

Preserving the Vital Pulp in Operative Dentistry: 4. Factors Influencing Successful Pulp Capping

PETER E. MURRAY, PHILIP J. LUMLEY, ABEER A. HAFEZ, CHARLES F. COX
AND ANTHONY J. SMITH

Abstract: The sequence of factors that mediate pulp inflammation and necrosis are unclear, and controversy surrounds the effects of different pulp capping materials on exposed pulps. Clinicians have few quantitative studies that rank the *in vivo* pulp capping effects of commonly used restorative materials.

Dent Update 2002; 29: 225–233

Clinical Relevance: Pulp capping with composite resins provides the lowest incidence of bacterial microleakage, lowest levels of pulp inflammation, and the least incidence of necrosis. To obtain the best outcome from pulp capping treatments, we recommend that practitioners avoid creating excessive dentine fragments and operative debris and choose pulp capping materials that minimize pulp injury and bacterial microleakage.

In this series of reviews we have considered the basic biology of tooth dentine and pulp, pulpal injury and repair responses to restorative procedures, and the pulpal responses to cavity remaining dentine thickness. This final review covers the most injurious and complex injury and repair response

– pulp exposure. The selection of treatment following a pulp exposure depends on many factors, including the patient's general condition and clinical history, future restoration plans, progression of caries, pre-existing pulp symptoms and response to stimuli, extent of pulp injury and size of exposure, degree of pulp haemorrhage, periodontal status and the time elapsed since exposure.

In primary teeth with open apices, pulpotomy may be undertaken to maintain the tooth in a physiologically healthy restorable state until natural exfoliation occurs. If the apex is already closed, endodontic procedures are indicated.

In permanent teeth with open apices, coronal pulpotomy is an acceptable endodontic procedure in children and

young adults for the treatment of fractured crowns exhibiting symptoms of infection and inflammation. Carious teeth presenting with symptomatic pulpitis may be treated by pulpotomy and analgesia, or by pulpotomy and root canal treatment, or extraction. If clinical and radiographic evidence suggests the possibility of healing, the pulp exposure can be direct pulp capped: studies *in vivo* have shown that pulp exposures will heal, and that dentine bridge formation does take place, provided the capping material prevents bacterial microleakage and avoids recurrence of inflammation and necrosis.¹

PULP-CAPPING THERAPY

Pulp exposures are more difficult to restore than non-exposed pulps for several reasons:

1. During surgery it is necessary to control haemorrhage, and prevent clot formation, whilst identifying and removing diseased or infected tissue.
2. Mechanical or traumatic injury compromises and reduces the pulp healing response.
3. The loss of the buffering effect of dentine renders the pulp tissue more sensitive to the potentially irritating or even cytotoxic action of the capping agent.

Peter E. Murray, BSc, PhD, Research Fellow in Oral Biology, **Philip J Lumley** PhD, BDS, MDentSci, FDS RCPS, Senior Lecturer in Restorative Dentistry, School of Dentistry, University of Birmingham, Birmingham, UK, **Abeer A. Hafez**, BDS, MSc, Postgraduate DDS student, USC, Los Angeles, **Charles F. Cox**, DMD, FADI, Professor of Dentistry, UCLA, Los Angeles, USA, and **Anthony J. Smith** BSc, PhD, Professor of Oral Biology, School of Dentistry, University of Birmingham, Birmingham, UK.

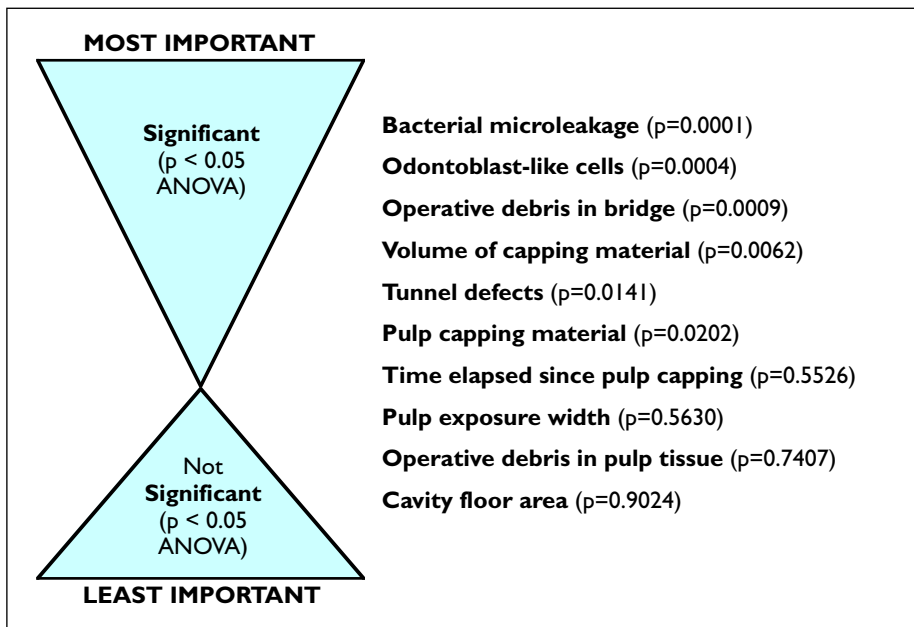


Figure 1. Sequence of variables correlated to pulp inflammatory activity.

