Preserving the Vital Pulp in Operative Dentistry: 3. Thickness of Remaining Cavity Dentine as a Key Mediator of Pulpal Injury and Repair Responses

PETER E. MURRAY, PHILIP J. LUMLEY AND ANTHONY J. SMITH

Abstract: **Confusion surrounds the pivotal role played by the remaining dentine thickness in a cavity in determining pulpal injury and repair response outcomes after restorative treatment. The multifactorial nature of the injury repair response requires that attention is focused on the most important factors, including remaining dentine thickness, to harness the natural regenerative properties of the pulp and to avoid postoperative treatment complications.**

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Clinical Relevance: **This information can be used to help minimize the onset of postoperative complications, without the need for a subjective approach, enabling practitioners to determine the success of treatment more accurately.**

The previous two papers in this series of articles on vital pulp therapy examined biological approaches to vital pulp therapy (Paper 1) and guidelines for successful restoration of unexposed dentinal lesions (Paper 2). This article focuses on the importance of the remaining dentine thickness (RDT) following cavity preparation. Understanding and being able to predict pulpal reactions to cavity preparation and restoration events has many advantages for the practitioner, one of the most obvious of which is the ability to define normal pulpal responses in terms of odontoblast cell survival, reactionary dentinal repair activity and pulpal inflammation. Deviations from these normal responses can help to identify postoperative complications. Identifying postoperative complications quickly is important as it provides the practitioner with the option to reattempt restorative treatment or prescribe a new course of therapy before the complications cause irreversible changes. The data used in this paper are derived from the sample of 383 restored human teeth described in the second article of this series.

***Peter E. Murray**, BSc, PhD, Research Fellow in Oral Biology, **Philip J Lumley** PhD, BDS, MDentSci, FDS RCPS, Senior Lecturer in Restorative Dentistry, and **Anthony J. Smith** BSc, PhD. Professor of Oral Biology, School of Dentistry, University of Birmingham.*

CAVITY PREPARATIONS AND PULPAL COMPLICATIONS

The importance of maximizing the RDT beneath cavity preparations in reducing pulpal damage has long been established, although the precise relationship between the degree of pulp injury and size of cavity preparation is unclear. The impact of this uncertainty on patient care has not been established, but with a recent longitudinal investigation showing a high rate of initially vital teeth becoming symptomatic after restoration and requiring endodontic treatment,[1] all aspects of tooth restoration must be examined. Recent evidence suggests that when creating cavity preparations the skill and expertise of the clinician in minimizing unnecessary iatrogenic dentine removal can reduce the probability of recurrent pulp complications.[2] However, many factors are reportedly associated with endodontic complications, and it is unclear how the combination of cavity preparation events, restoration, patient variables, microleakage and other pathological variables influence pulpal injury or repair responses.

SEQUENCE OF VARIABLES CORRELATED WITH CAVITY RDT

Our studies have shown that odontoblast survival and the extent of reactionary dentine secretion were the two pulpal responses most sensitive to cavity RDT (Figure 1). Pulp inflammation

