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EDITORIAL

Asepsis: A Plea to Manufacturers

The issue of asepsis and sterilization in the dental office has moved to the forefront of those facing the dental practitioner today. As concerns about hepatitis became more prevalent, we were compelled to think about changing some of our practices in the dental office, at least when treating patients who may have hepatitis. But it has been the rapid onset of the disease AIDS which has constrained the profession to take what many feel to be actions long overdue.

We are changing the way we clean and sterilize our instruments — at least that appears to be the trend in many offices. Until recent years, the average dentist has been unable to sterilize the dental handpiece; at best, most dentists have used only an alcohol wipe to clean it. Burs were frequently left in the handpiece from one patient to the next. Most handpieces are now autoclavable, yet many dentists are just now beginning to consider such a practice, in part because of the added cost of additional handpieces.

Dentists are now being forced by patients as well as by regulatory agencies to update the aseptic techniques employed in their offices, the type of sterilization used, and the protective barriers to be used, such as gloves and safety glasses.

Dentists have always called the treatment room an "operatory,"and yet in the past we have not treated it like it is one. In medical facilities, aseptic practices have in general been conducted at a much higher level than has been observed in dental treatment rooms. Gloves are worn, even for cleaning the simplest of wounds. Until now, dentists have rarely been known to wear gloves, even when doing periodontal or endodontic surgery, or for routine tooth removals.

Patients are not going to continue accepting dentistry's lackadaisical attitude toward these issues. Patients, when looking for a new dentist, frequently call ahead and ask if the doctor routinely uses gloves; and if not will go elsewhere. In dental school clinics, observant patients will watch faculty members move from chair to chair, and are quick to tell instructors they must change their gloves before touching them. Other patients inform the provider that gloves must be put on in their presence.

Whether we dentists like it or not, it is time to clean up our act. The question is: "How well can we do it?" Can we really do it well? I would suggest to you that we cannot, unless we can obtain unit-dose packaged materials. As an example of the problem, I will cite only two commonly used materials. These are Copalite varnish (H T Bosworth, Skokie, IL 60076) and the Prisma-Fil composite with its syringe delivery system (L D Caulk Co, Milford, DE 19963-0359). Copalite bottles are reopened with soiled hands or gloves, entered and re-entered with a cotton pliers. Even though studies have shown that the solution harbors few if any bacteria, there is no evidence that you cannot spread organisms with its use. With Prisma-Fil the same syringe is typically used on many patients: the compule of resin is inserted into one patient's tooth cavity, and then used on the next patient as well. At best, it might be wiped with alcohol. The dentist typically does not resterilize the compule syringe. There is too much expensive composite resin in the compule to throw away, so it is used over again. The manufacturer should be compelled to provide a smaller unit-dose so that the compule can be discarded after treating each patient. Copalite could also be packaged in a single unit-dose. Sometimes samples are provided in this manner, in a small plastic pouch which holds only a drop or two.

We will never solve the problems associated with asepsis as long as we continue to use drugs or materials which are used over and over again — unless we go to a unit-dose system. I have talked to several manufacturers about this. They say that there is lack of interest and it will raise the cost considerably.

Dentists use amalgam in a unit-dose system when they buy pre-encapsulated amalgam. Why not with other materials? A suggested list of materials would include cavity varnishes, liners and bases, composite resins for syringe delivery, and cements and other mixable filling materials.

Expensive, you say? Certainly! There can be no doubt that it would add significantly to the cost of providing dental care, but so do the use of gloves, clinic clothing, and other items related to sterilization and asepsis. The manufacturers are doing the profession a disservice by not considering the implementation of the unit-dose system for the products they market to us.

DAVID J BALES, DDS, MSD

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MICROLEAKAGE SYMPOSIUM

Microleakage of Restorative Materials

RICHARD E WALTON

Many of the cherished dogmas and historical concepts related to the biologic properties of materials are in doubt today. In recent years, serious questions have arisen as to whether materials or their by-products exert a direct action upon the dental pulp. Traditionally, the reaction of the dental pulp to the placement of various restorative materials has been attributed to the diffusion of chemicals from those materials passing through the dentinal tubules and coming in direct contact with the connective tissue of the pulp. Presumably, these chemicals, which have shown to be toxic to cells in studies in vitro (Autian, 1974; Tronstad, Hasselgren & Wennberg, 1977; Spangberg & others, 1973), were held directly responsible for the resultant inflammatory or other degenerative changes in the pulp (Zander, 1946; Lefkowitz, Seelig & Zachinsky, 1949; Langeland & others, 1966; Stanley, Swerdlow & Buonocore, 1967).

Is this concept of direct chemical action on the dental pulp really true? Are the chemical components of the materials important, or are we observing a phenomenon that may result from factors other than ingredients of the materials? The fact that the pulp may respond adversely to materials that are irritating does not prove a cause-and-effect relationship. In fact, the pulp may react from some other cause. One of the possible etiologies may be failure of the material to seal at its interface with the tooth structure, allowing microleakage of other irritants from the oral cavity between the restoration and the cavity interface (Brännström & Nyborg, 1971; Skogedal & Eriksen, 1976; Bergenholtz & others, 1982; Browne & others, 1983).

If microleakage is indeed the culprit, with what aspect of microleakage should we be concerned? Initially, bacteria and/or bacterial toxins were considered the key (Brännström & Nyborg, 1973). However, it could be speculated that bacterial contamination beneath a restoration may only indicate gross microleakage while other chemicals or substances that originate in the oral cavity, such as salivary by-products or food chemicals, may also be responsible for the phenomenon of pulp changes under leaking restorations. How can we identify these potential irritants, and what is their importance?

EXPERIMENTAL TECHNIQUES

What methods are available which will demonstrate the presence and degree of microleakage? The numerous methodologies employed have shown limitations by using techniques that depend on the material, on the situation, on a specific purpose, or

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on the technology available at the institution or in that laboratory. Obviously, different approaches may give different results. It would be most helpful to standardize the experimental methods for examining microleakage.

It is important to stress that we should not imply a cause-and-effect relationship between what the technique demonstrates and the response of the underlying pulp and/or dentin.

PROBLEMS OF MICROLEAKAGE

Although the focus of studies in microleakage has been on its impact upon the pulp and subsequent reactions, there are other important considerations. Microleakage of various substances in and around restorations and tooth structure may affect the property of the materials, result in recurrent caries, or cause hypersensitivity of exposed dentin. Many of these may be as, or more, important than the biological effects on the pulp alone. Obviously, the longevity or serviceability of the restoration may also be compromised.

BIOLOGICAL CONSIDERATIONS

Assuming that microleakage is, in effect, exposing dentin to the oral cavity, what will be the results? What should be further considered? It is possible that there may be a combination effect from chemicals released by the material as well as from the substances associated with microleakage. In a biological sense, nothing is simple; there usually are combinations of factors that elicit tissue responses.

PREVENTIVE MEASURES

Assuming that microleakage around restorative materials has clinical significance (Bergenholtz & others, 1982), how might the microleakage be prevented? What techniques are available? What preventive measures have been tested? Of course, absolute adhesion between the material and tooth structure in all areas and at all interfaces would be the most effective. Is this indeed possible in both the long and short term?

One approach would be to place a secondary material to occupy the interface, such as some type of liner or varnish. Is this feasible and available? Another alternative would be to seal the surface of both the restoration and tooth structure with a single material applied after the restoration is placed. Or, if microleakage is not totally preventable, are there base or lining materials available to place in the cavity to prevent passage of irritants into or through the tubules? In addition to being dentin sealers, these substances may be antibacterial and prevent colonization or proliferation of microbes on the cavity floor.

The five papers, immediately following, will provide useful data concerning the questions put forth in this paper.

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Are Adhesive Techniques Sufficient to Prevent Microleakage?

D HUGO RETIEF

INTRODUCTION

When considering the topic "Are Adhesive Techniques Sufficient to Prevent Microleakage?", the first question we should answer is: How do we define adhesion? According to the American Society for Testing and Materials (1964), adhesion, as a phenomenon, is defined as the state in which two surfaces are held together by interfacial forces, which may consist of chemical forces (primary or secondary valence) or interlocking forces (mechanical), or both, and an adhesive, as a material, is defined as a substance capable of holding materials together by surface attachment. The molecular forces of attraction are classified as chemical (or primary) and physical (or secondary) attractive forces. The chemical attractive forces arise as a result of electrostatic or ionic bonds, and polar and nonpolar covalent bonds. The physical attractive forces are derived from the Van der Waals forces which include the Keesom, Debye, and London forces, and from hydrogen bonds. Adhesion, therefore, may result from mechanical bonding and/or adhesive bonding where physicochemical interaction takes place between the adhesive and the substrate.

MECHANICAL BONDING TO ENAMEL

Mechanical bonding between dental resins and etched enamel is achieved by means of the acid-etch technique. This concept was first introduced by the late Michael Buonocore (1955). He demonstrated that the adhesion of acrylic restorative resins to enamel could be achieved by etching the enamel surface with orthophosphoric acid (H_3PO_4). I will briefly discuss the effects of H_3PO_4 etching on the surface morphology, the resin/etched enamel interface, and the wettability of enamel surfaces.

Changes in the surface morphology of enamel surfaces etched with H_3PO_4 can readily be studied by scanning electron microscopy (SEM). An unetched enamel surface has a featureless morphology (Fig 1).

FIG 1. *Unetched enamel surface. X700 (original magnification X2000)*

All photos are scanning electron micrographs.

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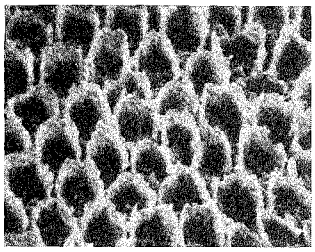
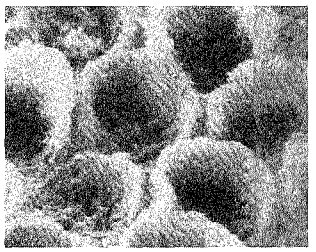
Etching the enamel surface with H_3PO_4 produces three types of etching patterns (Silverstone & others, 1975). In the Type 1 etching pattern, preferential dissolution of the prism cores occurs (Fig 2), which results in a typical "honeycomb" appearance (Fig 3).





FIGS 2 & 3. *Type 1 etching pattern. Figure 2 at X1050 (original X3000). Figure 3 shows honeycomb appearance. X2450 (original X7000)*

In the Type 2 etching pattern, preferential dissolution of the prism peripheries occurs, resulting in a typical "cobblestone" appearance (Fig 4). Type 1 and

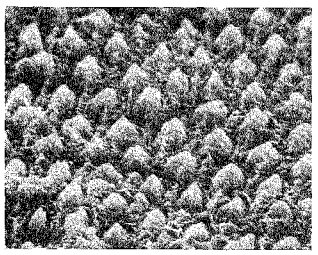
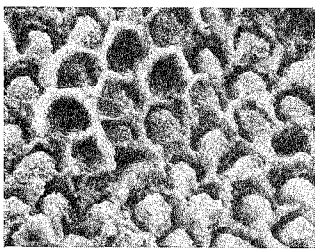
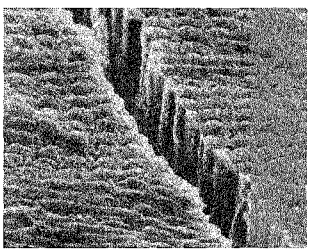


FIG 4. *Type 2 etching pattern. X700 (original X2000)*

Type 2 etching patterns are often obtained in adjacent areas of the same tooth (Fig 5). Type 1 and Type 2 etching patterns occur on enamel surfaces in which the prisms extend to the enamel surface (Fig 6). In the Type 3 etching pattern, surface loss occurs





FIGS 5 & 6. *Type 1 and Type 2 etching patterns. Fig 5: adjacent areas, same tooth. X875 (original X2500). Fig 6: on enamel surfaces in which the enamel prisms reach the surface. X700 (original X2000)*

without exposing the underlying prisms (Fig 7). Gwinnett (1973) demonstrated that this etching pattern is usually observed at the cervical aspects of teeth where the enamel prisms do not extend to the surface. The etching pattern is also dependent on the orientation of the enamel prisms. The etching

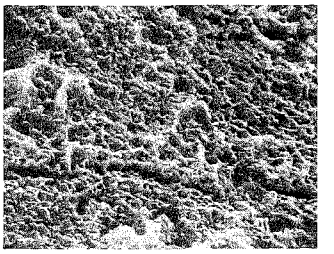
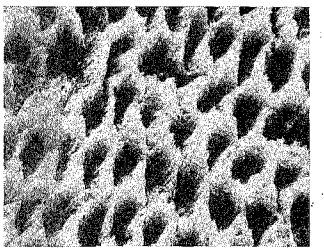
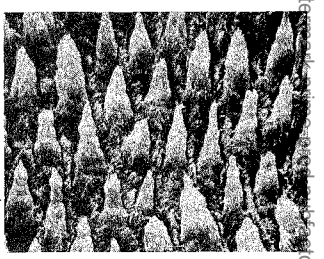


FIG 7. *Type 3 etching pattern. X875 (original X2500)*

effect is more severe on enamel prisms oriented perpendicular to acid attack (Fig 8) than on enamel prisms oriented parallel to acid attack (Fig 9).





FIGS 8 & 9: *Etching of enamel prisms oriented perpendicular to acid attack in Fig 8 at left; parallel to acid attack in Fig 9. X1050 (original X3000)*

The resin/etched enamel interface can be exposed for examination in the SEM by placing a resin on an etched enamel surface, preparing a cross section through the resin/enamel interface, and immersing the sectioned tooth in a weak hydrochloric acid solution. The resin tags which extended into the etched enamel surface are clearly visible (Fig 10).

