# Fate of vital pulps beneath a metal-ceramic crown or a bridge retainer

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## Abstract

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**Aim** To investigate the incidence of and factors associated with pulpal necrosis in vital teeth restored with metal-ceramic crowns (CMCs) or crowned as part of a fixed–fixed bridge.

**Methodology** Patients who had a CMC or bridge retainer (BR) placed on a tooth with no previous history of root canal treatment from 1981 to 1989 were retrieved from computer records. The collated patients were randomly selected and their clinical records examined. Those who satisfied the inclusion criteria were contacted and offered a review. After clinical examination, long-cone paralleling periapical radiographs were taken of the selected teeth, which were then assessed by two precalibrated operators to ascertain the pulpal status. Factors that might contribute to loss of pulp vitality and the tooth type were also recorded. The collected data were analysed statistically using the chi-square test and subject to Bonferroni adjustment where indicated.

**Results** The numbers of preoperatively vital teeth in the CMC and BR groups were 122 and 77, and the mean observation periods were 169 ± 25 (SD) and 187 ± 23 months, respectively. In the CMC group, 19 failed cases (15.6%) were due to an endodontic reason; total number of failures was 34. In the BR group, 25 (32.5%) showed signs of pulpal necrosis; a significant association with maxillary anterior teeth was noted. The survival rates for pulp vitality were 84.4% (CMC) and 70.8% (BR) after 10 years, and 81.2% (SC) and 66.2% (BR) after 15 years. The difference between the two groups was significant.

**Conclusion** The survival of the vital pulp in teeth restored with a single-unit CMC was significantly higher than those serving as an abutment of a fixed–fixed bridge. Maxillary anterior teeth used as bridge abutments had a higher rate of pulpal necrosis than any other tooth types.

**Keywords:** bridges, crowns, fixed prosthodontics, pulpal necrosis, survival analysis.

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## Introduction

Full coverage crowns have long been used to restore heavily damaged teeth and/or, in the case of metal-ceramic crowns (CMCs), to satisfy the patient's aesthetic demand. They are also frequently used as retainers for fixed prostheses to replace missing teeth. In either case, it is likely that the teeth involved might have suffered cumulative insults from caries, periodontal disease, or trauma, be it physical or due to restorative procedures, prior to the restoration (Ericson *et al.* 1966). Any history of dental disease and restorations could have an impact on the health of the dental pulp and further treatment might precipitate pulpal problems in the future (Seltzer & Bender 1984).

Diseases of endodontic origin affecting the abutment teeth have been regarded as a biological failure of the fixed prostheses; other biological reasons for failures include caries and periodontal disease (Selby 1994). Fixed prostheses may also fail mechanically, because of loss of retention, fracture of porcelain, failure of the

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metal framework or solder joints, wear, and fracture of the abutment tooth. Defective margins, poor contour and poor aesthetics may then account for the remaining failures. Many of these failures can have a detrimental effect on the health of the dental pulp.

Over the years, many reports on the longevity and reasons of failures in fixed prostheses have been published. But in many studies the (condition leading to) replacement of the restorations was regarded as the only criterion for failure (Scurria *et al.* 1998) whereas other types of complications, most notably endodontic, were not considered (e.g. Morrant 1956, Kantorowicz 1968, Roberts 1970, Glantz *et al.* 1984, Palmqvist & Swartz 1993, Smales & Hawthorne 1997). There have been a number of studies that gave an indication of pulpal necrosis after construction of fixed prostheses (Table 1). Bergenholtz & Nyman (1984) reviewed patients treated for advanced periodontal disease and found pulpal necrosis in 15% of abutment teeth, compared with only 3% in nonabutment teeth after a mean observation period of 8.7 years. In a survey where patients with fixed bridges were evaluated, 20 of 169 bridges examined had failed due to endodontic reasons, that is, some 57% of all failed bridges had one or both of their abutments so affected (Cheung *et al.* 1990). Unfortunately, the number of vital abutment teeth that had been affected was not reported. In contrast, three of 73 previously vital teeth restored with single crowns were deemed to have failed because they became periapically involved or had been root canal treated after a mean observation period of 34 months (Cheung 1991). That is, 4% of vital teeth developed pulpal necrosis after placement of single crowns. Jackson *et al.* (1992) reported that 5.7% of teeth had received root canal treatment some 1–6 years after cementation of a single crown or fixed bridge; unfortunately, the response rate of their study was just over 10%. Saunders & Saunders (1998) conducted a cross-sectional, radiographic survey of patients for whom a set of full-mouth periapical radiographs was available and reported that 19% of initially vital teeth developed periradicular radiolucency after (an unknown period of) crown placement. The influence of various factors, such as preoperative restorative and periodontal status, operators, gender and tooth type, has not been conclusive.

