Caries Removal and the Pulpo-dentinal Complex

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The Cartwright Prize is awarded every five years by the Faculty of Dental Surgery of the Royal College of Surgeons of England.

The prize was founded in 1884 by the Association of Surgeons practising Dental Surgery with the object of commemorating the services of Samuel Cartwright, FRCS in improving the status of the Dental Profession, not only by inducing many of those engaged in dental practice to become fully qualified Surgeons but also by assisting to obtain recognition of Dentistry as a special branch of Surgery by the Royal College of Surgeons of England. When the Association of Surgeons practising Dental Surgery was dissolved the administration of the Fund for the endowment of the prize was entrusted to the Royal College of Surgeons of England.

In 2000, a first and a second prize were awarded based on an essay submitted on any subjected relating to dental surgery. The first prize was awarded to Edwina Kidd: 'Caries Removal and the Pulpo-dentinal Complex', and the second prize to Martin Ashley: 'It's Only Teething...'.

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Edwina Kidd being awarded the Cartwright Prize 2000 by Mr David Barnard, Dean of the Faculty of Dental Surgery.

> "The complete divorcement of dental practice from studies of the pathology of dental caries, that existed in the past, is an anomaly in science that should not continue. It has the apparent tendency to make dentists mechanics only."
>
> G. V. Black,1908

Writing this essay is uncomfortable but taking shelter behind this quote from one of the Fathers of modern operative dentistry helps! When Black wrote his textbook of *Operative Dentistry* in 1908 he based it on his observations of the disease process and, indeed, devoted one of two volumes to describing the disease in detail.[1]

In the intervening years something has gone strangely wrong. In many dental schools the science of cariology and the technicalities of operative dentistry are taught and researched separately. Generations of students have passed through operative technique courses and phantom head rooms restoring caries-free natural teeth or, even worse, plastic counterfeits. The eventual appearance of the demineralized tissue in living patients on the clinic is a considerable inconvenience, ruining stereotyped outline forms and preconceptions of appropriate depths, widths and angles. For many years your essayist has tried to base her teaching of operative dentistry on the science of cariology and recently this approach appears to have precipitated something of an intellectual crisis over the question of caries removal. What follows is endless questioning and trawling of the literature in search of evidence to confirm or refute our current practice. These are ramblings of a troubled mind that has been uneasy for

some time and it is now challenged to confirm or refute current practice.

CURRENT PRACTICE IN CARIES REMOVAL

There is one textbook of operative dentistry that advises the student to remove the caries and then put the instruments down, look, think and design.[2] But, what is caries and what should be removed and why? The problem is about to be defined. *If dental caries is the tissue destruction caused by bacterial metabolism in the biofilm, or plaque, and if the process is arrestable simply by removing the biofilm, then why do the symptoms of this process (demineralized tissue) have to be removed at all? Why not remove the biofilm and seal the hole in the tooth so that the patient can clean?*

Current practice in caries removal takes away much more than just the biofilm. Enamel is cut back to expose softened, infected dentine. The enamel-dentine junction is further instrumented until it is hard and, in some countries, until it is also stain-free. Over the pulpal surface, softened, demineralized dentine is scooped away with sharp, small spoons called excavators. The point of terminating excavation varies according to country, dental school, the individual teacher's idiosyncrasy and the presumed proximity of the softened tissue to the pulp.

This essay will now try to assemble the biological evidence behind what needs to be removed. The discussion begins by considering what drives the demineralization process on various tooth surfaces.

WHAT DRIVES DEMINERALIZATION OF ENAMEL?

It is reassuring to start this discussion at a point in the carious process where there would be general agreement. The white spot lesion on enamel is the earliest macroscopically visible sign of demineralization. There is general agreement that this may be looked upon as a breakdown in microbial homeostasis in the plaque. It is a local ecological catastrophe.[3] This process is arrestable by re-establishment of plaque control. In time the outer demineralized surface may be worn away and the lustrous translucent appearance of the enamel restored.[4]

