How 'Clean' Must a Cavity Be before Restoration?

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Abstract

The metabolic activity in dental plaque, the biofilm at the tooth surface, is the driving force behind any loss of mineral from the tooth or cavity surface. The symptoms of the process (the lesion) reflect this activity and can be modified by altering the biofilm, most conveniently by disturbing it by brushing with a fluoride-containing toothpaste. The role of operative dentistry in caries management is to restore the integrity of the tooth surface so that the patient can clean. Thus, the question, 'how clean must a cavity be before restoration?' may be irrelevant. There is little evidence that infected dentine must be removed prior to sealing the tooth. Leaving infected dentine does not seem to result in caries progression, pulpitis or pulp death. However, some of the bacteria survive. What is their fate and if they are not damaging, why is this?

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Caries the Process, Caries the Lesion

The disease dental caries is a dynamic process taking place in dental plaque, the microbial deposit (biofilm) on the tooth surface, which results in a disturbance of the equilibrium between tooth substance and the overlying biofilm. Over time, there may be a net loss of mineral, leading to dissolution of the dental hard tissues and possibly a carious lesion that can be seen [Baelum and Fejerskov, 2003].

In this definition of the caries process, it is the metabolic activity in the biofilm that is the all-important driving force. The demineralization of the enamel and dentine beneath may be seen as a reflection of the dynamic events taking place in the biofilm. The implication of this definition is that the symptoms of the process (the lesion) can be modified by alteration of the biofilm; for instance, the lesion can be modified by regular disturbance of the plaque with a brush and a fluoride-containing toothpaste, the fluoride controlling the rate of lesion progression [ten Cate and Featherstone, 1996].

However, at an advanced stage of caries, sometimes a hole (cavity) in a tooth retains the biofilm and careful brushing cannot remove it. Now operative dentistry has a role to play in caries management to restore the integrity of the tooth so that the patient can clean effectively; but once the enamel is cavitated, the dentine becomes demineralized and infected and now the essential question is: what is driving the caries process? Is it the biofilm at

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the cavity surface or the infected dentine within the cavity, or both? If it is only the biofilm that drives the caries process, the question, 'how "clean" must a cavity be before restoration?' becomes an irrelevance because what matters is sealing the hole in the tooth so that the biofilm can be removed. And yet, the concept of removing infected, demineralized tissue and its replacement by a filling material has spawned a profession, a public and political paymasters who consider that the removal of infected tissue and filling teeth is an essential management of dental caries.

The discussion as to how much tissue must be removed in order to arrest the caries process is not new. In 1859, John Tomes [1859] wrote, 'it is better that a layer of discoloured dentine should be allowed to remain for the protection of the pulp rather than run the risk of sacrificing the tooth', but in 1908, G.V. Black [1908] disagreed claiming '... it will often be a question of whether or not the pulp will be exposed when all decayed dentine overlaying it is removed ... it is better to expose the pulp of a tooth than to leave it covered only with softened dentine'.

The following discussion will look for evidence to confirm or refute the current practice of cleaning infected tissue out of the cavity prior to placing a restoration. This review of evidence must be preceded by a brief description of caries pathology.

Pulpo-Dentinal Reactions in Response to Caries

The shape of the enamel lesion is governed by the activity of the bacteria in the overlying biofilm and the orientation of the enamel crystals. The corresponding pulpo-dentinal reactions are similarly influenced by the biofilm with transmission of the stimulus through the enamel being in the direction of the prisms [Thylstrup and Qvist, 1986]. The implication of this is that when acid production ceases at the surface, due to regular disturbance or removal of the biofilm, lesion progression arrests [Bjørndal, 2002]. The demineralized enamel and dentine remain as scars in the tissue. In non-cavitated enamel lesions, the level of bacterial invasion is very low, if present at all. But once the demineralized enamel crumbles and a cavity forms, the biofilm will form in the hole and the dentine becomes infected. Once the biofilm is directly on the dentine, the lesion spreads laterally along the enamel-dentine junction at the edges of the cavity, as well as back through the sound, undermined enamel.