As most reports in the literature were cross-sectional in nature and only a mean observation period was reported, their study methodology thus has assumed a uniform rate of development of pulpal or periapical complications, which might not be the case. There also appears to be a lack of information that highlights any difference on the pulpal status of teeth supporting single crowns or conventional fixed–fixed bridge retainers. The aim of this study was to investigate the incidence and the factors that might be associated with the development of pulpal necrosis in vital abutment teeth that had been crowned either singly or as part of a fixed–fixed bridge.

## Materials and methods

The study population consisted of patients who had received a CMC or a conventional fixed–fixed bridge on vital tooth/teeth from 1981 to 1989 at the Prince Philip Dental Hospital (PPDH), which is a dental teaching hospital in Hong Kong. All treatment provided had been recorded on a computer database, from which a total of 872 CMCs and 241 bridges with no history of root canal treatment of the abutment teeth were identified. About half of each type of restoration, i.e. 440 CMCs and 124 bridges, were randomly selected. This was done by choosing every other patient who was on the list, arranged in ascending order of the hospital registration number, for that particular type of restoration. The written clinical records of selected patients were checked to ensure that all samples fulfilled the following inclusion criteria:

1. The tooth (or, in the case of a bridge, at least one of the abutment teeth) had not received any form of root canal treatment prior to the construction of the restoration; and
2. The tooth either received a CMC or bridge retainer (BR) for a fixed–fixed bridge. In other words, all resin-bonded retainers were excluded.

### Clinical assessments

A total of 284 CMCs and 102 bridges satisfying the inclusion criteria were identified. The patients were invited to return for a review. Prior to his/her attendance, the patient's record was studied for any possible pre- and intra-operative factors that might contribute to the development of pulpal necrosis: preexisting DMFT score, presence of dental pins or pulp capping, status of the preexisting restoration filling and the alveolar bone level, the reason for crown/bridge construction, period of temporization and the luting cement used. Patients who declined to attend the recall were interviewed over the phone whether the tooth in question had received any