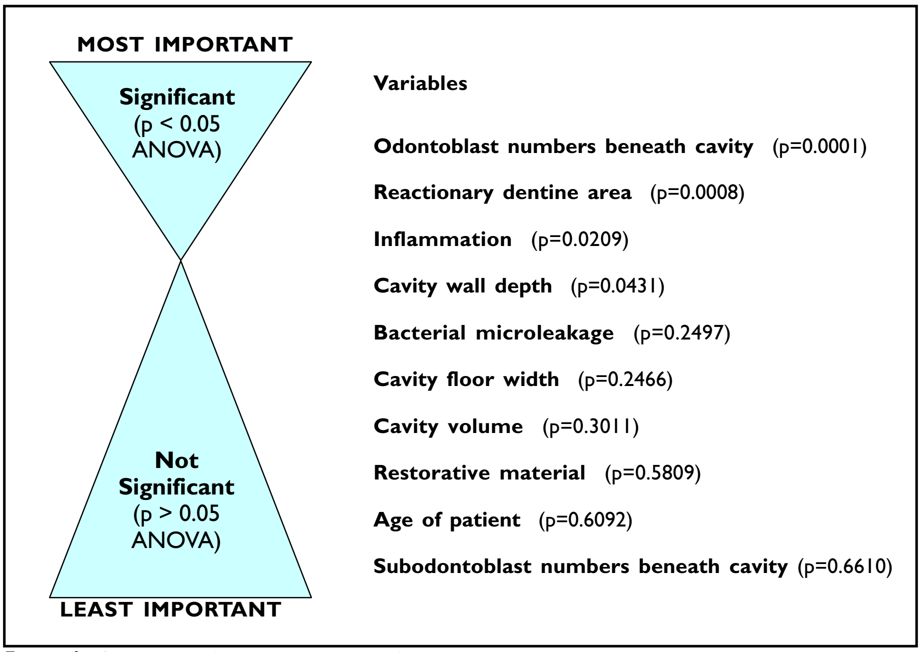

Figure 1. *Correlation of variables in order of importance according to the remaining dentine thickness of cavity preparations (ANOVA p value in brackets after each variable).*

and cavity wall depth were influenced to a lesser degree, and restorative factors were much less influenced by RDT (Figure 1). (The cavity wall depth depends on cavity positioning and tooth dentine thickness; this explains why cavity wall depth varies slightly in relation to RDT.) The effects of RDT on odontoblast survival, reactionary dentine repair activity and the severity of inflammation can be attributed to the degree of cellular injury to the odontoblast and to the protective buffering properties of dentine, which can modify the severity of the injury stimulus.

Dentine protects pulp from injury: deep cavities with small RDTs leave the pulp tissue less protected from preparation trauma, and from the chemical activity of dental materials. Over the years, estimates of the minimal cavity RDT that does not cause unequivocal pulp injury have been decreasing:

- in 1984, Stanley[3] suggested that a RDT of 2 mm would protect the pulp from most restorative procedures;
- in 1991, Pameijer, Stanley and Ecker[4] reported that a RDT of 1 mm or more would protect the pulp tissue from the cytotoxic effects of zinc phosphate and resin-modified glass ionomer materials during the luting process;
- within the past year, it has been suggested that these were cautious estimates, and deeper preparations, carefully cut down to 0.5 mm, appeared to have only limited effect on underlying odontoblast survival.[5]

ODONTOBLAST SURVIVAL AND CAVITY RDT

A decrease in cavity RDT tends to reduce the numbers of odontoblasts underlying the cavity preparations (Figure 2). For instance, in our study the numbers of odontoblasts beneath preparations cut with RDT of 1–3 mm were 17.5% higher than in deeper cavity preparation forms with a RDT of 0.5–1.0 mm (Figure 2). In deeper preparations, with RDT of 0.25–0.50 mm, odontoblast numbers were reduced by a further 11.1% (Figure 2), and in very deep cavity preparations, with RDT between 0.04 and 0.25 mm, odontoblast numbers fell by a further 31.7%. Overall, the reduction in odontoblast numbers beneath shallow and very deep cavity preparation forms was 49.8% (Figure 2). Consequently, to preserve odontoblast numbers (and possibly, by extrapolation, pulpal vitality) the depth of cavity preparation forms should minimize iatrogenic dentine removal. Minimizing