In these deep lesions, there may be large variations and changes within the lesion environment. The central part of the lesion may be cavitated, open and accessible to plaque removal, by chewing and cleaning. In this area, the lesion may progress slowly. However, peripheral parts of the same cavity may still be protected by undermined enamel with heavy plaque accumulation. In these areas, the lesion may progress more rapidly. Thus, it is possible to have slowly and rapidly progressing parts within the same lesion. It is also possible that an entire lesion is rapidly or slowly progressing and the responses of the pulp-dentine complex to these two speeds of progression vary. As Massler [1967] pointed out a long time ago, it is essential to differentiate active from arrested lesions if one is to make any sense of the biological reactions.

In slowly progressing lesions, increased mineralization of the dentine beneath the enamel lesion is normal. Formation of highly mineralized peritubular dentine corresponding to the affected dentinal tubules reduces the diameter of the tubules. Furthermore, tertiary dentine forms at the pulpal end of the affected tubules (reactionary dentine). The more active the lesion, the more irregular the structure of this dentine. Together, these processes serve to protect the pulp against exogenous destructive stimuli [Bjørndal and Mjör, 2001].

In rapidly progressing lesions, there may be destruction of the odontoblasts and a lack of formation of tertiary dentine. Now the pulpal tissue will react to the transmission of microbial products through a permeable dentine. There will be inflammatory changes in the pulp leading to either reversible or irreversible pulpitis, sometimes associated with sensitivity or pain. Even though the odontoblasts have been destroyed, new odontoblast-like cells may differentiate from the pulp to form tertiary dentine (reparative dentine) if the cariogenic environment is removed or altered [Bjørndal and Mjör, 2001].

It can be seen from the preceding discussion that the lesion entirely reflects the activity in the biofilm, so it is hardly surprising that modifying the biofilm will modify the lesion. How should this understanding of caries pathology influence the operative dentist?

Root Caries

Root caries lesions, accessible to cleaning, are of particular relevance to this discussion. The dentine in such lesions is infected at a relatively early stage in lesion progression [Nyvad and Fejerskov, 1990]. Despite this, active lesions can be converted to inactive lesions over a period of months by regular cleaning and fluoride application [Nyvad and Fejerskov, 1986]. Thus, in these lesions,

Table 1. Chronological overview of studies placing sealants over carious dentine

Study	Treatment	Period	Control	Indication of caries activity	Result and conclusion
Jeronimus et al. [1975]	occlusal lesions of varying depths on bitewing etching, sealant (n = 33) etching, sealant (n = 33) etching, sealant (n = 30) etching, sealant (n = 25)	10 min 2 weeks 3 weeks 4 weeks		gross observation of carious dentine micro-organisms (% positive cultures)	many sealants lost; where sealant was intact, dentine became dry, dark, leathery; decrease in micro-organisms in shallow lesions, but persist in deeper lesions
Handelman et al. [1976]	etching, sealant (n = 60)	0–2 years	untreated (n = 29)	clinical observation, radiography, micro-organisms	no increase in radiographic lesion depth; large reduction of micro-organisms by comparison to controls, increased with time
Going et al. [1978]	etching, sealant (n = 46)	5 years	untreated (n = 21)	clinical observation, micro-organisms	sealed teeth caries arrested; on re-entry either sterile or large reduction in micro-organisms in comparison to controls, but *Streptoccocus mutans* and lactobacilli survived
Mertz-Fairhurst et al. [1979a]	occlusal lesions at DEJ on X-ray; etching, sealant (n = 4)	6–12 months	untreated (n = 4)	lesion depth measurements, micro-organisms	no increase in lesion depth in test; control lesions increased in depth; absence of micro-organisms in test sealed teeth
Mertz-Fairhurst et al. [1979b]	occlusal lesions at DEJ on X-ray; etching, sealant (n = 4)	6–12 months	untreated (n = 6)	clinical observations, radiographs	under sealant dentine powdery, dry, white with hard, glassy, smooth dentine beneath, control dentine spongy, soft, yellow; sealed teeth – no or small increase in depth; control – increase in depth
Jensen and Handelman [1980]	etching, sealant (n = 106)	0–12 months	unsealed, unsealed and etched	micro-organisms	etching alone reduced micro-organisms by 75%; in sealed teeth, bacterial counts reduced with time
Handelman et al. [1981]	etching, sealant (n = 108)	2–5 years	contralateral routine amalgam	radiographic lesion depth	decrease in lesion depth provided sealant intact
Mertz-Fairhurst et al. [1986]	etching, sealant (n = 14)	1–17 months	unsealed (n = 14)	direct lesion depth measurements and radiographs; micro-organisms	unsealed lesions got deeper but sealed lesions did not; all but I sealed lesion, no micro-organisms
Weerheijm et al. [1992]	teeth already etched and sealed but occlusal radiolucency in dentine (n = 30)			micro-organisms: total colony forming units lactobacilli, mutans streptococci, non-mutans strepto-cocci; clinical observation of dentine	cariogenic micro-organisms found in 50% of teeth despite sealant; dentine soft, moist, dark (not leathery, dry)