**Table 1** Summary of some reports that gave an indication of the pulpal status after construction of crowns or bridges

| Reference | Subjects | Response rate | Observation period | Restorations or teeth examined | Assessment method | Results related to pulpal status |
|---|---|---|---|---|---|---|
| Ericson *et al.* (1966) | 272 patients with 668 vital teeth in a student clinic | Yr 1: 97%<br>Yr 2: 93% of first year<br>Yr 4: 95.8% of second year | 4 years | 642 teeth (1 year)<br>573 teeth (2 years)<br>544 teeth (4 years) | Radiographs | Pulp necrosis 2% in first year review, 2.4% after 2 years and 2.8% after 4 years (another 1.6, 1.2 and 0.4% borderline cases, respectively) |
| Bergenholtz & Nyman (1984) | 52 patients with extensive treatment for periodontal disease in a teaching hospital | Retrospective study | Mean 8.7 years (range 4–13 years) | 255 bridge abutments and 417 single crowns (total 627 vital pulps) | Clinical symptoms and radiographs | Pulp necrosis in 15% abutment teeth and 3% beneath single crowns; half of necrotic pulp was diagnosed 7–12 years postoperative |
| Karlsson (1986) | 164 patients in a private clinic | 26% | 10 years | 238 bridges with 944 abutments | Questionnaire, clinical and radiographic exams | 10% vital abutment had radiographically visible periapical lesions |
| Cheung *et al.* (1990) | 143 patients with 169 bridges in a teaching hospital | 77% | Mean 35 months | Fixed–fixed bridges | Clinical and radiographic exams | 57.1% of bridges had one or both abutments with endodontic involvement (no. of initially vital abutment not known) |
| Cheung (1991) | 132 patients with 152 crowns in a teaching hospital | 38% (no. of teeth) | Mean 34 months | Single crowns; total 73 initially vital teeth | Clinical and radiographic exams | Pulp necrosis in 4.1% of vital teeth |
| Jackson *et al.* (1992) | 130 patients in a teaching hospital | 10.6% | 2–6 years | 437 vital teeth: 235 bridge retainers and 202 single crowns | Clinical examination, cold test and radiographs | 25 teeth (5.7%) had had RCT |
| Valderhaug *et al.* (1997) | 32 patients with 101 restored teeth in a teaching hospital | 28% (patients)<br>24% (teeth) | 25 years | Single crowns ($n = 46$)<br>Bridges ($n = 112$) up to 14 units | Radiographs | 10% of all failures were due to pulp necrosis (estimated survival of intact pulp was 98% after 5 years; 92% after 10 years; 87% after 20 years; 83% after 25 years) |
| Saunders & Saunders (1998) | 202 patients with 802 crowns in two teaching hospitals | Cross-sectional study (only on patients requiring full-month periapical radiographs) | Unknown | 802 crowns with 458 on vital teeth | Radiographs | 87 (19%) showed signs of radiographic peri-radicular disease |
| Walton (1999) | 239 patients in a specialist prosthodontic practice | 69% | 52% 5–10 years<br>48% less than 5 years but more than 1 year | 688 CMCs, 67% on vital teeth | Clinical examination | 27 (2.7%) units with periapical involvement |

further dental treatment and, if so, the time and nature. At the review appointment, the selected restoration(s) was examined in detail clinically. The presence of caries (recurrent or new), and the quality and status of the restoration were noted. Percussion test, palpation of the corresponding attached mucosa, and cold and electric pulp tests (where possible) were carried out. A long-cone, paralleling periapical radiograph was then taken. The restoration was deemed to have failed if any one or a combination of the following complications were noted: (i) technical or mechanical – including fracture of any part of the restoration or the tooth, and loss of retention of the restoration; (ii) aesthetic; (iii) presence of secondary caries; (iv) endodontic; and (v) any other reason, such as tooth extraction (Cheung 1991). But, for purpose of this study, only the endodontic reason was considered; that is, the abutment tooth became pulpally or periapically involved, or had been root filled after the restoration. During the review, the diagnosis of the pulpal status of the crowned teeth often had to rely on clinical symptomatology and radiographic assessment, because pulp testing was not always possible. Here, the presence of a periapical radiolucent area was taken as an indicator of nonvitality of the dental pulp.

### Radiographic assessment

Two precalibrated, independent observers examined the radiographs and categorized the periapical status of the selected teeth according to a written set of criteria (Table 2). Inter-examiner agreement on the radiographic assessment was determined by computing the Cohen's $\kappa$ value, which was found to be 0.79. If there was any disagreement, a third observer was recruited as an arbitrator before a final score was reached.

### Statistics

Cases already recorded to have failed because of endodontic reason and those diagnosed to be nonvital at the recall constituted the group that had developed pulpal necrosis after the restoration. Chi-square test and two-sample *t*-test were used, where appropriate, at $\alpha = 0.05$. If multiple comparisons were made, then the significance level was subject to Bonferroni adjustment and was set at $P = 0.005$. As the subjects of this study were a cohort of patients receiving the treatment, the data on pulpal status were examined by survival analysis using the Kaplan–Meier estimator. For entry into the Kaplan–Meier analysis, the time to development of pulpal necrosis was taken as the mid-point between the date of diagnosis of failure and the last known date of an intact restoration without pulpal complications (Cheung 2002). If the date of diagnosis could not be found in the record, the patient was asked to provide the date when he/she first noticed a problem that led to further treatment, which date would serve as the date of diagnosis.