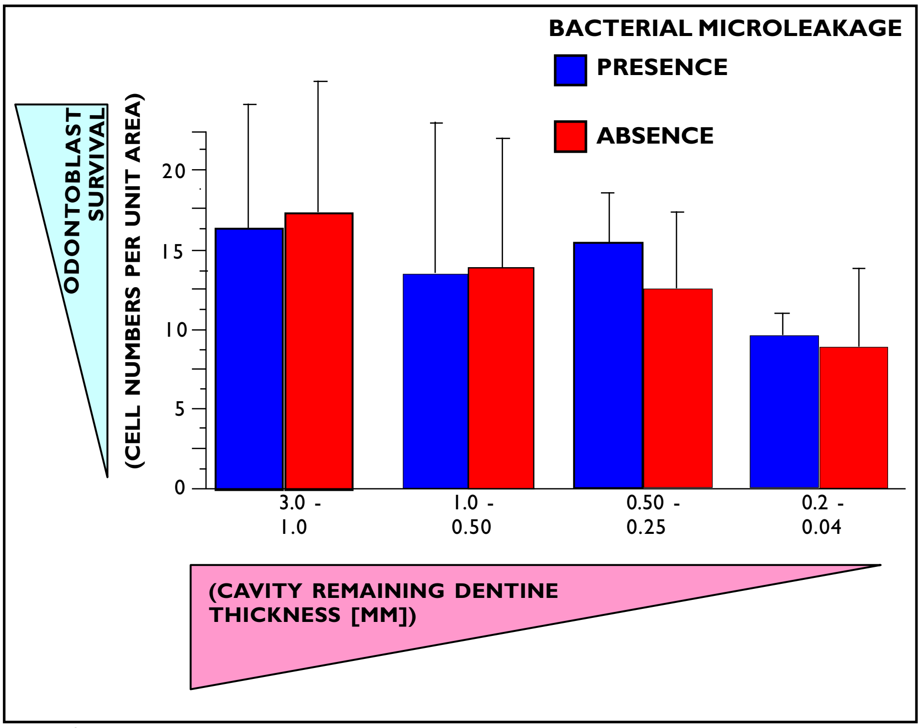

Figure 2. *Cavity remaining dentine thickness and odontoblast survival.*

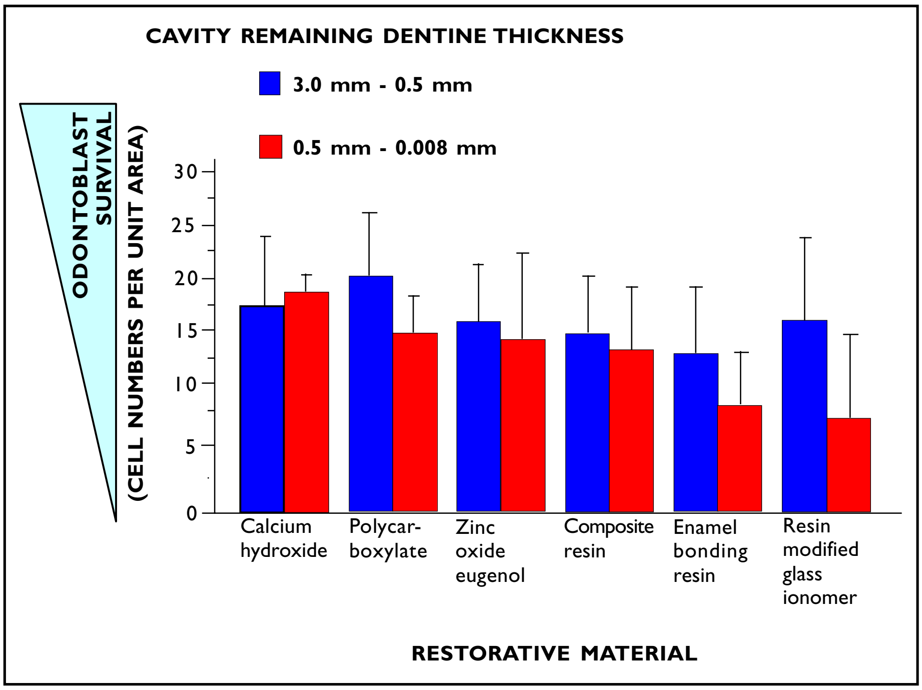

***Figure 3.** Cavity restoration materials and odontoblast survival.*

pulp injury will reduce the need for repair responses, which will reduce the probability of complications.

ODONTOBLAST NUMBERS AND RESTORATIVE MATERIALS

The type of restorative material appears to be a critical factor in mediating underlying odontoblast survival, in combination with the RDT (ANOVA p = 0.0001). We found that, using calcium hydroxide as a baseline measure of odontoblast survival, the ability of the materials tested to maintain odontoblast survival with RDT below 0.5 mm (see Figure 3) was as follows:

- calcium hydroxide 100%;
- polycarboxylate 81.1%;
- zinc oxide eugenol 78.4%;
- composite resin 74.2%;
- enamel-bonded resin 48.3%;
- resin-modified glass ionomer 43.1%.

These reductions in odontoblast numbers are in agreement with observations of pulpal injury following the restoration of very deep cavity preparations with resin-modified glass ionomers.[3] Because of these findings, it would appear sensible to consider using alternatives to resin-modified glass ionomers for filling when the RDT is small, in order to limit pulp injury.

