Resin bonding to cervical sclerotic dentin: A review

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Summary
Several reports have indicated that resin bond strengths to noncarious sclerotic cervical dentine are lower than bonds made to normal dentine. This is thought to be due to tubule occlusion by mineral salts, preventing resin tag formation. The purpose of this review was to critically examine what is known about the structure of this type of dentine. Recent transmission electron microscopy revealed that in addition to occlusion of the tubules by mineral crystals, many parts of wedge-shaped cervical lesions contain a hypermineralised surface that resists the etching action of both self-etching primers and phosphoric acid. This layer prevents hybridisation of the underlying sclerotic dentine. In addition, bacteria are often detected on top of the hypermineralised layer. Sometimes the bacteria were embedded in a partially mineralised matrix. Acidic conditioners and resins penetrate variable distances into these multilayered structures. Examination of both sides of the failed bonds revealed a wide variation in fracture patterns that involved all of these structures. Microtensile bond strengths to the occlusal, gingival and deepest portions of these wedge-shaped lesions were significantly lower than similar areas artificially prepared in normal teeth. When resin bonds to sclerotic dentine are extended to include peripheral sound dentine, their bond strengths are probably high enough to permit retention of class V restorations by adhesion, without additional retention.

Introduction
The efficacy of current adhesive systems is often evaluated based upon their ability to bond to sound dentine. Although many dentists bond to sound dentine, a variety of pathological dentin substrates are encountered in the clinical practice, which include carious and sclerotic dentin. It is ironic that our current knowledge on the variability of clinical bonding substrates is so limited compared with the progress achieved in adhesive technology. This review examined the ultrastructure and bonding characteristics of one type of abnormal bonding substrate – noncarious sclerotic dentine. Essentially, it compared the obstacles in bonding to sound vs. sclerotic dentine. It will be seen that there is a tremendous difference in substrate morphology as well as that of the resin–dentine interfaces created by bonding to such substrates. The recent introduction of contemporary self-etching primers and...
the timely all-in-one adhesives are redefining adhesive dentistry. Because these simplified adhesive systems are easy to use, we have placed extra emphasis on the interfaces created by these agents on sclerotic cervical dentine.

Noncarious cervical sclerotic dentine

Noncarious cervical sclerotic lesions was described by Zsigmondy in 1894 as angular defects, and by Miller in 1907 as 'wasting of tooth tissue' that was characterised by a slow and gradual loss of tooth substances resulting in smooth, wedge-shaped defects along the cemento-enamel junction. The multifactorial etiology of these cervical lesions has been extensively reviewed. There is increasing evidence of the possible role of eccentric occlusal stress in the pathogenesis of these hard tissue defects. Recent studies on simulation of wedge-shaped cervical lesions using various finite element analytical models of cuspal flexure confirmed the contribution of stress induction in these so-called 'abfraction lesions'. Unrestored, angular, wedge-shaped lesions demonstrated severe stress concentration that varied with the location of the teeth in the oral cavity, with the highest stress contractions along the maxillary incisors and premolars. These stresses were only partially relieved after these lesions were restored.

Sclerotic dentine is a clinically relevant bonding substrate in which the dentine has been physiologically and pathologically altered, partly as the body's natural defense mechanism to insult, and partly as a consequence of colonisation by the oral microflora. Partial or complete obliteration of the dentinal tubules with tube- or rod-like sclerotic casts is commonly observed. Depending on the degree of clinical sensitivity of the lesion, various levels of tubular patency were observed, with most dentinal tubules being occluded within the insensitve transparent dentine regions.

In the absence of undercut retention, cervical sclerotic dentine was found to be more difficult to adhere to than normal dentin both in vitro and in vivo, even with increased etching time. Recent studies showed that the sclerotic casts that obliterated the dentinal tubules were still present after acid-conditioning of the sclerotic dentine, resulting in minimal or no resin tag formation. Furthermore, the zone of resin-impregnated sclerotic dentine was found to be thinner than those observed in normal dentine.

Due to the thinness of hybrid layers in sclerotic dentine, and the complexity of the resin-bonded interface, regional tensile bond strength to cervical sclerotic root dentine with some contemporary adhesives was found to be 20–45% lower than those bonded to artificial wedge-shaped lesions created in normal cervical root dentine. This was attributed to: (a) the presence of sclerotic casts within dentinal tubules that precluded optimal resin infiltration into the dentinal tubules, and/or (b) the presence of a surface hypermineralised layer that is more resistant to acid-etching. It was postulated that an adhesive strategy that involved micromechanical interlocking by the formation of a resin–dentine interdiffusion zone combined with resin-tag formation into the dentinal tubules would be less effective when applied to the hypermineralised sclerotic dentine. Contrary to these findings, a recent study suggested that phosphoric acid-etching was detrimental to the bonding of sclerotic dentine, and that sclerotic dentine that was treated with a hydrophilic primer exhibited better marginal adaptation of resin composites than similarly primed normal dentine. These authors recommended that the layer of sclerotic dentine be preserved for optimal bonding in cervical lesions.