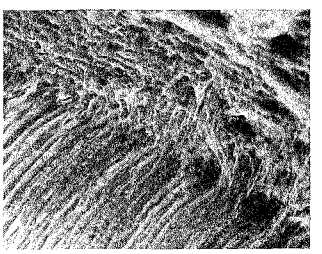


FIG 10. *Resin tags extending into etched enamel. X350 (original X1000)*

The effect of phosphoric acid etching on the wettability of the enamel surface can be evaluated by determining the contact angle of a dental resin on enamel surfaces. When a liquid drop is placed on a solid substrate, the surface of the liquid will, at equilibrium, exhibit a definite angle with the surface of the substrate. This angle is called the contact angle and it is a useful inverse measure of the wettability of the substrate by the resin. The cosine of the contact angle is a direct measure of wettability. The contact angle of Concise Enamel Bond (3M Co, St Paul, MN 55144) on unetched enamel is $20.2 \pm 6.0°$ and on enamel surfaces etched with H_3PO_4, $7.2 \pm 1.0°$.

In summary, etching of enamel surfaces produces:

- Increase in surface area available for bonding
- Microspaces in the enamel resulting from the preferential etching patterns
- Increased wettability of the etched enamel surface which facilitates the penetration of the curing resin into the microspaces
- Resin tags extending into the etched enamel, bonding the cured resin mechanically to enamel

It is believed that physicochemical interaction between the resin and etched enamel plays a small part in the bonding of dental resins to etched enamel, and that the main mechanism is that of mechanical bonding (Retief, 1978).

Numerous publications deal with microleakage at the dental resin/etched enamel interface. In one study the effect of cavosurface treatment on marginal leakage in class 5 composite resin restorations was evaluated by Retief, Woods, and Jamison (1982). Sixty class 5 preparations were made on the facial surfaces of extracted human maxillary canine teeth, thirty butt preparations with 90-degree cavosurface angles, and 30 beveled preparations with 135-degree cavosurface angles. In each group the enamel margins of the preparations were not etched, etched with H_3PO_4 for 60 seconds, or a low-viscosity bonding resin was applied to the etched enamel surface prior to restoration with Adaptic (Johnson & Johnson Dental Products, East Windsor, NJ 08520). The filled teeth were stored in water at 37 °C and final finishing was done seven days after restoration. The teeth were then subjected to 100 cyclic temperature changes between 4 °C and 55 °C in Blak-Ray fluorescent dye (Ultra-Violet Products Inc, San Gabriel, CA 91778) with a dwell time of one minute in each bath. The teeth were embedded in epoxy resin (Miller-Stephenson Chemical Co, Inc, Danbury, CT 06810) and at least three faciolingual sections prepared from each tooth. Microleakage was evaluated at both the incisal and cervical aspects of the restorations.

The following results were obtained. Microleakage at the cervical aspects of the restorations was significantly greater than at the incisal aspects. At the cervical aspects of the restorations placed in butt preparations, microleakage was not significantly different ($P = 0.10$) but significantly different microleakage was observed at the incisal aspects ($P = 0.02$). Similar results were obtained in the restorations placed in bevel preparations with P-values of 0.14 and 0.001, respectively.

It was concluded that acid etching of the enamel walls of class 5 butt and bevel preparations effectively reduced microleakage at the incisal aspects but not at the cervical margins of the restorations. In a subsequent publication it was shown that microleakage at the cervical aspects of such restorations could be significantly reduced by extending the composite resin over the etched margins of the preparations (Gross, Retief & Bradley, 1984).

ADHESIVE BONDING TO DENTIN

Although acid etching of dentin opens the dentinal tubules which leads to extensive resin penetration into the opened tubules, the bond strengths of dental resins to etched dentin is negligible (Asmussen, 1985). The application of H_3PO_4 to freshly cut dentin is not recommended because severe pulpal responses may be elicited (Retief, Austin & Fatti, 1974; Stanley, Going & Chauncey, 1975).

Adhesive dental restorative materials which bond to tooth structure by physical and/or chemical interaction have recently become available to the dental profession. These are the glass-ionomer cements and the dentin bonding agents.

Glass-ionomer Cements

The glass-ionomer cements were developed by Wilson and Kent (1972) in the Laboratory of the Government Chemist in London. The adhesive mechanism of these cements has been described (McLean & Wilson, 1977). The microleakage of glass-ionomer cements has been evaluated in several studies and I wish to refer to the publication by Alperstein, Graver, and Herold (1983). Class 5 cavities were prepared on extracted human anterior teeth and the teeth stored in a moist chamber. Final finishing of the restorations was done after 24 hours and the teeth placed in a 20% solution of a fluorescent dye for one hour. The teeth were then sectioned faciolingually and the sections viewed by fluorescent light microscopy. Microleakage was scored from 0 (no leakage) to 4 (most severe leakage) and the results are presented in Table 1. Note that all the glass-ionomer cement restorations showed some leakage and that the treatment of the cavity walls with 50% citric acid before insertion of the restorations did not reduce marginal leakage.

Table 1. Microleakage of Glass-ionomer Cement Restorations

Restorative Material and Procedure	Degree of Marginal Leakage					
	n	0	1	2	3	4
Glass-ionomer cement — ASPA Cavity walls treated with 50% citric acid	20	0	8	12	0	0
Glass-ionomer cement — ASPA Cavity walls untreated	22	0	9	10	3	0

Drs Thornton, Bradley and I have recently completed a study evaluating the microleakage of two glass-ionomer cements, Ketac-Fil and Ketac-Silver (ESPE, Seefeld/Obergay, West Germany) (Thornton, Retief & Bradley, 1986). Sixty class 5 preparations were made at the cementoenamel junction of extracted human permanent premolar teeth. Durelon liquid was applied to the cut dentin surfaces for five seconds and then thoroughly rinsed with water for 60 seconds. The Ketac-Fil and Ketac-Silver were mixed in a Vari-Mix II amalgamator (L D Caulk Company, Milford, DE 19963) for 10 seconds and transferred to the preparations. A matrix was firmly held over the restorations for two minutes and Visiobond (ESPE, Seefeld/ Obergay, West Germany) was applied around the margins of the restorations. The matrix was removed after five minutes, the excess cement removed with hand instruments, and Visiobond applied over the restorations. Final finishing was done after 10 minutes and Visiobond again applied over the restorations. The restored teeth were stored in physiological saline at 37 °C for 24 hours. The apices of the teeth were cut off and sealed with Copalite varnish and amalgam. Two coats of nail varnish were applied to the tooth surfaces leaving a 1 mm space adjacent to the restorations free of varnish. The teeth were exposed to 100 or 200 cyclic temperature changes between 5 °C ± 1 °C and 55 °C ± 1 °C in a 1% methylene blue solution in a temperature cycling machine (Transtemp, W B O'Neal, Birmingham, AL 35223) with a dwell time of one minute in each bath. The teeth were embedded in epoxy resin and four mesial sections cut through the restorations with an Isomet low-speed saw (Buehler Ltd, Evanston, IL 60204). The sections were viewed microscopically and microleakage was evaluated at both the enamel and cementum margins of the restorations. Microleakage extending to the floor of the restorations was observed at both aspects of all the restorations.

Dentin Bonding Agents

Several commercial dentin adhesive bonding agents have recently become available and several others are in the experimental stage of development. Basically the dentin bonding agents are bifunctional molecules with a methacrylate group which bonds to the restorative resin by chemical interaction and a functional group which bonds to either the inorganic or organic constituents of dentin (Asmussen, 1985).

In a recent study, the microleakage of posterior composite resin restorations used in conjunction with a dentin bonding agent, Scotchbond (3M Company, St Paul, MN 55144), was evaluated (Gross, Retief & Bradley, 1985). Scotchbond contains a bis-GMA monomer in which the hydroxyl group is esterified with chlorophosphoric acid. Sixty class 2 preparations extending 1 mm above and 1 mm below the cementoenamel junction were made in extracted human mandibular molar teeth. Four restorative procedures were used, as shown in Table 2.

1. Acid-etch enamel, enamel bond on enamel only, restored with P-10 (3M Co)
2. Acid-etch enamel, Scotchbond on enamel and dentin, restored with P-10
3. Acid-etch enamel, Scotchbond on enamel and dentin, restored with P-30 (3M Co)
4. Acid-etch enamel, citric acid applied to dentin surfaces, Scotchbond on enamel and dentin, restored with P-30

The teeth were stored in water at 37 °C 15 minutes after restoration, and final finishing was done after 24 hours. The teeth were subjected to 100 temperature cycles between 5 °C ± 1 °C and 55 °C ± 1 °C in a ^{45}Ca solution with a dwell time of one minute in each bath. The teeth were sectioned mesiodistally and buccolingually and autoradiographs prepared. Microleakage was evaluated both above and below the cementoenamel junction.

Table 2. Microleakage of Posterior Composite Resin Restorations (n = 10)

Procedure with Acid-etch Enamel	Restored with	Leakage per Number of Specimens* (1 mm above or below CE junction) Gingival Floor	Axial Wall	Pulpal Floor
Enamel bond on enamel only	P-10	7 (above) 1 (below)	0 6	3 3
Scotchbond on enamel and dentin	P-10	8 (above) 6 (below)	1 3	1 1
Scotchbond on enamel and dentin	P-30	6 (above) 4 (below)	0 1	4 5
Citric acid applied to dentin surfaces; Scotchbond on enamel and dentin	P-30	9 (above) 4 (below)	1 6	0 0

*All specimens demonstrated leakage.

The results are presented in Table 2. Microleakage occurred both above and below the cementoenamel junction in all of the restorations prepared with the four restorative procedures. Microleakage occurring 1 mm above the CE junction was not as severe as at 1 mm below it. Microleakage at the occlusal aspects of the restorations was effectively inhibited by Scotchbond.

There is a paucity of information in the literature on the microleakage of other dentin bonding agents, but a recently published study could have a direct effect on microleakage (Munksgaard, Hansen & Asmussen, 1984). After extraction, the teeth were cleaned and stored in physiological saline for a maximum period of five weeks. The surfaces of the roots were ground flat and cavities with 90-degree cavosurface angles prepared in dentin. The cavity depth was 1.5 mm and the diameter ranged from 1.8 mm to 6.5 mm. The five dentin bonding agents were applied as instructed by the manufacturers and the cavities were restored with a visible-light-cured microfill composite resin, Silux (3M Co). The teeth were stored in a physiological saline solution and ground wet to remove 0.1 mm of the dentin and the restorations. The ground surfaces were micropolished and the largest gap along the periphery measured. The restoration diameters in millimeters were plotted against the maximal contraction gaps. The results obtained in restorations with a 4 mm diameter are presented in Table 3. The smallest con-

Table 3. Contraction Gaps of Silux Restorations Utilizing Different Bonding Agents

Dentin Bonding System		Contraction Gap (4 mm diameter) % of diameter	μm
Untreated (control)	*	0.38 ± 0.021	15.2
Clearfil		0.38 ± 0.021	15.2
Scotchbond		0.24 ± 0.015	6.6
Bowen System		0.19 ± 0.014	7.6
Superbond		0.08 ± 0.008	0.32
GLUMA		0.05 ± 0.008	0.20

*Not significantly different

traction gaps were found in restorations with which the GLUMA dentin bonding system (Bayer, Leverkusen, West Germany) was used. The bonding agent consists of an aqueous solution of glutaraldehyde and HEMA (hydroxyethylmethacrylate). Bonding occurs via chemical interaction with the collagen of dentin (Asmussen, 1985).

Munksgaard and his co-workers (1984) found that for a given adhesive the contraction gap increased with the diameter of the cavity, and that the main determinant of the size of the contraction gaps was the ratio of the volume of the restoration to the area of the walls of the cavity — the higher the ratio the larger the contraction gap. These findings are of great clinical significance. Asmussen (1985, pp 71-72) recently suggested that: "After excavation of a carious lesion on a root, one should not prepare it further if the retention of the restorative resin can be ensured with an effective adhesive."

CONCLUSIONS

The following general conclusions are made:

1. Acid etching of enamel effectively eliminates microleakage of composite restorative resin restorations provided that sufficient enamel is present, particularly at the gingival aspects of restorations.
2. Microleakage can be expected to occur around glass-ionomer cement restorations.
3. Although some of the newer dentin bonding agents have the potential to prevent microleakage, further studies are required to establish this.

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Biocompatibility of Dental Materials in the Absence of Bacterial Infection

CHARLES F COX

INTRODUCTION

For several decades, the enigma between testing dental materials in vitro and in vivo has created a perplexing consideration within the dental research community as well as within the literature. Many tests in vitro (tissue culture) have shown that various dental materials, such as silicate cements and composite resins, are nontoxic to cultured cells while zinc oxide and eugenol (ZOE) and IRM are toxic to the same cell culture lines. On the other hand, tests done in vivo have shown that silicate cements, zinc phosphate, and various composite resins are toxic to the pulp, and that ZOE and IRM are nonirritating when tested according to the specifications of the ADA, FDI, and ISO. If ZOE were introduced to the dental profession as a new restorative material today, would it pass the initial tests in vitro before studies in vivo were pursued? According to today's criteria for testing in vitro, it would not. This presentation has been developed to provide insight into this enigma and point to the real biological issues.

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CONSIDERATION OF PULP INFLAMMATION

Two main hypotheses have been presented to explain pulp inflammation in usage tests: material toxicity and bacterial infection.

Material Toxicity

Pulp inflammation as a direct consequence of chemical toxicity, such as material pH, osmotic balance, unset resin monomers, or other chemical agents when placed directly on either dentin and/or pulp tissues, has been reported in several studies (Zander, 1946; Zander & Pejko, 1947; Schroff, 1947; Lefkowitz, Seelig & Zachinsky, 1949; Mitchell, Buonocore & Shazer, 1962; Bowen, 1976). The chemical toxicity hypothesis has been strongly reflected in the development of the various international schemes for testing in vivo. The usage tests require placement of silicate cements, thought to cause inflammation due to their pH constituents, into a test cavity as the control irritant.

Bacterial Infection

The hypothesis that inflammation of the pulp underneath various restorations is the result of bacterial infection is supported by data from a number of studies. An early report by Crowell (1927) speculated that bacterial infection underneath silicate was responsible for pulp inflammation; and a number of more recent studies (Brännström & Nyborg, 1971; Brännström & Nordenvall, 1978; Qvist, 1980; Bergenholtz & others, 1982; Brännström, 1982, 1984; Cox & others, 1985) have demonstrated that the

presence of bacteria within the restoration gap and adjacent dentin is the major factor leading to pulp inflammation and eventual necrosis.