## Results

All teeth that presented with pulpal symptoms at the review also showed a periapical radiolucent lesion or had been root-filled; they were classified as failures due to endodontic reason. The quality of the crown or bridge at the date of examination was acceptable, except for those that were deemed to have failed due to various reasons (Tables 3 and 4).

### Metal-ceramic crowns

Of the 284 CMCs that met the inclusion criteria, 114 CMCs in 79 patients were examined clinically. Another

**Table 2** Radiographic categorization of pulpal status

| Category | Status[a] | Description |
|---|---|---|
| 0 | Intact pulp | No evidence of radiopaque foreign material in pulp chamber and/or root canal(s), and no periradicular radiolucent area |
| 1 | Widening of the PDL space | Widening of the apical part of the periodontal ligament space, not exceeding two times the width of the lateral periodontal ligament space |
| 2 | Periapical radiolucency | Radiolucency in connection with the apical part of the root, the diameter exceeding two times the width of the lateral periodontal ligament space |
| 3 | Endodontically treated tooth | Tooth with radiopaque material in pulp chamber (if discernible) and/or root canal(s) |

[a]Categories 1, 2 or 3 were deemed to be associated with a nonvital pulp.

**Table 3** Reasons for failure of CMCs

| Reasons of failure | Frequency | Percentage of total no. examined (% of all failed cases) |
|---|---|---|
| Endodontic | 19 | 15.6 (55.9) |
| Aesthetic | 3 | 2.5 (8.8) |
| Prosthetic | 3 | 2.5 (8.8) |
| Fracture of porcelain | 2 | 1.6 (5.9) |
| Tooth fracture | 3 | 2.5 (8.8) |
| Root caries | 2 | 1.6 (5.9) |
| Others | 2 | 1.6 (5.9) |
| All failed CMCs | 34 | 27.9 (100.0) |
| Total no. of CMCs analysed | 122 | 100.0 (–) |

**Table 4** Reasons for bridge failures

| Reasons of failure | Frequency | Percentage of total no. examined (% of all failed bridges) |
|---|---|---|
| Endodontic | 14 | 34.0 (53.3) |
| Endodontic + debond | 6 | 12.8 (20.0) |
| Pain | 2 | 4.3 (6.7) |
| Loss of retention (include debond of one of the retainers) | 6 | 8.5 (13.3) |
| Others | 2 | 4.3 (6.7) |
| All failed bridges | 30 | 63.8 (100.0) |
| Total no. of bridges analysed | 47 | 100.0 (–) |

eight CMCs were found to have failed according to the patients' records, which were also included in the analysis. The sample thus comprised (114 + 8 =) 122 CMCs, with a mean observation period of 169 months (SD = 25) up to the date of recall. Of the 34 CMCs (28%) that were deemed to have failed, 19 were classified as endodontic, giving an overall incidence of pulpal necrosis of 16% (Table 3). The shortest interval to the development of pulpal necrosis was 6 months after cementation whilst the longest was 188 months. As the cumulative survival had not dropped below 50%, a medium survival time for maintenance of pulp vitality could not be calculated.

No significant association was found between the development of pulpal necrosis and gender, presence of pins, tooth type, presence and type of preexisting restoration, preoperative alveolar bone level, reasons for crown construction, types of cement used, function of the crown, operators, the pre- or postoperative DMFT score, or period of temporization.

## Conventional bridges

Of the 102 bridges selected, 38 bridges in 33 patients were examined. Including another nine bridges that had already failed according to the patients' records, a total of 47 bridges and 77 preoperatively vital abutments were available for analysis. The mean observation period was 187 months (SD = 23). Of the 30 bridges (64%) that were deemed to have failed, 22 had at least one of their abutments affected by some form of pulpal or endodontic complications (Table 4). Twenty-five of the 77 initially vital abutments had been treated endodontically or developed pulpal necrosis, an incidence of 33% (Table 5). The shortest time to the development of pulpal necrosis was 2 months whilst the longest was 176 months after cementation of the bridge.