Scanning electron microscopy of such surfaces, even with the use of field emission-type microscopes, does not provide sufficient detail to understand the complex subsurface structures, or to reveal how well these structures are demineralised during etching. Information is best obtained using transmission electron microscopy (TEM). In this review, extensive TEM examinations were used to evaluate biologic variations in sclerotic cervical dentine. This is not an exhaustive review of the literature on noncarious cervical lesions or in resin-bonding to sclerotic dentine. Rather, it is an attempt to summarise a number of studies that provide a rationale for why resin bonds to sclerotic, noncarious, wedge-shape cervical lesions are lower than those made to normal dentine at those same sites. This review will be divided into two sections. In Section 1, microstructural changes that exist in noncarious, sclerotic cervical dentine will be summarised. This is followed by a review on the application of adhesive resins to this altered bonding substrate.

Microstructural changes in sclerotic dentine

Tubular occlusion

In dentine sclerosis, tubular obliteration with rhombohedral, whitlockite crystallites (Fig. 1A)
has been well documented at an ultrastructural level. A high degree of variation could be observed even within a single lesion. While some tubules may be completely devoid of, or sparsely occluded with crystallites, others may be heavily obliterated with peritubular dentine. Toward the surface of the lesion, these crystallites were reduced in size and formed columns of agglomerates that completely plug the tubular orifices. They were often referred to as sclerotic casts. At an ultrastructural level, these tiny, electron-dense crystallites were surrounded by a tube-like membranous structure that probably represented a mineralised form of the lamina limitans of the dentinal tubule.

Hypermineralised surface layer in shiny sclerotic lesions

Unlike tubular occlusion, the presence of a surface hypermineralised layer in natural cervical sclerotic wedge-shaped lesions has only been elucidated through the use of microradiography and FTIR photoacoustic spectroscopic analysis. Although it has been speculated that the hypermineralised...
layer is devoid of collagen fibrils, the ultrastructural features of this layer were only verified recently. Fig. 3A is a toluidine blue-stained, undemineralised thin section taken from the deepest part of a wedge-shaped defect that is least accessible to tooth brushing. In the preparation of the specimen, the surface of the lesion was not cleaned and was fixed in Karnovsky’s fixative prior to the embedding protocol for TEM preparation. A surface layer of stained, unmineralised filamentous bacteria could be seen, beneath which was an approximately 15 \( \mu \text{m} \) thick hypermineralised layer.

Mineralised bacteria could vaguely be discerned within this layer. The corresponding undemineralised TEM image (Fig. 3B) shows that both the mineralised plaque zone and the surface layer of the lesion are hypermineralised with respect to the underlying sclerotic dentine. At a higher magnification (Fig. 4), plate-like minerals could be recognised within intermicrobial matrix.

The ultrastructure of the surface hypermineralised layer is highly variable within the deepest part of the wedge-shaped lesions. This can be better discerned using demineralised TEM sections. It is interesting to observe that such a layer is still retained after complete demineralisation of the underlying sclerotic dentine. Fig. 5A shows a thick, continuous, dense hypermineralised layer that contained two different species of bacteria. One species was trapped within this layer, while the other was found to grow on the surface of this layer. The hypermineralised layer in Fig. 5B, on the other hand, consists of several thin, discontinuous layers that were sandwiched among different species of bacteria. This suggests that changes in the microecology of the oral environment may have resulted in the colonisation of different species of bacteria at sequential periods. Each colony of bacteria was, in turn, mineralised prior to the deposition of the next colony. It is pertinent to point out that both specimens, from which Fig. 5A and B were taken, were brushed cleaned with a mixture of chlorhexidine and pumice prior to laboratory processing. Both appeared as clean, highly shiny lesions when examined under magnification lens.

Figure 3 (A) Light microscopic image of an undemineralised section taken from the deepest part of an untreated noncarious cervical sclerotic lesion. B: stained, unmineralised bacteria; HM: hypermineralised surface layer of the lesion; SD: intact sclerotic dentine; Pointers: mineralised bacteria ghosts. (B) Corresponding unstained TEM micrograph of the undemineralised section. The hypermineralised nature of the surface layer (HM) could be recognised by comparing its electron density with the underlying sclerotic dentine (SD) (from Tay et al., 2000, with permission).

Figure 4 (A) Undemineralised TEM micrograph of a bacterium (B) within the hypermineralised layer (HM). The intermicrobial matrix (i.e. the material between adjacent bacteria) was mineralised and contained plate-like crystallites (pointer) (from Tay et al., 2000, with permission).

