

Preserving the Vital Pulp in Operative Dentistry: 2. Guidelines for Successful Restoration of Unexposed Dentinal Lesions

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

Abstract: The exciting treatment possibilities arising from tissue engineering approaches are still some years away from involvement in dentistry. Meanwhile, it is important to optimize conventional treatments, although precise information on pulp responses to cavity preparation and restoration variables are limited. Odontoblast survival, pulp inflammation, and tertiary dentine area are used as measures of pulp injury and repair.

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Clinical Relevance: This information can be used to help minimize the onset of postoperative complications and focus practitioner attention on aspects of treatment that are most likely to benefit outcomes.

Tissue engineering approaches are viewed as having enormous clinical potential (reviewed in the first paper of this series). The everyday transplantation of cultured tissues and the use of bioactive molecules and gene therapy are anticipated to have a considerable impact on dental practice over the next 10 years, with the repair and replacement of mineralized tissues, the promotion of oral wound healing and the use of gene transfer therapy being likely to produce the greatest benefits.¹ However, before these approaches can be used routinely, a number of ethical, safety and cost

implications must be dealt with. In the meantime, we should consider how aspects of more traditional restorative treatment can lead to success or failure.

CLINICAL GUIDANCE ON EFFECTS OF RESTORATIVE VARIABLES

The biological and clinical aspects of vital pulp therapy have been investigated over the past four decades, although precise information on pulp responses to preparation and restoration events remain limited.² This situation is indicative of the complexity of pulp responses, which can be sensitive to caries and pathological events, trauma of cavity preparation, the chemical activity of dental materials, bacterial microleakage and postoperative inflammation, patient factors and treatment history.

The relationships between these factors are not well understood; nevertheless, the successful outcome of restorative treatments depends on making treatment decisions that are congruent with the natural repair responses of the tooth.³ To identify the variables with greatest influence on mediating postoperative complications, we have investigated aspects of cavity preparation and restoration events³⁻⁵ in a large sample of restored human teeth (383). We have focused on pulpal reactions, including injury, repair, bacterial microleakage and inflammation over an extended postoperative period (7-381 days) to observe a broad picture of the overall responses. In quantifying these pulpal reactions, we have performed statistical correlations with cavity preparation, restoration and patient factor variables to provide a basis for treatment planning. This should allow attention to be focused on the factors that are most important in reducing the frequency and severity of short-term postoperative complications.

QUANTIFICATION OF PULP INJURY

It is difficult to quantify pulp injury in response to cavity preparation and restoration events, and qualitative approaches have commonly been adopted. However, these methods are imprecise and have largely prevented

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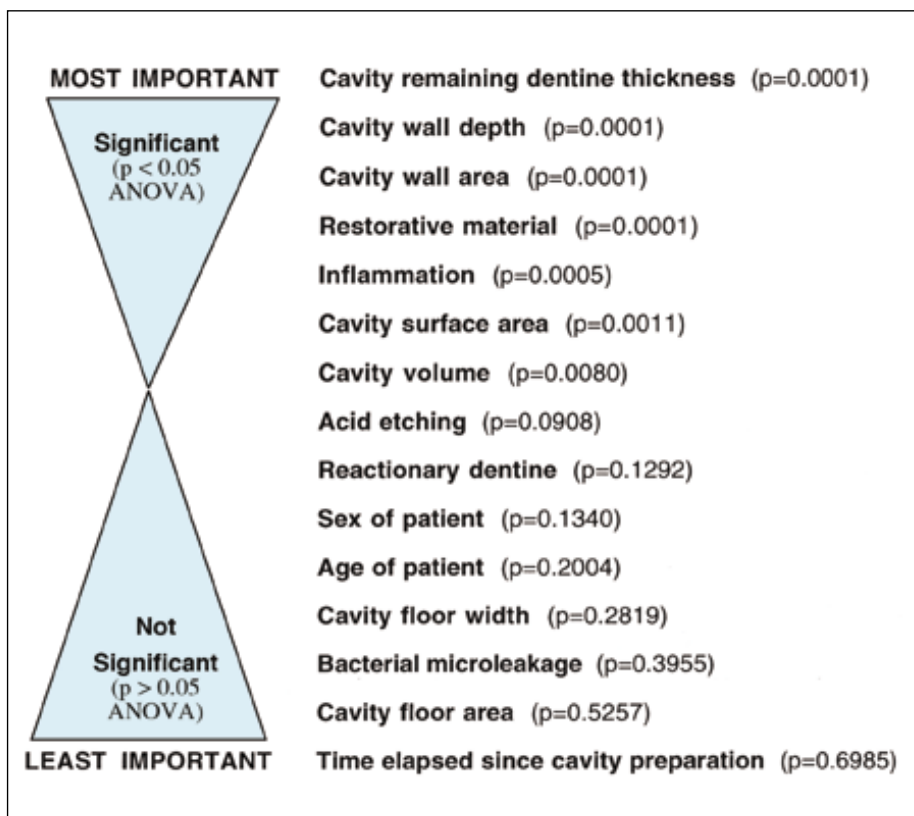


