inner tubule density = 40,691 +/- 7107 tubules/mm^2 ($\bar{X}$+/-SD). Outer tubule density = 20,895 +/- 2817 tubules/mm^2 ($\bar{X}$+/-SD). Student's t Test of difference between means: t=12.4643; df=23, 18; $p<0.0001$. Note: missing entries in table represent fields in which no tubules were visible.

DENTINAL TUBULE DIAMETERS

SAMPLE	INNER SLAB	OUTER SLAB
1	7.21	5.99
	8.21	5.81
	7.26	6.13
	7.53	4.80
	6.95	6.38
	8.92	2.62
	9.51	-
	8.49	-
2	10.31	5.25
	9.08	5.05
	8.72	5.21
	8.85	5.56
	8.86	6.07
	10.37	6.45
	10.04	6.37
	8.21	-
	10.85	-
	9.20	-
	9.94	-
	9.91	-
	6.69	-
3	6.31	5.70
	5.76	3.63
	7.23	5.38
	7.25	4.82
	6.92	5.63
	6.52	5.84
	7.44	5.15
	8.36	5.14
	5.81	5.93
	7.22	6.09
	8.02	4.92
	7.36	5.27
	6.80	3.78
	6.36	-

continued...

Table 3. See next page for legend.

Sample	INNER SLAB	OUTER SLAB
	6.14	-
	6.32	-
	6.23	-
	8.03	-
	8.06	-
	6.36	-
	8.28	-
	5.86	-
4	7.26	-
	9.38	-
	9.08	-
	8.59	-
	9.27	-
	9.26	-
	9.24	-
	9.65	-
	9.30	-
	7.74	-
	4.31	-
	8.07	-
	5.18	-
	7.46	-
	4.17	-
	8.52	-
	6.83	-
	6.40	-
	8.66	-
	7.93	-
	6.80	-
	7.17	-
	6.59	-
	7.60	-
	9.41	-

Table 3. Dentinal tubule diameters in fractured human radicular dentin. Measurements are in mm at magnification 5000X and were converted to um. Inner tubule diameter = 1.56 +/- 0.29 µm ($\bar{X}$+/-SD). Outer tubule diameter = 1.07 +/- 0.18 µm ($\bar{X}$+/-SD). Student's t Test of difference between means: $t = 8.0763$; $df = 92$; $p = 2.31 \times 10^{-7}$.

SAMPLE	CORONAL Lp	INNER ROOT Lp	OUTER ROOT Lp
1	1.18×10^{-1}	1.01×10^{-2}	1.11×10^{-3}
2	2.81×10^{-2}	1.44×10^{-2}	7.32×10^{-4}
3	7.10×10^{-2}	1.07×10^{-2}	5.75×10^{-4}
4	3.20×10^{-1}	7.84×10^{-3}	6.53×10^{-3}
5	9.81×10^{-2}	8.62×10^{-3}	5.23×10^{-3}
6	1.54×10^{-1}	1.52×10^{-2}	6.80×10^{-3}
mean	1.32×10^{-1}	1.12×10^{-2}	3.50×10^{-3}
SD	9.28×10^{-2}	8.13×10^{-3}	8.13×10^{-3}

Table 4. Hydraulic conductance (Lp) of human coronal vs radicular dentin. Student's t Test of differences between means: $p<0.05$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

STUDY	Lp
Pashley et al (1981a)	5.7×10^{-2}
Pashley et al (1981b)	9.8×10^{-2}
Pashley et al (1983b)	7.59×10^{-2}
Pashley et al (1986)	3.45×10^{-1}
Pashley et al (1984b)	1.07×10^{-1}
Reeder et al (1978)	7.02×10^{-2}
Mean of above	1.26×10^{-1}
Present Study	1.32×10^{-1}
Mean of above	1.29×10^{-1}

Table 5. Hydraulic conductance (Lp) of human coronal dentin. Comparison of results of present study to those of previous studies. Lp is in units of $\mu L\ cm^{-2} min^{-1} cm\ H_2O^{-1}$.