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The presence of colonising bacteria on the surface of sclerotic wedge-shaped lesions is consistent with the report of Spranger. The microecology beneath bacterial plaque changes over time depending on the metabolism of the microorganisms. This results in substantial pH fluctuations along the tooth surface. Bacterial products may trigger gingival inflammation with an increased rate of sulcular fluid flow, which in turn, provides nutrition for the microorganisms. If a plentiful supply of fermentable carbohydrates is available, the microbes release organic acids that will lower the plaque pH and tend to demineralise the underlying dental hard tissue. When the carbohydrate source is depleted, the local pH rises due to salivary buffering, and remineralisation of the dental hard tissue and mineralisation of the dental plaque can proceed. In the absence of carbohydrates, these bacteria may remain viable for prolonged periods, utilising glyogen-like intracellular polysaccharide as a metabolisable source of carbon during periods of nutrient deprivation. They may also metabolise amino acids and other nitrogenous substrates, creating ammonia and other basic chemicals that may elevate plaque pH and promote mineralisation and, perhaps, hypermineralisation.

Along the occlusal wall of the wedge-shaped lesions, both the surface bacterial layer and the hypermineralised layer are usually thinner, with the latter between 1 and 2 μm thick. Similarly, the gingival wall of a wedge-shaped lesion, which is usually more accessible to tooth brushing, is usually devoid of bacteria. However, a very thin surface hypermineralised layer may sometimes be observed. Such a layer, if present is around 200-300 nm thick and may be readily discerned in undemineralised sections by its increased electron density, as well as the characteristic arrangement of the crystallites, which will be discussed in Section 3.3.

**Mineral distribution**

Minerals present within the hypermineralised layer are larger in size compared with those within the underlying sclerotic dentin (Fig. 6A). Unlike crystallites within the underlying dentine that are randomly arranged, those that are found in the hypermineralised layer are longitudinally aligned along the c-axis of the crystallites. This is clearly demonstrated in Fig. 6B. Orientation of the crystallites along their c-axis in the surface hypermineralised layer is analogous to the induction of cellular orientation by low-level electrical currents and the alignment of filler particles within resin composites under
the application of an electric field. Dentine is only mildly piezoelectric compared with bone. However, piezoelectric potentials have been reported to be generated when teeth are subjected to parafunc-tional loading. Some have speculated that polarisation of the remineralised crystallites caused by piezoelectric potentials created during eccentric tooth flexure results in their attraction and repulsion by dipole interaction. Dipole interaction competes with randomisation caused by Brownian motion and alignment occurs as the dipole interaction predominates.

**STEM/EDX analysis**

Hypermineralisation implies that the density of the mineral within the surface layer of the defect is higher than that of the underlying sclerotic dentine. This is confirmed by a qualitative STEM/EDX line scan of the calcium and phosphorus distribution longitudinally across the surface layer of the defect into the underlying sclerotic dentine (Fig. 7A and B). Also evident in the line scan is the presence of a region of decreasing calcium and phosphorus counts along the top 500 nm of the hypermineralised layer that is attributed to partial

**Figure 6** (A) Crystallites (pointer) with the hypermineralised layer (HM) were plate-like and were aligned along the c-axis. They were also larger than those (arrowhead) present within the underlying sclerotic dentine (SD). (B) The characteristic orientation of crystallites along their c-axis was also evident in a thick hypermineralised layer taken from the deepest part of another wedge-shaped lesion. The lesion surface was etched by a self-etching primer, with blunting and rounding of the partially dissolved crystallites.

**Figure 7** (A) Brightfield STEM image showing the location of a line scan that was performed across the surface hypermineralised layer of a noncarious cervical sclerotic lesion (from Tay et al., 2000, with permission). (B) Qualitative energy dispersive line scans showing the distribution of calcium and phosphorus along the adhesive, the surface hypermineralised layer of the lesion and the underlying intact sclerotic dentine (from Tay et al., 2000, with permission).
demineralisation by a self-etching primer (Clearfil Liner Bond 2V; Kuraray Medical, Inc., Tokyo, Japan) that was used to bond to sclerotic lesions.57

Quantitative energy dispersive X-ray spectra of the elemental content of crystallites present within (a) the surface hypermineralised layer, (b) the underlying intertubular dentine, and (c) within the dentinal tubules of the wedge-shape defect are shown in Fig. 8.57 As the analysis was performed using 70 nm thick undemineralised sections, this enabled estimation of the calcium/phosphate ratio of these crystallites without additional ZAF correction. The Ca/P ratios of the crystallites within the hypermineralised layer and the underlying dentine approach the theoretical value of 1.67 calculated for hydroxyapatite.58 The larger apatite crystallites observed in the surface hypermineralised layer are similar to larger apatite crystallites reported in remineralised carious dentin59,60 and cementum.61 Conversely, the Ca/P ratio of crystallites from the sclerotic casts within the dentinal tubules is slightly lower than the calculated value of 1.50 for