4. Operative debris including dentine fragments (sometimes called ‘chipitis’ – a term used to describe the dentine fragments) and particles of capping materials may infiltrate the pulp, causing injury and inflammatory reactions.

The failure to form dentine bridges – or the opposite effect, where the pulp has become occluded by excessive dentine bridge formation – can be associated with postoperative complications such as hypersensitivity and necrosis.

Among the variables that affect pulp repair by dentine bridge formation are the capping materials used, degree of mechanical injury, creation of dentine chips during operative procedures,

inflammation and bacterial microleakage. However, quantitative data is lacking, and the relative importance of these pulp capping variables which mediate pulp repair activities and pulpal inflammation is not well understood.

To identify the relative importance of these factors, we investigated 142 standardized pulp exposed cavities in non-human primate teeth according to ISO usage guidelines, following the pulp capping of exposures with calcium hydroxide or composite resins.

PULP CAPPING VARIABLES CORRELATED TO PULP INFLAMMATION

The slow development and acceptance

of new pulp capping materials might be partially attributable to the lack of information on the interactions between pulp exposure variables and pulp repair activity. An understanding of the variables that modify, stimulate or impede dentine bridge formation may help to explain how potentially pathological postoperative complications can arise, and provide guidance on the aspects of pulp capping that are most likely to minimize the incidence and severity of pulp inflammation and other associated postoperative complications.

The most important variables correlated to pulp inflammation are postulated to be bacterial microleakage and factors relating to the microleakage of bacteria, which include:

- the presence of operative dentine debris (which includes dentine fragments and particles of pulp capping material);
- ‘tunnel defects’, the term used to describe imperfections in dentine bridge continuity; and
- the type and volume of the pulp capping material (Figure 1.)

In this study, pulpal necrosis and severe inflammatory activity appeared to be limited primarily to infected, pulp capped exposures, not as a response to vital pulp etching (Table 1). The microleakage of bacteria was highly correlated to pulp inflammation (Figure 2). Composite resin and calcium hydroxide pulp capping had similar effects on pulp inflammation in the presence and absence of bacterial

Pulp capping material	No.	Inflammation (%)				Tunnel defects (%)	Bacterial microleakage (%)
		Absent	Slight	Moderate	Severe		
Resin composite (+ Bac)*	15	40.0	26.7	0.0	33.3	1.3	19.7
Resin composite (- Bac)	61	82.0	9.8	6.6	1.6		
Calcium hydroxide (+ Bac)	31	29.0	22.6	29.0	10.4	47.0	47.0
Calcium hydroxide (- Bac)	35	60.0	31.4	5.7	2.9		

*(+Bac = microleakage)

Table 1. Frequency of bacterial microleakage, tunnel defects and pulp inflammation following pulp capping.