Figure 1. Correlation of variables in order of importance for reducing odontoblast numbers and causing pulp injury (ANOVA p value in brackets after each variable).

the establishment of correlations between the severity of pulp injury, individual cavity preparation variables and restoration factors. Because of this, we have employed the numbers of odontoblasts beneath the site of cavity preparation as a measure of pulp injury. This type of analysis has allowed the degree of pulp injury to be measured on a quantitative scale, and the accuracy of this approach has made it possible to correlate pulp injury with individual cavity preparation and restorative events (Figure 1).

ODONTOBLASTS AND PULPAL CELL POPULATIONS

A key aspect of successful restorative treatment is the ability to use the natural repair responses of the pulpal cell populations as a part of treatment.⁴ The repair of dentine is accomplished by the secretory activity

of odontoblast cells. These are highly differentiated post-mitotic cells which regulate dentine synthesis, secretion and mineralization throughout life. The two other major pulp cell populations, the cells of the sub-odontoblast layer and the fibroblasts of the pulp core, are important in supporting odontoblast activity but do not appear to play a direct role in the secretion of dentine matrix.

SEQUENCE OF VARIABLES MEDIATING ODONTOBLAST NUMBERS

In the event of dentine damage by pathological, traumatic or restorative events, the intrinsic capacity of the dental pulp to repair lost or damaged dentine, and to increase the barrier between itself and the injurious stimuli, is dependent on the vitality of the odontoblast cell layer.⁴ The relationship between odontoblast numbers, pulp inflammation and

restorative materials shows that odontoblast numbers are most conserved in the absence of, or with only slight, pulp inflammation and cavity restoration with calcium hydroxide (Figure 2). In contrast, odontoblast numbers are least conserved with moderate or severe pulp inflammation, following restoration with resin-modified glass ionomer (Figure 2). Avoidance of inflammation and selection of restorative materials that minimize pulp injury are therefore important goals during cavity restoration.

Odontoblast survival is sensitive to:

- the cavity remaining dentine thickness;
- cavity wall depth;
- cavity wall area;
- restorative material used;
- inflammation;
- cavity surface area; and
- cavity volume (Figure 1).

This sequence of correlations indicates that these variables are more important in causing a loss of odontoblast survival than some other cavity preparation and patient variables (Figure 1). Increasing the scale of cavity preparation has a tendency to reduce the odontoblast survival. The reduction in odontoblast numbers with increasing cavity dimensions might suggest that it may be due to the increasing diffusion of potentially injurious cytotoxins from restorative materials in a pulpward direction. However, the limited influence of the cavity floor width and cavity floor area does not appear to support this hypothesis (Figure 1). Consequently, consideration must be given to the adverse effects of trauma during cavity cutting, of which heat has been suggested to be the most injurious. The amount of intrapulpal heat generated during cavity preparation is determined by:

- the drill rotation speed;
- size, type and shape of the cutting instrument;
- length of time the instrument is in