![Energy spectra from different locations of a noncarious cervical sclerotic lesion. (a) Spectrum showing composition of crystallites within the surface hypermineralised layer. (b) Spectrum of crystallites within the underlying intact sclerotic dentine. (c) Spectrum of crystallites occupying the lumen of a dentinal tubule. Spectra were obtained using a 7 nm probe for 200 live seconds at 200 kV. The relative concentration of Ca, P and Mg, and the calculated Ca/P ratios are shown in the table beneath the spectra (modified from Tay et al., 2000,57 with permission).](image)

<table>
<thead>
<tr>
<th>Region from which crystallites were analysed</th>
<th>Mg (wt %)</th>
<th>Ca (wt %)</th>
<th>P (wt %)</th>
<th>Ca/P ratio*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surface hypermineralised layer</td>
<td>0</td>
<td>62.5 0.9</td>
<td>37.5 0.9</td>
<td>1.7 0.1</td>
</tr>
<tr>
<td>Underlying sclerotic dentine</td>
<td>0</td>
<td>63.1 0.5</td>
<td>36.9 0.5</td>
<td>1.7 0.1</td>
</tr>
<tr>
<td>Sclerotic casts within dentinal tubules</td>
<td>4.6 1.0</td>
<td>55.3 1.8</td>
<td>40.2 1.3</td>
<td>1.4 0.1</td>
</tr>
</tbody>
</table>
tricalcium phosphate. The additional presence of about 5% magnesium suggests that these crystallites are whitlockite (Mg substituted β-tricalcium phosphate).

**Status of the collagen fibrils within the surface hypermineralised layer**

An intriguing question that has remained unanswered is whether the surface hypermineralised layer is devoid of collagen. Using special staining for collagen, it can be seen that the supporting matrix for the crystallites within the hypermineralised layer consists of a bed of denatured collagen (Fig. 9A). The transition from denatured collagen (gelatin) to intact collagen with cross-banding is evident at the base of the hypermineralised layer, where some of the collagen fibrils are observed to unravel into microfibrillar subunits. This transition from banded collagen to denatured microfibrils is further illustrated beneath a layer of bacteria in Fig. 9B, in which unraveling of collagen fibrils created a network of microfibrillar strands that no longer showed cross-banding.

It is possible that colonisation of bacteria along the surface of the wedge-shaped defect results in the production of acidic and enzyme by-products that demineralise and denature the collagen fibrils (Fig. 9B). Diffusion of the enzymes from the demineralised surface downwards probably accounts for the transition from completely denatured collagen to unravelled microfibrils and finally intact collagen within the underlying sclerotic dentine. The loss of phosphoproteins and subsequent remineralisation of the surface bed of denatured collagen under the possible influence of piezoelectric charges generated from eccentric flexural deformation may result in the characteristic orientation of the crystallites within the denatured microfibrillar matrix of the surface hypermineralised layer.

**Summary of the microstructural changes in noncarious sclerotic cervical dentine**

Sclerotic dentine is an abnormal bonding substrate that exhibits a high degree of variability both in terms of occlusion of the dentinal tubules as well as the thickness of the surface hypermineralised layer. The latter, in particular is invariably associated with bacteria. It is possible that bacteria are involved in the pathogenesis of the hypermineralised layer. That is, dentine may first require demineralisation before it can be hypermineralised. Also, mineral crystallites cannot accumulate without a scaffold. The presence of bacteria, apart from demineralising the dentine, also denatures the existing collagen matrix, resulting in a bed of denatured collagen. This may act as a scaffold upon which the demineralised dentin may be subsequently remineralised. The formation of the hypermineralised layer is probably also enhanced by the presence of high concentrations (10 ppm) of fluoride ions. As denaturing of the collagen fibrils probably removes some of the restrictions on the size of
crystallite formation, this may enable larger crystallites to be formed during the process of remineralisation. The unique arrangement of the crystallites further suggests the complex role of parafunctional stress on the formation of the surface hypermineralised layer in these natural wedge-shaped lesions. The presence of bacteria, mineralised bacterial matrices, hypermineralised surfaces and mineral occluded tubules makes sclerotic cervical dentine a unique multi-layered bonding substrate. This implies that bonding studies that attempt to generate hypermineralised dentine in vitro by immersing partially demineralised dentine in a remineralising solution containing a high fluoride content do not simulate the actual bonding conditions that clinicians are likely to encounter when bonding to noncarious cervical sclerotic dentine.

**Bonding adhesive resins to sclerotic dentine**

Current dentine adhesives employ two different means to achieve the goal of micromechanical retention between resin and dentine. The first method, the total-etch or etch and rinse technique, attempts to remove the smear layer completely via acid-etching and rinsing. The second approach, the self-etch technique, aims at incorporating the smear layer as a bonding substrate.