ISSUES IN TESTING FOR BIOCOMPATIBILITY

The real issue of biocompatibility of dental materials seems to be divided between material toxicity versus bacterial infection. The literature is replete with many studies which have considered these two issues (Manley, 1942; Langeland & others, 1966; Swerdlow & Stanley, 1962; Stanley, Swerdlow & Buonocore, 1967; Stanley & others, 1969; Heldridge & Jensen, 1966; Sayegh & Reed, 1969; Heys & others, 1979; Mjör & others, 1977; Brännström, 1984; Skogedal & Eriksen, 1976; Browne & others, 1983). Since the late 1970s, well-controlled studies of pulp biology have supported the hypothesis of bacterial infection, as first demonstrated in the studies of Brännström and his colleagues. However, it is interesting to note that, among the many considered factors within biocompatibility testing, bacterial infection is not strongly reflected in most current schemes in dental materials testing.

ROLE OF BACTERIAL INFECTION IN PULP INFLAMMATION

Convincing evidence for the role of bacteria and pulpal inflammation under restorations was presented by Brännström and Nyborg in 1971. Silicate cement and composite resin were surface-sealed with ZOE to prevent oral bacteria from entering the restoration/dentin interface and the reactions of the pulp tissue were none to minimal. Their unsealed silicate controls invariably showed stained bacteria on the test cavity walls with subjacent pulp inflammation ranging from moderate to severe. More recent studies combining microbiologic and histopathologic factors (Qvist, 1980; Bergenholtz & others, 1982) have shown that cultures of bacteria recovered from beneath cavities were directly associated with pulp inflammation and necrosis. The ZOE test cavities showed no bacterial growth and the underlying pulp tissue was normal with no inflammation.

RECENT BIOCOMPATIBILITY DATA

Several factors precipitated the development of our first biocompatibility study. First, we realized the potential to provide a bacterially tight seal with ZOE following the model of Brännström (1982) and Bergenholtz and others (1982). Secondly, discussions with Dr P L Fan of the ADA in 1984 gave us additional incentive to study the enigma between testing data in vivo and in vitro. Our study was developed to observe the biocompatibility of various dental materials in vivo when mechanically exposed pulps were capped with a spectrum of so-called toxic materials. Half of the mechanical pulp exposures were capped with one of the selected materials. The other half of the teeth were capped and the outer portion of the cavity was surface-sealed to the cavosurface margin with ZOE (Fig 1) to exclude microleakage of oral

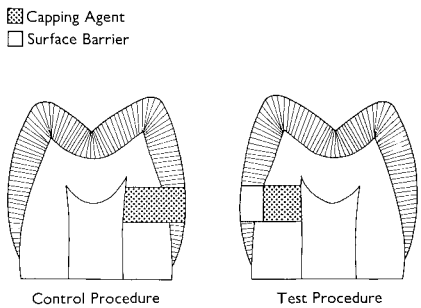

FIG 1. *A diagram showing the experimental sealing model. Direct capping with the various materials is seen on the left and surface sealing of the materials with ZOE is seen on the right.*

bacteria. The pulp responses were assessed at seven days and 21 days. For the control phase, the mechanically exposed pulps were direct-capped to the cavosurface margin with a ZOE surface seal. These unsealed restorations showed pulp inflammation ranging from moderate to severe. Exposed pulps that were direct-capped with ZOE at seven days showed soft tissue healing with migration of pulpoblasts to the interface (Fig 2). All 21-day pulps

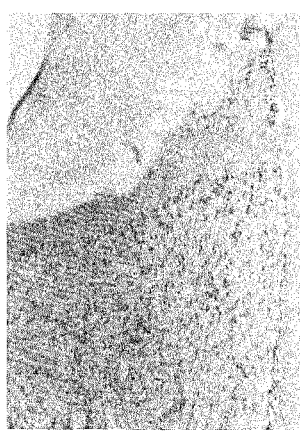

FIG 2. *Direct capping with a hand mix of ZOE after seven days reveals a thin eosinophilic zone and subjacent soft tissue healing. No inflammation is present in the deeper pulp tissue.*

without a ZOE surface seal showed the pulps to be inflamed to necrotic in varying degrees. Pulps that were capped with the various materials and then surface-sealed with ZOE showed little to no inflammation, migration of the pulpoblasts to the medicament interface with formation of a new dentin matrix, and little to no pulp inflammation.

Results of the data from this study were found to be focused upon two important issues. First, that mechanically exposed pulps present an inherent capacity to heal soft tissue when they are surface-sealed to prevent bacterial infection. Secondly, our serendipitous observation was even more provocative, showing new dentin matrix directly adjacent to the interface of zinc phosphate cement, silicate cement, and test materials of light-cured composite resin when surface-sealed against microleakage.

As a result of this data (Keall & others, 1985; Cox & others, 1987), we undertook a second study to gain additional information employing the surface-seal model. For verification of our experimental model, we followed the sequential soft-tissue healing and compared it to the healing pattern as reported against a hard-set calcium hydroxide medicament (Fitzgerald, 1979; Cox & Bergenholtz, 1986). We wanted to observe if new dentin matrix would form against ZOE-sealed amalgam when observed at longer time intervals. Preliminary data had shown that connective tissue-healing patterns of the pulp follow the same scheme as had been demonstrated for hard-set $Ca(OH)_2$ pulp-capping agents (Cox & Bergenholtz, 1986). Pulps that had been mechanically exposed and had been capped with a light-cured composite without a ZOE seal for 10 days presented a necrotic pulp with a dense staining infiltrate of bacteria throughout the pulp chamber (Fig 3).

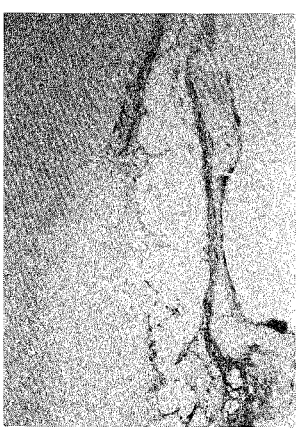

FIG 3. *Direct-capped pulp with light-cured composite and no ZOE seal after 10 days. The necrotic pulp is stained for bacteria which are seen as small dark profiles throughout the tissues.*

The same was observed in mechanically exposed pulps that were capped and filled to the cavosurface with silicate, showing necrotic pulps (Fig 4) with a dense staining bacterial infiltrate in adjacent serial sections (Fig 5). Mechanically exposed pulps that were capped with amalgam and surface-sealed with ZOE showed connective tissue healing at seven days (Fig 6) as well as at five weeks; however, no new dentin matrix formation was seen. Pulp healing as well as new dentin formation was shown directly

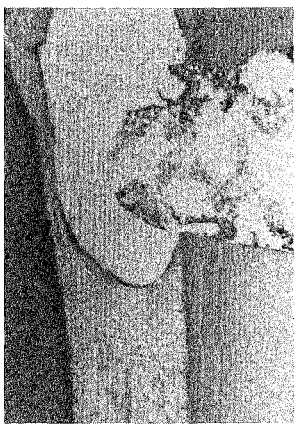

FIG 4. *A necrotic pulp that was direct capped with silicate for 21 days. No ZOE seal was placed.*

FIG 5. *A high magnification from an adjacent tissue section of the tooth described in Figure 4. The internal dentin wall is seen on the right with large foci of dark-staining bacteria lying within the silicate.*

adjacent to the material interface after 14 days using the ZOE surface-sealed silicate, zinc phosphate, and an experimental light-cured $Ca(OH)_2$ material (Figs 7, 8, 9).

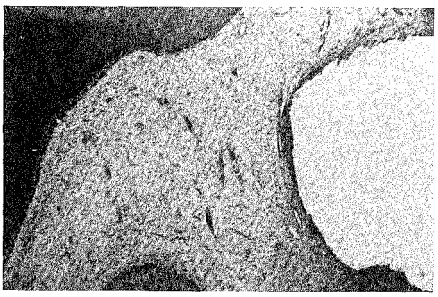

FIG 6. *Amalgam direct-capped pulp after seven days. A surface seal of ZOE was present to the cavosurface margin. The soft tissues of the pulp have migrated to the amalgam interface (clear area) on the right center field.*

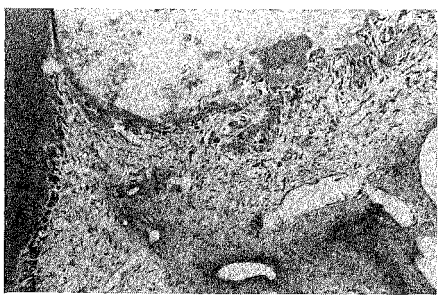

FIG 7. *A silicate-capped pulp after 14 days. A ZOE surface seal was present to the cavosurface margin. Note the silicate in the upper center with deposition of a new dentin matrix at the material interface. Healing is present with vessels and only mild inflammation.*

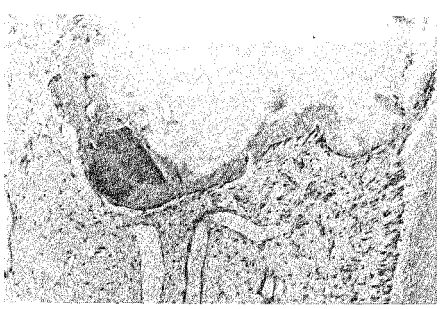

FIG 8. *A zinc phosphate-capped pulp after 14 days. A ZOE surface seal was present to the cavosurface margin. New dentin matrix is present at the medicament interface with healed pulp tissue below.*

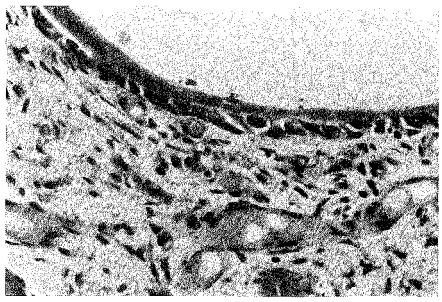

FIG 9. *A light-cured $Ca(OH)_2$-capped pulp after 14 days. A ZOE surface seal was placed at the cavosurface margin. The material is seen in the upper center with a new dentin matrix, odontoblastic zone, and healed pulp tissue below.*

These data serve to reinforce the previous biocompatibility study (Cox & others, 1987), indicating that healing progresses at the same cellular level for all materials tested in the absence of bacterial infection. In addition, new dentin matrix formation occurs when bacterial infection is eliminated with a ZOE surface seal for the five-week testing interval.

THE ISSUES OF PULP HEALING

This report has focused upon a controversial aspect of pulp healing and new dentin matrix formation. Recent data (Cox & others, 1987) demonstrate that restorative materials previously reported as "toxic"do not cause pulp inflammation or necrosis when placed directly on exposed pulps and surfaces sealed from bacterial infection. Generally, two distinct types of pulp tissue reactions were seen. First, the pulps either displayed severe inflammation and necrosis, or the pulp tissue presented reorganization and healing with dentin matrix repair. Second, neither calcium nor hydroxyl ions, nor a specific material pH, appears directly responsible for initiating pulp healing and hard tissue (dentin) formation. The latter was frequently seen when the outer portion of the restoration had been covered with ZOE, whereas pulp tissue inflammation and necrosis were associated with non-ZOE-sealed restorations. These patterns were observed irrespective of the type of capping material employed. Thus, pulp healing and cell repair occurred not only with the hard-set $Ca(OH)_2$ but also with amalgam, composite, zinc phosphate cement, and silicate cement. These findings contradict a large number of previous studies reporting that silicate and zinc phosphate cements and composites are particularly injurious to the pulp by virtue of their acid content (Zander, 1946; Zander

& Pejko, 1947; Schroff, 1947; Mitchell & others, 1962; Lefkowitz & others, 1949; Langeland & others, 1966; Stanley & others, 1967; Heldridge & Jensen, 1966; Sayegh & Reed, 1969).

CELL TOXICITY FROM EUGENOL

Placement of hand-mixed ZOE on exposed pulps at seven days presented a spectrum of pulp inflammation ranging from none to severe. The 21-day ZOE-capped exposures showed chronic inflammation, paralleling studies (Watts & Paterson, 1977; Watts & others, 1985) that demonstrated calcific bridge formation in 27 of 28 ZOE-capped exposed pulps in germ-free rats. However, after 28 days inflammation and necrosis was observed. These reports suggest why the literature has reported lack of repair and chronic inflammation with ZOE capping in vivo. The same toxicity mechanisms in vitro are seen in usage tests in vivo where free eugenol is available to pulp cells, interfering with respiration and healing, thus resulting in sterile necrosis. Hume (1984) analyzed the diffusion of eugenol through dentin from a hand mix of ZOE. He reported that the concentration of eugenol was highest adjacent to the medicament and less at the dentin-pulp surface. Dentin acts as a buffer, reducing eugenol concentrations at the pulpal interface to nontoxic levels. However, when pulps are directly capped with ZOE without a dentin buffer, eugenol concentrations remain high over longer time intervals, resulting in depression of cellular respiration.

PRESSURE FROM INSERTION OF MATERIAL

Our observations regarding placement of amalgam directly on exposed pulps are at variance with those of Möller (1979). With amalgam-capped ZOE-sealed restorations, we only saw one of eight pulps with a mild inflammatory response at seven and 21 days, whereas Möller reported moderate to severe pulp inflammation at seven and 30 days. He concluded that chemical irritation, cavity depth, and silver amalgam plasticity (insertion pressure) are major factors in pulp inflammation and that clinical microleakage of bacterial contaminants is not. The disparity between the data may simply be a function of comparing two different alloy systems. In addition, Möller's procedure of rubbing calcium hydroxide onto the cavity walls probably reduced the accessibility of bacteria and filtrates to the pulp. This reduction in dentin permeability is explained by a study of Bergenholtz and Reit (1980). Although we saw no dentin bridging against amalgam capping, we observed soft tissue reorganization and cell migration directly adjacent to the amalgam interface in ZOE-sealed restorations at all time periods.