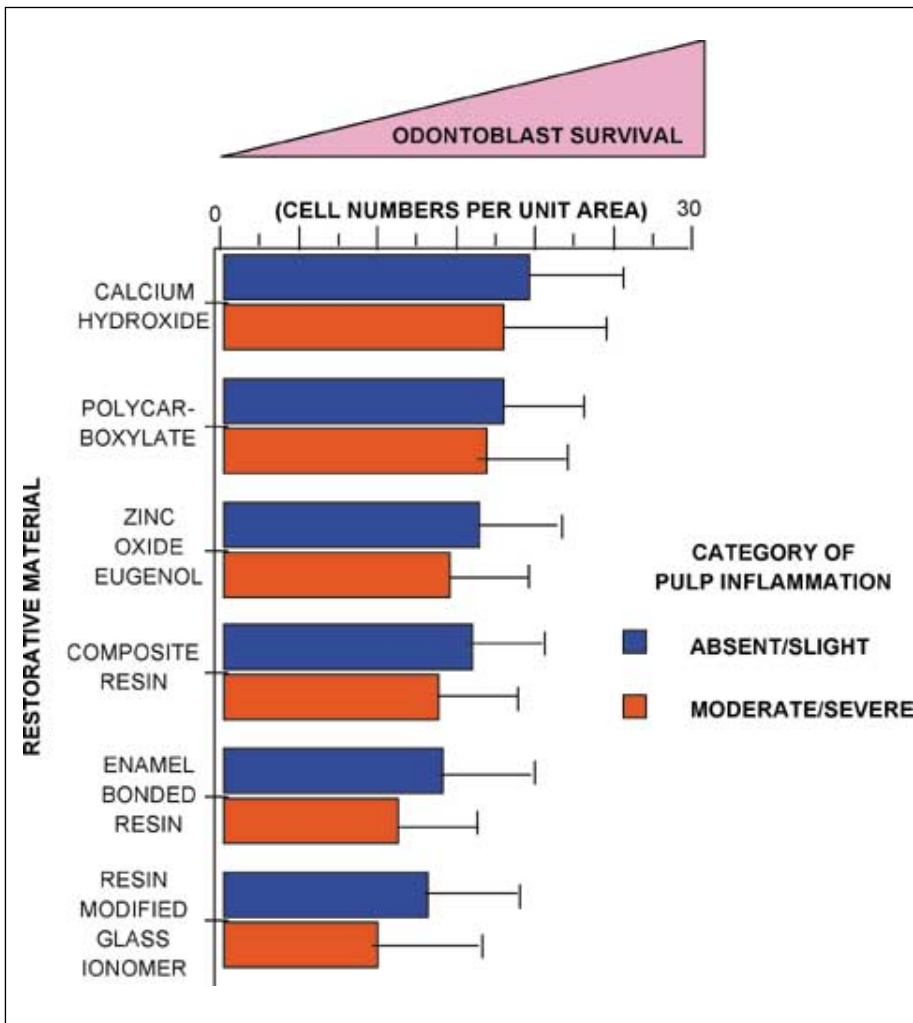


Figure 2. Odontoblast survival and restorative materials.

- contact with dentine;
- the amount of pressure exerted on the handpiece;
- cutting technique; and
- the use of coolants.

DENTINOGENIC REPAIR ACTIVITY

Dentine matrix secretion can be classified as primary, secondary or tertiary in origin according to the chronology and circumstances of its secretion by odontoblasts. In humans, primary dentine is secreted at a rate of 4 microns per day during tooth development, until the completion of root formation. Thereafter, physiological secondary dentine is laid down at a reducing rate of approximately 0.5 microns per day

along the pulp–dentine border throughout life. In the event of primary and secondary dentine injury, the intrinsic capacity of the dental pulp to repair lost or damaged dentine, and to increase the barrier between itself and the injurious stimuli, depends on the ability of the odontoblast cell layer to secrete tertiary dentine. Tertiary dentine can be classified as being reactionary in origin, depending on the severity of the initial response and conditions under which the dentine matrix was secreted (see previous paper).⁶ If odontoblasts survive the trauma of primary and secondary dentine injury, these cells secrete new tertiary dentine matrix, termed reactionary dentine.⁷ If the odontoblasts are destroyed by injury, they are replaced by a new

generation of odontoblast-like cells which secrete a dentine matrix termed reparative dentine⁷ (this will be discussed further in the fourth paper of this series). Reactionary dentine repair activity appears to be influenced by:

- cavity surface area;
- cavity volume;
- cavity wall area;
- remaining thickness of dentine in the cavity;
- cavity floor area;
- cavity floor width;
- cavity wall depth;
- the presence of bacterial microleakage; and
- acid etching treatment (Figure 3).

Other cavity restoration events and patient factors appear to be less influential (Figure 3).

The sequence of effects correlated with reactionary dentine area also appears to be closely related to the sequence of events related to odontoblast survival and pulp injury (Figure 1), which suggests that reactionary dentine secretion is regulated to respond to the severity of pulp injury. All the most influential mediators of pulp repair appear to be related to the size of cavity cutting. Following cavity restoration, the area of reactionary dentine also appears to be correlated with different restorative materials (Figure 4). Calcium hydroxide was associated with the greatest area of reactionary dentine, whilst no reactionary dentine deposition was observed following cavity restoration with zinc polycarboxylate (Figure 4).