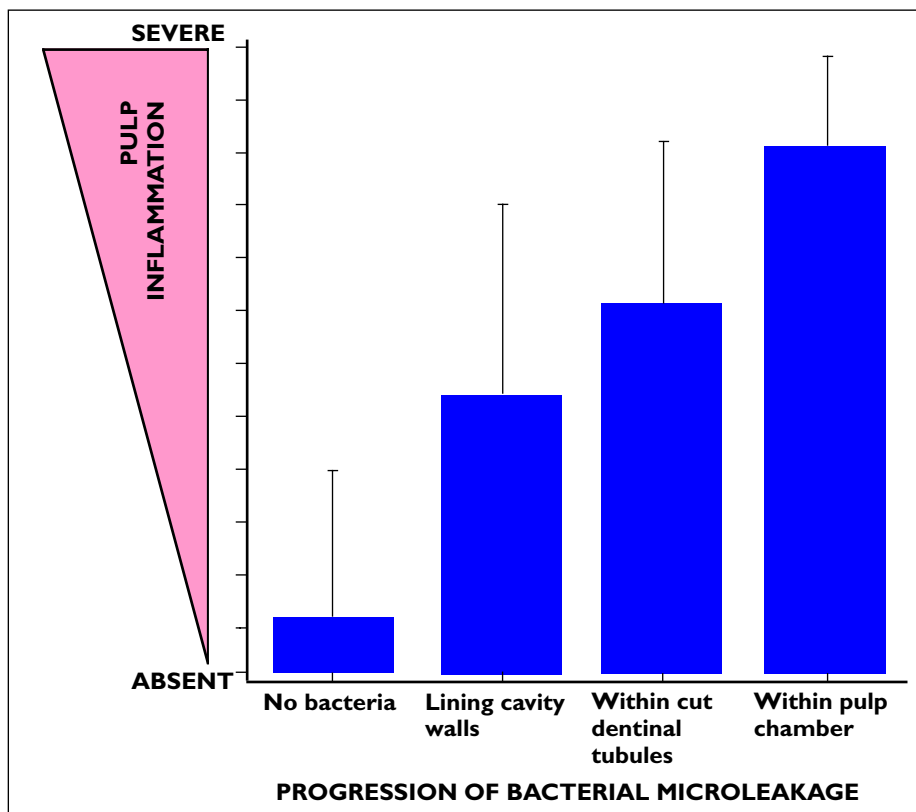


Figure 2. Pulp inflammation and progression of bacterial microleakage.

microleakage (Figure 3).

The most important ways to minimize pulp inflammation are to avoid creating excessive operative dentine debris during cavity cutting, and to select a pulp capping material with the ability to seal the pulp exposure and prevent bacterial microleakage.

DENTINE BRIDGE FORMATION

Treatments for the exposed dental pulp have changed greatly in the last 200 years, recognizing that it is not easy to promote healing following pulp exposure. The pulp repair events may be summarized as formation of a dentine bridge beneath pulp capping materials: dentine bridges have been observed in 90% of cases following mechanical pulp exposures.² The presence of a dentine bridge provides natural protection for the pulp against the microleakage of bacteria, and prevents particles leaching from capping materials and infiltrating into pulp tissues.³

By furthering our knowledge of the

sequence of important variables that modify, stimulate or impede dentine

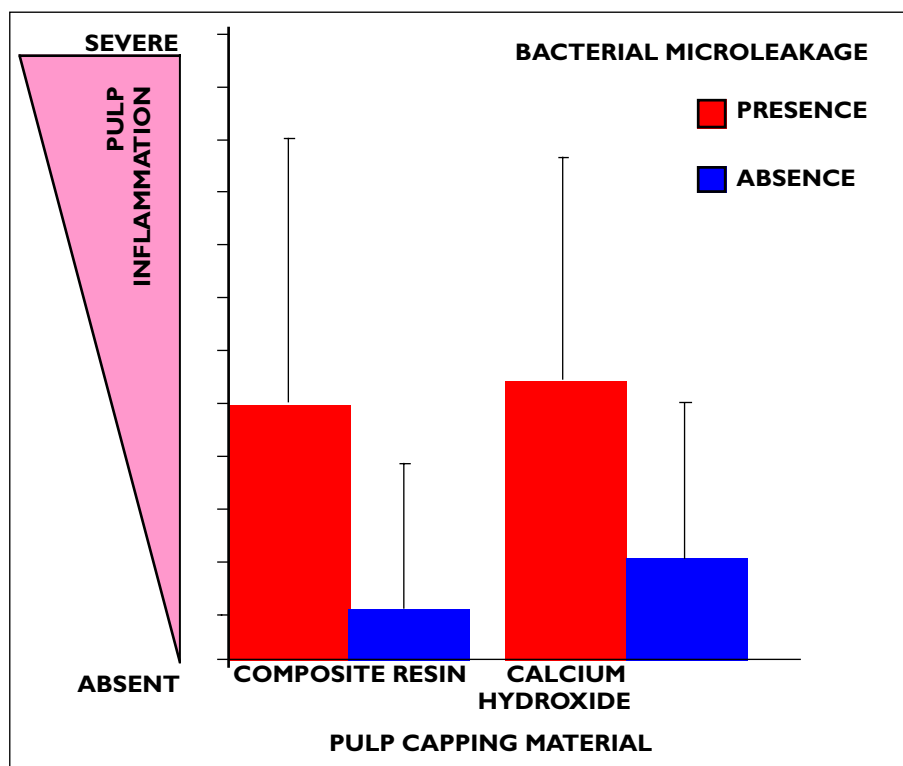


Figure 3. Pulp inflammation and pulp capping material.

bridge formation, we may begin to understand how pathological postoperative complications can arise, and learn how to minimize the incidence and severity of complications. The most important of these factors are:

- the density of odontoblast-like cells that synthesize and secrete dentine bridges;
- the time elapsed since pulp capping; and
- the presence of operative debris, including dentine fragments and material particles (Figure 4).