Reductions in odontoblast survival appear to be correlated to the chemical activity of the lining or restoration material, because some materials are more cytotoxic to pulp tissue than others.[6] These observations highlight the importance of avoiding the placement of cytotoxic materials in very deep preparations, where unnecessary injury and possible necrosis of underlying vital pulp tissue must be prevented.

Although some reduction in odontoblast numbers may be largely unavoidable following the trauma of cavity preparation (Figure 2), the placement of enamel-bonded resin and resin-modified glass ionomers within 0.5 mm of the pulp tissue appears to reduce odontoblast numbers by more than 50% (Figure 3). Consequently, we would recommend the use of less cytotoxic materials, and the placement of a thin lining of calcium hydroxide to the cavity floor of deep preparations before applying a resin-modified glass ionomer as being the best way of avoiding needless odontoblast injury.

PULPAL INFLAMMATION

Several investigators have reported that the severity of pulp inflammation increases in response to a decrease in RDT, and the cutting of cavity preparations with a RDT below 0.3 mm has been reported to be associated with persistent pulp inflammation (pulpitis).[7] Pulpitis can injure the pulp cell populations, and may be linked to hypersensitivity and necrosis; thus it is important to understand the effect of RDT on levels of pulp inflammation.

The severity of pulpal inflammatory activity was always observed to be greater in the presence of bacterial microleakage than in non-infected cavity restorations (Figure 4). Furthermore, preparations with RDTs below 0.25 mm have more severe forms of inflammatory activity than preparations with greater RDTs (Figure 4). In cavity preparations contaminated by bacterial microleakage, and with RDTs below 0.25 mm, severe inflammation is always observed (Figure 4). Increasing inflammatory activity with decreasing RDT may well be due to severe levels of cavity preparation trauma, together with a lack of dentine to provide pulp protection from the chemical effects of restorative materials and microbial metabolism.

These findings support the hypothesis that inflammation is not solely due to bacterial microleakage: sensory nerve fibres, chemical inflammatory mediators and other biological processes are also likely to be involved.[8]

REACTIONARY DENTINE ACTIVITY

There is no artificial material that can be placed into a tooth which provides better protection for the pulp than dentine.[9] For this reason, attention continues to focus on the stimulation of reactionary dentine (see Paper 1). Dentine repair is clinically advantageous in terms of reducing or avoiding recurrent pulpal complications and offers considerable advantages over the use of artificial materials.[2] An understanding of the factors influencing reactionary dentinogenesis is critical to