FACTORS PROMOTING NEW DENTIN MATRIX FORMATION

Of equal consideration are stimulatory factors thought to promote pulp healing and initiate new dentin matrix formation. Torneck, Moe, and Howley (1983) speculated that calcium ions stimulated cell proliferation whereas Hanks, Bergenholtz, and Kim (1983) reported that certain $Ca(OH)_2$ cements caused depression of cell synthesis. Studies in vivo by Schröder and Granath (1971) reported that a slurry of $Ca(OH)_2$ promoted tissue destruction and necrosis. However, other studies (Heys & others, 1980, 1981; Cox & others, 1982, 1986, 1987) have reported favorable pulp healing and new hard-tissue formation against hard-set $Ca(OH)_2$ materials. A recent review by Schröder (1985) suggested biologic variation as the reason for the success or failure of calcium hydroxide containing compounds to stimulate bridging. Her speculation ranged from availability of either the calcium or hydroxyl ions to the formation of a calcium carbonate interface at the cell healing zone.

A report by Gresham (1945) stated that release of low pH substances from silicate cements was responsible for pulp inflammation and eventual necrosis. Recent studies (Phaneuf, Frankl & Ruben, 1968; Shubich & others, 1978; Heys & others, 1980) have speculated that the pH of the medicament is the primary factor in pulp healing and bridging. Our recent data agrees with that of Kozlov and Massler (1960) and Yamamura (1985), who have indicated that pH is not the major factor in stimulating new dentin matrix formation.

Cox and others (1987) reported that a thick hand mix of ZOE effectively seals cavities against bacterial infection, thereby allowing pulp tissue healing and new dentin matrix formation. These observations are in agreement with other investigators (Kozlov & Massler, 1960; Cox & others, 1982, 1985, 1986; Watts & others, 1985; Yamamura, 1985) who have reported that exposed pulps will heal when capped with a variety of materials. Regarding material toxicity, chronic inflammation was seen only when ZOE was placed directly onto exposed pulps and maintained for 21 days. This was due to the suppression of cell physiology by the free eugenol in the ZOE. All other inflamed unsealed restorations were associated only with bacterial infection. Our data supports the many studies of Brännström and Nyborg (1971), Brännström (1984), and Bergenholtz and others (1982), who have shown that the presence of oral bacteria is directly related to pulp inflammation.

CONCLUSIONS

What appears to be the reason for disparity between testing in vivo and testing in vitro? A careful consideration of the literature reveals that the current criteria for testing in vitro do not consider factors of bacterial infection and dentin buffering. What are the implications for current testing in vitro? The sponsoring testing organizations must now re-evaluate current criteria for testing in vitro and consider changes for incorporating the factor of bacterial infection in vivo.

A modified dentin filtration test in vitro (Hanks & others, 1987) has the potential to provide evaluation of the bacterial component in concert with cell and material toxicity. Such tests in vitro must be set up to enable evaluation of the bacterial infection component before expensive tests in vivo are proposed and implemented. In order for this to come to fruition, current international standards and criteria for testing in vivo must be modified to reflect the factor of bacterial infection in relation to material toxicity.

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When is Microleakage a Real Clinical Problem?

H TRIADAN

INTRODUCTION

The formation of fissures between filling and tooth is generally considered a serious deficiency in restorations. If such defects extend to the dentin, they become the main cause of irritation of the pulp (Brännström, 1984). This paper is concerned with composite resin systems using the principles of acid-etch and adhesive techniques.

Ever since the first descriptions of the acid-etch restorative technique by Buonocore (1955) and Buonocore, Wileman, and Brudevold (1956), the problem of establishing a durable bond between the hard substances of the tooth, especially the enamel, and the resin filling has been repeatedly discussed. Hundreds of publications, the majority of them of a biotechnical nature, have evaluated the type of bond produced, the factors which could contribute to bond failure, and the consequences of such failures.

The enamel-composite bond may be purely mechanical (macro or micro), chemical, or a combination of the two, mechanical/chemical. Today the macro/micromechanical anchoring of the composite resin to enamel is for the most part considered to be the prime factor in the bonding process.

The purpose of this paper is to address the clinical problems associated with microleakage, their causes, and perhaps some solutions.

Factors which can affect the original bond between enamel and resin are: (1) mechanical forces, especially shear forces; (2) temperature fluctuations (thermocycling); (3) the biological decomposition of the composite's margin through the action of saliva and/or plaque constituents (bacteria).

The partial loss of the bond between tooth and composite does not necessarily lead to adverse clinical consequences, particularly if the restoration was placed using accepted principles of beveling and etching of the cavity margins (Fig 1).

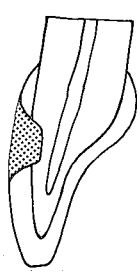

FIG 1. *Illustration of properly beveled and acid-etched, composite resin*

In cases of partial bond failure, a microscopic fissure may develop which, at first, is clinically tolerable. Moreover, the "leak" need not necessarily reach the dentine and result in clinically significant problems. Understandably, the physical properties, such as strength values, dimensional stability, poly-

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merization shrinkage of the polymers in resin systems, modulus of elasticity, coefficient of thermal expansion, chemical solubility, and biological compatibility, are all important parameters and each is subject to widely differing assessment by experts with respect to its clinical significance (Vanherle & Smith, 1985).

In the conditions commonly encountered in practice, four situations in which microleakage presents a serious clinical problem are detailed below.

FOUR CLINICAL SITUATIONS

Esthetics

The esthetics of restorations is of prime importance in anterior teeth, particularly in class 3, class 4, and class 5 cavities. Yellow and dark yellow to brownish discolorations at the periphery of the restoration can, apart from the discoloration of the entire filling, classify the entire restoration as a failure and result in its replacement (Fig 2). These marginal discolorations are located in the microfissure between the edge of the composite and the originally etched enamel, after the etched bond has deteriorated (Fig 3).

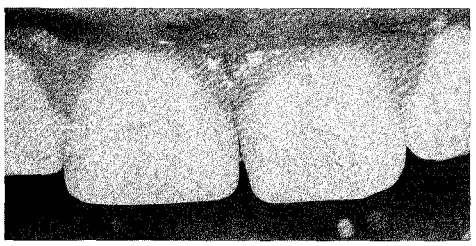

FIG 2. *Marginal discoloration of upper central incisors with class 4 composite restorations*

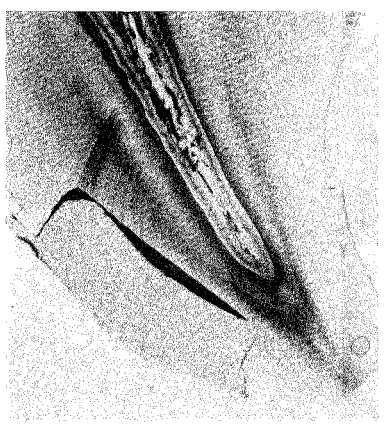

FIG 3. *Composite restoration in an upper central incisor, with loss of adhesion of composite resin at the left margin. Ground section (X6; original magnification X10)*

Such discolorations are predominantly caused by exogenous food and drink deposits, ingrown bacterial plaque, or possibly the biological by-products from the decomposition of the resin filling itself. Our observations, made over the years, indicate that unattractive marginal discolorations of this kind appear frequently in neglected teeth, which also generally show an above average incidence of caries or gingivitis, reinforcing the supposition that the problem has a biological, plaque-related genesis.

Due to the location of the pigment material, the discoloration cannot be polished away. The restoration is clinically defective and must be completely replaced.

Sensitivity

Patients who have flawless or acceptable restorations sometimes complain of temporary pain sensations during the consumption of sweet, sour, or salty beverages or foods. This phenomenon is encountered frequently in class 5 restorations and indicates the loss of the leakproof bond between the tooth and the restoration. Solutions of low molecular weight penetrate, by capillary action, through the minute cleft which has developed into dentine and may cause a painful osmotic irritation. This condition, which is very unpleasant for patients, is sometimes reversible but can persist and develop into a pulpitis necessitating endodontic treatment which in turn leads to the loss of vitality of the afflicted tooth. For this reason, the replacement of imperfect restorations in sensitive teeth should not be postponed. As a rule, the repair of the leaky margin by sealing does not achieve long-term success.

Percolation

Rhythmic deformations of the restorations can cause separation of the restoration from the cavity (Staninec & others, 1986; Schwickerath & Nolden, 1982). Contractions and expansions resulting from temperature changes then pump saliva and bacteria through gaps in the periphery into the internal form of a restored cavity. This process is called percolation. The form of the dimensional changes is determined by the geometry of the restoration, by the modulus of elasticity, and by the coefficient of thermal expansion. An extreme example of this would be

a MOD restoration in molars, consisting of a relatively elastic resin (that is, with a low modulus of elasticity) and with a high coefficient of thermal expansion (Fig 4), resulting in a bending upward, a

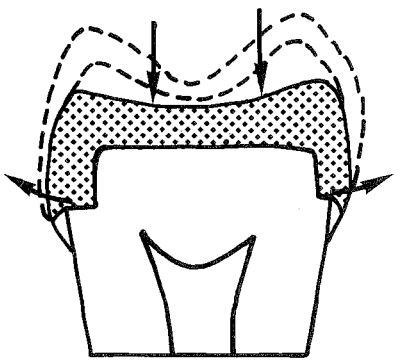

FIG 4. *Composite resin in MOD restoration illustrates distortion of restoration when occlusal loads are applied.*

relaxation of the restoration under mechanical stress, expansion when heated, and shrinkage when cooled.

Recent research in our clinic has shown that the coefficient of thermal expansion drops not only with an increase in the mass of inorganic filler, but is also dependent upon the nature of the organic matrix (Pötters, 1984).

While clinical importance is indisputably attributed to mechanical percolation, the effect of thermal percolation is contested. According to the latest investigations by Steinke (1985) in our clinic, the heat conductivity of filling materials is very important for the consequences of stress produced by cyclic temperature stresses. The range of dimensional changes caused by thermocycling remains within acceptable limits for composites with dentine-like heat conductivity. The heat conductivity decreased after the commercial grade composites were thinned by the addition of organic resin. Conversely, it increased for materials containing a relatively large proportion of inorganic filler particles.

The fluctuations of temperature, since they are greater on the surface than in the internal section of a filling, are important in achieving firmness of the bond between the composite and the periphery of the cavity. Percolation may be responsible for the pain sensations resulting from sweet, sour, or salty substances passing through the fissure between the cavity and the restoration. Moreover, it is possible that those cavity lining materials which are unstable and less "mouth resistant" will be dissolved in the course of time. The fissure between the filling and the more centrally located wall of the cavity then extends even further. This result may be one explanation for the "disappearance" of the cavity liners, which are known to have been set, a phenomenon often described in literature and not yet completely clarified.

Materials such as a low viscosity liner with a calcium hydroxide base are not resistant to percolation.

Experiments originally conducted and reported by Masuhara, Hirasawa, and Tarumi (1960) involving the application of adhesive to the dentine are worth mentioning. These authors attempted to form a direct chemical bond between a composite filling and the collagen of the dentine using Tri-n-butylborane. Recent developments provide for the application of a separate dentine adhesive agent, such as Dentin-Adhesit (Vivadent Schaan, Liechtenstein), Panavia Ex (Kuraray, Osaka, Japan), or Scotchbond (3M, St Paul, MN 55144). The objective is to achieve adhesion of a restoration not only at the etched border but to the entire cavity, and thus impede percolation. However, priming systems such as these have not yet been proven in the long term (Triadan, 1985).

Secondary Marginal Caries

The secondary caries of the margin of a defective composite filling is considered to be the end result of all peripheral leaks. It is especially emphasized, in addition to marginal discoloration, as a criterion for the clinical assessment of restoration material in ISO or FDI standard recommendations (Stanford & Ryge, 1977) (Fig 5).

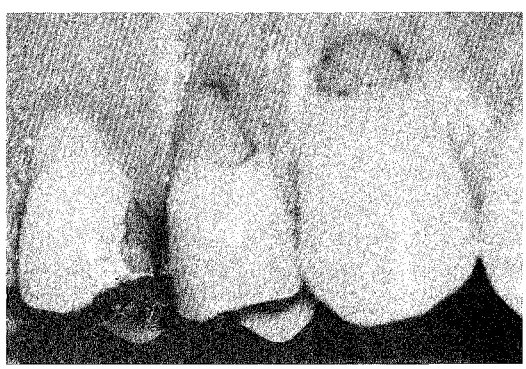

FIG 5. *Class 5 composite restorations with secondary marginal caries in upper right caninus and first premolar*

In my opinion, the clinical danger of secondary caries is far greater than the extent of the peripheral imperfection of restorations that have been placed using the acid-etch technique. For example, even if

as much as 90% of the periphery of a filling is leak-proof, a carious lesion, at first limited to the enamel, can later creep through the gap and develop from the point of the leak outward (Figs 6 & 7).

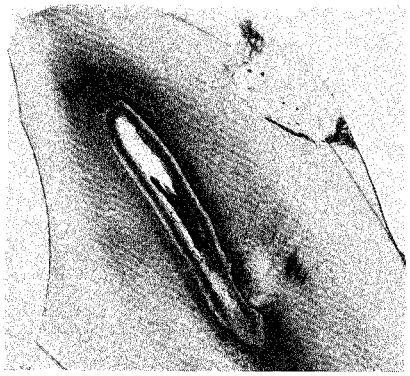

FIG 6. *Composite restoration with marginal caries of the enamel in lower right first premolar. Ground section (X6; original magnification X10)*

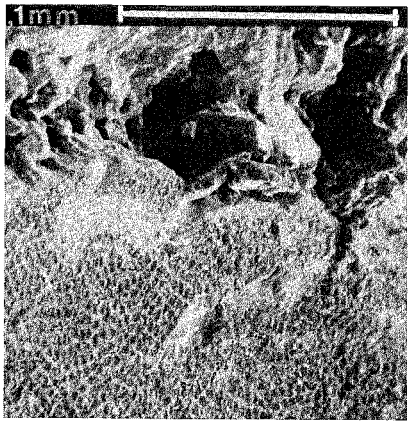

FIG 7. *Scanning electron micrograph shows small marginal defects and loss of composite material above etched enamel surface.*

The carious process, occurring principally along the minute canals of the collagenous fibers, which run at right angles to the dentine tubules (Fig 8), spreads out unchecked as as soon as the dentine is reached. The initial peripheral defect is often so small that the patient, and sometimes the dentist, does not notice it. The onset of pulpitis pain serves to notify, unfortunately too late, the patient and dentist of the condition.