By minimizing the amount of operative debris and carefully placing pulp capping materials, practitioners may encourage the formation of dentine bridges.

PULP CAPPING MATERIALS

The selection of pulp capping materials is extremely controversial. Pulp capping products and treatments have changed little in the past 30 years, whilst adhesive dentistry has revolutionized indirect restorative practice.⁴

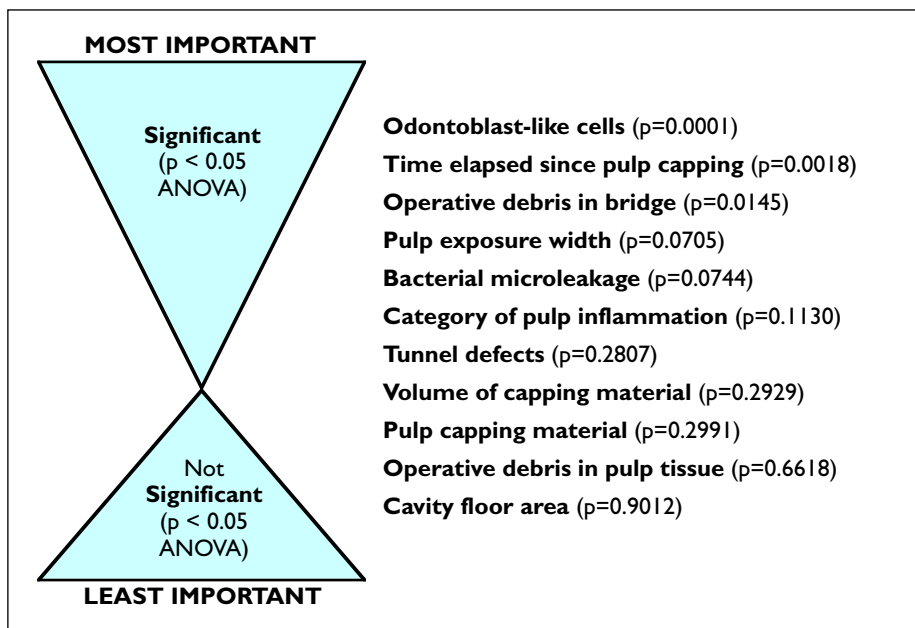


Figure 4. Sequence of variables correlated to the area of dentine bridge formation.

Calcium Hydroxide

Calcium hydroxide is the most commonly used pulp-capping agent, although its use is largely based on empirically grounded evidence such as inducing healing of periradicular lesions, promoting apical closure in incompletely developed teeth, and preventing or arresting root resorption. Calcium hydroxide is also cost-effective to use and relatively easy to apply.

The high pH of calcium hydroxide provides bactericidal activity, and encourages tissue repair by promoting dentine bridge formation. In our study, the area of dentine bridges forming beneath calcium hydroxide was 62.3% greater than that forming beneath composite resin pulp caps (Figure 5).

However, the therapeutic advantages of calcium hydroxide appear short term, because the continuous stimulation of dentine bridge formation often leads to occlusion of the pulp chamber and root canals, making future endodontic treatment difficult.

Alternative Materials

Alternative pulp capping materials have been available since the early 1980s, and the use of adhesive systems for direct pulp capping has stimulated a

number of histological and clinical investigations.

Acid etching of vital pulp is necessary for the adhesive bonding of resin composite to tooth structure, and there was initial resistance to accept that this does not cause significant pulp injury. However, clinical and usage reports have confirmed that capping with composite resin materials does not cause pulp inflammation or necrosis.⁴ In the study reported here, resin composite was associated with a lower frequency of bacterial microleakage and tunnel defects than calcium hydroxide (19.7% versus 47.0% and 1.3% versus 47.0%, respectively), as well as the absence (73.7% versus 45.4%) of pulp inflammation (Table 1). These findings agree with reports suggesting that new composite resin products are better than their predecessors, having improved bacteriometric sealing properties.

Calcium hydroxide is therefore no longer the most suitable pulp capping material available.

TUNNEL DEFECTS

The presence of a dentine bridge, and its area, may be of less importance than the quality of the dentine bridge – to

function as a bacteriometric barrier, and prevent microleakage, a dentine bridge must have no tunnel defects.⁵ Tunnel defects permit the microleakage of bacteria and migration of calcium hydroxide particles or resin globules. The 47.0% incidence of bacterial microleakage in calcium hydroxide capped pulps may thus be explained by observation of tunnel defects in 47.0% of these dentine bridges, and the lower incidence of tunnel defects in dentine bridges beneath composite resins could partially explain the lower incidence of microleakage observed following pulp capping with these materials (Table 1).