CAUSES OF RESTORATION FAILURE

There are various reasons for the failure of restorations, such as tooth fracture, marginal fracture, degradation by abrasion, attrition, erosion, non-carious defects, diet and oral hygiene characteristics of each patient. Recent surveys in the USA, Europe and the UK have overwhelmingly shown that the most frequent reasons for failure are surgical trauma and bacterial

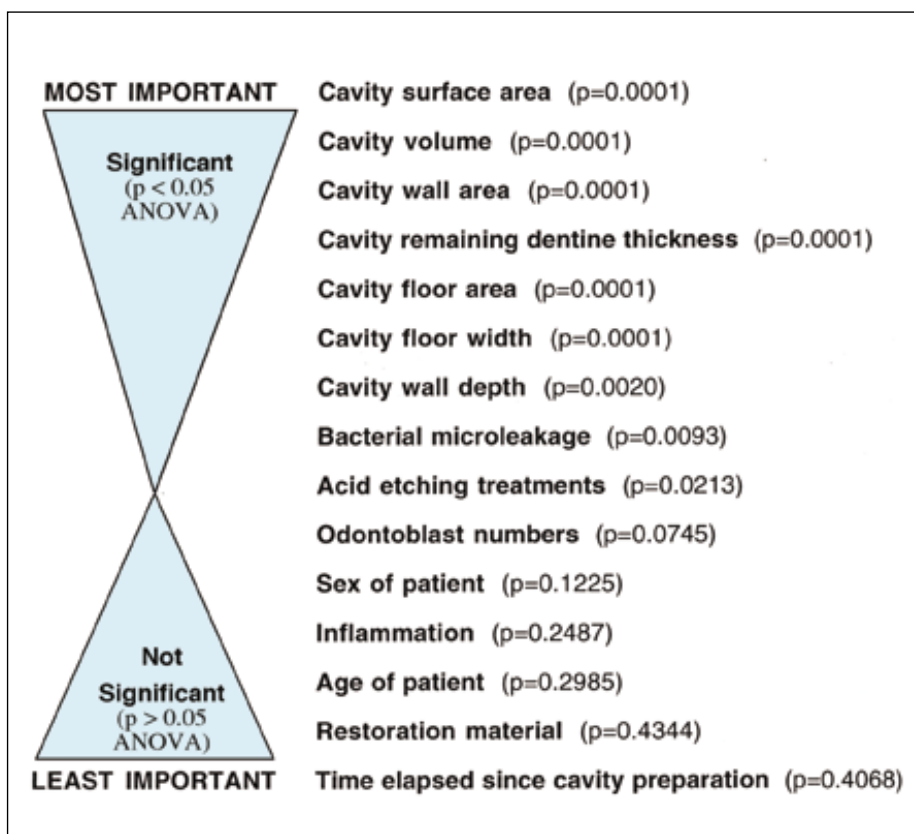


Figure 3. Correlation of variables in order of importance for influencing reactionary dentine and pulp repair activity (ANOVA p value in brackets after each variable).

microleakage into cavity margins.⁸

Microleakage complications include:

- postoperative sensitivity;
- marginal discoloration;
- recurrent caries;
- pulp inflammation;
- pulp necrosis; and
- the eventual need for endodontic therapy.⁹

Although the diet and oral hygiene characteristics of patients play a considerable role in restoration longevity, several surveys have shown that the selection of one restorative material in preference to another can make the critical difference between success and failure within a few years.⁸

BACTERIAL MICROLEAKAGE

Patient confidence is promoted by the placement of long-lasting restorations

that do not cause recurrent pulp inflammation, allergic hypersensitivity, or symptoms requiring endodontic treatment. Recognition that the largest proportion of restoration failures and postoperative complications result from bacterial microleakage has led to developments in dental materials that improve their antimicroleakage characteristics and longevity. In general, this has made the placement of new generations of restorative materials more complex and, contrary to some expectations, issues of technique sensitivity and surgical skill remain extremely important for influencing treatment outcomes.

The dimensions of cavity preparation appear to be correlated to the incidence of bacterial microleakage (Figure 5): fewer numbers of bacteria were detected in cavities with smaller restoration volumes (Figure 6). This demonstrates that the likelihood of detecting bacteria increases as the cavity preparation size increases, and

may be why replacement fillings have a shorter life expectancy than initial fillings – the increased dimensions of replacement restorations may increase the risk of bacterial microleakage. In agreement with these observations, Osborne and Gale¹⁰ postulated that tooth deflection under mastication may play a role in the onset of defects in the cavosurface margin, with larger restorations being most at risk from bacterial microleakage.