SAMPLE	INNER ROOT SLABS THICKNESS (mm)	INNER ROOT SLABS Lp ($x10^{-2}$)	OUTER ROOT SLABS THICKNESS (mm)	OUTER ROOT SLABS Lp ($x10^{-2}$)
1	0.46	4.65	0.33	2.35
	0.50	3.92	0.39	1.52
	0.55	3.39	0.44	1.10
	0.58	1.93	0.47	0.77
			0.59	0.42
			0.68	0.21
			0.75	0.21
2	0.15	8.36	0.22	4.50
	0.21	7.58	0.29	1.36
	0.42	4.70	0.35	0.68
	0.61	2.82	0.41	0.54
	0.76	1.78	0.56	0.29
3	0.48	6.43	0.48	2.40
	0.59	4.70	0.58	1.31
	0.68	3.45	0.68	0.84
	0.78	2.46	0.80	0.63
	0.86	1.88	0.87	0.42
			0.91	0.21
			1.07	0.21
4	0.30	3.35	0.29	1.67
	0.44	1.52	0.36	1.05
	0.51	1.25	0.43	0.63
	0.58	1.05	0.51	0.52
			0.62	0.31
			0.73	0.26
5	0.32	10.70	0.20	3.19
	0.37	8.15	0.39	0.84
	0.44	6.27	0.48	0.58
	0.51	5.44	0.59	0.37
	0.58	4.81	0.66	0.16
	0.61	4.60	0.74	0.11

Table 6. Effect of thickness on hydraulic conductance (Lp) of human radicular dentin slabs. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	ACID-ETCHED Lp	ΔTHICKNESS (mm)	% Δ Lp
1	3.24×10^{-3}	8.36×10^{-3}	3.29×10^{-2}	1×10^{-2}	74.6
2	7.32×10^{-3}	2.61×10^{-3}	8.89×10^{-3}	2×10^{-2}	70.6
3	9.41×10^{-3}	3.51×10^{-3}	9.41×10^{-3}	2×10^{-2}	62.7
4	1.10×10^{-2}	2.09×10^{-3}	1.25×10^{-2}	3×10^{-2}	83.3
5	1.07×10^{-2}	4.70×10^{-2}	1.07×10^{-2}	1×10^{-2}	56.1
				mean % Δ Lp =	69.5
				SD =	10.5

Table 7. Effect of sharp curettes on the hydraulic conductance (Lp) of inner root slabs. Student's Paired t Test: $t=14.7512$; $df=4$; $p=7.034 \times 10^{-4}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	ACID-ETCHED Lp	Δ THICKNESS (mm)	% Δ Lp
1	8.89×10^{-3}	2.09×10^{-3}	9.15×10^{-3}	3×10^{-2}	77.2
2	9.41×10^{-3}	3.55×10^{-3}	9.41×10^{-3}	2×10^{-2}	62.3
3	1.25×10^{-2}	5.49×10^{-3}	1.91×10^{-2}	3×10^{-2}	71.3
4	1.07×10^{-2}	7.32×10^{-3}	1.96×10^{-2}	3×10^{-2}	62.7
5	1.05×10^{-2}	4.18×10^{-3}	1.10×10^{-2}	3×10^{-2}	61.9

mean % Δ Lp = 67.1
SD = 6.9

Table 8. Effect of dull curettes on the hydraulic conductance (Lp) of inner root slabs. Student's paired t Test: $t=21.8157$; $df=4$; $p=4.057 \times 10^{-4}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1} cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	ACID-ETCHED Lp	Δ THICKESS (mm)	% Δ Lp
1	5.77×10^{-4}	4.18×10^{-4}	5.77×10^{-4}	9×10^{-2}	27.6
2	6.27×10^{-4}	7.84×10^{-4}	1.10×10^{-3}	3×10^{-1}	28.8
3	3.66×10^{-4}	4.70×10^{-4}	6.80×10^{-4}	1.8×10^{-1}	30.9
4	8.36×10^{-4}	8.36×10^{-4}	1.25×10^{-3}	2.2×10^{-1}	33.1
5	7.32×10^{-4}	5.75×10^{-4}	7.84×10^{-4}	7×10^{-2}	26.7

mean % ΔLp = 29.4
SD = 2.6

Table 9. Effect of sharp curettes on the hydraulic conductance (Lp) of outer root slabs. Student's Paired t Test: t=25.4008; df=4; $p=3.416 \times 10^{-4}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	ACID-ETCHED Lp	Δ THICKNESS (mm)	% Δ Lp
1	1.10×10^{-3}	7.84×10^{-4}	1.46×10^{-3}	8×10^{-2}	46.3
2	6.80×10^{-4}	5.23×10^{-4}	8.36×10^{-4}	9×10^{-2}	37.5
3	1.11×10^{-3}	7.32×10^{-4}	1.15×10^{-3}	1.5×10^{-1}	36.6
4	1.52×10^{-3}	1.10×10^{-3}	1.57×10^{-3}	2×10^{-2}	30.0
5	7.84×10^{-4}	5.75×10^{-4}	8.89×10^{-4}	4×10^{-2}	35.3
6	5.75×10^{-4}	4.18×10^{-4}	5.75×10^{-4}	3×10^{-2}	27.3
				mean % Δ Lp =	35.5
				SD =	6.6