A further insidious possibility in the progression of secondary marginal caries is that cariogenic bacteria can multiply practically undisturbed in a narrow fissure, even when there is very little oxygen. In addition, sweet solutions flow into the fissure. As a result, the carious process can develop as though in an incubator.

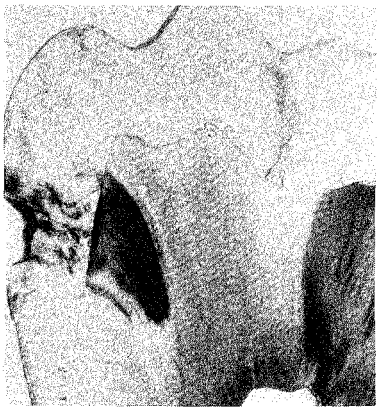

FIG 8. *Enamel caries causing undermining caries of the dentine is shown in a lower first molar, with loss of the main part of a small composite restoration that was exclusively placed in the enamel layer.*

Secondary marginal caries is thus much more dangerous than the large, open defects clearly evident between a conventional nonadhesive filling and a cavity. A large open defect is not only recognized earlier but is also more likely to be exposed to air, to receive the cleansing and neutralizing action of the saliva, and to be cleansed superficially, such as with a toothbrush.

In addition, the x-ray diagnosis of secondary marginal caries is severely limited, at least in the initial stages of the lesion, since the x-rays tend to be perpendicular to the fissure between filling and tooth.

Penetrant dyes could therefore prove to be convenient for clinical and diagnostic purposes, as, for example, recommended by Fusayama (1980).

Fluoridation is another method for prevention against secondary marginal caries of adhesive restorations, either by touch-coating the beveled, etched enamel border prior to restoration of a cavity, or through the mixing of fluorides with the sealers, or with the filling composite itself.

CONCLUSIONS

The formation of microfissures on the margin of an adhesive restoration does not necessarily have serious clinical consequences. Sometimes microfissures can be eliminated using the alternative

methods of finishing and sealing. As a rule, total replacement of the filling is necessary in cases presenting such clinical problems as esthetics (peripheral discoloration), increased sensitivity of the tooth, percolation (possibly connected with the dissolution of the cavity lining materials), and secondary marginal caries.

The prevention of the formation of microfissures between adhesive restoration and the surface of the tooth can be achieved by the following measures:

- Design of a cavity which takes into consideration its location and stresses, with appropriate preparation to avoid the creation of breaking forces at the periphery of a filling
- Beveling of the enamel border
- Proper etching
- Choice in each case of the most suitable materials
- Regular examinations and, if necessary, finishing of the adhesive restoration approximately twice every year
- Monitoring the oral hygiene of the patient and, if necessary, renewed and repeated instruction, information, and motivation

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Infection beneath Composite Resin Restorations: Can It Be Avoided?

MARTIN BRÄNNSTRÖM

INTRODUCTION

In any vital tooth there is a slow, continuous, outward flow of fluid around the restoration due to the higher fluid pressure in the pulp. Consequently, under restorations the fluid leakage is directed outward. With time, the flow may be reduced by the accumulation of solids, but the outward flow will not stop. There is also normal outward fluid flow in dentin covered by intact enamel.

Microleakage studies have clearly demonstrated the presence of a fluid-filled gap at the interface between the restoration and the cavity wall. However, considering the pulp, I have not seen any experimental evidence that toxic products from bacteria, the most important factor, may exist on the tooth surface and at the margin of the filling in concentrations great enough to reach the pulp by diffusion to any appreciable extent. Saliva circulates at the surface and flows outward from the gap. Neither mechanical stress nor any temporary reduction or increase of the gap width will significantly alter this situation.

For a few seconds or minutes, acid is present in the oral cavity or is produced in plaque. However, it may be buffered rather rapidly. Except for the fact that acids may prevent the development of a calcified pellicle, which may partly block the margin, acids probably have little clinical significance and certainly no direct influence on the pulp (Hume & Wang, 1986).

Small molecules of sugar existing in high concentration at the surface margin may diffuse inward, feeding new cariogenic microbes as well as those left behind in a gap at the dentinoenamel junction. Sugar may also increase the outward flow in gaps and in the corresponding tubules and this may elicit pain (Lindén & Brännström, 1967; Brännström, 1982).

SOURCES OF INFECTION IN GAPS

Our experiments in vivo have demonstrated that bacteria enter and occupy the contraction gap under composites by multiplication. We eliminated other sources of infection and found that bacteria had entered under 16 out of 17 fillings (Brännström &

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Nyborg, 1973). In laboratory studies we have observed that due to the higher fluid pressure in the pulp, theoretically an open tubule can be emptied about 10 times a day (Johnson, Olgart & Brännström, 1973). With similar techniques we have also found that bacteria such as Streptococcus mutans may enter dentinal tubules despite the presence of a slow outward flow (Olgart, Brännström & Johnson, 1974). This inward penetration of microbes is also true for the fluid-filled gap around a restoration.

In our discussion of leakage we have singled out the aspects of communication from the oral cavity and the ingrowth of bacteria from the surface. Clinically, however, we may have other sources of infection.

The Gap at the Dentinoenamel Junction

In several studies of early caries in enamel and dentin (Brännström & others, 1977; Brännström & others, 1980; Brännström, 1982; Mejàre & Brännström, 1985; Seppä, Alakuijala & Karvonen, 1985) we have seen an extensive lateral spread of infection in a narrow gap at the dentinoenamel junction. In that gap we observed bacteria penetrating deep into the intact enamel in an outward direction, as well as into the dentin. This space, which is not visible clinically, is often left behind after preparation, and bacteria from this area may also occupy the fluid gap around the filling.

Dentinal Tubules

Another source of infection is bacteria which remain deep in the tubules of mineralized dentin. I have seen too many bacteria in mineralized tissue, including dentin beneath caries, to accept earlier concepts that infection is present only in decalcified dentin. Microbes present in mineralized dentin may multiply and penetrate in an outward direction. This can be seen in root dentin tubules of teeth with necrotic pulps and also in the area of dentinal cracks (Brännström, 1982).

The Smear Layer

The next and rather common source of infection in the contraction gap is the smear layer. When surface sealing of composites was first used in 1973 to avoid bacterial ingrowth from the oral cavity, we found in our study (Brännström & Nyborg, 1973) that a bacterial layer had replaced the smear layer in 17 out of 20 cavities. These cavities had been cleaned with water spray only. In the contralateral cavities the superficial smear layer had been removed and the cavity treated antibacterially with Tubulicid (Dental Therapeutics AB, Nacka, Sweden). No bacteria were seen in these cavities. The cavities were prepared in intact teeth but not under sterile conditions. Had the cavities been prepared in carious dentin, the situation is likely to have been worse.

This observation supports the thesis that treatment of infected dentinal wounds should follow the same principles as for treatment of infected wounds in other parts of the body. An antibacterial detergent should remove the superficial smear layer which would facilitate disinfection of the remaining smear plugs of the dentinal tubules. The plugs should also be impregnated by fluorides which might contribute to denser plugs. The smear plugs should remain to reduce permeability and facilitate desiccation of the dentin. This may also lead to better adhesion of a resin lining and luting cements (Stangel, Ostro & Benko, 1986).

In addition, our study from 1973 and numerous studies since then have demonstrated that single microbes, entrapped within the smear layer, could multiply and replace most of the smear layer within four weeks. This occurred despite a rather good seal at the cavosurface margin. Apparently the microbes get sufficient nutrients from the tissue fluid seeping outward from the pulp.

Additional experimental studies (Brännström, 1982) employing inlays cemented with luting cements have supported this view. When the superficial smear layer was removed with an antibacterial detergent (Tubulicid) before cementation, no bacteria were present and no reaction was observed in the pulp, not even when an experimental lesion was present. On the other hand, when the smear layer was left after water spray cleansing, many teeth revealed a severe pulpal inflammation and a thin bacterial layer could be seen on the cavity wall (Brännström & Nyborg, 1960, 1974, 1977).

These observations support the following consideration: bacteria from the smear layer had multiplied and replaced this layer. As there was no appreciable contraction gap or communication to the oral cavity, this indicates that the original infection on the cavity walls can be serious despite the absence of large gaps along the inner surface of the restoration.

Contamination

The last source of cavity infection is from contamination of the cavity walls after cleansing and disinfection, such as from contaminants in an air blast.

To summarize the sources of infection and the means by which we may try to avoid them:

- Invasion from the tooth surface. This risk may be reduced by proper sealing of margins and contraction gaps.

- Bacteria present in the smear layer. These can be eliminated by the use of an antibacterial cleanser such as Tubulicid which has been investigated experimentally (Brännström, Nordenvall & Glantz, 1980).
- Three other sources. From dentinal tubules in mineralized dentin, from the gap at the dentino-enamel junction, or from a few microbes recontaminating the surface after cleansing.

Linings

These sources may be eliminated by application of a thin antibacterial liner attached to all cavity walls. We have tested a double lining (Brännström, 1983) consisting of an antibacterial hydrophilic shellac resin spread to a thin film with an air blast, over which a polystyrene liner was applied in the same way (Tubulitec Primer and Liner, Dental Therapeutics AB, Nacka, Sweden). The enamel walls were not etched, which means that a gap was present around all the enamel margins so that bacteria could enter. After several weeks, no bacteria were observed on any cavity walls under any of the 24 composites. However, the contralateral cavities of teeth lined with a double application of Copalite (H J Bosworth Co, Skokie, IL 60076) showed a bacterial layer in 20 of 24 cavities (Brännström & others, 1983), almost the same as having no lining at all. Obviously, the Copalite film was ineffective as a liner or sealer under composites, as it had become detached from the cavity walls. The results from our other studies (Torstenson, Nordenvall & Brännström, 1982; Torstenson & Brännström, 1986) indicate that the same could be true for any resin bonding agent and pure resin linings such as the Palfique (Tokuyama Soda Co, Kanagawa, Japan) lining system. It is possible that the positive effect observed for the combination of shellac resin and polystyrene liner was due to a better flow and retention of the polystyrene liner which in turn prevented a detachment of the resin film.

Only a few investigations have been made on thin liners under composites, perhaps because thick bases are generally recommended. Clinically it has been observed that thick bases do not prevent sensitivity and that secondary caries has been reported. The clinical assumption that the use of thick bases with high concentrations of calcium hydroxide or other agents placed along the pulpal or axial wall, the wall often least at risk (Brännström, 1982), will solve these problems may be erroneous. Thick bases cannot be placed on the important cervical wall and cover the surface to the outer margin (the dentin 0.2 mm from the cavosurface may contain 15 000 cross-cut dentinal tubules per square millimeter). Another disadvantage of using thick bases is that they cannot be placed on the lateral walls, which are important for retention. Without a liner on the lateral walls we may increase the risk of secondary caries. When a large fluid gap develops, the calcium hydroxide base will disappear to a large extent and the remaining space will be invaded by bacteria. This condition occurs beneath amalgams (Grajower, Bielak & Eidelman, 1984; Cox & others, 1985), and is perhaps even more common for composites with large gaps remaining without corrosion products partly blocking the gap.

THE GAP SIZE UNDER COMPOSITES

As indicated from our previous studies, the size of the gap may be less important than other factors when considering its effect upon the pulp. Some composites are known to expand hygroscopically, and even when the gap and the bacterial layer are thin the pulpal inflammation may be severe. On the other hand, large gaps in communication with the oral cavity may provide more nutrients to microbes within the gap, resulting in recurrence of caries. In addition, as previously discussed, parts of a lining material may wash out.

In a more recent study in vitro (Brännström, Torstenson & Nordenvall, 1984), we added a fluorescent dye to the Enamel Bond resin (3M Co, St Paul, MN 55144) used to impregnate the gap in approximal composite restorations, allowing us to measure the gap size and reveal failure in bonding. This method was superior to that used in an earlier study (Bergvall & Brännström, 1971), avoiding artificial gaps and blockage of gaps caused by specimen preparation. The profile was examined in the microscope under UV-light (see figure). Within a few

Cervical wall of approximal filling. Occlusin composite (ICI, Macclefield, England) above, dentin below. The size of the cervical gap is approximately 20 μm.

seconds the resin penetrated, by capillary action, from the cervical margin and occupied at least the gap present at the cervical wall and sometimes filling the entire gap existing from the cervical to the occlusal wall.

Gaps were present varying from 6 to 30 μm when various composites and bonding procedures, including the GLUMA technique, were examined (Torstenson & Brännström, 1986; 1987). If we cannot eliminate the formation of gaps in our laboratory studies, working under optimal conditions, we cannot expect to avoid gaps clinically.

For some composites we measured the gap after the tooth had been stored in water for two weeks. Composites recommended for posterior teeth and the hybrid composites had very little if any hygroscopic expansion (Torstenson & Brännström, 1987).

An incremental technique did not eliminate the contraction gap. After three applications using either the GLUMA technique or Scotchbond and P-30, we found cervical gaps of more than 10 μm. We also observed, using this technique, that thermal stress does not change the gap size to any appreciable extent (Torstenson & Brännström, 1987).

Furthermore, clinically relevant thermal stress involves a change of about 30 °C applied for 3 - 4 seconds only. If a fluid gap exists, normal temperature changes do not have much clinical significance. This may be true especially if we consider that a continuous, slow, outward flow of fluid persists around the restoration. In microleakage studies with thermocycling, extreme temperature variations, and for long application periods up to one minute are generally used. The experimental model does not seem to relate well to the clinical situation.