Choice of restorative material also has an important influence on bacterial microleakage (Table 1). Zinc oxide eugenol and resin-modified glass ionomer were found to prevent bacterial microleakage into all cavity restorations for up to one year following treatment, which can be attributed to antibacterial activity and direct sealing with cavity walls. However, the placement of enamel-bonded resin composite and adhesive-bonded resin composite did not seem to result in a perfect seal with cavity walls, because bacteria have been detected in 22% and 10%, respectively, of these restorations (Table 1). These observations confirm earlier reports that zinc oxide eugenol restorations have significantly lower bacterial

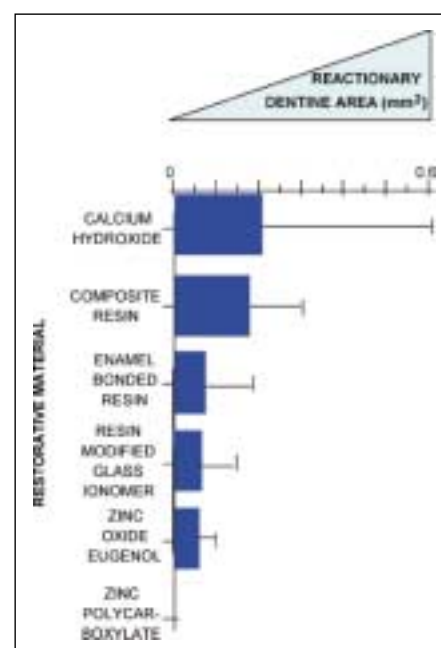


Figure 4. Reactionary dentine area and restorative materials.

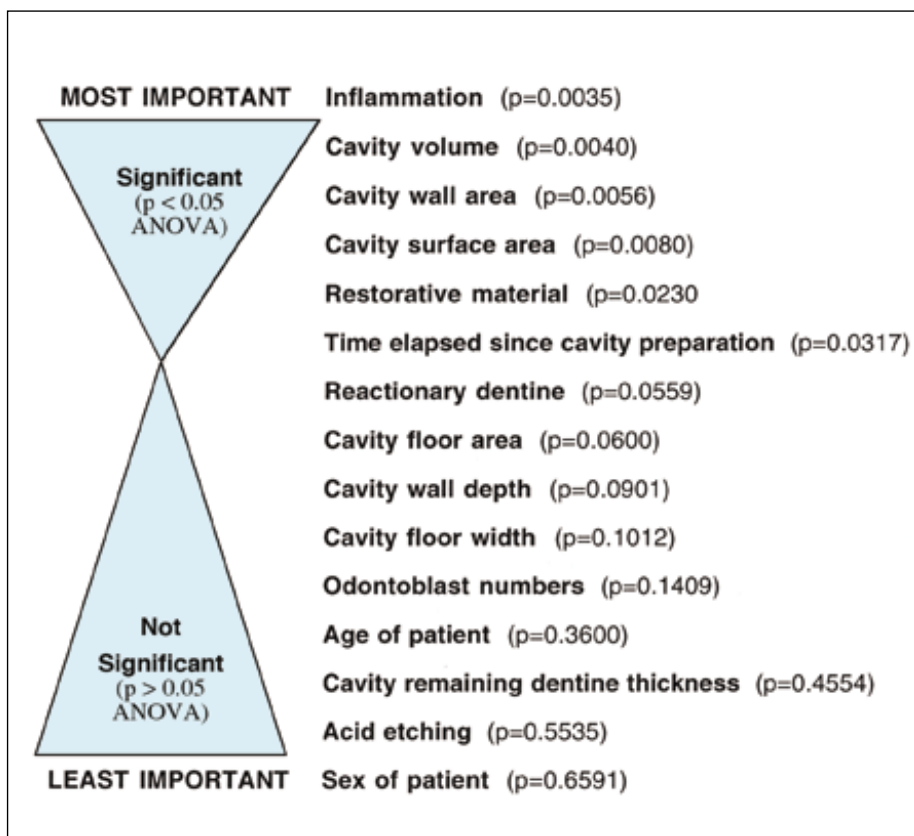


Figure 5. Correlation of variables in order of importance to bacterial microleakage (ANOVA p value in brackets after each variable).

counts than other comparable dental materials.⁵

In summary:

- The favourable sealing characteristics of resin-modified glass ionomer explain why it is particularly recommended for Class V cavities in caries-prone patients.
- The detection of bacteria beneath composite resin restorations demonstrates the continuing need to make improvements to the adherence and marginal sealing of these materials, by using sandwich placement techniques to reduce the effect of shrinkage during polymerization.
- The greater prevalence of bacteria in cavities following use of enamel-bonded resin composite suggests that inadequate or incomplete bonding to dentine may allow increased microleakage of bacteria through cavity margins.