Table 10. Effect of dull curettes on the hydraulic conductance (Lp) of outer root slabs. Student's paired t Test: $t=13.1267$; $df=5$; $p=2.883 \times 10^{-4}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	ACID-ETCHED Lp	Δ THICKNESS (mm)	% Δ Lp
1	$8.36X10^{-3}$	$3.66X10^{-3}$	$8.36X10^{-3}$	$0X10^{-2}$	56.2
2	$1.93X10^{-2}$	$1.52X10^{-2}$	$3.24X10^{-2}$	$9X10^{-2}$	53.1
3	$5.65X10^{-2}$	$4.91X10^{-2}$	$6.79X10^{-2}$	$4X10^{-2}$	27.3
4	$7.32X10^{-3}$	$3.56X10^{-3}$	$7.32X10^{-3}$	$4X10^{-2}$	51.4
5	$7.84X10^{-3}$	$3.66X10^{-3}$	$8.10X10^{-3}$	$3X10^{-2}$	54.8

mean % Δ Lp = 48.6
SD = 12.0

Table 11. Effect of endodontic files on hydraulic conductance (Lp) of inner root slabs. Student's Paired t Test: t=9.0331; df=4; $p=1.832x10^{-3}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	ACID-ETCHED Lp	Δ THICKNESS (mm)	% Δ Lp
1	6.79×10^{-3}	6.27×10^{-3}	8.36×10^{-3}	4×10^{-2}	25.0
2	5.77×10^{-4}	4.70×10^{-4}	6.27×10^{-4}	7×10^{-2}	25.0
3	6.27×10^{-4}	5.75×10^{-4}	6.27×10^{-4}	7×10^{-2}	8.3
4	1.15×10^{-3}	7.84×10^{-4}	1.41×10^{-3}	6×10^{-2}	44.4
5	8.89×10^{-4}	6.80×10^{-4}	8.89×10^{-4}	4×10^{-2}	23.5
				mean % Δ Lp =	25.2
				SD =	12.8

Table 12. Effect of endodontic files on hydraulic conductance (Lp) of outer root slabs. Student's Paired t Test: $t=4.4030$; $df=4$; $p=1.287 \times 10^{-2}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE #	Lp BEFORE	Lp AFTER	% Δ Lp
1	8.36×10^{-3}	1.04×10^{-3}	87.6
2	6.79×10^{-2}	2.61×10^{-3}	96.2
3	9.15×10^{-3}	1.05×10^{-3}	88.5
4	1.10×10^{-2}	1.04×10^{-3}	90.5
5	1.91×10^{-2}	4.44×10^{-3}	76.9
6	8.62×10^{-3}	6.27×10^{-4}	92.7
		mean % Δ Lp =	88.7
		SD =	6.6

Table 13. Effect of 3% monopotassium oxalate application on hydraulic conductance (Lp) of inner root slabs (inner surface). Student's Paired t Test: t=33.0702; df=5; $p=7.126 \times 10^{-5}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	% Δ Lp
1	1.25×10^{-3}	4.18×10^{-4}	66.6
2	1.62×10^{-3}	8.89×10^{-4}	45.1
3	8.89×10^{-4}	4.18×10^{-4}	53.0
4	9.15×10^{-3}	2.35×10^{-4}	74.3
5	1.36×10^{-2}	4.44×10^{-3}	67.4
		mean % Δ Lp =	61.3
		SD =	11.8

Table 14. Effect of 3% monopotassium oxalate application on hydraulic conductance (Lp) of outer root slabs (outer surface). Student's paired t Test: t=11.523; df=4; $p=1.094 \times 10^{-3}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	% Δ Lp
1	1.18×10^{-1}	1.19×10^{-3}	99.0
2	2.81×10^{-2}	5.23×10^{-4}	98.1
3	7.10×10^{-2}	6.03×10^{-4}	99.2
4	3.20×10^{-1}	5.23×10^{-4}	99.8
5	9.81×10^{-2}	1.61×10^{-4}	99.8
6	1.54×10^{-1}	7.24×10^{-4}	99.5
		mean % Δ Lp =	99.2
		SD =	0.6