However, the presence of tunnel defects did not necessarily increase pulp inflammation (Figure 6), suggesting that not all tunnel defects allow bacterial contamination. Microleakage of bacteria and their products causes pulp reactions which may persist, owing to continuing irritation, and may finally lead to necrosis.

Unexpectedly, the presence of tunnel defects did not correlate with the area of dentine bridge formation ($p = 0.8367$), suggesting that tunnel defects occur during dentine secretion, rather than being like ‘stress-fractures’ due to the proportions of the dentine bridge itself.

It is possible that the capping

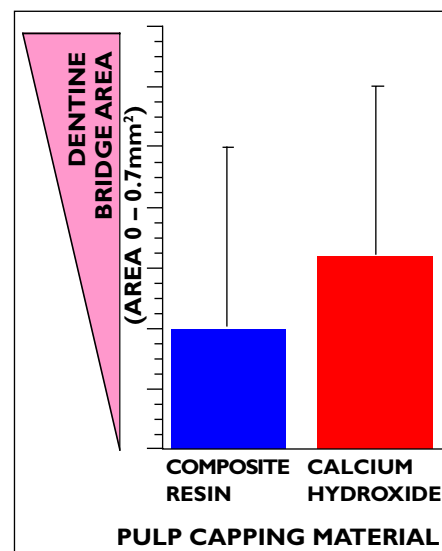


Figure 5. Dentine bridge area and pulp capping material.

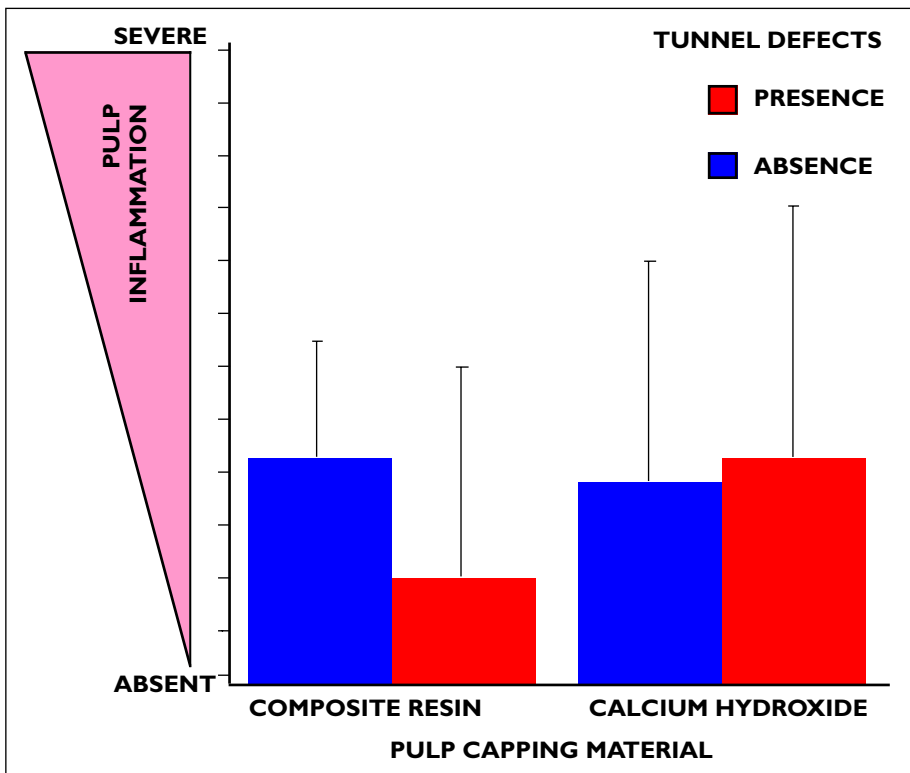


Figure 6. Pulp inflammation and tunnel defects.

material used may influence the reparative dentinogenic response, which may affect the degree of dysplasia in the matrix secreted and incidence of tunnel defects. Consequently, a reduced frequency of tunnel defects must be a goal for successful pulp-capping materials.

BACTERIAL MICROLEAKAGE

The prevention of bacterial microleakage into cavity preparations is important, as it will contribute to the longevity of cavity restorations. Complications due to bacterial microleakage include:

- postoperative sensitivity;
- marginal discoloration;
- recurrent caries;
- periodontal disease; and
- eventual need for endodontic therapy.