PULPAL INFLAMMATION

The immune system triggers inflammatory reactions to limit tissue damage from invading or foreign molecules. Paradoxically, these inflammatory reactions can injure the pulpal cell populations, and lead to pulp complications in response to cavity restorations that may initially appear to be successful. Severe forms of inflammatory activity can develop into total pulpal necrosis, and periapical lesion development with local bone destruction. In less severe cases the inflamed pulp is associated with hypersensitivity, so that thermal, mechanical or osmotic stimuli encountered in normal function can cause intense pain. These observations explain why immunological inflammatory activity is associated with the high rates of primarily vital teeth exhibiting pulpal complications following cavity restoration.¹¹

The presence of bacterial microleakage appears to cause more

severe pulp inflammatory activity than in non-infected cavity restorations, at times between 3 and 364 days (Figure 7). Zinc oxide eugenol and resin-modified glass ionomer were able to prevent bacterial microleakage into cavity restorations, and no severe inflammatory activity was observed with these materials (Table 1). Bacteria were observed in cavities restored with resin composites, and associated with 7 and 11% of severe grades of inflammation (Table 1). Pulp inflammation was found to be influenced by most cavity preparation and restoration variables, only patient variables and reactionary dentine being not significantly correlated with inflammation (Figure 4). The most important variables (restorative material, cavity wall area, acid etching) all appeared to be related to bacterial microleakage, suggesting that the ability of restorative materials or acid etching techniques to prevent or minimize the progression of microleakage is extremely important.

RESTORATIVE MATERIALS

Selecting the type of restorative materials to optimize treatment

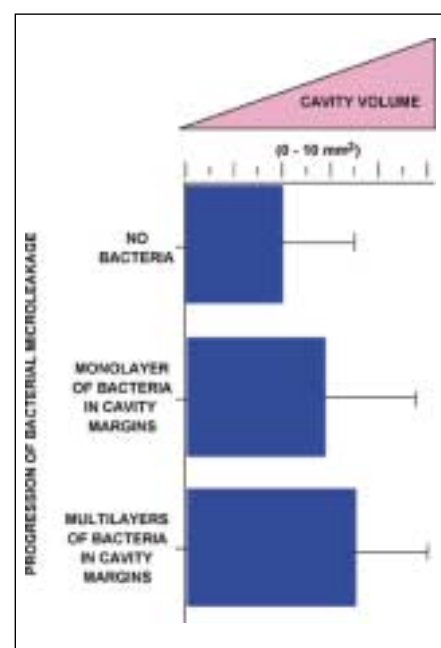


Figure 6. Cavity volume and progression of bacterial microleakage.

Restoration material	Cavities containing bacteria (%)	Grade of pulpal inflammation (%)		
		Slight	Moderate	Severe
Zinc oxide eugenol	0	91	9	0
Resin-modified glass ionomer	0	44	46	0
Enamel-bonded resin composite	22	34	59	7
Adhesive-bonded resin composite	10	35	54	11
Calcium hydroxide	ND	83	17	0

Table 1. Cavities restored with different classes of materials showing the presence of bacterial microleakage and different grades of inflammation.

outcomes on the basis of limited scientific data often proves problematic. Information on the success of materials is normally based on longevity surveys – but in these surveys the mode of restoration failure is often poorly characterized, and it is not possible to understand how cavity preparation and restoration events in addition to pulp reactions contributed to the development of complications and eventual restoration failure.

CONCLUSION

The methods presented here have shown how individual cavity preparation and restoration events and patient factors can be correlated to odontoblast numbers and pulp injury (Figure 1), reactionary dentine repair activity (Figure 3), bacterial microleakage (Figure 5), and pulp inflammation (Table 1). It has become clear that pulp responses are the summation of multifactorial events (Figures 8 and 9); however, the effects of individual variables are not uniform, which explains why we have presented each of the variables as a sequence. All of the work presented (and on which guidelines are based) has been conducted on initially caries-free teeth, although the guidelines should be interpreted in the context of the practitioner’s experience of treatment of caries. We postulate that concentrating attention

on the more important restoration variables will lead to the most productive improvements in at least short-term treatment outcomes.