Table 15. Effect of 3% monopotassium oxalate application on hydraulic conductance (Lp) of coronal dentin. Student's Paired t Test: $t=377.0926$; $df=5$; $p=2.486 \times 10^{-5}$. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

SAMPLE	Lp BEFORE	Lp AFTER	% Δ LP
1	$6.27X10^{-3}$	$6.27X10^{-3}$	0
2	$2.09x10^{-3}$	$2.09x10^{-3}$	0
3	$1.25x10^{-3}$	$1.25x10^{-3}$	0
4	$1.05x10^{-3}$	$1.05x10^{-3}$	0
5	$7.84x10^{-4}$	$7.84x10^{-4}$	0

mean % Δ Lp = 0

Table 16. Effect of sodium hypochlorite on hydraulic conductance of radicular dentin slabs with endodontic smear layers. Lp is in units of $\mu L\ cm^{-2}\ min^{-1}\ cm\ H_2O^{-1}$.

APPENDIX 1

ABBREVIATIONS

CEJ	cemento-enamel junction
CMCP	camphorated monochlorophenol
CPCP	camphorated parachlorophenol
DEJ	dentino-enamel junction
EDTA	ethylene-diamine-tetraacetic acid
Jv	fluid flow rate, $\mu L\ min^{-1}$
Lp	hydraulic conductance, $\mu L\ min^{-1}\ cm^{-2}\ cm\ H_2O^{-1}$
M	molar
min	minutes
mm	millimeters
NaOCl	sodium hypochlorite
PDL	periodontal ligament
PCP	parachlorophenol
psi	pounds per square inch
r	radius
SEM	scanning electron microscope/micrograph
SD	standard deviation
TEM	transmission electron microscope/micrograph

µL	microliter
µm	micrometer
vs	versus
$\bar{X}$	mean

APPENDIX 2

EXPERIMENTAL MATERIALS, EQUIPMENT AND SOURCES

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chamber - Norway Tool & Engineering Co, 2110 Pontius Ave, Los Angeles, CA, 90025, (213) 478-2228; Ask for Dr. Pashley's "Diffusion Cell Assembly", Drawing #NT-8214 A made of Plexiglass (unless organic solvents are to be used, then specify "Delrin")

connectors - Small Parts Inc, P.O. Box 381736, Miami FL, 33238, (305) 751-0856; 4 port, 18 ga. connectors, cat #TC-18/4

citric acid monohydrate - Fischer Scientific, Fairlawn, New Jersey, 07410

EDTA - REDTA Irrigating Solution, Roth Drug Co, Chicago, IL, 60610

endodontic files - #60 K-files, Union Broach, 1107 Boadway, New York, NY, 10010

Epoxy - Extra Fast Setting Epoxy, Cole Parmer Instrument Co, Chicago, IL, 60648; reorder #8778

Methylene blue dye - Elkins-Sinn Inc., Cherry Hill, NJ, 08002

micrometer - Mitutoyo Mfg Co Ltd, Tokyo, Japan

micropipettes - microcaps, Drummond Scientific Co, Broomall, PA

microsyringe - Gilmont Instruments Inc, 401 Great Neck Road, Great Neck, NY 11021, (516) 487-0120; cat #S-1200, 2 ml capacity

Millipore filter - Millex-GS 0.22 um filter, Millipore Corp, Bedford, MA, 01730; serial #SLGS0250S

NaOCl - "Clorox", The Clorox Company, Oakland, CA, 94612

PE 160 & 90 tubing - Clay Adams, Division of Becton Dickinson & Co, Parsippany, NJ, 07054; reorder #7431 (PE160), #7421 (PE90)

periodontal curettes - Hu Friedy, 3232 N. Rockwell St., Chicago, Ill., 60618

potassium oxalate - Eastman Kodak Co., Rochester, NY, 14650

Saline - 0.9% sodium chloride, American McGraw, Division of American Hospital Suply Corp, Irvine, CA, 92714

Saw - Slow Speed Saw, Buehler, Isomet, 41 Waukegan Rd., Lake Bluff, Ill., 60044

timer - Cole Parmer Instrument Co, Chicago, IL, 60648

valve for nitrogen bottle - Victor Equipment Co., San Francisco, Calif.

vials for sample storage - 20 ml scintillation vials, Kimble, Division of Owens-Illinois, Toledo, Ohio, 40666

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