At a later stage in the demineralization process, when the carious lesion, although not cavitated, has penetrated further through the enamel, it is still possible to arrest the process by plaque control alone.[5] However, the enamel surface of the arrested lesion still appears white or brown, owing to adsorption of pigments from the mouth. However, the matt appearance of the active lesion changes to the lustrous surface of the arrested lesion. Indeed, plaque removal alone will continue to arrest lesions that appear active even when the outer enamel surface has broken down, provided the cleaning aid can access the cavity to remove the biofilm. It should be noted that this arrest takes place despite the presence of a few pioneer organisms within the enamel.[6] Notice that operative intervention is not required and this was discussed as long ago as 1908 by G.V. Black when he suggested that simple cleaning of buccal surfaces would arrest decay in the enamel.[1]

WHAT DRIVES DEMINERALIZATION ON THE ROOT SURFACE?

The active root surface lesion is plaque-covered, usually close to the gingival margin and the tissue feels soft on gentle probing. These changes may be seen with or without cavitation.[7,8] These lesions do not extend apically as the gingival margin recedes. New lesions develop at the level of the gingival margin in a plaque stagnation area.

The original active lesion may now be left high and dry by the receding gingival margin. If regular plaque removal is established, the lesion changes with its surface becoming shiny, smooth and hard on probing. Indeed, despite the fact that cavitation may have occurred, the root surface may take on a glossy appearance with only discoloration suggesting previous carious activity. These changes show that, just as in the case of an enamel lesion, the root surface is reacting to the dynamic process taking place in the biofilm at the tooth surface. Indeed, this fact is used in the management of root caries where, with time and regular plaque removal, active lesions may become arrested and converted to inactive lesions.[9]

To extend the argument it is now important to consider the different levels of infection in active and arrested root surface lesions.[8] Active lesions are heavily infected with organisms entering the demineralized tissue (cementum initially) at a relatively early stage of tissue destruction. The bacterial invasion takes place along the exposed collagen fibres of the cementum before the demineralization and the infection spreads into the underlying dentine.[10] Arrested lesions, on the other hand, are minimally infected. So what drives the demineralization process? Is it the bacteria in the biofilm at the tooth surface, the bacteria within the cementum and dentine, or both? Since the lesion can be arrested simply by modifying the biofilm,[9] it seems logical to suggest that it is these micro-organisms that are the major players.

If it is accepted that active root surface lesions may be arrested by plaque control, without recourse to operative dentistry, it would appear that leaving infected dentine in this situation is not deleterious to the tooth. This argument may come hard to those taught to manage caries by cutting infected dentine away. The discussion must now be extended to consider lesions where the enamel is cavitated and active lesions have established in the dentine beneath.

WHAT DRIVES DEMINERALIZATION OF CAVITATED CORONAL LESIONS?

In a cavitated coronal lesion the plaque is within the cavity and, if it cannot be removed with a toothbrush or dental floss, the lesion cannot be arrested.[11]

Operative dentistry now has a role to

play. However, again the question must be asked: what is driving the carious process? Is it the microbial plaque in the cavity or the microbial populations within the infected dentine, or both? It is known that these lesions can be arrested by opening up the overlying enamel so that the patient can access the plaque. Indeed, this management was suggested as long ago as 1938 when Anderson was able to promote arrest in large, active lesions of first molars by removing the undermined enamel and making the occlusal surface self-cleansing. He found that the soft surface layer of the lesion was worn away, leaving a hard, darkly pigmented, shiny surface.[12] This is just as is described in arrested root surface caries and, logically, there is no reason why the two processes should differ.

PULPAL REACTIONS TO DENTAL CARIES

Dentine is a vital, cellular tissue, containing the cell processes of the odontoblasts. Thus dentine and pulp must be considered together. The ecological catastrophe in the biofilm is an assault on this vital tissue and it is capable of defending itself. Indeed, the reader may come to the conclusion that the pulpo-dentinal complex might get on better with rather less assistance than the operative dentist currently lavishes upon it!

In 1967, Massler elegantly distilled current scientific knowledge on this subject[13] including describing his own research work carried out over a period of 11 years on more than 800 human teeth. His sense of frustration at some of his colleagues jumps from the page as he writes:

> "It is somewhat disturbing to the biologically orientated clinical teacher to witness the overly focused attention of some dentists upon the operative and restorative phases of dentistry, the 'drilling and filling' of teeth, to the neglect of the disease process which causes the lesion (cariology) and the preoperative treatment of the wounded tooth-bone".