However, a sudden small change of the fluid volume within the gap may occur and explain occasional sensitivity to cold (Brännström, 1986). Due to capillary forces, the cooling and contraction of the fluid may lead to rapid outward flow of several micrometers in the gap and in the corresponding tubules. Fluid gaps within the tooth seem to favor hypersensitivity to cold, especially if they are combined with infection as is the case when there is a communication to the oral cavity. When the gap and the infection are eliminated — for example, by replacing the restoration with ZOE cement — the sensitivity usually disappears. An analogous situation is seen in the dentinal crack syndrome (Brännström, 1982). When the patient fractures the involved cusp including the crack, the hypersensitivity to cold also disappears.

Sensitive dentin means that tubules are open all the way to the pulp, as is often encountered in cavities. We must realize that any kind of acidic treatment or high concentration of EDTA or acidic salts will increase the communication to the pulp many fold. In the case of microbial contamination, not only is there a high risk of bacterial invasion in dentinal tubules (Vojinović, Nyborg, & Brännström, 1973), but it is also difficult to stop the outward flow of fluid from the dentinal tubules even with an air blast of 10 seconds (Brännström & Nordenvall, 1977; Nordenvall, 1978). There will be no adhesion to dentin with fluid seeping outward under a slow-setting resin, lining, or a slow-setting cement.

PROTECTIVE FACTORS TO REDUCE THE RISK OF INFECTION

Large gaps remaining along the cervical wall may lead to serious problems. Our experiments in vitro and in vivo (Torstenson, Brännström & Mattsson, 1985) have shown that resin such as Enamel Bond can penetrate the contraction gap. In order to reduce the risk of infection under composites, the following protective factors should be considered:

(1) Removal of superficial smear layer and disinfection

(2) Proper desiccation for about 10 seconds

(3) A thin antibacterial liner applied to all cavity walls, and which is not detached by during polymerization shrinkage of the restorative material

(4) Etching of beveled enamel

(5) Sealing of gaps with resin impregnation

(6) Finally, we may also expect some hygroscopic expansion.

One of these factors may fail, but surely not all of them. We may also hope for the defense mechanisms, such as a calcified pellicle blocking the cavosurface margin and irregular dentin blocking the pulpal ends of dentinal tubules.

Glass-ionomer Cements

Recently glass-ionomer cements have been suggested as a base material beneath composite resins. However, infection and inflammation in the pulp under such restorations have been observed in two studies (Nordenvall, Brännström & Torstenson, 1979; Langeland & Pascon, 1986). Is the use of chemical bonding with this cement overemphasized? Contraction forces may detach the cement from cavity walls.

Shallow cavities also need a liner to protect against infection. In the clinic, can we easily place any cement with a thickness of around 200-400 μm on all cavity walls and the outer cervical margin without impairing retention, strength of the filling, and invasion of microbes? Can this cement be more easily handled than a thin lining system, spread and quickly dried with an air blast? More research is needed to prove that this material can be accepted as a lining material. In this context it should be pointed out that there is no evidence that "thermal shocks"

may cause damage to the pulp under normal conditions. Dentin is an insulator, equally as good as cements, and therefore there is no need for thick liners or cements beneath restorations as a protection against thermal changes. Tooth sensitivity and associated pulpal complications following cementation have been associated with the use of glass-ionomer cements (H Trowbridge, personal communication). The reason for this is as yet unclear, but gap formation and bacterial infection may possibly play a part.

Are Filling Materials Irritating the Pulp?

Full support must be given to the results presented by Cox (1987; in this issue of *Operative Dentistry*). In numerous studies since 1970, involving several hundred pairs of human teeth, our results have pointed to the same conclusion: namely, when infection is avoided, there is no appreciable irritation to the pulp. When silicate-, phosphate-, carboxylate-, and ASPA-cements and composites were placed on exposed pulps the same also proved true. (For description of the methods, illustrations, and references, see Brännström, 1982.) Other workers have also found a high correlation between pulpal inflammation and the presence of bacteria on cavity walls (Bergenholtz & others, 1982; Tobias, Plant & Browne, 1982: Hörsted, Simonsen & Larsen, 1986).

CONCLUSIONS

One reason for conflicting opinions is the introduction of experimental errors. For instance, when shorter follow-up periods are used, bacteria have not yet replaced the smear layer. In laboratory decalcification, not only the smear layer disappears completely, but so do the microbes within the smear layer and those present in the fluid-filled contraction gap. When the restoration is removed in the laboratory after decalcification, bacteria can be detached from the cavity walls and then only a limited area of the cavity is examined in the sections. Thus, infection has not been observed, although pulpal inflammation is found to be present.

Such a combination of errors occurs frequently and explains why many investigators fail to understand the role of bacteria in pulpal inflammation. This problem in experimental research has been discussed elsewhere and a better design of experiments has been suggested (Brännström, 1982).

In the future we may hope for better materials and more honest and careful research and recommendations from the manufacturers. We must also remember that we need simple techniques which can be easily and successfully used, not only by a few, but by the majority of dentists.

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Model Systems for Determining Biologic Effects of Microleakage

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INTRODUCTION

Microleakage may be defined as the ingress of oral fluids into the space between tooth structure and restorative materials. It has long been recognized that in general dental materials do not bind or adapt to tooth structure well enough to provide a perfect seal and the resultant flaw provides a pathway for penetration of various solutes and solvents. Evidence suggests that microleakage, in addition to establishing an environment for the development of caries, may result in pulp pathology and postoperative tooth sensitivity. Microleakage also contributes to the corrosion, dissolution, or discoloration of certain dental materials.

The purpose of this paper is (1) to discuss the causes of microleakage, (2) to examine the conditions under which microleakage may be increased or decreased, (3) to briefly review the research techniques that have been developed to examine flaws that form around restorations, and (4) to consider the adverse effects of microleakage on the enamel, dentin, and pulp.

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CREATION AND MODIFICATION OF MICROLEAKAGE

A major cause of microleakage is poor adaptation of restorative materials to tooth structure, which may be due to inferior adaptability on the part of the material or inexpert insertion of the material by the operator. Another major cause is shrinkage of the material resulting from chemical or physical changes after it is inserted. Polymerization contraction of acrylic resins and the initial contraction of amalgams exemplify chemical shrinkage, whereas thermal change may introduce gaps as a result of physical alterations in restorative materials. Long-term dimensional changes in restorations may result from disintegration or corrosion of the material. In the case of silicate cement, dehydration produces contraction. Hansen and Bruun (1971) found that after the initial response to placement of silicate cement had subsided, pulpal inflammation tended to become more severe. This was attributed to an increase in microleakage resulting from contraction of the cement.

Granath and Möller (1975) have shown that elastic deformation of tooth substance by masticatory forces is capable of increasing the space between tooth structure and restorative materials. They found that in MO, DO, and MOD cavity preparations the buccal and lingual cavity walls can be bent outward by mechanical loading of restored teeth. Qvist (1983) found a marked effect of masticatory forces on the development of marginal leakage of composite restorations. The frequency of marginal leakage was significantly greater in teeth that were in functional occlusion than in similar teeth without antagonists.

All restorative materials are more or less sensitive to manipulation, so the skill of the operator is a critical factor in determining the extent of microleak-

age. Thus, the surface quality of the cavity walls and the method by which the material is inserted will affect the sealing properties of the restorative material. In the case of amalgam, Mahler and Nelson (1984) found that condensation technique and plasticity of the material were much more influential than burnishing the margins in determining the extent of microleakage. Granath (1971) had shown earlier that the degree of plasticity of amalgam at the time of insertion is a critical factor in determining the sealing quality of amalgam. Moisture control is also of importance, as moisture adversely affects a number of restorative materials, particularly silicate and glass-ionomer cements.

Cavity depth may have a profound influence on the extent of leakage. Granath and Möller (1975) found that in MOD restorations the cavity depth was of great importance in determining the degree to which the gap between tooth structure and restoration was increased. Additionally, a relationship exists between cavity depth and the extent to which leakage affects the pulp. The diameter of the dentinal tubules and the number of tubules per surface unit increase as the tubules converge toward the pulp (Fig 1). Thus, in deep cavities a greater proportion of

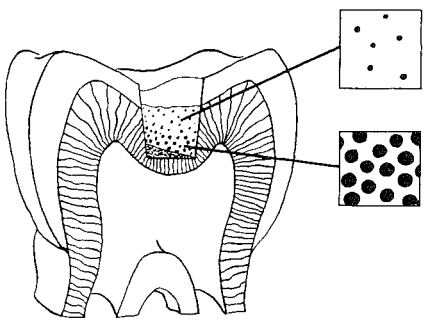

FIG 1. *Diagram illustrating differences in the diameter and number of dentinal tubules in the floor of a shallow (top) and deep (bottom) cavity. (From Trowbridge (1982)* Dentistry **82** *2, 22-32)*

tubule surface area is available for the ingress of solutes and solvents than is the case in shallow cavities.

Location of the cavity walls may also influence microleakage, particularly if the margin of the cavity is located in cementum. While acid etching reduces leakage where composite restorations abut enamel, it has little effect on leakage at the cementum margin, a serious weakness of resin restorations (Causton & others, 1984).

The restoration's environment is also of paramount importance in determining the extent of marginal leakage. In the oral cavity, both restoration and surrounding tooth substance are subjected to mechanical loading and temperature variations and are in contact with food, saliva, and micro-organisms. Gaps around restorations may provide pathways for the ingress of oral micro-organisms and/or their metabolic products. Numerous studies have shown that bacteria can successfully colonize the walls of cavities (for review see Brännström, 1982).

How large must the gap be for bacterial penetration to occur? Since the size of a bacterium is approximately 2 μm, bacteria should be able to enter a very narrow space. However, a larger marginal defect may be required for plaque to form. Jørgensen and Wakumoto (1968) found no secondary caries where the space between the surface margin of amalgam fillings and the adjacent cavity wall was less than 50 μm. However, these investigators did not attempt to determine the extent of bacterial penetration into the underlying crevice, an aspect that requires further clarification.

Microleakage is a dynamic process that can increase or decrease with time. As a result of long-term exposure to saliva, pellicle, and bacterial plaque, changes may occur that will serve to obturate the space between tooth substance and restoration. Amalgam restorations placed in unlined cavities exhibit an initial leakage that tends to decrease with time (Going, 1972). The accumulation of products of corrosion are thought to contribute to this reduction (Phillips & others, 1961). Deposition of mineral salts in the pellicle may also play an important role in obturating flaws around restorations (Brännström, 1984). The initial contraction of acrylic resins is somewhat compensated for by absorption of water that causes them to expand (Asmussen & Jorgensen, 1972).

METHODS OF STUDY

By far the greatest problem facing the investigator is the susceptibility of the interface between tooth structure and restoration to dimensional changes resulting from experimental manipulation of specimens. Most model systems in vitro fail to simulate conditions in the oral cavity, so the interface may be affected by artifactual alterations. Granath and Svensson (1970) recognized the importance of maintaining mouth temperature and humidity in developing an air pressure method of assessing insufficiencies in obturation in vitro. The presence of saliva, masticatory forces, and the pressure differential between the pulp and the oral cavity are other factors that affect diffusion processes under conditions in vivo. Consequently, most studies in vitro have been of little clinical relevance.

Space limitations preclude detailed discussion of

all of the various methods that been developed for assessing the interface between tooth structure and restorative materials. However, consideration will be given to representative examples of experimental model systems that have made significant contributions to our present knowledge of microleakage.

By definition, microleakage is a phenomenon that involves diffusion, so most studies have focused primarily on that aspect of the problem. However, recognition of the dynamic relationship between tooth structure and restorative material has led to other investigative approaches, some of which will be mentioned here.

In general, methods that have been developed fall into three major categories: (1) laboratory tests involving passage of dyes, radioactive isotopes, bacteria, or air through flaws around restorations; (2) direct visualization of the marginal gap; (3) clinical tests in which biologic effects are assessed.

Diffusion of Dyes and Isotopes

Dye and isotope studies, in spite of their shortcomings, have provided much of the information upon which our present knowledge of microleakage is based.

The clinical relevance of studies using ions or small molecules has been questioned, as there is no evidence that diffusion of dyes or isotopes in laboratory studies is proof that fillings will fail clinically. The vast majority of restorations are successful, yet laboratory studies indicate that the boundary of most restorations can be penetrated by isotopes, leading to the conclusion that leakage of isotopes is not a crucial measure.

Most dye and isotope diffusion methods lend themselves only poorly to quantification. To overcome this difficulty, Douglas and Zakariasen (1981) developed a volumetric method with which the dye that has diffused into a gap can be recovered and measured spectrophotometrically. Another problem is that isotopes such as Ca^{45} may have an affinity for tooth structure or for restorative materials, and thus distribution of the isotope may be misleading. Severity of leakage as determined by dyes and isotopes is probably a critical factor, as bacteria are likely to enter the gap only if leakage is pronounced.

Bacterial Penetration

Invasion of marginal crevices by bacteria provides a method of studying microleakage that would seem to be clinically more realistic than dye and isotope diffusion methods. This is because the size of most dye molecules and isotopes that have been used in microleakage experiments is infinitesimal in comparison with bacteria. A restorative material can be tested by placing the tooth in a broth culture and determining whether bacteria have penetrated flaws around the restoration.

Compressed Air

A method utilizing compressed air to assess microleakage has been developed by Granath and Svensson (1970). Measurement of a constant flow of an inert medium between a restoration and a mold of dentin provides a means of assessing insufficiencies in obturation with great accuracy. Ideally, a noble gas should be used. This method proved to be a valuable technique for comparing the sealing properties of different amalgams as well as cements (Möller, Schröder & Granath, 1983). However, the flow of gas furnishes very little information about clinical leakage and therefore provides only supporting data for explaining differences in biologic effects between restorative materials.

Neutron Activation Analysis

Neutron activation analysis is a quantitative method first developed by Going, Myers, and Prussin (1968). Using this method, restorations can be placed in the oral environment for varying intervals before testing, thus providing longitudinal data. The teeth are exposed to a solution of ^{56}Mn in vivo and subsequently extracted and subjected to neutron activation. Douglas, Chen, and Craig (1980) modified this method to assess the sealing qualities of restorations following varying postoperative intervals in vivo. However, the method is quite expensive and probably too complex for use in most laboratories.