Chi-square analysis failed to reveal any association between the development of pulpal necrosis in vital bridge abutments and gender, operators, presence of preexisting restoration or pin(s), pulp capping, reasons for bridge construction, preoperative alveolar bone level, types of cement used, design of the bridge, or period of temporization. An association was found between pulpal necrosis and tooth type ($\chi^2 = 15.77$, df = 5, $P = 0.008$) with maxillary anterior teeth being most often affected (Table 6).

## Comparison between CMCs and fixed bridges

Vital abutments in the BR group were distributed in different regions of the dentition, whereas CMCs were placed mostly in the maxillary anterior segment

**Table 5** Types of retainers used and the development of pulpal necrosis in bridge abutments

| Type of retainers | Number of abutments involved (a) | Number of initially vital abutments (b) | Number showing signs of pulpal necrosis (c) | Percentage of (c) in (b) |
|---|---|---|---|---|
| Metal-ceramic crown | 60 | 45 | 19 | 42.2 |
| Full gold crown | 31 | 28 | 6 | 21.4 |
| Partial veneer crown | 3 | 3 | 0 | – |
| Onlay | 1 | 1 | 0 | – |
| Total | 95 | 77 | 25 | 32.5 |

**Table 6** Frequency of tooth types and the development of pulpal necrosis in the two groups

| | Presence of necrosis in BR group (% of subtotal) | | | Presence of necrosis in CMC group (% of subtotal) | | |
|---|---|---|---|---|---|---|
| Tooth type | No | Yes (%) | Subtotal | No | Yes (%) | Subtotal |
| Maxillary anteriors | 15 | 18 (54.5) | 33 | 59 | 14 (19.2) | 73 |
| Maxillary premolars | 5 | 2 (28.6) | 7 | 16 | 2 (11.1) | 18 |
| Maxillary molars | 7 | 3 (30.0) | 10 | 1 | 2 (66.7) | 3 |
| Mandibular anteriors | 2 | 0 | 2 | 4 | 0 | 4 |
| Mandibular premolars | 12 | 2 (14.3) | 14 | 18 | 1 (5.3) | 19 |
| Mandibular molars | 11 | 0 | 11 | 5 | 0 | 5 |
| Total | 52 | 25 (32.5) | 77 | 103 | 19 (15.6) | 122 |

(Table 6). Significantly greater amount of bridge abutments developed pulp necrosis than teeth restored with CMC ($\chi^2 = 7.82$, df $= 1$, $P = 0.005$).

The survival of pulpal vitality of teeth restored with CMCs was compared with that of bridge abutments; the steeper slope of the survival curve for conventional bridges indicated a greater probability of finding a necrotic pulp in bridge abutments compared with CMCs at the same period of observation (Fig. 1). Using the Kaplan–Meier estimator, the survival rates for pulp vitality were 84.4% (CMC) and 70.8% (BR) after 10 years, and 81.2% (CMC) and 66.2% (BR) after 15 years. The difference was statistically significant between the CMC and BR groups (log rank test, $P < 0.005$).

## Discussion

This is a retrospective study on the incidence of pulpal necrosis, or more accurately, of periapical lesions after placement of a fixed restoration on vital abutment teeth in a cohort of patients treated some years ago. Some 79 patients (37.4% of the total number of patients in this group) with 114 CMCs (40.4% of total number of crowns), and 33 patients (32.4%) with 38 bridges (34.5%) were examined in this study. These response rates were comparable to other studies with similar or longer period of observation (e.g. Karlsson 1986, Valderhaug *et al.* 1997) (see Table 1). The characteristics of the nonrespondents ($n = 24$) from the telephone interview are summarized in Table 7. Ten of them reported that the restoration had dislodged ($n = 2$), had been replaced ($n = 7$), or the tooth extracted ($n = 1$); it remains unknown if these teeth involved were pulpally involved (Table 7). Hence, the result reported here might be an underestimation of the actual incidence. For those other patients who did not respond or could not be contacted by phone or in writing, it is uncertain whether they might have influenced the results.