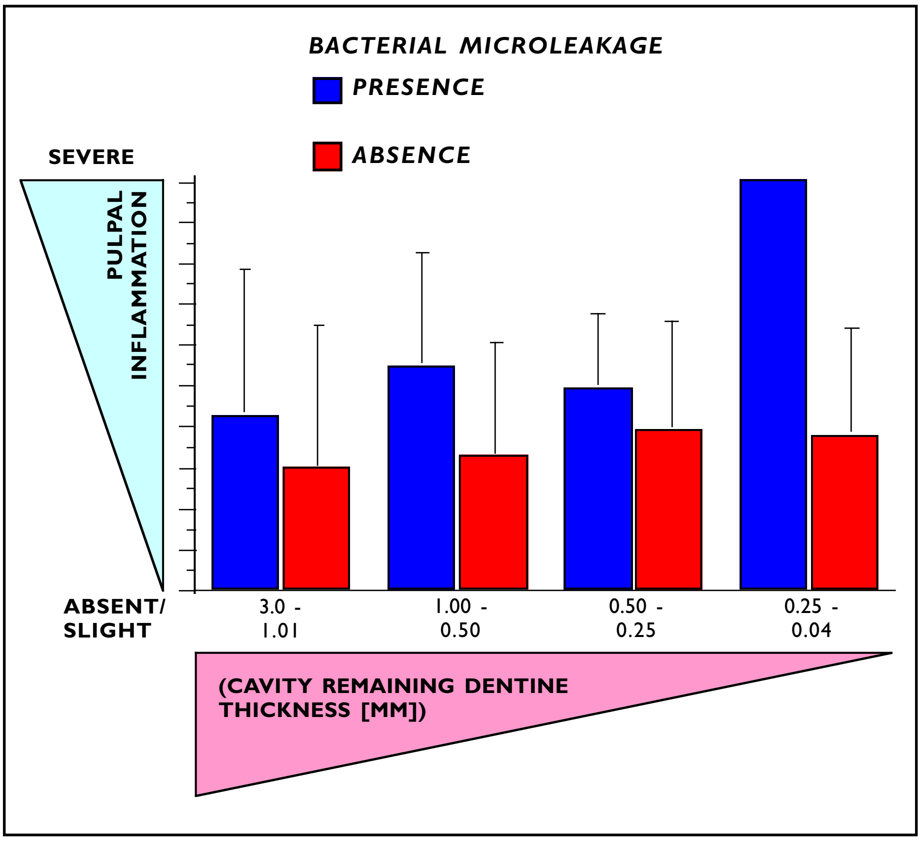

***Figure 4.** Cavity remaining dentine thickness and pulp inflammation.*

ensuring that restorative treatments are congruent with the natural repair processes in the tooth. Several mechanisms have been postulated to stimulate formation of reactionary dentine:

- cavity preparation trauma;
- restorative dental procedures;
- operator hand instrumentation;
- pathogenic diseases;
- caries;
- attrition;
- erosion;
- chemicals;
- restorative materials; and
- bacterial microleakage.

A common factor in all of these observations has been an injury, chemical or disease stimulus advancing towards the pulp. In contrast to the lack of consensus on the stimulation of reactionary dentine, the role of reactionary dentine matrix in protecting the pulp from injury is clear: deposition of reactionary dentine increases the distance of the pulp from the noxious stimulus, causing an overall decrease in permeability. This assists in providing pulp protection and maintains pulp vitality from the injurious effects of stimuli.

The relationship between RDT and the secretion of reactionary dentine has become a source of controversy: some studies have found that the area of reactionary dentine deposited following cavity restoration increases gradually in response to reduction in RDT;[2] some have found uniform dentine repair activity in response to various RDTs;[10] yet others still have observed reactionary dentine repair only beneath cavities cut into the inner third of the dentine matrix, close to pulp tissue.[10] This range of observations may appear disparate, and irreconcilable; however, this is not the case. Depending on the RDTs selected for analysis, it is possible to identify reactionary dentine secretion at a uniform rate, decreasing with a reducing RDT, and increasing only within the inner third of the dentine matrix (Figure 5).

The RDT and secretion of reactionary dentine appear to be related (Table 1). The most important initiating factor in the secretion of reactionary dentine appears to be a cavity RDT between 0.25 mm and 0.50 mm (Figure 5): the mean rate of reactionary dentine in such a restoration contaminated with bacteria has been estimated to be nearly five times greater than the amount formed beneath similar preparations with other RDTs (Figure 5). In a similar comparison of uncontaminated preparations, and with RDT of 0.25—0.50 mm, the increase in reactionary dentine was found to be 29.5% (Figure 5).

The exact reason for this pattern of reactionary dentine secretion with RDT is not clear, but it provides an insight into the mechanisms that trigger odontoblast secretion of reactionary dentine. The mechanisms that may have a role in communicating dentine injury, or advancing disease or chemicals, are the odontoblast process or the contents of the tubule lumen,[2] although the most likely mechanism involves the solubilization of growth factors and bioactive molecules sequestered within the dentine matrix[2] (see article 1 in this series). Dentine injury, particularly following cavity preparation and acid etching, can solubilize endogenous growth factors from the matrix, which diffuse through the dentinal tubules to the odontoblast cells. The odontoblast cells respond to these growth factors by increasing the rate of secretion of dentine matrix, resulting in the appearance of reactionary dentine. The greatest areas of reactionary dentine in our studies were observed with RDTs between 0.25 mm and 0.50 mm. At these low RDTs, bioactive molecules appear to be able to diffuse to odontoblast cells more readily than with thicknesses above 0.5 mm. The presence of bacteria may exacerbate release of growth factors from the matrix through their acidogenic activity.