Electrical Current

Delivanis and Chapman (1982) devised a quantitative method of assessing microleakage by utilizing the flow of electricity. When flaws around restorations result in the establishment of a continuous electrolytic pathway, the amount of electrical current passing through the flaw can be measured. It was found that microleakage assessed by this method was in close agreement with results obtained with dye penetration and autoradiographic techniques involving the use of radioactive tracers.

Direct Visualization

Direct visualization of the marginal gap, either with the light microscope or by scanning electron

microscopy, is an indirect method of studying microleakage, but it has yielded information regarding the dimension of openings through which leakage occurs. For example, Asmussen and Jørgensen (1972) employed direct visualization to assess the effects of shrinkage and expansion of resin restorations on the size of the gap between cavity wall and restorative material. Unfortunately, manipulation of the specimens during histologic processing may result in artifacts.

Bacterial Staining

Histologic staining methods have been employed to demonstrate bacteria under restorations (for review see Brännström, 1982, 1984). The results of such studies have revealed a positive correlation between the presence of bacteria on the walls of cavities and pulpal pathology beneath the cut tubules. Mejàre, Mejàre, and Edwardsson (1979) employed a combination of culturing and bacterial staining techniques to assess bacterial ingress into gaps around restorations and to characterize the plaque that formed on cavity walls. They found that the flora beneath composite fillings more closely resembles dental plaque than the bacterial components of either saliva or carious dentin.

Artificial Caries

Bacterial plaques growing in systems in vitro or in an acidified gel have been used to test the cavity-sealing properties of restorations. Demineralization of the cavity wall is evidence of microleakage. This method has proved to be useful in exploring the relationship between microleakage and secondary caries. Using polarizing light microscopy, Jensen and Chan (1985) determined the extent of demineralization of cavity walls adjacent to composite resin restorations following application of an acidified gelatin gel to the tooth surface to simulate caries. Grieve and Glyn Jones (1980) utilized a similar system to demonstrate that beveling of the cavity margins before acid etching greatly reduces the leakage of composite resin restorations. It remains to be established how closely artificial caries systems resemble caries activity in vivo.

Thermocycling

If the coefficient of thermal expansion of a restorative material differs significantly from that of tooth structure, the dimensions of the space around the filling material will change as the tooth is subjected to temperature variations. Together with thermal expansion of fluid occupying the crevice between the tooth and the restoration, this difference in the coefficients of thermal expansion will result in fluid exchange between the tooth and the restoration (Nelson, Wolcott & Paffenbarger, 1952). For this reason, in microleakage experiments it has become common practice to subject specimens to thermocycling. However, the clinical significance of thermocycling has been challenged, particularly when specimens are subjected to temperature variations for periods longer than several seconds. In most studies, samples have been cycled through baths varying from 4 to 60 °C with dwell times of one to three minutes. Since teeth are in contact with hot and cold foods for only a few seconds, a longer dwell time would seem to be inappropriate. Cycling regimes using a short dwell time may be more realistic clinically (Causton & others, 1984). The number of cycles employed varies, but it is fairly common practice to use several thousand cycles.

Crim, Swartz, and Phillips (1985) found no significant difference in microleakage between four different thermocycling techniques. Their nonthermocycled control specimens exhibited significantly less leakage than that of the experimental groups, indicating that thermocycling is capable of increasing microleakage.

An example of how thermocycling can promote marginal leakage of materials is our experiments with zinc oxide-eugenol (ZOE). ZOE has been shown to have superior sealing properties when used as a cement for restorations, and for this reason it has been employed in surface-sealing experiments to exclude bacteria from the cavity (Brännström, 1982; Cox & others, 1987). Using a dye diffusion method, we found that 50 cycles from 4 to 60 °C with a dwell time of one minute produced severe leakage when ZOE was used to cement castings (Graver, Alperstein & Trowbridge, 1986) (Fig 2). No dye diffusion was observed in specimens that were not thermo-

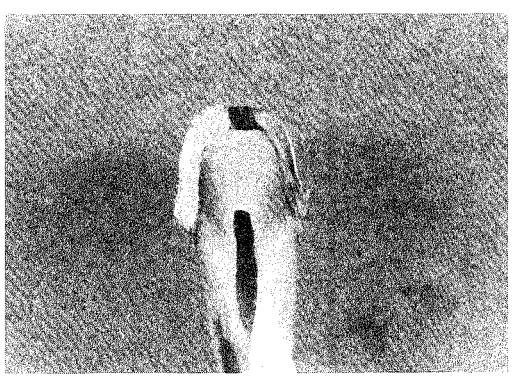

FIG 2. *Casting cemented with ZOE. Note marked diffusion of dye into dentinal tubules in thermocycled specimen.*

cycled. The extent of microleakage observed in the thermocycled specimens almost certainly does not occur under clinical conditions.

CAVITY VARNISHES, LINERS, AND BASES

Because leakage is to a greater or lesser extent an inherent weakness of most dental restorative materials, cavity varnishes, liners, and bases have been developed to protect the pulp. The main purpose of such agents is to block the dentinal tubules and thus limit diffusion of irritants to the pulp. The effectiveness of various materials that have been developed for this purpose has been the subject of numerous studies. Most of these studies have been qualitative or semiquantitative and have involved histologic evaluation of the pulp, diffusion of dyes and silver nitrate, and autoradiographic techniques employing radioactive tracers. More recently, a quantitative method of assessing the ability of cavity varnishes, liners, and bases to reduce dentin permeability, as measured by changes in the hydraulic conductance of dentin, has been developed by Pashley and others (1985). They found that most bases were more effective than varnishes and liners at reducing dentin permeability.

BIOLOGIC BARRIERS

We need to know more about the natural barriers that tend to reduce the permeability of dentin and thus limit diffusion of substances to the pulp. These include reparative (irregular) dentin and dentinal sclerosis. It has been reported that formation of reparative dentin contributes to pulp protection against the ingress of irritants (El-Kafrawy & Mitchell, 1963; Bergenholtz & Reit, 1980). Figures 3 and 4 depict a very mild pulp response beneath a deep cavity that had extended into preformed reparative dentin. The cavity was filled with ZOE, and the tooth was extracted 21 days postoperatively. Thomas, Stanley, and Gilmore (1969) found that the presence of irregular dentin beneath test cavities provided the underlying pulp with good protection against the placement of direct filling gold.

Less is known about the ability of dentinal sclerosis to protect the pulp from the effects of microleakage. Dentinal sclerosis is a reaction occurring in dentin that involves deposition of calcium phosphate crystals in the dentinal tubules. This reaction occurs beneath nearly all carious lesions (Stanley & others, 1983), and it is possible that the high success rate of restorative procedures is due in part to the protective effects of sclerotic dentin.

CALCIUM HYDROXIDE

Möller (1975) found that treatment of the cavity floor with calcium hydroxide protects the pulp from the irritating effects of amalgam. Similarly, Bergenholtz and Reit (1980) have reported that treatment of dentin with calcium hydroxide greatly reduces pulp irritation caused by microbial products placed in test cavities. Mjör, Finn, and Quigley (1961) found that coronal dentin of young permanent teeth exposed to

FIG 3. *Cavity preparation extending into preformed reparative dentin. Hematoxylin and eosin stain. (X6; original X10)*

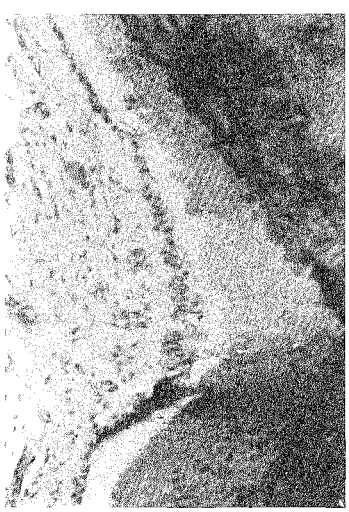

FIG 4. *High power view of tooth shown in Figure 3. Note very mild inflammatory response. Hematoxylin and eosin stain. (X60; original X100)*

calcium hydroxide for various periods of time developed increased hardness and density, suggesting that a form of dentinal sclerosis may have evolved. However, Brännström, Isacsson, and Johnson (1976) found no areas of mineralization in the tubules of calcium hydroxide-treated dentin. Furthermore, microradiographic examination of the dentin revealed no significant differences in the degree of mineralization between calcium hydroxide-treated and untreated dentin. Thus, while treatment of dentin with calcium hydroxide does not produce true dentinal sclerosis, it does appear to have the capacity to obturate dentinal tubules, perhaps by occluding the tubules or denaturing their contents (Bergenholtz & Reit, 1980).

DENTIN PERMEABILITY

Grinding debris (smear layer) that is deposited on cavity walls during operative procedures has been shown to reduce dentin permeability markedly (Pashley, 1984). It appears that the smear layer is also capable of preventing bacteria from entering the dentinal tubules (Olgart, Brännström & Johnson, 1974; Michelich, Schuster, & Pashley, 1980). It has been shown that removal of the smear layer by acid etching greatly increases dentin permeability by decreasing surface resistance (Pashley, 1985). Stanley, Going, and Chauncey (1975) reported that, in the case of deep cavities, acid etching with 50% phosphoric acid for 60 seconds significantly increased pulpal inflammation beneath restorations.

In vital teeth, bacteria do not readily pass through dentinal tubules into the pulp. Michelich, Schuster, and Pashley (1980) found that irregularities within the dentinal tubules were capable of arresting 99.8% of bacteria that enter the tubules. However, it has been hypothesized that in the presence of severe microleakage, hydraulic pressure generated during mastication may cause a restoration to act as a hydraulic plunger, thus driving substances, including bacteria, through the dentinal tubules and into the pulp (Pashley, 1985).

Pashley and others (1983) have shown that dentin permeability decreases approximately 75% within the first six hours following cavity preparation. In a subsequent study it was found that adhesion of plasma proteins to the walls of the dentinal tubules is probably responsible for the decrease in permeability (Pashley & others, 1984). Theoretically, trauma brought about by cavity preparation evokes an inflammatory reaction in the pulp, and this in turn causes venules in the underlying pulp to become more permeable. As a result, plasma proteins leave the venules and pass into the dentinal tubules where they accumulate and contribute to the increased resistance to fluid flow.

BIOLOGIC EFFECTS OF MICROLEAKAGE

While early studies on microleakage were concerned primarily with assessing the ability of restorations to obturate the flaws around restorations, in recent years there has been increased interest in the biologic impact of microleakage. Although methods in vitro still have value as a means of screening materials and providing quantitative data on a comparative basis, the emphasis has shifted from diffusion and sealing studies to studies on mechanisms involved in demineralization of cavity walls, pulp pathology, and postoperative sensitivity. Much progress has been made in elucidating the role of microorganisms in these conditions, but it still remains to be established precisely how microleakage adversely affects the dental tissues.

The most important biologic manifestations of microleakage are development of caries beneath restorations and pulp pathology. Microleakage may also be the cause of postoperative sensitivity if diffusion of bacterial products into the pulp results in hyperalgesia.

Caries around restorations involves the formation of a primary lesion on the outer surface of the tooth from which diffusion of acid into gaps around the restoration may result in the development of a "cavity wall lesion" (Hals & Simonsen, 1972). Development of wall lesions is favored by microleakage in connection with a cariogenic milieu. As in primary carious lesions, wall lesions may undergo remineralization if the ingress of acid ceases or the caries process is arrested.

Some years ago the notion that toxic ingredients of filling materials are primarily responsible for pulpal injury resulting from restorative procedures was called into question (Vojinović, Nyborg & Brännström, 1973). Diffusion of bacterial products to the pulp is now regarded as a major cause of pulpal injury associated with microleakage (Bergenholtz & others, 1982). The results of numerous experiments suggest that microleakage will not seriously affect the pulp unless bacterial plaque becomes established on the walls of the cavity. In studies involving germ-free animals (Kakehashi, Stanley & Fitzgerald, 1965; Watts, 1979), as well as in experiments in which surface-sealing agents were employed to exclude bacteria from the cavity (Brännström, 1982; Cox & others, 1987), the importance of bacteria in pulpal injury has been well documented.

In assessing the effect of restorative materials on the pulp, most investigators have placed test materials in cavities prepared in healthy human or animal teeth and examined the pulp reaction after various postoperative time intervals. As pointed out by Bergenholtz and others (1982), there are many inherent variables associated with this model system which make it difficult to distinguish the effect of the mate-

rial being tested from the effects of cavity preparation, desiccation, microleakage, and bacterial growth beneath the restoration. These researchers designed an experiment that enabled them to directly correlate pulpal histopathology with the presence of bacterial colonies under the fillings. Rather than employing traditional bacterial staining methods, filter paper disks were inserted beneath restorative materials and later recovered and cultured. They found that pulpal histopathology correlated directly to the presence of bacterial colonies beneath the restorative materials.

The origin of bacteria found beneath restorations is still unclear. Brännström (1984) believes that bacteria trapped within the smear layer have the capacity to proliferate. On the other hand, Bergenholtz and others (1982) have provided evidence that bacteria enter the cavity via leakage from the oral environment. Their findings suggest that organisms contaminating the cavity at the time of preparation would have little chance of surviving in the absence of microleakage.

Brännström (1982) and Cox and others (1987) have shown that in the absence of bacteria, various materials placed near or directly on the pulp produce a relatively mild tissue response, frequently followed by dentinal bridging. In pulp-capping experiments in germ-free rats, Watts (1979) found that silicate and zinc phosphate cements did not evoke an inflammatory response. Both materials evoked a low-grade tissue reaction, manifested only by contact necrosis. Leakage that permits bacteria to grow beneath a restoration may result in a pulp response that varies from suppuration and abscess formation (Fig 5) to a chronic inflammatory reaction characterized by the presence of lymphocytes, plasma cells, and collagen formation (Fig 6). Thus, there is a marked similarity between inflammatory reactions beneath restorations and carious lesions (Trowbridge, 1981). The presence of immunologically competent lymphocytes and plasma cells suggests that introduction of bacterial antigens into the pulp may play an important role in evoking a response.