Guidelines for successful pulp preservation appear to be dependent on the following treatment strategies:

1. Appropriate treatment planning according to patient age, health and history.
2. Cavity preparation using an intermittent cutting technique with light handpiece pressure, and directing water coolant at the site of cutting.

3. Removal of diseased and infected tissue to protect the pulp from additional pathological injury, but avoidance of iatrogenic dentine removal. This should maximize both the remaining dentine thickness beneath the cavity floor and the cavity width, to limit dentine-pulp injury from operative procedures.
4. Use of dams and techniques to avoid infection during the restorative phase of dental surgery.
5. Selection of materials that provide restoration longevity, and bacteriometric sealing with tooth structure, to avoid allergic hypersensitivity, recurrent pulp inflammation or symptoms that require endodontic treatment.
6. Placing of materials with an understanding of the importance of technique sensitivity, particularly with adhesive composite resins.

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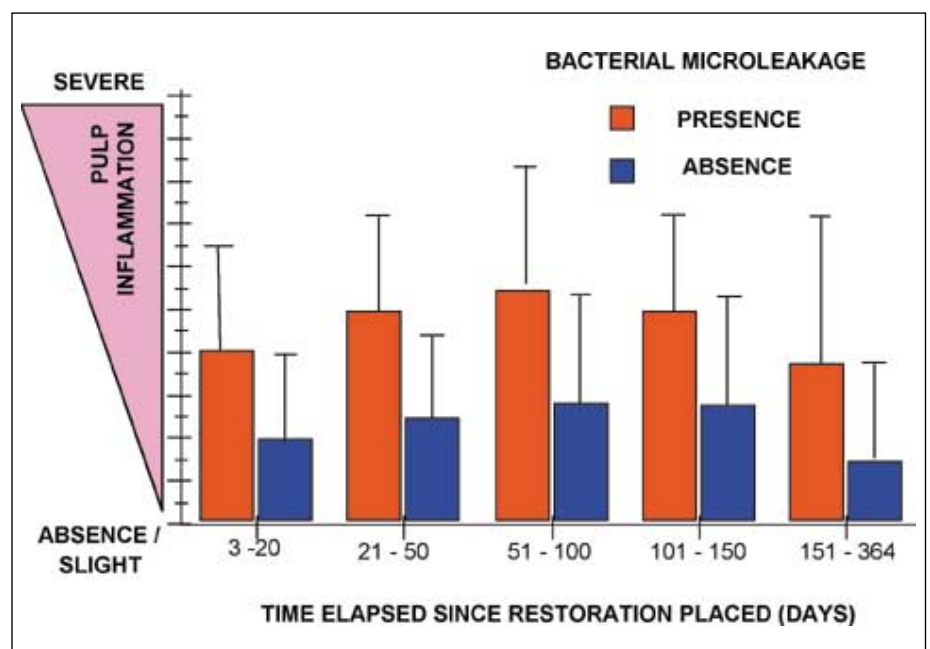


Figure 7. Pulp inflammation and the time elapsed since restoration.