**Total-etch technique**

Most self-priming, single-bottle adhesives available to-date attempt to bond to dentine that is etched with inorganic or organic acids. Following rinsing of the conditioners, retention is accomplished by means of resin-infiltration into the exposed, demineralised collagen matrix to form a hybrid layer of resin-impregnated dentine. Systems containing hydrophilic primer resins solvated in acetone or ethanol were found to produce higher bond strength when acid-conditioned dentine was left visibly moist prior to bonding. Pioneered by Kanca, this technique is often referred to as ‘wet bonding’. The benefit of wet bonding stems from the ability of water to keep the interfibrillar channels within the collagen network from collapsing during resin-infiltration. These channels, which are about 20 nm wide when fully extended, must be maintained open to facilitate optimal diffusion of resin monomers into the demineralised intertubular dentine.

**Self-etch technique**

Recent developments in dentine bonding have reintroduced the concept of utilising the smear layer as a bonding substrate, but with improved formulations that could etch through the smear layer and beyond, into the underlying dentine matrix. Failure to etch beyond the smear layer, exemplified by some of the early adhesives that were applied directly to the smear layer, resulted in weak bonds due to the complete absence of a hybrid layer in underlying intertubular dentine. Contempor-ary self-etch adhesives have been developed by replacing the separate acid-conditioning step with increased concentrations of acidic resin monomers. Two-step self-etching primers combine etching and priming into a single step. The primed surfaces are subsequently covered with a more hydrophobic adhesive layer that is light-cured. In the presence of water as an ionising medium, these adhesives that etch through smear layers and bond to the underlying intact dentine. The recent introduction of single-step (all-in-one) self-etch adhesives represents a further reduction in bonding steps that eliminates some of the technique sensitivity and practitioner variability that are associated with the use of total-etch adhesives.

When applied to sound dentine, the milder self-etch adhesives produce a hybridised complex (Fig. 11) that consists of a surface zone of hybridised smear layer and a thin, subsurface hybrid layer in the underlying intertubular dentine. Despite the presence of a hybrid layer that was generally below 2 μm thick, high initial bond strengths have
been reported for sound dentine. The more aggressive self-etch adhesives completely dissolve smear plugs and demineralise dentine to the extent that is comparable with phosphoric acid-etching. There has been some concern that mild self-etch adhesives may not be able to penetrate through thick smear layers, such as those produced clinically by rough diamond burs. The use of adjunctive phosphoric acid pre-conditioning has been suggested as a means to improve bonding of self-etching primers to sound dentine with thick smear layers.

Problems in bonding to sclerotic dentine

Irrespective of the use of a total-etch or a self-etch technique, bonding to pathologically altered substrates such as sclerotic dentine from noncarious cervical lesions generally led to compromised bonding. Reduced bonding efficacy was attributed to a combination of factors that include the obliteration of dentinal tubules with sclerotic casts, the presence of an acid-resistant hypermineralised layer, and the presence of bacteria on the lesion surface. The presence of the hypermineralised layer, bacteria and the tubular mineral casts in sclerotic dentin are analogous to the presence of the smear layer and smear plugs in sound dentine, being potential diffusion barriers for primer and resin-infiltration. The concern that a self-etching primer may not etch through the superficial layers on sound dentine may likewise be applicable to sclerotic dentine. Acid pre-conditioning prior to the application of a self-etching primer may thus be a viable technique when bonding to sclerotic dentine. It has been shown that the hybrid layer morphology after self-etching or wet bonding in uninstrumented sclerotic dentine is substantially different from those observed in sound, abraded dentine.

Obstacles in bonding to acid-etched, sound dentine

Adhesive strategies that rely mostly on micromechanical retention are hampered by obstacles that jeopardise effective infiltration of resin into dental tissues. In abraded sound dentine, the smear layer is effectively removed in adhesive systems that utilise a separate acid-conditioning and rinsing step (Fig. 12). In order to achieve optimal resin-infiltration, acid-etched demineralised dentine must be suspended in water to prevent collapsing of the interfibrillar spaces. This is effectively accomplished using the wet bonding technique. Interfacial strength is dependent upon the ability of resins to engage the leading edge of demineralised intertubular dentine. The collagen matrix may thus be viewed as a diffusion barrier or obstacle for resin-infiltration in acid-etched dentine.

Figure 11 The etching effect of a self-etching primer (Clearfil SE Bond, Kuraray) through a thick smear layer produced in sound dentine. The substructure of the smear layer consisted of loose, globular subunits (arrow) and channels that were filled with water prior to resin (R) infiltration (asterisk). The hybridised complex consisted of a hybridised smear layer (Hs) and a thin hybrid layer (Ha) in intact dentine (D).