the operative dentist has no need to cut away the infected dentine in order to arrest the lesion. Subsequently, the arrested root caries lesion is only superficially colonized [Beighton et al., 1993] presumably because the soft, infected dentine has been brushed away.

Does this mean that it is not necessary to remove infected dentine when preparing coronal cavities to receive fillings? Once the restoration is in place, there is no chance for the patient to brush the infected material away. What is the fate of these micro-organisms, entombed by the restorative dentist? Do these lesions remain active or are they arrested?

Fissure Sealant Studies

Table 1 gives a chronological overview of studies investigating the consequences of placing sealants over carious dentine. All studies, with the exception of Weerheijm et al. [1992], were prospective and in many there were unsealed, control, lesions. Caries activity was assessed in a number of ways including clinical observation, lesion depth measurement, radiographic lesion depth measurement and microbiological sampling. Observation periods varied from 2 weeks to 5 years.

The disparity of methodologies militates against a systematic review of the studies, but some uniform themes emerge. Sealed lesions appeared to arrest both clinically and radiographically. Investigations of the fate of the sealed bacteria showed a decrease in micro-organisms with time or their complete elimination. There was no pulpitis reported in sealed teeth. On the other hand, lesions progressed where sealants were lost and in unsealed, control teeth.

The study of Weerheijm et al. [1992] is an interesting outlier. This work was a retrospective examination of sealed teeth where radiographs showed radiolucency in dentine beneath a sealant that was clinically intact. This methodology precluded microbiological sampling before the sealant was placed, which is unfortunate because there can be no comparison of microbial counts before and after sealing. Nevertheless, it is worrying that cariogenic micro-organisms were found in 50% of the teeth and the dentine was often soft and moist, rather than leathery or dry. This would seem to indicate active lesions. The microbiological examination in this work was more detailed than in many other studies examining for lactobacilli, mutans streptococci and non-mutans streptococci. Since there was no preoperative sample, it is impossible to know whether sealing had changed the numbers or the distribution of the microflora.

Classical Caries Excavation

The operative tradition is to remove softened dentine in order to eliminate infected tissue. This approach assumes that both the biofilm and the micro-organisms within the carious dentine drive the caries process. In fact, it is not possible to eliminate all the micro-organisms because a few will remain even if all soft dentine is removed [Lager et al., 2003].

At the enamel-dentine junction, some schools teach that the area should be stain-free as well as hard, but a few bacteria remain whatever approach is adopted and thus it seems logical to leave stain in this area as a more conservative approach [Kidd et al., 1996].

Over the pulpal surface, contemporary teaching recommends that carious dentine that is 'firm and leathery' should be left where its removal might expose the pulp [Hilton and Summitt, 2000]. A calcium hydroxide liner is placed over the demineralized area of dentine and this medicament has been shown to significantly reduce the number of remaining bacteria [Leung et al., 1980]. This procedure is called indirect pulp capping. Vigorous exca-

vation is positively contraindicated, but the student will find that one teacher's definition of 'firm and leathery' is another's 'rather soft' interpretation.