Mejàre, Mejàre, and Edwardsson (1979) found the flora beneath restorations was similar to that of the gingival crevice and suggested that patients may have been sensitized to plaque antigens previously, in which case the pulpal response to filling materials may in fact represent a form of hypersensitivity reaction. They further speculated that activation of the complement system by lipopolysaccharides from gram-negative anaerobic bacteria in plaques beneath restorations could play a role in the initiation of inflammation.

Pulpal inflammation may result in hyperalgesia, a condition in which nociceptive nerve fibers become more responsive to stimulation (for review see Trowbridge, 1985). Lundy and Stanley (1969) observed that exposure of freshly cut dentin to saliva results in severe sensitivity and reversible inflammation in the underlying pulp. Thus, postoperative tooth sensitivity may be indicative of pulpal inflammation secondary to severe microleakage.

In assessing the role of microleakage, more information is needed regarding the nature, concentration, and biologic activity of substances reaching the pulp. New methods will have to be developed in order to achieve these objectives. Because of the intervening dentin, diffusion of substances from a cavity preparation to the pulp may be quite limited. Hume (1984), for example, reported that there is a fairly substantial release of unreacted eugenol from

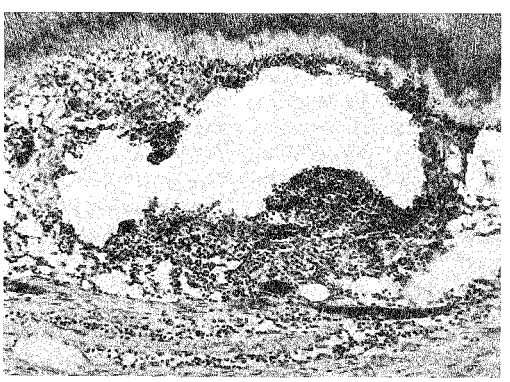

FIG 5. *Pulp response three weeks after insertion of zinc phosphate cement in deep cavity. Photomicrograph shows area of suppuration with abscess formation. Hematoxylin and eosin stain. (X60; original X100)*

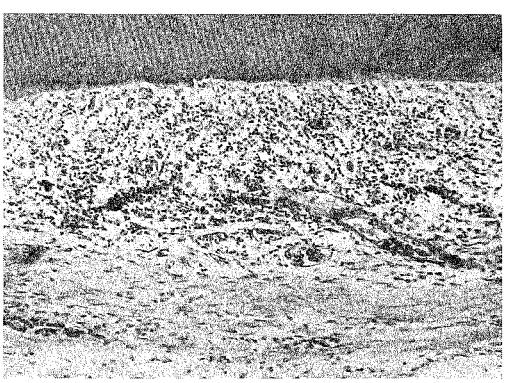

FIG 6. *Pulp response 13 weeks following insertion of zinc phosphate cement in deep cavity. Photomicrograph shows chronic inflammatory cell infiltrate with collagen deposition in peripheral area of lesion. Hematoxylin and eosin stain. (X60; original X100)*

ZOE placed in a cavity but reasoned that it is unlikely that eugenol would reach concentrations in excess of 10^{-4}M in the underlying pulp tissue.

If bacterial growth beneath restorations is the primary cause of pulpal injury, the antibacterial properties of restorative materials should be of considerable interest. Updegraff, Chang, and Joos (1971) found that some restorative materials, when freshly prepared, exhibited marked antibacterial activity. However, the antibacterial activity of all materials except copper cement was abolished by leaching in water for seven days. Glassman and Miller (1984) studied several types of amalgam and found them to have antibacterial activity, some more than others. However, their tests were conducted over the first 24 hours after the amalgams had set, so long-term antibacterial activity was not established. Myers (1966) reported that amalgam restorations increase the Zn, Hg, Ag, and Cu concentration of enamel and dentin. He speculated that the latter two elements have a strong oligodynamic antibacterial action and could be effective in arresting secondary caries. This would help to explain the very low incidence of recurrent caries over a five-year recall period reported by Leinfelder and others (1980). Only 3 out of 418 restorations they examined showed evidence of recurrent caries.

Ørstavik and Hensten-Pettersen (1978) found that a number of freshly mixed restorative resins inhibited bacterial growth in a test system in vitro. However, storage in saline for 24 hours greatly reduced or entirely abolished the antibacterial activity of these materials. Other materials reported as having antibacterial properties include calcium hydroxide and ZOE. It is obvious that if pulpal injury is to be avoided, materials with long-term bacteriostatic activity will have to be developed.

CONCLUSIONS

It is obvious that the causes and consequences of microleakage require further characterization. In assessing the role of microleakage as an etiologic factor in pulp disease, more information is needed regarding the nature, concentration, and toxicity of substances reaching the pulp through the dentinal tubules. Growing evidence supports the theory that unless microleakage allows bacteria to become established on the cavity walls, most restorative materials are well tolerated by the pulp.

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LITERATURE REVIEW

Posterior Composite Resins: A Status Report for the Academy of Operative Dentistry

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HAROLD LASWELL

INTRODUCTION

According to a recent survey, posterior composite resins are used by 80% of the dentists practicing in the United States (Trends in Dentistry, 1986). In spite of their popularity, however, the indications and limitations for the use of composite resin as a posterior restorative material are not well defined, and this has led to inappropriate clinical applications in many cases.

Although one study (Phillips & others, 1971), describing an early clinical trial of posterior composites, reported favorable results with the material, subsequent reports by the same investigators described excessive wear associated with those restorations (Phillips & others, 1972, 1973). Additional studies have also described the major problem associated with posterior composites as a loss of anatomic form due to significant wear occurring after about one year of clinical function (Osborne, Gale & Ferguson, 1973; Eames & others, 1974; Leinfelder & others, 1975).

Manufacturers have significantly improved composite resins since the 1970s. Filler particles have evolved from macro (20 μm) quartz fillers to softer micro (.04 μm) fillers, to combination or hybrid fillers. Accompanying the changes in the filler particles, significant improvements in the physical properties of the resin matrix have been realized. Improved polymerization resulted from the introduction of the visible-light-cured system (Pollack & Blitzer, 1982). Further improvements in composite resins were achieved through increased filler loading and with the introduction of bimodal fillers (Söderholm, 1985).

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To date, four composite resins have met the American Dental Association's requirements for provisional acceptance as posterior direct composite filling materials (Phillips, Lutz & Setcos, 1986). The ADA guidelines for provisional acceptance of composite resins for class 1 and class 2 restorations require at least two independent studies with a minimum observation time of three years. Full acceptance requires an observation period of five years. Evaluations are made to determine color stability, interfacial staining, anatomic form, recurrent caries, surface roughness, and interproximal dimension. Wear is also measured indirectly from casts of the restorations. No posterior composite material has achieved full acceptance as of this date.

As a restorative material, composite resins have many attractive features. Beyond their obvious esthetic superiority to metallic restorative materials, other desirable features include low thermal conductivity, the ability to mechanically bond to properly treated enamel, and the elimination of metallic galvanic currents. Resins, especially visible-light-cured composites, develop high early strength and are therefore resistant to early fracture due to the forces of mastication. Prior to polymerization, visible-light-cured composites can be shaped and reformed to obtain ideal contour. Furthermore, these restorations may be repaired if necessary. Although numerous studies have reported that properly placed resin restorations can strengthen remaining tooth structure (Bell, Smith & dePont, 1982; Share, Mishell & Nathanson, 1982; Simonsen, Barouch & Gelb, 1983; Landy & Simonsen, 1984; Mishell, Share & Nathanson, 1984; Newman & Pisko-Dubienski, 1984; Eakle, 1985; Bakke & others, 1985; Morin, DeLong & Douglas, 1984; Joynt & others, 1985; Moulder, Ogle & Hood, 1985; Stampalia & others, 1986; Herrin, 1986; Watts, 1986), it has recently been reported that this reinforcement may decrease with time and thermocycling (Eakle, 1986).

In spite of the advances that have been made, many clinical and material limitations have restricted the universal use of composite resin as a posterior restorative material. Resin restorations are technically difficult and require more time for placement than similar amalgam restorations (Dilley & others, 1985). Approximal contacts are difficult to obtain; in fact, two recent independent studies reported weak or defective approximal contacts in 20% of the posterior composite restorations placed (Boksman & others, 1986; Wilson, Wilson & Smith, 1985). The entire adhesive technique requires strict isolation from moisture contamination since any contamination of the etched surface reduces bond strength (Silverstone, Hicks & Featherstone, 1985). Tooth sensitivity following placement of composites further complicates their clinical use and may be due to improper placement of the acid during etching or to the polymerization shrinkage which leads to opening at the gingival margin of class 2 restorations (Boksman & others, 1986). Inadequate marginal seal is a more serious problem with composites than amalgams since resins do not corrode and seal at the marginal interface as amalgams do.

Composite resin as a posterior restorative material has several limitations. One of the most serious is a large polymerization shrinkage which averages two volume per cent for visible-light-cured composites (Goldman, 1983). This shrinkage contributes to internal stress in the resin, causes gap formation at the gingival wall of class 2 and class 5 restorations, and can produce cracks in the enamel at the margin (Bowen, 1967; Asmussen & Jørgensen, 1972; Bowen, Nemoto & Rapson, 1983; Davidson & deGee, 1984; Hansen, 1982). Although somewhat controversial, the incremental curing technique may decrease marginal opening occurring as a result of polymerization shrinkage (Podshadley, Gullett & Binkley, 1985; Crim & Chapman, 1986; Lutz, Krejci & Oldenburg, 1986; Gordon & others, 1986; Podshadley, Gullett & Crim, 1985; Eick & Welch, 1986; McConnell & others, 1986; Leclaire & others, 1986; Krejci, Lutz & Zwicky, 1987). Water sorption of composite resins may reduce the wear resistance of the restoration since the water absorbed by the polymer matrix can cause filler debonding or hydrolytic filler degradation (Söderholm, 1983). Wear may also be related to polymerization shrinkage (Jensen & Chan, 1985), fatigue (McCabe & Ogden, 1987), chemical degradation of the resin matrix (McKinney & Wu, 1985), porosity of the composite (Ogden, 1985), and finishing technique (Ratanapridakul, Leinfelder & Thomas, 1987).

Although accurate measurement of this wear has been difficult, many different methods have been found to reach the same conclusion: com-

posite resin restorations exhibit greater loss of anatomical form than metallic restorations. The US Public Health Service method of evaluation (Cvar & Ryge, 1971) was the first means of direct evaluation of composite wear. Since the Ryge criteria for evaluating wear are relatively insensitive, more recently developed indirect methods are currently used (Taylor, Turnbull & Leinfelder, 1984; Bangerter, Christensen & Christensen, 1987; Boksman & others, 1987; Taylor & others, 1987). The indirect methods have demonstrated that the wear of the composites Isopast, Silar, P-10, Ful-Fil, Occlusin, and Miradapt ranged from 12 - 105 μm per year, while amalgam wear varied from 6 - 58 μm per year, and enamel wear ranged from 3 - 54 μm per year (Leinfelder, Wilder & Teixeira, 1986; Heymann & others, 1986, 1987; Braem & others, 1986; Ameye, Roulet & Lutz, 1984; Dogon & Van Leeuwen, 1983; Mitchem & Gronas, 1985; Sturdevant & others, 1986; Lambrechts, Braem & Vanherle, 1987). These studies clearly demonstrate increased wear for composite restorations. Additional wear occurs with increased restoration size, with more distal placement in the dental arch (Bayne & others, 1987), and in stress-bearing areas (Braem & others, 1987).

Posterior composite restorations may require the use of new cavity preparations to increase their potential for success. Most techniques for the placement of posterior composite resin advocate a conservative cavity preparation with an isthmus width of one-fourth the intercuspal distance or less. Recently, several new preparation techniques have been advocated which are designed to utilize more fully the properties of composite resin restorations (Lutz, Krejci & Oldenburg, 1986; Oliveira, Covey & Denehy, 1986).

The use of composite resin has expanded as new areas for its use have evolved. The value of resin sealants in preventing pit-and-fissure caries is well documented (Mertz-Fairhurst, Schuster & Fairhurst, 1986). Resins may be also be applied to pits and fissures with shallow enamel preparations using the "preventive resin restoration" (Simonsen, 1978, 1980; Swift & Quiroz, 1987). Recently this technique has been expanded to include the restoration of incipient lesions, areas of enamel decalcification, and enamel craze fractures on smooth surfaces; this technique combines resin sealants or resin dentin-enamel bonding agents with highly filled composite resin (Houpt & others, 1986; Croll, 1987). These cosmetic restorations, which are applied to prepared tooth surfaces, have increased wear resistance. However, since their exact longevity is not known, continued follow-up is necessary to ensure their success.

In spite of its problems, composite resin may be indicated in the following selected posterior areas:

1. Preventive resin restoration. Certainly one of the greatest uses of composite resins is as a preventive sealant for pits, fissures, and decalcified enamel (Simonsen, 1982).

2. Class 1 and class 2 restorations in primary teeth (Leinfelder & Vann, 1982; Hills, 1986).

3. As a temporary restoration. Resin may be used when temporarily restoring teeth which have incomplete tooth fractures or have been treated endodontically.

4. Conservative class 1 or class 2 restorations in the permanent dentition. May be used when esthetics is an important primary consideration of the patient and centric stops can be maintained on enamel.

These recommendations do not imply that composite resins should be used as an unlimited substitute for amalgam. Clinical results to date do not substantiate the use of resins in large restorations or in heavy stress-bearing areas. Patients should be advised that composite resin restorations are not substitutes for metallic restorations.

Posterior composite materials have improved greatly in recent years and their future is promising; however, improvements remain to be made. The use of these materials requires learning and applying new techniques. Clearly these materials are not universal substitutes for amalgam or cast metal restorations. Clinical judgment must be used in case selection, and esthetics should be the primary reason for their use.

The opinions expressed herein are those of the authors and do not necessarily reflect the opinions of the United States Air Force or the Department of Defense.

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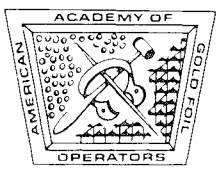

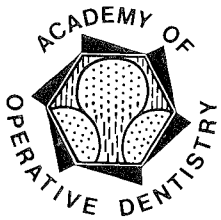

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