In our study, odontoblast-like secretion of dentine bridges was 42.86% lower in the presence of

bacterial microleakage (Figure 7). These findings suggest that an important property of a pulp capping material is its capacity to prevent bacterial microleakage. Bacterial microleakage can impede bridge formation (Figure 7), particularly following pulp capping with composite resin, which may lead to the development of postoperative complications.

The density of odontoblast-like cells was not particularly influenced by the effects of pulp-capping variables, which suggests that complications with dentine bridges are due to inhibition of odontoblast-like dentine bridge secretory activity, rather than inhibition of odontoblast-like cell density (Figure 8).

Although all these variables require further investigation to establish the precise nature of their effects, the outcomes of pulp capping treatments could be beneficially influenced by preventing bacterial microleakage as far as possible.

OPERATIVE DEBRIS

Results of studies of the effects of

operative debris have been ambiguous, and quantitative information on the effects of operative debris on pulp repair activity, pulp inflammation or dentine bridge formation is scarce. The presence of operative dentine debris appears to increase dentine bridge area (Figure 9), which at first sight may be construed as beneficial – but larger dentine bridges increase the possibility of pulp strangulation and occlusion of the pulp chamber. The persistence of operative debris may cause immediate pulp inflammation, which can progress to pulp necrosis,⁶ although in our study only severe forms of inflammation appeared to prevent dentine bridge formation (Figure 9).

The lack of correlation between presence of operative debris and bacterial microleakage may indicate that not all operative debris can be assumed to be contaminated with bacteria. However, the presence of operative debris below the exposure site certainly appears to complicate healing and bridging, and our observations suggest its presence may be associated with the development of postoperative complications such as inflammation, microleakage and necrosis.

We recommend that minimizing the presence of operative debris during pulp capping will improve treatment outcomes.

ODONTOBLAST-LIKE CELLS

Following pulp exposure the underlying

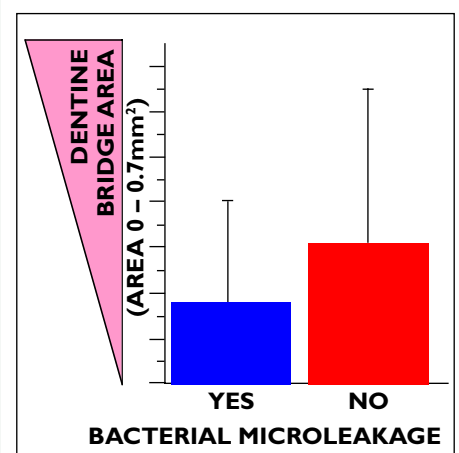


Figure 7. Dentine bridge area and bacterial microleakage.

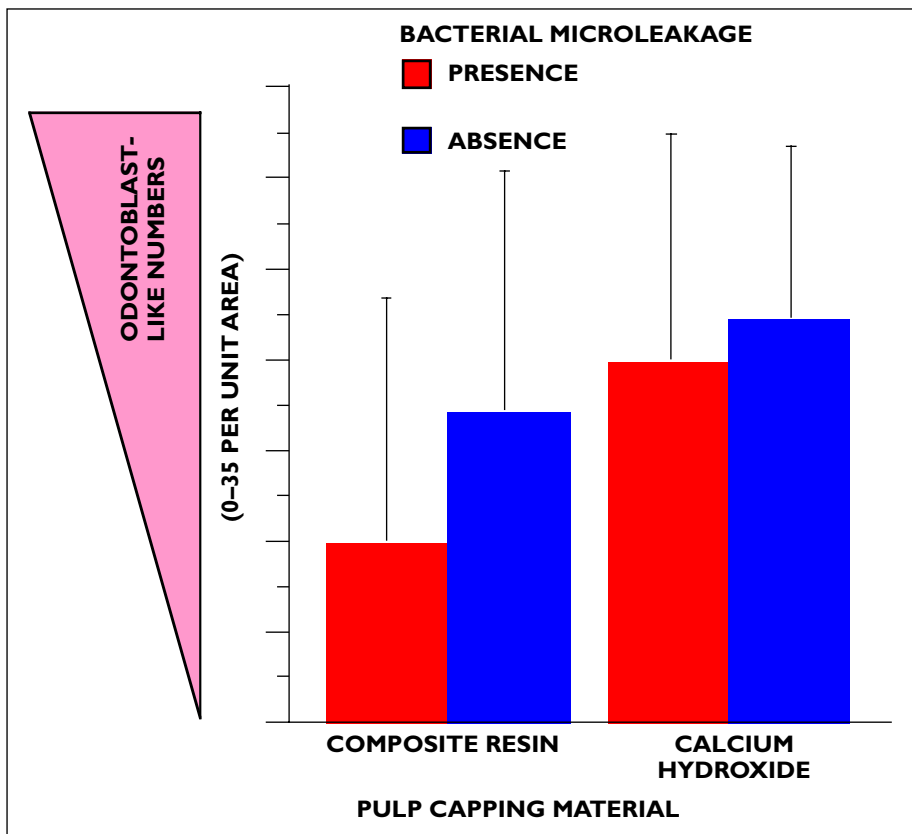


Figure 8. Odontoblast-like-cell numbers and pulp capping material.