However, this hypothesis of the regulation of reactionary dentine secretion does not explain why the

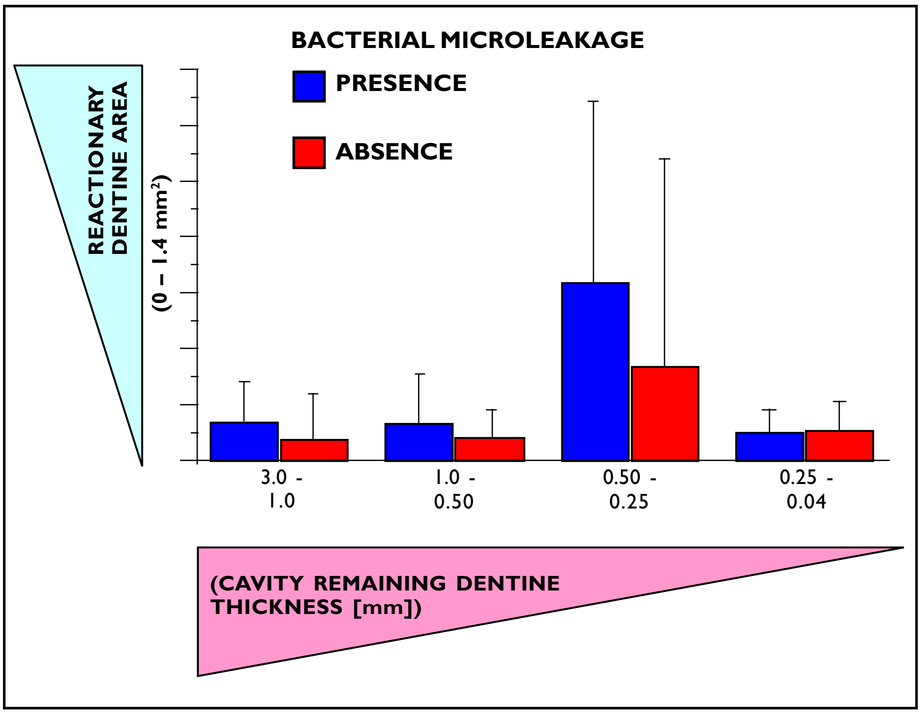

Figure 5. *Cavity remaining dentine thickness and reactionary dentine area.*

maximal rate of reactionary dentine does not take place with RDTs below 0.25 mm, because bioactive molecules should be able to diffuse to odontoblasts more readily through this shorter tubule distance. The most likely reason for reduction in secretion of reactionary dentine with RDTs below 0.25 mm is that odontoblasts have been injured to a greater extent by the cutting of these deep cavity preparations, which impairs dentine secretory activity. Evidence of odontoblast injury following deep cavity preparation is given in Figure 1, where it is clear that RDTs below 0.25 mm are associated with the greatest loss of odontoblast cell numbers. This suggests that the cutting of very deep cavity preparations injures the underlying odontoblasts, impairing their ability to secrete reactionary dentine.

The effect of cavity restoration material on reactionary dentine activity has similarly proved controversial. Some materials, such as calcium hydroxide, have been reported to stimulate increased reactionary dentine deposition,[3] whereas other studies suggest that the effects of the restorative material are minimal.[11] Our present study has demonstrated a relationship between the extent of reactionary dentine secretion and cavity restoration materials; however, this relationship appears to be heavily influenced by the RDT of the cavity preparations (Figure 6). In general, most restorative materials appeared to cause greater reactionary dentine secretion with a cavity RDT below 0.5 mm (Figure 6). The rank order of reactionary dentine secreted in preparations with an RDT below 0.5 mm was:

- calcium hydroxide;
- composite resin;
- enamel-bonded resin;
- resin-modified glass ionomers;
- zinc oxide eugenol;
- polycarboxylate.

This finding is in agreement with previous studies that have shown that calcium hydroxide is associated with greater deposition of reactionary dentine than other restorative materials such as zinc oxide eugenol.[12]

The reasons why some restorative materials are associated with larger areas of reactionary dentine secretion is still unclear. The difference in degree of stimulation of odontoblasts and pulp tissue, caused by differences in the cytotoxicity and chemical activity of restorative materials (see paper 1 in this series)[6] may provide an explanation, but much further investigation is required.