The subjective clinical assessment of carious dentine led Fusayama [1988] to develop a caries dye (acid red in propylene glycol) to differentiate clinically 'infected' from 'affected' dentine. He reported that the more superficial zone of infected dentine was an irreversibly damaged, bacterially infected layer that would never remineralize. The deepest affected dentine was shown to harden as a result of remineralization [Eidelman et al., 1965]. Fusayama's group suggested the dye staining front coincided with the bacterial invasion of the dentine.

However, several studies have reported that the dye does not discretely discriminate the bacterially infected from softened affected tissues [Anderson et al., 1985; Boston and Graver, 1989; Kidd et al., 1993]. Consequently, its injudicious use may lead to over-preparation of the tissues, encouraging excess removal at the enamel-dentine junction [Kidd et al., 1993] as well as unnecessary removal of dentine over the pulpal surface [Yip et al., 1994].

Soft dentine is usually wet but sometimes, particularly when an old restoration has been removed, the dentine may appear crumbly and dry. This dry dentine has been shown to be minimally infected [Kidd et al., 1995] and it may represent residual caries that a previous dentist left during cavity preparation. This may indicate that there is no need to remove soft, wet dentine. The process may be arrestable by simply sealing it in place.

Stepwise Excavation

Stepwise excavation, described by Bodecker [1939], differs from the classical excavation of carious lesions described above. Only part of the soft, dentine caries is removed at the first visit during the acute phase of caries progression. The cavity is restored and re-opened after a period of weeks. Further excavation is now carried out prior to a definitive restoration. The objective of the exercise is to arrest lesion progression and allow the formation of tertiary dentine before final excavation, making pulpal exposure less likely.

This procedure has been investigated scientifically for more than 30 years. These studies have involved baseline investigations of carious dentine and then a re-analysis after a period of sealing it in the tooth. This work is important evidence of the consequences of sealing infected dentine into teeth.

Table 2. Chronological overview of stepwise excavation studies

Study	Toothtype, lesion depth	Treatment	Control	Time to re-entry	Indication of carious activity	Result and conclusion
Law and Lewis [1961]	deciduous and permanent, deep lesions	access to caries then Ca(OH)$_2$ + H$_2$O on dentine; amalgam (n = 66); re-entry at 6 months; excavation completed (n = 57)		6–24 months	clinical observation; observation dentine on re-entry, radio-graphs	76% clinically (no exposure) and radiographically (no pathology) successful
Schouboe and Macdonald [1962]	molars with occlusal caries	access, carious dentine sampled; gold plate over dentine, then amalgam (n = 17)		69–139 days	micro-organisms	positive cultures in 14 cases, on re-entry a different flora
King et al. [1965]	? deciduous, deep lesions, no pulpitis	only deepest layer decayed, dentine left; capped with Ca(OH)$_2$ or ZnO/Eug or amalgam; restored amalgam (n = 51)		25–206 days	observation dentine on re-entry, micro-organisms	initial samples of deep, soft dentine, infected dentine harder on re-entry with Ca(OH)$_2$ and ZnO/Eug but *not* with amalgam; 3/8 teeth exposed after further caries removal with amalgam; micro-organisms on re-entry; Ca(OH)$_2$ teeth 61.4% sterile; ZnO/Eug teeth 81.8% sterile; amalgam teeth 0% sterile but numbers of organisms reduced
Kerkhove et al. [1967]	deciduous and permanent, deep lesions	only deepest layer decayed, dentine left; 41 teeth Ca(OH)$_2$ and amalgam, 35 teeth ZnO/Eug and amalgam		3–12 months	observation of dentine on re-entry, radio-opacity relative to control area assessed visually and densi-tometrically	92% clinical success; on re-entry dentine dry, hard, brownish yellow; increased radio-opacity; very slight time but not material dependant
Magnusson and Sundell [1977]	deciduous, deep lesions, no pulpitis	partial excavation; calcium hydroxide, zinc oxide and eugenol cement; at re-entry all soft carious dentine excavated (n = 55)	full excavation (n = 55)	4–6 weeks	clinical observation, observation dentine on re-entry	15% treatment group pulp exposed, 53% control group pulp exposed
Weerheijm et al. [1993]	permanent molars, small visible occlusal lesions	part of lesion opened to dentine; this filled glass ionomer cement (GIC); remainder sealed GIC; at re-entry all caries removed and composite placed (n = 20)	as treatment but Delton sealant used (n = 4)	7 months	clinical observation of dentine on re-entry micro-organisms	poor retention GIC sealant, micro-organisms 100 times less in re-entry sample but still found in 90% of second samples
Leskell et al. [1996]	permanent, deep, no pulpitis	bulk carious dentine excavated; calcium hydroxide, zinc oxide and eugenol cement; at re-entry all soft dentine removed, excavators or burs (n = 57)	all soft caries removed, Ca(OH)$_2$, ZnO/Eug cement, then GIC composite or amalgam (n = 57)	8–24 weeks	clinical observation	17.5% treatment group exposed, 40% control group exposed
Kreulen et al. [1997]	permanent molars, occlusal caries on radiograph	lesions opened to dentine, filled resin modified glass ionomer (n = 40)	as treatment but filled amalgam (n = 40)	6 months	clinical observation of dentine on re-entry micro-organisms	dentine darker and harder on re-entry; substantial decrease in total viable count, mutans strep-tococci and lactobacilli; more reduction with resin modified glass ionomer than amalgam
Weerheijm et al. [1999]	permanent molars, occlusal caries on radiograph	lesions opened to dentine, filled resin modified glass ionomer (n = 33)	as treatment but filled amalgam (n = 33)	2 years	micro-organisms	25 patients reviewed; substan-tial decrease in total viable count, mutans streptococci and lactobacilli; more decrease in glass ionomer than amalgam; micro-organisms not cultured in 11 out of 50 cases