Figure 12 Application of Clearfil Liner Bond 2V (Kuraray) to sound dentine (from an artificial wedge-shaped lesion) that was etched with 40% phosphoric acid for 15 s. The smear layer was completely removed. The hybrid layer (H) was about 5 μm thick. Arrow: base of hybrid layer; A: filled adhesive containing nanofillers; D: sound dentine (from Kwong et al., 2000, with permission).
Obstacles in bonding to acid-etched sclerotic dentine

Unlike sound dentine, application of the same adhesive strategy to sclerotic dentine results in substantial variation in both hybrid layer and resin tag morphology. Potential obstacles of resin-infiltration into uninstrumented natural lesions include the hypermineralised surface layer, an additional partially mineralised surface bacterial layer and intratubular mineral casts that are comparatively more acid-resistant.23,25,27 As these inclusions vary considerably along the occlusal, gingival, and the deepest part of a wedge-shaped lesion, variation in the ultrastructure of the resin-sclerotic dentine interfaces are possible in these different regions. While it is reasonable to assume that the extent of tubular occlusion (Fig. 13A) would vary according to the severity of dentine sclerosis,103 both the superficial bacterial and hypermineralised layers are found to vary from site to site, being thicker along the deepest part of the wedge-shaped lesions.42 The surface hypermineralised layer was usually thinner in gingival and occlusal surfaces than in the apical or deepest part of the wedge-shaped defects, and was often partially or completely dissolved when phosphoric acid is applied to sclerotic dentine (Fig. 13A). As a result, the thickness of the hybrid layers in the gingival and occlusal sites were similar to those seen in acid-etched sound dentin and remained fairly consistent at about 5 μm. Bacteria, if present, tended to be tenaciously attached to the dentine surfaces and in the dentinal tubules, and were retained even after rinsing (Fig. 13B).

Thicker diffusion barriers were found within the deepest part of wedge-shaped lesions that hampered the penetration of acids through the underlying intact sclerotic dentine. As a result, alterations in hybrid layer morphology and thickness were seen in these regions. Considering that the mean thickness of the hybrid layer was about 5 μm in sound dentine as well as the gingival/occlusal aspects of natural sclerotic lesions, hybrid layer morphology in the deepest part of the wedge-shaped lesions may be described as ‘erratic’ in appearance.

One example is shown in Fig. 14A. The surface hypermineralised layer from the deepest part of a wedge-shaped lesion was about 3 μm thick and was trapped within the resin-sclerotic dentine interface. The phosphoric acid apparently etched through the hypermineralised layer and created a hybrid layer of about 2 μm thick within the underlying intact sclerotic dentine. Rhombohedral whitlockite crystallites from the sclerotic casts may also be identified within dentinal tubules in the underlying sclerotic dentine.

Fig. 14B is a stained, demineralised TEM micrograph taken from the deepest part of another acid-etched, wedge-shaped natural sclerotic lesion. The surface obstacle layers were either absent or completely dissolved. Within the 50 μm wide region of the micrograph, the thickness of the hybrid layer...
in sclerotic dentine changed abruptly from 2 to 5 μm. This aspect of uneven etching may also be seen in areas in which the acid etches laterally within the subsurface sclerotic dentine, producing lateral hybrid layer extensions that are separated from areas above that are not infiltrated with resin. This feature is distinct from incompletely resin-infiltrated hybrid layers that are observed in air-dried, acid-etched sound dentine.

Unlike sound dentine in which the morphology of the hybrid layer remains fairly consistent as long as a wet bonding technique is used, extensive variations are found in different specimens or locations within cervical sclerotic lesions. Fig. 15A and B are

Figure 14  (A) Undemineralised section taken from the deepest part of a wedge-shaped natural lesion that was etched with 40% phosphoric acid and bonded using Clearfil Liner Bond 2V. Hh: hybridised hypermineralised layer that contained bacteria remnants (pointer). Hd: hybridised sclerotic dentine that was about 2 μm thick. A: adhesive containing nanofillers; SD: sclerotic dentine. (B) Demineralised TEM micrograph of an ‘erratic’ hybrid layer from the apex of a wedge-shaped lesion that was etched with 40% phosphoric acid and bonded using Clearfil Liner Bond 2V. The thickness of the hybrid layer varied from 2 μm (pointers) to 5 μm (Hd). A lateral extension of the hybrid layer could be seen below an area that was not infiltrated with adhesive resin (asterisk). A: adhesive; SD: sclerotic dentine.

These ‘eccentric’ hybrid layers in abnormal dentin substrates have never been observed in bonded sound dentine.