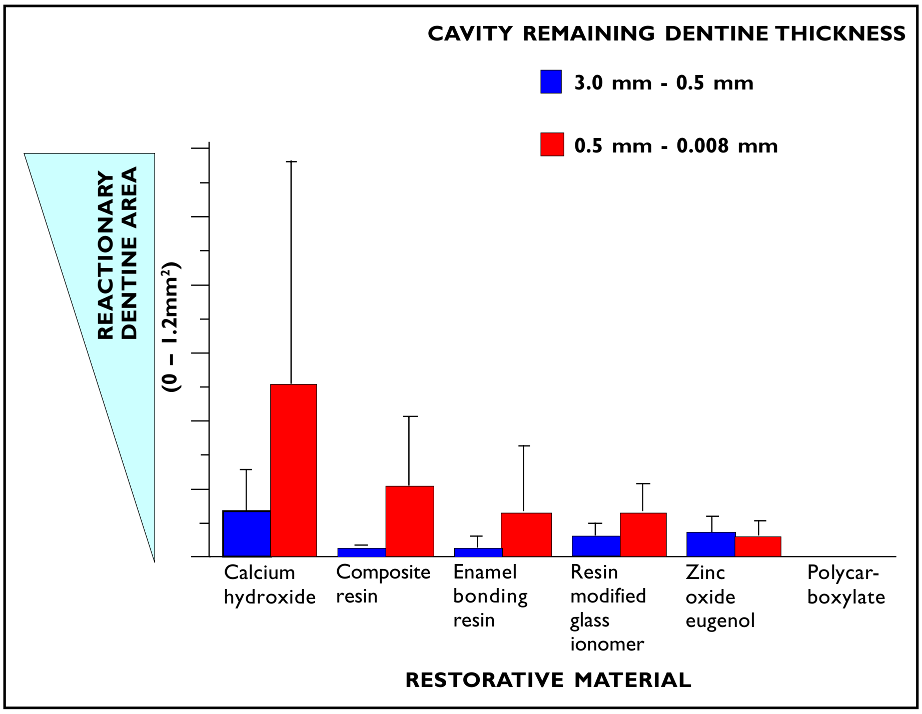

Figure 6. *Reactionary dentine and restorative materials.*

REMOVAL OF CARIES

In the management of caries it is difficult to achieve a balance between over-prescription and supervised neglect. This is an issue for treatment planning. Regardless of treatment strategy, the success of capping of exposed pulp is in the range of 44—97%, while the success of capping of non-exposed teeth is generally much higher, varying with different types of materials and conditions. The sizeable difference between the success of exposed and non-exposed pulp capping restorations means that it is worth while making every effort to avoid pulp exposure.

Pulp exposure may be avoided during caries removal by using indirect pulp capping in symptomless vital teeth, rather than removing all discoloured carious dentine and sound tooth structure. This method involves completely removing caries at the enamel/dentine junction because no restorative material forms a perfect cavity seal and the disease may progress more readily if caries remains in this area. Stained but firm dentine should be left in the preparation floor, to avoid pulp exposure.

FUTURE DIRECTIONS

This study has highlighted the complex interplay between cavity RDT, restorative materials, bacterial microleakage and pulp inflammation. Cavity RDT plays a central role in determining the extent of pulpal injury and repair responses.

Although the mechanisms of secretion of odontoblast reactionary dentine remain incompletely understood, the ability to anticipate and manipulate the activity of this material should form part of treatment planning. The basis for a biological approach to clinical practice would be to identify and attempt to reduce sources of tissue injury, and also the dentine repair capacity of teeth intentionally as part of treatment. This should help to minimize early postoperative complications following restorative treatment. Long-term complications are more difficult to predict and readers should interpret this data with some caution. To aid appreciation of pulp dentine responses to the RDT of preparations, we have shown a summary in Table 1.

Cavity RDT	Cavity type	Odontoblast survival (%)	Reactionary dentine formation	Pulp inflammation activity
>1 mm	Shallow	100	Slight	Minimal
0.5–1.0 mm	Moderate	88.9	Slight	Minimal
0.25–0.50 mm	Deep	82.5	Significant (292% increase)	Increasing
<0.25 mm	Very deep	68.3	Slight	Most severe with bacterial microleakage

Table 1. *The effect of the cavity remaining dentine thickness on odontoblast survival, reactionary dentine activity and pulp inflammation.*

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