primary odontoblasts are often irreversibly damaged. These post mitotic terminally differentiated cells cannot proliferate to replace subjacent irreversibly injured odontoblasts. Consequently, the origin of odontoblast-like cells that secrete dentine bridges following pulp exposure is a source of much speculation. Autoradiographic studies have indicated that new odontoblast-like cells derive from within other pulp cell populations by a process of differentiation and migrate toward the site of pulp exposure, where dentine bridges are secreted.⁷

Possible progenitor cell populations for the new odontoblast-like cells include cells of the subodontoblast layer, pulp fibroblasts, vascular and undifferentiated mesenchymal cells. The nature of these cell populations may influence the quality of dentine bridge formation.

The ability of bacterial microleakage to reduce the rate of dentine bridge formation beneath composite resin

capped pulps is of interest because the delay in providing pulp protection may further aid the penetration of bacteria and their toxins into pulp tissue.⁸ The

prevention of bacterial microleakage is thus important in allowing pulp repair activity to proceed normally.

CONCLUSION

We propose the following guidelines for avoiding pulp exposures, and for capping success, at least short-term:

1. Caries lesions should be monitored to distinguish active lesions requiring restorative treatment.
2. The possibility of bacterial infection during restorative procedures from air or water supplies should be assessed, and consideration given to use of rubber dams, wearing masks and a sterile water supply.
3. Restoration of non-pulp exposed cavity preparations rather than pulp exposed preparations will greatly avoid injury to underlying pulp. Minimally invasive restorative procedures should be used to reduce the amount of caries-affected dentine removed beneath preparations.
4. Direct pulp capping should be used only to restore teeth with a good prognosis; pulpotomy or extraction should always be

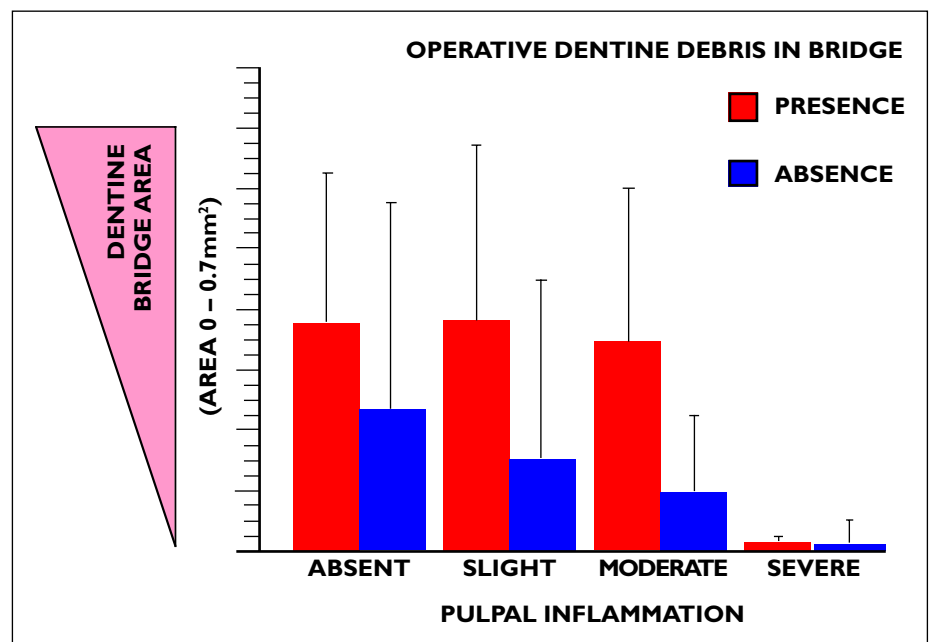


Figure 9. Dentine bridge area and pulpal inflammation.

considered (pulpotomy should be performed on carious pulp exposures, for example).