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Table 2 (continued)

Study	Toothtype, lesion depth	Treatment	Control	Time to re-entry	Indication of carious activity	Result and conclusion
Bjørndal et al. [1997]	permanent teeth, no pulpitis, deep lesions	peripheral excavation and excavation 'cariogenic biomass' and superficial demineralized dentine; calcium hydroxide and temporary filling; at re-entry, complete excavation (n = 31)		6–12 months	clinical observation of dentine on re-entry; micro-organisms	no pulpal exposures at final excavation; at re-entry dentine darker, harder, dryer; substantial reduction in colony-forming units – not time-dependent
Bjørndal and Thylstrup [1998]	permanent teeth, no pulpitis, deep lesions	peripheral excavation and excavation 'cariogenic biomass' and superficial demineralized dentine; calcium hydroxide and temporary restoration (n = 94)		2–9 months	clinical observation of dentine on re-entry; follow up clinical and radiographic examination 1 year after final restoration	dentine harder and darker on re-entry; 5 exposures on final excavation (2 sensitive to pressure, 2 inadequate seal): 88 cases symptomless at 1 year; 1 case lost temporarily and needed root treatment
Bjørndal and Larsen [2000]	permanent teeth, no pulpitis, deep lesions	as above + microbiological sampling (n = 9)		4–6 months	clinical observation of dentine on re-entry; micro-organisms	dentine harder and darker on re-entry; colony-forming units and proportion lactobacilli substantially reduced; gram-negative rods declined; flora dominated by *Actinomyces naeslundii* and various streptococci
Maltz et al. [2002]	permanent teeth, no pulpitis, deep lesions	cavity walls made hard; incomplete caries removal pulpally; calcium hydroxide and zinc oxide and eugenol cement		6–7 months	clinical observation of dentine before and after re-entry; radiographic examination; microorganisms	dentine dryer, harder, darker on re-entry; increase in radio-opacity during study period; bacterial counts decreased significantly

Table 2 gives a chronological overview of stepwise excavation studies. The majority of these studies have no control. Most have been done on permanent teeth with deep lesions. The amount of carious dentine removed at the initial excavation varies from access to caries only, to removing the bulk of the carious dentine.

The restorative materials are also very variable. They include calcium hydroxide, zinc oxide and eugenol, amalgam, glass ionomer cement and composite resin. Times to re-entry are also very variable, the shortest being 3 weeks, the longest 2 years.