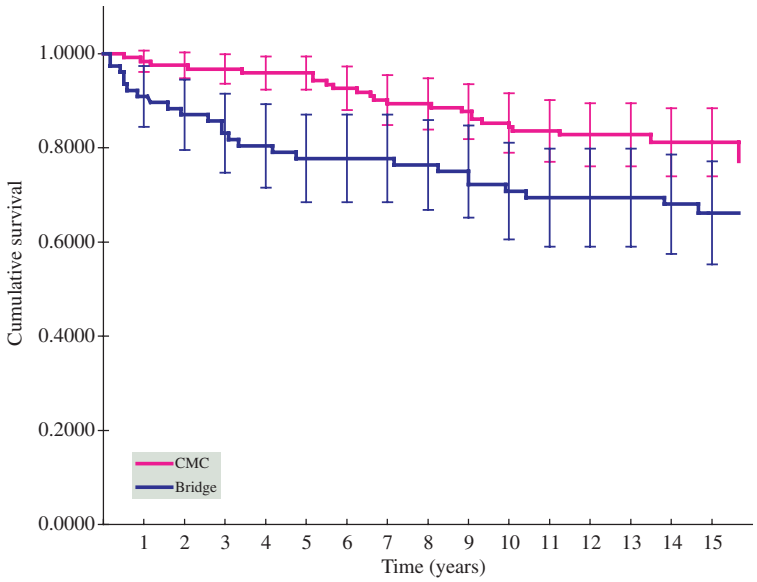


**Figure 1** Survival curves of pulp vitality for teeth restored with CMCs or served as bridge abutment (BR) with the 95% confidence intervals indicated.

**Table 7** Characteristics of the nonrespondents

| Reasons for not coming back | No. of patients | % |
|---|---|---|
| Not interested (interviewed over the phone) | | |
| The crown/bridge was dislodged | 2 | 8.3 |
| A new crown/bridge had been made | 7 | 29.2 |
| The tooth had been extracted | 1 | 4.2 |
| No time | 6 | 25.0 |
| Don't want to come back | 7 | 29.2 |
| Disable or ill | 1 | 4.2 |
| Total no. of nonrespondents | 24 | 100.0 |
| Total no. of patients examined (respondents) | 117 | – |

In the present study, the diagnosis of pulpal necrosis was based on patient's presenting history, clinical signs and symptoms, and the radiographic findings. Pulp testing was not used in most cases because it was not always practicable on teeth with full coverage restorations. Moreover, pulp-testing results might not indicate the state of the pulp correctly (Seltzer *et al.* 1963). As the margins of most crowns were placed equi-gingivally (which was the prevailing teaching at PPDH), it made strict moisture control and application of the electric pulp tester difficult. Some methods to expose the tooth substance apical to the margin of the crown were attempted, such as retracting the free marginal gingivae with a flat plastic instrument, using a piece of gingival retraction cord, or placing a surgical suction tip next to the electrode probe. However, a false-positive result from the gingival tissue still could not be excluded. The use of a mini-probe did not improve the situation either. Cold test with application of ethyl chloride-impregnated cotton pellet or solid carbon dioxide (dry ice) on the metallic portion of the crown/retainer had been tried, but the response was variable. Dry ice appeared to elicit a 'response' more often than ethyl chloride, but, unfortunately, the apparatus for producing dry ice was not available for the whole period of the study. Thus, except for those with discernible root canal filling materials or periapical radiolucent areas, the determination of pulp vitality was based on a careful study of any presenting symptoms and their history and of the radiographs. It was possible that pulpal necrosis could have developed but remained undetected in the present study due to a lack of radiographic change and an absence of clinical sign or symptom. As a result, the prevalence of pulpal necrosis could be regarded as the 'best case' scenario here. On the other hand, any periapical changes as a consequence of pulpal necrosis were likely to have developed and become detectable radiographically in view of the long observation time in this study.