Here is the re-incarnation of G.V. Black's plea, written some 60 years earlier and reproduced at the beginning of this essay.

A combination of defence and degenerative reactions characterize the carious process in the pulpo-dentinal complex. Massler's particular contribution was to point out how essential it is to differentiate active from arrested lesions if one is to make any sense of the biological reactions. From this a logical management follows that seeks to convert an active lesion into an inactive or arrested lesion thus aiding the defence and healing processes in dentine and pulp *before* restorative procedures are attempted.

ACTIVE AND ARRESTED ENAMEL LESIONS

Massler showed that under an active lesion the dentinal tubules were permeable to dyes placed in the pulp and to isotopes placed on the enamel surface.[13,14] Under arrested lesions, however, there were sclerotic zones in the dentine that were impermeable to dyes and isotopes. He pointed out evidence going back to 1929 to show that active lesions could be arrested by merely removing plaque[13] and applying fluoride.

ACTIVE AND ARRESTED DENTINAL CARIES

Massler described an active lesion as characterized by an active bacterial colony on the surface (the infected layer) and a very wide layer of demineralized dentine beneath, containing few pathogenic micro-organisms (the affected dentine).[15] However, the dentine tubules in active lesions are very permeable to dye tracers[16] and the lesions are painful, presumably because the tubular contents are still vital.[17]

Arrested lesions can be identified by a hard, leathery and deeply pigmented surface layer beneath which a layer of sclerotic dentine is invariably found plus a layer of reparative dentine (within the pulp). Spontaneous pain and painful reactions to sweets and acids are generally absent. He subsequently pointed out that most lesions found clinically were a combination of active and arrested lesions. At the periphery of the lesion an active lesion is often spreading under the overhanging enamel, along the enamel-dentine junction, while the central, more easily cleaned area is hard and partially remineralized. This argument is almost identical to the argument presented earlier in this essay where it was suggested that it is the biomass at the surface of the lesion which drives the carious process.

DEFENCE REACTIONS UNDER DENTAL CARIES

Massler points out that the destruction of dentine appears to require both an acidogenic phase to demineralize and a proteolytic phase to break down the demineralized organic matrix. He claims the acidogenic phase precedes the proteolytic phase but questions whether it is acid or bacterial enzymes that are responsible for proteolysis.

He shows that defence reactions probably commence at the end of the acidogenic phase. As far as sclerotic dentine is concerned, he shows this comprises two layers. The more superficial probably results from a reprecipitation of dissolved mineral salts.[18] This area may receive calcium salts from saliva and from calcium hydroxide when this is placed over carious dentine.[19] The second sclerotic layer lies deeper, within the normal viable dentine and probably acquires its calcium via the pulp.[20,21]

The plugging of the tubules forms a very effective barrier against further penetration of toxic materials towards the pulp – a barrier that is probably more effective against penetration by isotopes and dyes than any base or filling material.[15,16] As we shall see later, this a zone that some dentists would advocate attacking with a bur and this, biologically, must be crazy.

With respect to reparative dentine, a relationship has been shown between the trauma of cavity preparation and the

amount of dentine formed centrally[22] – a cute mechanism if ever there was one!

DEEP DENTINAL LESIONS AND PULP EXPOSURES

Massler's work shows that deep and extensive dentinal lesions are the result of a number of carious attacks and repair. Although rapidly penetrating carious lesions occur, these are not the norm. He suggests that the frequency of pulp exposures in deep caries is often the result of too vigorous removal of affected dentine and he advocates gentle pre-operative treatment of the dentinal lesion to promote sclerosis and repair prior to operative procedures. This he describes as 'indirect pulp capping'[19, 23,24] but 'stepwise excavation'[25] would also come under this heading.

What is perhaps surprising is that this logical dentist suggested gentle operative procedures *prior to* operative dentistry. He did not suggest such procedures *instead of* conventional caries removal and this possibility will be discussed as this essay unfolds.

CAN AND SHOULD INFECTED DENTINE BE REMOVED?