Caries activity has been assessed clinically, radiographically and often by microbiological examination at initial entry and on re-entry. With such differing methodologies, a systematic review is not possible but some themes emerge. (1) The clinical success rate appears high. Exposure is usually avoided using the stepwise technique and symptoms rarely arise between excavations. Control lesions are often exposed by conventional excavation. (2) Some studies report the dentine is altered on re-entry, being dryer, harder and darker. (3) Microbiological monitoring indicates substantial reductions in cultivable flora.

Some teeth appear sterile, but in most some micro-organisms survive. Two studies [Bjørndal and Larsen, 2000; Maltz et al., 2002] suggest that the cultivable flora is altered on re-entry to a less cariogenic flora. (4) There is a possibility that there may be an effect from the dental material on the outcome, but very few studies have addressed this in a controlled manner.

Why Re-Enter?

The studies in table 2 seem to show that the depth of the first excavation is not relevant to the level of infection of the soft, dry dentine that is found on re-entry. The final excavation allows the dentist to be sure there is no exposure and removes the remaining infected dentine. The logic here is that the carious process may continue, albeit slowly, in this infected tissue.

However, perhaps there is no need to re-enter and indeed this is the basis of the indirect pulp capping technique [Hilton and Summitt, 2000], although most of the demineralized tissues is removed in this procedure. In

Table 3. Randomized controlled clinical trials of 'complete' versus 'incomplete' caries removal

Study	Tooth type, lesion depth	Treatment	Control	Observation period	Results and conclusions
Magnusson and Sundell [1977]	deciduous, deep but no pulpitis	cavity washed microbiocidal solution; partial excavation, calcium hydroxide, zinc oxide and eugenol cement; at re-entry, all soft carious dentine excavated (n = 55)	all softened dentine excavated regardless of risk of exposure (n = 55)	re-entry: 4–6 weeks in treatment group	treatment: 2 cases pulpitis between visits, dentine 'altered' on re-entry; 15% pulps exposed; control: 53% pulps exposed
Leskell et al. [1996]	permanent, deep but no pulpitis	bulk carious tissue excavated, calcium hydroxide, zinc oxide and eugenol cement; at re-entry all soft dentine removed with excavator or burs (n = 57)	all softened dentine removed; if no exposure, calcium hydroxide, zinc oxide and eugenol cement, glass ionomer cement; in some teeth composite or amalgam on top of this (n = 70)	re-entry: 8–24 weeks in treatment group	treatment: 17.5% pulps exposed; easy to distinguish 'soft' and 'hard' dentine on re-entry; control: 40% pulps exposed
Mertz-Fairhurst et al. [1998]	permanent; occlusal lesions no deeper than halfway into dentine on radiograph	DEJ not made caries free; moist, soft, infected dentine left at DEJ and over pulp; restored bonded, sealed, composite (n = 156)	complete caries removal; amalgam + sealant group (n = 77); conventional amalgam group (n = 79)	no re-entry; 10-year follow-up	no exposure during caries removal; treatment: 85 teeth reviewed at 10 years, caries apparently arrested, 1 lesion 'caved in'; control: some conventional amalgam rest failed with new caries at margin
Ribeiro et al. [1999]	deciduous, no pulpitis, no exposure expected	DEJ made caries free with round bur but moist, soft, infected dentine left over pulp; restored dentine bonding agent and composite (n = 24)	caries removal with slow round bur guided by caries dye; all dye stained dentine removed; restored dentine bonding agent and composite (n = 24)	no re-entry: followed for 1 year; assessed on radiograph and histology	treatment: all restorations retained; excellent marginal integrity after 1 year; on radiograph: 46% regressed, 25% progressed, 29% unchanged; adhesive system formed altered hybrid layer histologically; control: pulpal necrosis in 1 tooth, all other restorations retained; excellent marginal integrity, adhesive system formed hybrid layer

stepwise excavation, on the other hand, soft, wet dentine is left in place. Is it now necessary to re-enter? After all, if the caries process is driven by the activity in the biofilm, the process should be arrested simply by sealing the cavity. The persistence of a few micro-organisms may be irrelevant. Perhaps they are just opportunistic squatters adapted to the new environment in which they find themselves.