The study design included only those data that indicated the development of a necrotic pulp or the maintenance of pulp vitality over a reasonable period of time. Teeth that did not have any review status, because the patients had failed to attend any recall since the cementation visit, would not provide any censored data in the survival analysis here. Those restorations and the pulps that were diagnosed or recorded as failed and necrotic, respectively, were included for analysis. Thus, there might be a possibility of 'favouritism' for the inclusion of failure cases, which might explain a higher rate of failure for the restorations compared with other studies.

For the survival analysis, the time of failure was taken as the mid-point between the date of diagnosis of failure and the last known date of an intact restoration. Such an estimation is believed to be a better representation of the failure date than using such other time as the date of recall, because most endodontic complications would have taken some time to develop (Cheung 2002). Also, it would take some time before lesions became radiographically discernible, and they might remain undiagnosed until another routine examination or onset of acute symptoms. The time to the development of pulpal necrosis was estimated in a similar fashion for entry into the Kaplan–Meier analysis. Such estimation was believed to be more accurate than assuming the necrosis to have occurred on the day of examination. Only a few long-term studies on the survival or longevity of single-unit extra-coronal restorations were available (Smales & Hawthorne 1997, Walton 1999). These two studies had not used radiographs during their review, which could underestimate the incidence of (asymptomatic) endodontic complications. Another study was in the form of radiographic evaluation without clinical examination (Valderhaug *et al.* 1997). It has been pointed out that many studies have used different survival criteria and analyses, and have presented the survival findings of crowns as various solitary crown types and/or abutments for fixed bridges, making it impossible to compare the study results with any confidence (Leempoel *et al.* 1989).

It is noteworthy that the main reason for failure of both types of restoration examined in the present study was endodontic involvement (Tables 3 and 4). The pulp could have lost its vitality due to a multitude of reasons. Mechanical and chemical insults due to tooth preparation and other clinical procedures, such as the

use of pins and impression taking, and the temporary or permanent luting cements used during the construction of the restoration can lead to pulpal inflammation. But that usually resolves in time if there is no bacterial contamination (Olgart & Bergenholtz 2003). The presence of a preexisting filling may suggest a compromised pulp as a result of previous carious attack and restorative procedures. The period of temporization and the type of cement would have a bearing on the pulp vitality, if marginal leakage of the temporary or permanent restoration was not excluded. It has been the practice at PPDH that all abutment teeth for crowns or bridges are assessed for their vitality status using radiographs and pulp tests prior to the fixed restoration. These tests, however, might fail to indicate the histologic state of pulp with certainty (Seltzer *et al.* 1963, Chambers 1982). Thus, certain teeth might already be suffering from asymptomatic pulpitis preoperatively and further tooth reduction could lead to pulpal necrosis. That may explain why some teeth developed pulpal necrosis shortly after cementation of the crown or bridge.

Using the Kaplan–Meier method, the survival rates for pulp vitality were estimated to be 84.4% (CMC) and 70.8% (BR) after 10 years, and 81.2% (SC) and 66.2% (BR) after 15 years. In a previous survey carried out in PPDH, 57% of all failures of fixed–fixed bridges were endodontic in origin, that is, loss of pulp vitality or presence of a periapical radiolucent area at one or both abutments (Cheung *et al.* 1990). But that study also included, as failures, those previously root filled abutment teeth presenting with periapical lesions and hence a direct comparison with the BR group here could not be made. Another survey in the same hospital reported some 4% of crowned, previously vital teeth developed a periapical lesion after about 3 years (Cheung 1991). The figure seems to agree with the current finding by referring to the survival curve for pulp vitality (Fig. 1), but the present study strongly suggested that the development of pulpal complications was not limited to the first few years after cementation of the restoration, although the rate tended to slow down in time. Valderhaug *et al.* (1997) have also estimated the probability of pulpal complications leading to endodontic treatment in teeth with an initially vital pulp using the Kaplan–Meier method, and reported survival rates of 92% after 10 years and 87% after 20 years. These rates, which were collective of both single crowns and bridge abutments, compare favourably to the findings of this study. However, the method of determining the failure date, i.e. the date of the development of complications, for entry into the survival analysis was not mentioned. If they had taken it as the date of diagnosis or recall (which seemed to be the case as a means of adjustment was not described), it would have the effect of shifting the survival curve to the right on the time-axis and there would be a steeper drop towards the end of the curve when observation was made (Cheung & Chan 2003). The result would be a higher survival rate at any particular point in time on that survival curve, which might explain the difference in the results here.