Thus far the evidence presented has argued strongly that it is the biofilm at the lesion surface that drives the carious process and that only this layer must be removed in order to arrest the lesion. Supposing, however, a clinician disagrees with this interpretation of the evidence and wishes to remove all infected dentine; can this be achieved?

The studies that addressed this question were reviewed by Shovelton in1968.[26] His review showed that softening of dentine generally precedes the organisms responsible for it[27] but a few organisms will remain even if all soft dentine is removed. These organisms remain viable beneath restorations without apparently causing any detrimental effect. It is thought-provoking that MacGregor reported as early as 1962 that the area of low pH in the dentine was actually deep to the softened area. This implies that even complete removal of the softened layer would not remove this acidic front.[28]

WHAT CRITERIA WOULD GUIDE A CLINICIAN WHO WISHED TO REMOVE INFECTED DENTINE?

The author of this essay is now in deep trouble! She has yet to produce any evidence that it is necessary to remove infected dentine and indeed has shown it is not possible to do this. So why does she now subject the reader to the criteria that should be used to remove infected dentine? She confesses a lack of logic and explains herself by rather pathetically claiming that the removal of infected dentine is still being taught, and by her![2,29]

The most commonly used criterion for the removal of infected dentine is 'to scoop out the soft stuff' with an excavator. At the enamel-dentine junction, some schools teach, the area should be made stain-free as well as hard, others just say hard and ignore stain. Since staining is an unreliable guide to the level of infection of the dentine, and since a few bacteria will remain whatever approach is adopted, the more conservative approach of leaving stain seems the less illogical of the two.[29]

Over the pulpal surface, stained dentine should remain as long as it is reasonably hard. Provided a tooth is symptomless and responds as vital to pulp testing, vigorous excavation over the pulpal surface seems positively contraindicated once the cavity floor is reasonably firm. (The student will find that one teacher's definition of 'reasonably firm' is another teacher's 'rather soft' and since there is no evidence to support or refute either approach, it is difficult to be more specific).

The subjectivity of these assessments led to the development by Fusayama[30,31] of red dyes to be used clinically to differentiate 'infected' from 'affected' dentine. Infected dentine was shown to be an irreversibly damaged layer while affected dentine was the inner, remineralizable zone. The same authors tentatively suggested that the dye staining front coincided with the bacterial invasion front.

Thus, in theory, this dye could be used to identify the carious tissue which is infected with bacteria and thus needs to be excavated. Subsequently, a number of studies[32,33,34] showed that the dye does not necessarily discriminate infected tissue and use of the dye could lead to the over-preparation of cavities encouraging removal of excess tissue at the enamel-dentine junction[34] and removal of sclerotic and reparative dentine over the pulpal surface.[35]

STEPWISE EXCAVATION

Stepwise excavation differs from the classical excavation of carious lesions described above. Only the necrotic layer of dentine is removed at the first visit during the acute phase of caries progression. The demineralized dentine is covered with calcium hydroxide or zinc oxide and eugenol[25,36-39] before placing a temporary restoration, although amalgam and resin-modified glass ionomer cement have also been placed directly on the demineralized dentine.[40,41]

After a period of weeks, cavities are re-opened and further excavation carried out prior to definitive restoration. The initial approach is very similar to removal of the biofilm except that a small amount of space is made for the restorative material that will seal the cavity.[25,36-43] Where the stepwise approach has been compared to conventional complete excavation, two controlled trials have shown many more pulpal exposures in the latter groups.[38,39]

WHAT HAPPENS TO THE BACTERIA?

Several studies have examined bacterial survival in these incompletely excavated cavities after varying periods with temporary fillings.[25,36,40,41] The common trend in these reports is the observation of marked reductions in bacterial growth with one controlled study showing calcium hydroxide and zinc oxide and eugenol to be more effective in this

respect than amalgam.[36] Can these residual bacteria continue the carious process, albeit at a slower rate? There is no evidence that they can. How do they survive? Presumably from pulpal blood flow or perhaps they derive sufficient nutrient from the tissue in which they remain.

CLINICAL DENTINE ALTERATIONS

An intriguing feature of these studies was that, on re-entry, the dentine was found to be darker, harder and dryer after the treatment interval.[25,36-38] Although the authors note this, they do not attempt to explain it. Why should these changes occur?