Figure 15  (A) Undemineralised TEM micrograph from the deepest part of an acid-etched, natural lesion. The hybrid layer (Hd) in intact sclerotic dentine (SD) was 5 μm thick. This was reduced to 2 μm thick beneath the hypermineralised layer (HM). The separation of the hypermineralised layer (arrow) was an artefact produced during specimen processing. A: filled adhesive. (B) Demineralised TEM micrograph of an ‘erratic’ hybrid layer from the apex of a wedge-shaped lesion that was etched with 40% phosphoric acid and bonded using Clearfil Liner Bond 2V. The thickness of the hybrid layer varied from being absent (arrow) when a hypermineralised layer (HM) was present, to 5 μm (Hd) where the latter was thin and was eroded by bacteria (B). A: adhesive; SD: sclerotic dentine.
demineralised, stained TEM micrographs taken from the deepest part of the same natural lesion. Whereas a thin hypermineralised layer and the presence of bacteria did not prevent the penetration of acid or resin into the underlying sclerotic dentine, the thickness of the hybrid layer was greatly reduced in the presence of a thick hypermineralised layer (Fig. 15A). In some areas, the hybrid layer in sclerotic dentine is reduced to the extent that it is almost nonexistent (Fig. 15B). These thick hypermineralised layers serve as obstacles to diffusion and prevent the penetration of even phosphoric acid. Reduced hybrid layer thickness may have no correlation with reduced regional microtensile bond strength in natural sclerotic dentine. However, the presence of thin hybrid layers should clearly be differentiated from the total absence of hybrid layer formation in the bonding substrate. Under such circumstances, the adhesive will be bonding directly to the diffusion barriers that impede acid-etching. The resultant bond strength will depend on the strength of the attachment of such obstacles to the underlying sclerotic dentine. It is remarkable that these morphological fluctuations are continuous and vary within a very small region of a lesion that is covered by these micrographs. Such extreme variations in hybrid layer morphology are probably responsible for the large standard deviations in microtensile bond strength measurements of bonding to sclerotic dentine. There was concern, in normal dentine, that thick and rough smear layers may interfere with diffusion of self-etching primers into the underlying intact dentine. This may be due to the physical presence of thick smear layers as a diffusion barrier, or their ability to buffer the acidic monomers, making the pH too high to demineralise the underlying intertubular dentine.

Obstacles to bonding in sound dentin treated with a self-etching primer alone

The use of two-step and single-step self-etch adhesives represents an alternative means to acquire micromechanical retention in dentine. They are attractive in that they may be used on dry dentine and, after mixing, require only one primer application, which is subsequently air-dried rather than rinsed. The latest single-step self-etch adhesives further incorporates all the resin monomers, photoinitiator and tertiary amine accelerator into a single bottle (iBond, Heraeus Kulzer, Hanau, Germany) and eliminates an additional mixing step. Despite the physical appearance of thin hybrid layers and short, hybridised smear plugs in a two-step self-etch adhesive (Fig. 16), high initial bond strength has been reported. This suggests that there is no correlation between hybrid layer thickness and bond strength as along as a uniform demineralisation front is created in sound intertubular dentine. There is concern, in normal dentine, that thick and rough smear layers may interfere with diffusion of self-etching primers into the underlying intact dentine. This may be due to the physical presence of thick smear layers as a diffusion barrier, or their ability to buffer the acidic monomers, making the pH too high to demineralise the underlying intertubular dentine. Recent studies showed that mildly aggressive self-etching adhesives penetrated through smear layers up to 3-4 μm thick and still retained sufficient acidity to demineralise the underlying intertubular dentine to a depth of 0.4-0.5 μm. This suggests that either the buffering capacity of the smear layer is weak, or the smear layer does not impose much of a physical barrier to the primer compared with the underlying mineralised dentine matrix. The looseness of the surface portion of the smear layer and/or the presence of diffusion channels between its constituents may facilitate diffusion of the self-etching primer through the smear layer. Disaggregation of the smear layer into globular subunits further provides microchannels for diffusion of the self-etching adhesives. These microchannels, in theory, should be more permeable to resin monomers than the interfibrillar spaces (ca. 20 nm) of demineralised intertubular dentine.
Obstacles in bonding to self-etching primer treated sclerotic dentine

The smear layers in sound, cut dentine do not impose much of a restriction to the bonding of contemporary self-etch adhesives because of their loose organisation. Unless they are instrumented, shiny sclerotic cervical lesions are free of smear layers. In lieu of smear layers, other diffusion barriers in shiny cervical sclerotic lesions include the much denser surface hypermineralised layer, as well as the more loosely arranged, partially mineralised bacterial clusters. Some of the hypermineralised layers in sclerotic dentine are so thick that they restrict the penetration of strong inorganic acids such as phosphoric acid that usually etches 5 μm or beyond into sound dentine. Being less acidic in nature, a mild self-etching primer such as Clearfil Liner Bond 2V (Kuraray) etches only 0.5 μm into sound dentine that are covered with smear layers. As sclerotic dentine is highly variable in its ultrastructure, different morphologic expressions of the resin–dentine interfaces can be anticipated when a mild self-etching primer is applied to this abnormal bonding substrate. This may be classified into three categories, depending on the in terms of its thickness and continuity of the surface hypermineralised layer at different locations of the wedge-shaped lesion.