5. Provided the blood flow from an exposure is not excessive, which indicates an unfavourable pulp condition, efforts should be made to minimize bleeding – prolonged continuous flow of blood will injure pulp tissue.
6. The creation of operative debris during cavity preparation should be minimized, which will reduce pulp injury and permit increased continuity of dentine bridge formation. This will help to avoid postoperative complications and the need for endodontic treatment.
7. The cavities around pulp exposures should be cut with a low-speed bur to minimize operative debris, and all visible operative debris should be removed before capping.
8. Pulp capping materials should have excellent sealing properties (to adhere to tooth substance and prevent bacterial microleakage) as well as good physical characteristics. The performance of composite resin materials suffers from a degree of operator sensitivity but our data has shown that these materials, if used correctly, can produce better results for pulp capping than calcium hydroxide.
9. Unless practitioners are experiencing problems with their existing restorative materials, they should continue with their tried and tested approaches.
10. Practitioners should always consider all aspects of their pulp capping methods before switching to alternative capping materials or methods – success may be more a function of operator handling than differences between materials.

FUTURE DIRECTIONS

This review series has shown how current cavity preparation and restoration techniques can be used to maximize dental treatment outcomes.

Although the technical basis of many of these procedures is well recognized, we are only just starting to recognize the manner in which they impinge on biological events in the dentine–pulp complex. Recognition that dentine is not just an inert mineralized matrix, and that it contains many bioactive molecules capable of signalling cellular events, requires reconsideration of its treatment. Release of these molecules through tissue injury, cavity preparation or restoration could have important consequences for cellular injury and repair responses in the pulp. The ability of pulpal cells to respond to these molecules will reflect their survival following tooth injury, cavity preparation and restoration. Thus, careful monitoring of the disease process could limit the initiating injury responsible for the restoration, thereby providing a better prognosis for the tooth. Attention to cavity preparation may allow pulpal injury and repair responses to be optimized in relation to cavity depth, the presence of operative debris and other factors. Consideration of the action of cavity etchants can be exploited for release of bio-active molecules from the dentine matrix and their diffusion along dentinal tubules to the pulpal cells. Appropriate selection of restorative materials can allow the practitioner to avoid bacterial microleakage, hypersensitivity and pulp necrosis. All of these measures will contribute to minimizing pulp injury and harnessing the natural regenerative capacity of the dentine–pulp complex. Importantly, they should lead to an improvement in treatment outcomes without the need for radical change to treatment modality. Such biological approaches to the treatment of tooth injury can provide immediate benefits, allowing the continuing development of more far reaching technologies in the future. These might include the organ culture of dental tissues⁹ and transdental stimulation of repair with exogenous bioactive molecules¹⁰ in the shorter term and gene therapy, stem cell therapy and tissue engineering in the longer term.

ACKNOWLEDGEMENTS

We are grateful to the Wellcome Trust for a Sir Henry Wellcome Commemorative Award for Innovative Research (ref : 057820) for the support of P.E.M. We also warmly acknowledge our many collaborators who have contributed to the wider aspects of the programme of research described in this series of papers.

REFERENCES

1. Hafez AA, Kopel HM, Cox CF. Pulpotomy reconsidered: Application of an adhesive system to pulpotomized permanent primate pulps. *Quintess Int* 2000; **31**: 579–589.
2. Baume LJ, Holtz L. Long term clinical assessment of direct pulp capping. *Int Dent J* 1981; **31**: 251–260.
3. Stanley HR. Criteria for standardizing and increasing credibility of direct pulp capping studies. *Am J Dent* 1998; **11**: 17–34.
4. Kugel G, Ferrari M. The science of bonding: from first to sixth generation. *J Am Dent Assoc* 2000; **131**: 20S–25S.
5. Cox CF, Sübay RK, Ostro E, Suzuki S. Tunnel defects in dentin bridges: Their formation following direct pulp capping. *Operat Dent* 1996; **21**: 4–11.
6. Bergholtz G. Effects of bacterial products on inflammatory reactions in the dental pulp. *Scand J Dent Res* 1982; **85**: 122–129.
7. Fitzgerald M, Chiego JD, Heys R. Autoradiographic analysis of odontoblast replacement following pulp exposure in primate teeth. *Arch Oral Biol* 1990; **35**: 707–715.
8. Murray PE, Hafez AA, Smith AJ, Cox CF. Hierarchy of pulp capping and repair activities responsible for dentin bridge formation. *Am J Dent* 2002 (in press).
9. Murray PE, Lumley PJ, Ross HF, Smith AJ. Tooth slice organ culture for cytotoxicity assessment of dental materials. *Biomaterials* 2000; **21**: 1711–1721.
10. Smith AJ, Tobias RS, Murray PE. Transdental stimulation of reactionary dentinogenesis in ferrets by dentine matrix components. *J Dent* 2001; **29**: 341–346.

MAY
Self-Assessment
Answers

1. D	6. C
2. B, C	7. A, C, D
3. A, D	8. A, C, D
4. A, B, C, D	9. B, C
5. D	10. A, B, C