Why should the lesion be darker? Since it is not even clear why demineralized dentine is coloured brown, this is difficult to explain. One possibility is that the brown colour is caused by the Maillard reaction which is the formation of brown pigments when protein is denatured in the presence of sugar.[44] However, it would seem more logical that stepwise excavation would stop this reaction, not start it! Another possible reason that demineralized dentine is brown is that the colour is exogenous and taken up by the porous tissue.[45] It would seem unlikely that the temporary restorative materials leak so, unless the colour is coming from the pulpal side, this cause of the colour change seems unlikely. One other possibility is that the bacteria remaining are producing the pigments. None of these explanations seem really convincing.

Why should the lesion be harder and dryer? One possibility is that reprecipitation of mineral has occurred as the lesion becomes less acid. Alternatively, perhaps a medicament such as calcium hydroxide encourages remineralization. However, where does the liquid go? Perhaps it is taken up by the restorative material or perhaps it goes into the pulp. The latter seems unlikely since pulpal hydrostatic pressure is outwards and freshly cut dentine oozes moisture. Thus the dentine should get wetter not drier!

WHY RE-ENTER?

It seems remarkable that the need to re-enter has rarely been questioned. In the face of the evidence presented in this chapter, re-entry seems akin to digging up bulbs to see if they are growing! Two studies have had the courage not to re-enter[46,47] and the work of Mertz-Fairhurst *et al.* is of particular interest because they have now presented 10 years' results of a series of occlusal restorations where soft, wet, demineralized tissue was allowed to remain.[47] In this study, a divergent bevel, at least 1 mm wide, was placed in the sound enamel surrounding a frankly cavitated lesion. The undermined enamel and the soft demineralized dentine below the bevel was not removed. Shreds of carious dentine or other material were frequently hanging below the bevel where the soft and wet pulpal floor of the cavity could be seen. There was absolutely no instrumentation below the enamel bevel. The cavity was now washed and dried, etched, bonded and restored with composite resin. The adjacent enamel and residual fissure system were now sealed. There were two control groups of restorations. In these teeth all chalky enamel and soft demineralized dentine was removed leaving only dentine or enamel that was stained and hard. In one of these groups the fissures were removed with a bur and the teeth restored with amalgam. In the other group, an amalgam was placed and the remaining fissures were protected with a fissure sealant. The bonded and sealed composite restorations placed over frankly cavitated lesions arrested the progress of these lesions over a period of 10 years.

WHY NOT JUST SEAL THE CAVITY?

If infected dentine may safely be left, might it be possible to arrest the carious process by simply sealing the cavity in the tooth? This was investigated in clinical trials by Handelman who reviewed his work in 1991.[48] Occlusal lesions were sealed for time periods up to two years and samples of carious dentine were taken for microbiological examination from both sealed and unsealed control teeth. Results showed that a major reduction in cultivable bacteria occurred after two weeks, with a gradual reduction in the total count thereafter. Another study re-entered a number of fissure-sealed teeth with occlusal lesions that were radiographically in dentine. Results showed cultivable bacteria in many of these.[41,49]

However, perhaps the more important discussion concerns whether demineralization will progress beneath sealants. Handelman used radiographs to evaluate whether the carious process would progress beneath sealed occlusal lesions for periods of up to four years. The results showed that, radiographically, the lesions appeared to regress, provided the sealant remained intact. Even when there was some sealant loss there was no radiographic evidence of caries progression in a two-year study.[50]

CONCLUSION

The discussion in the previous pages can be likened to some military exercise. The author follows contemporary operative practice and advances into the infected and affected dentine only to be forced to retreat to the tooth surface again, beaten back by weight of evidence. Academic retreat seems as ignominious as military failure and the conclusion is an uncomfortable one for someone who has taught operative dentistry for 30 years. There would appear to be little logic in the current practice of caries removal. Biologically, it would appear to be potentially damaging even to attempt to remove all infected dentine. It is not even possible to achieve this. The evidence would seem to show that, provided a restoration is placed that seals the cavity, infected dentine may be left. It does not prejudice pulpal health and the carious process does not continue. These statements appear logical and predictable if it is accepted that it is the biofilm at the tooth surface that drives the carious process.

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