Randomized Controlled Clinical Trails

Are there deleterious consequences after incomplete caries removal? Only randomized controlled clinical trials will answer this question and table 3 documents 4 such studies.

Two of these selected deep lesions in deciduous [Magnusson and Sundell, 1977] or permanent [Leskell et al., 1996] teeth where exposure seemed likely following conventional caries removal. Both studies strongly support a stepwise approach (using calcium hydroxide after initial excavation) if pulp exposure is to be avoided. In these cases, conventional caries removal was deleterious; both studies re-entered.

The other two studies in table 3 selected less advanced lesions and did not re-enter to remove the remaining soft dentine in the treatment groups. Both studies sealed incompletely excavated cavities with dentine bonding agents and composite resins. The work of Ribeiro et al. [1999] on deciduous teeth concluded that the clinical performance of the restorations was not adversely affected by

the incomplete caries removal after 1 year. The study by Mertz-Fairhurst et al. [1998] was remarkable for a 10-year follow-up of occlusal restorations placed over moist, soft, infected dentine left both at the enamel-dentine junction and over the pulp. Lesion progression was arrested and there were no more clinical failures in this group than in control groups with conventional caries removal.

What Does the Evidence Tell Us about Our Current Operative Approach?

This review makes uncomfortable reading for those of us teaching operative dentistry. There is no clear evidence that it is deleterious to leave infected dentine, even if it is soft and wet, prior to sealing the cavity. Indeed, this cautious approach may be preferable to vigorous excavation because fewer pulps will be exposed and sealing the dentine from the oral environment encourages arrest of lesion progression. The reparative processes of tubular sclerosis and tertiary dentine are encouraged, thus reducing the permeability of the remaining dentine. The residual micro-organisms are now in a very different environment. They are entombed by the seal of the restoration on one side and the reduced permeability of the remaining dentine on the other. The apparent irrelevance of the infected dentine is biologically logical if it is accepted that the caries process is driven by the biofilm and its reflection is the lesion in the dental hard tissues.

Further Research

One of the most intriguing aspects of this review is the fate of the residual micro-organisms. How do they survive? Is their survival time dependant? Do they change, either phenotypically or genotypically? Do they continue to demineralize the dentine, albeit very slowly? How does the pulp react to their presence in the short and long terms? In view of the numerous studies that show the pulp can be compromised by leakage of bacteria around restorations [Bergenholtz et al., 1982], it is remarkable that their presence does not result in pulpitis and pulp death. It is probably highly relevant that the studies relating bacterial leakage around restorations to pulp pathology are done on caries-free teeth. Thus, cavity preparation will open up millions of tubules, each one a pathway to the pulp. There is a dearth of research that relates the activity of a carious lesion to the histological changes in the underlying pulp. Is it really necessary to extract a tooth to examine pulpal pathology? There seems a need to find a way of monitoring what is going on in vivo.

The stepwise excavation studies in table 2 show many disparate methodologies. Randomized, controlled clinical trials should be designed to compare: the results of the stepwise technique in shallow and deep lesions; superficial caries removal with a deeper excavation; the relevance of the medicament (e.g. calcium hydroxide, zinc oxide and eugenol) and the filling material (amalgam, composite, glass ionomer cement) to the outcome, and the relevance of the time before re-entry to the clinical and microbiological outcome. In addition, this methodology might examine techniques designed to kill bacteria in infected dentine such as ozone treatment [Baysan et al., 2000] photo-activated disinfection [Burns et al., 1995; Williams et al., 2003]. Would these techniques help, hinder or be irrelevant to the clinical outcome?

Further long-term, randomized, controlled clinical trails will be important, but those who have attempted such work must look at the 10-year results of Mertz-Fairhurst et al. [1998] with admiration. The problem in clinical trials is usually an unacceptable loss of patients, but they seemed able to recall many of their patients. Stable populations will be required for these essential long-term studies.

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