Whilst many factors could lead to insults to the dental pulp during the construction of a full coverage restoration (Langeland 1961), tooth type was the only significant factor associated with the loss of vitality in the bridge abutments. Over 70% of all pulpal necrosis developed in the maxillary anterior segment in the BR group. It was true that for anterior teeth, a CMC had been used as the retainer, which required more amount of tooth reduction than posterior teeth where a full gold crown might have been used. But the same high rate of pulpal necrosis was not found in the CMC group (Table 6). This significantly higher incidence of pulpal necrosis in bridge abutments might be related to the need of additional tooth reduction to align the preparations for a common path of insertion, hence greater amount of operative trauma (Cheung *et al.* 1990). The deeper and more extensive tooth preparation would result in a greater degree of inflammatory pulpal response (Kim & Trowbridge 1998). Although the underlying pulps were deemed to have failed (as evidenced by the development of periapical rarefaction) in the present study, many of the overlying restorations were still present and functioning in the patients' mouths after endodontic treatment was carried out via access through the crown or bridge retainer. So long as the restoration is present, such endodontic 'mishaps' have not been considered as failures by some investigators (Roberts 1970, Glantz *et al.* 1984, Palmqvist & Swartz 1993). It has been reported that the reduction in retention of crowns, through which an endodontic access had been created, can be regained or surpassed by simple restoration of the access cavity with amalgam (McMullen *et al.* 1990, Mulvay & Abbott 1996). However, little information on the influence of marginal leakage, if any, at the restoration–crown interface is available to date (Trautmann *et al.* 2000).

Preparation of teeth to receive crowns or conventional bridge retainers could involve a large degree of tooth reduction and inflict considerable trauma to the dental pulp. These teeth might already have a range of restorative procedures before, leaving the dental pulps

 

with an impaired ability to recover from trauma from further dental procedures. In order to prevent endodontic complications arising after provision of crowns or conventional fixed–fixed bridges, the operator should undertake the following steps: (1) careful evaluation of the preoperative status of the tooth/teeth; (2) consider alternative treatment options, such as adhesive bridges or implants to replace missing teeth, if the potential abutments are caries-free, vital and have not received any restoration before; (3) avoid over-reduction, overheating or dehydration of the tooth and carry out the tooth preparation with the least amount of trauma to the dental pulps; and (4) provide adequate protection to the dental pulps whilst the final restoration is being fabricated. It is uncertain whether results in this study can be extrapolated to general dental practice. On one hand, these restorations were placed by operators with a wide range of clinical expertise (from dental students to professors) that simulates the general practice situation. On the other hand, some restorations placed by students were likely to have taken more time or number of visits to complete. Regardless of the operator factor, the result on long-term survival of an initially vital pulp beneath these restorations seems to warrant more thoughtful protective measure to maintain the pulpal vitality of the abutments.

## Conclusion

The survival probability of the pulp in vital teeth restored with a single-unit CMC was significantly higher than in those teeth serving as an abutment of a fixed–fixed bridge. Greater number of maxillary anterior teeth serving as bridge abutments developed pulpal necrosis than any other tooth types. The survival rates for pulp vitality were estimated to be 84.4% (CMC) and 70.8% (BR) after 10 years, and 81.2% (CMC) and 66.2% (BR) after 15 years.

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