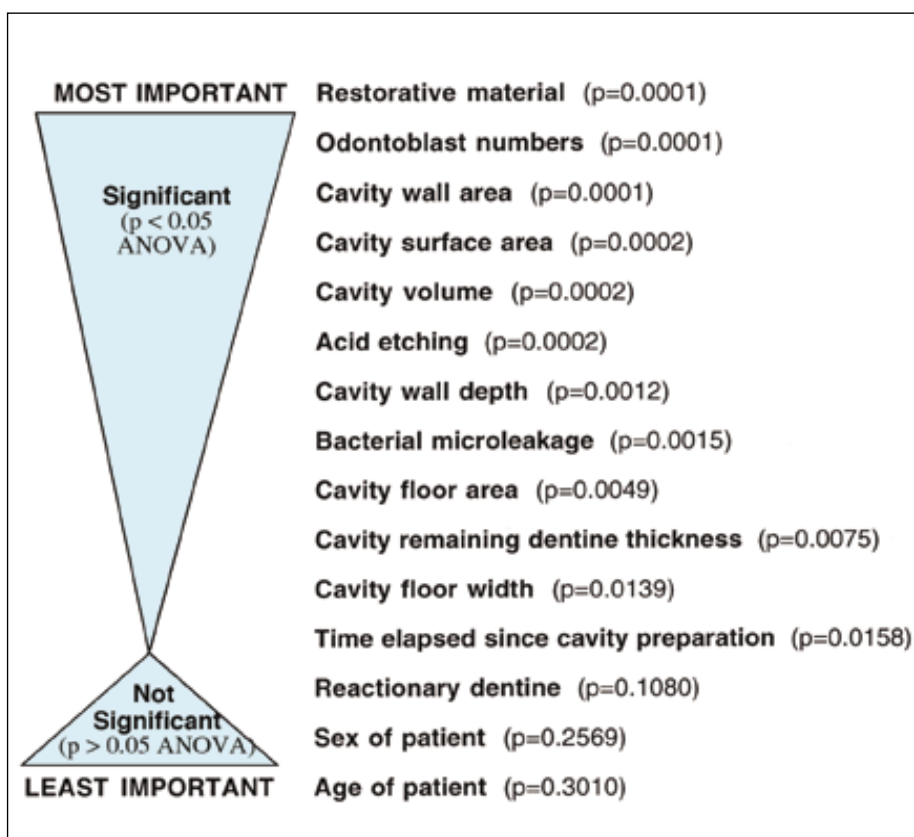


Figure 8. Correlation of variables in order of importance to pulp inflammatory activity.



Figure 9. Variables influencing pulp injury and postoperative activity.

our many collaborators who have contributed to the wider aspects of the programme of research described in this series of papers.

REFERENCES

- Baum BJ, Mooney DJ. The impact of tissue engineering on dentistry. *J Am Dent Assoc* 2000; **131**: 309–318.
- Tziafas D, Smith AJ, Lesot H. Designing new treatment strategies in vital pulp therapy. *J Dent* 2000; **28**: 77–92.
- Murray PE, About I, Lumley PJ, Smith G, Franquin J-C, Smith AJ. Postoperative pulp and repair responses. *J Am Dent Assoc* 2000; **131**: 321–329.
- Murray PE, About I, Lumley PJ, Smith G, Franquin J-C, Smith AJ. Human odontoblast cell numbers following dental injury. *J Dent* 2000; **28**: 277–285.
- Murray PE, About I, Lumley PJ, Smith G, Franquin JC, Smith AJ. Pulpal repair responses. *J Am Dent Assoc* 2001; **132**: 482–491.
- Smith AJ, Murray PE, Lumley PJ. Preserving the vital pulp in operative dentistry: I. A biological approach. *Dent Update* 2002; **29**: 64–69.

- Smith AJ, Cassidy N, Perry H, Begue-Kirn C, Ruch JV, Lesot H. Reactionary dentinogenesis. *Int J Dev Biol* 1995; **39**: 273–280.
- Burke FJ, Cheung SW, Mjör IA, Wilson NH. Reasons for the placement and replacement of restorations in vocational training practices. *Prim Dent Care* 1999; **6**: 17–20.
- Cox CF. Microleakage related to restorative procedures. *Proc Fin Dent Soc* 1992; **88**: (Suppl 1): 83–93.
- Osborne JW, Gale EN. Relationship of restoration width, tooth position, and alloy to fracture at the margins of 13- to 14-year-old amalgams. *J Dent Res* 1990; **69**: 1599–1601.
- Zollner A, Gaengler P. Pulp reactions to different preparation techniques on teeth exhibiting periodontal disease. *J Oral Rehabil* 2000; **27**: 93–102.

ABSTRACT

ARE ELECTRIC TOOTHBRUSHES REALLY BETTER?

Safety, Efficacy and Acceptability of a New Power Toothbrush: A 3-month Comparative Clinical Investigation. P.R. Warren, M. Cugini, P. Marks and D.W. King. *American Journal of Dentistry* 2001; **14**: 3–7.

How often do patients ask whether it really is worth buying an electric toothbrush? Here is a scientific report to which you can refer. These workers

compared a new electric toothbrush with a standard manual toothbrush, examining the hard and soft tissues for safety, and measuring plaque, gingivitis and bleeding scores. The study was randomized and examiner-blind, and measurements were made at the start, and after one and three months. Nine of the initial 110 subjects withdrew for various reasons, for example the unrelated prescription of antibiotic therapy, which may have affected plaque scores.

No detrimental soft or hard tissue abrasion was found at any point in the

study in either group. However, after both one and three months there were significantly lower plaque scores in the group using the electric toothbrush, and the gingival index was found to be significantly reduced after three months. The bleeding index was reduced at both time intervals, but this was not statistically significant. The workers were able to conclude that the power toothbrush was safe, and more effective at reducing plaque and gingivitis than a standard brush.

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