Sclerotic dentine with a thin hypermineralised layer (<0.5 μm thick)

Thin hypermineralised layers are usually located along the gingival and occlusal aspects of wedge-shaped lesions. Fig. 17A represents a straightforward situation in which a very thin hypermineralised layer is present, without bacteria, along the occlusal aspect of a natural wedge-shaped lesion. This layer may be recognised by the characteristic arrangement of the partially dissolved crystallite remnants. The difference in hybrid layer morphology that results from uneven etching is readily apparent. On the left side, the effect of the self-etching primer is restricted to the surface layer alone, producing a 0.1 μm thick, hybridised hypermineralised layer. On the right, the primer etched beyond the surface layer to form an additional layer of hybridised dentine that was about 0.5 μm thick.

A similar uneven etching effect may be seen in thin hypermineralised layers that contain additional surface bacterial attachments (Fig. 17B). This complicates etching by the fact that the self-etching primer must first infiltrate through the matrix between the bacteria, and then through the hypermineralised layer in order to demineralise the underlying intact sclerotic dentine. Unlike the hypermineralised layer that consists of densely arranged crystallites, the intermicrobial matrix is more easily penetrable by the self-etching primer. The hybridised complex, thus consisted of three components: the hybridised intermicrobial matrix, the hybridised hypermineralised layer and the layer

Figure 17 (A) Demineralised TEM micrograph of the gingival aspect of a wedge-shaped lesion that was treated with Clearfil Liner Bond 2V (A). Uneven etching was evident. On the left, only a hybridised hypermineralised layer (Hh) was produced, within which were plate-like crystallite remnants (pointers). On the right, a 500 nm thick layer of hybridised sclerotic dentine (Hd) was formed beneath. SD: sclerotic dentine. (B) The bonded interface from the occlusal aspect of a sclerotic lesion that contained a thin hypermineralised layer but heavy surface bacterial deposits. The hybridised complex consisted of: (1) a hybridised intermicrobial matrix (Him); (2) a hybridised hypermineralised layer; and (3) a hybridised layer of intact sclerotic dentine. (Hd). The latter was absent on the right side of the micrograph (arrows). SD: sclerotic dentine.
of hybridised dentine. In this micrograph, the layer of hybridised dentine was about 0.5 - 0.8 μm on the left, but was completely absent on the right. This illustrates the kind of biological variation that may be expected along the entire lesion surface. The features are not the rare, one of a kind phenomenon that is observed only in one single lesion. Fig. 18A and B are high magnifications of similar features observed in another specimen. There were taken from the same 1 mm x 1 mm section of the gingival aspect of a bonded sclerotic lesion.

Sclerotic dentine with a thick, continuous hypermineralised layer (>0.5 μm)

Thicker hypermineralised layers are found along the occlusal aspects and sometimes within the deepest part of wedge-shaped, sclerotic lesions. As the etching effect of a self-etching primer is limited, it cannot etch beyond the hypermineralised layer into the underlying sclerotic dentine (Fig. 19A). At a higher magnification, the crystallites within the bonded hypermineralised layer are shorter and more sparsely arranged, when compared with the underlying unaffected hypermineralised layer (Fig. 19B). This partially demineralised zone was about 0.5 μm in depth. Porosities created for resin infiltration within this zone are reminiscent of acid-etched, aprismatic enamel. It has been shown that bonding to the surface hypermineralised layer alone resulted in relatively high bond strength. The ultimate strength of the entire bonded assembly, however, depends on the strength of the attachment of the hypermineralised layer to the underlying sclerotic dentine.

Partial demineralisation of the surface hypermineralised layer also occurs in the presence of bacteria inclusions. In Fig. 19C, an unstained undemineralised TEM micrograph, silhouettes of the unstained bacteria could be seen above the partially demineralised, hypermineralised layer. The depth of demineralisation within this layer is comparable to the action of this self-etching primer on sound dentine, and was about 0.5 μm thick. It is reasonable to assume that a layer of hybridised dentine cannot be formed in the underlying dentine when the surface hypermineralised layer is thicker than 0.5 μm. It appears that the self-etching primer can easily diffuse through the intermicrobial matrix. When such a complex is subjected to tensile stress, it remains to be seen whether bond failure would occur between the resin infiltrated bacteria, the partially infiltrated hypermineralised layer, or between the base of the hypermineralised layer and the underlying dentine.

Sclerotic dentine with a thick, discontinuous hypermineralised layer (>0.5 μm)

These thick hypermineralised layers are found along the apex or deepest part of wedge-shaped lesions. As previously described, they consist of thin, discontinuous hypermineralised layers that are dispersed among several different colonies of bacteria. These hypermineralised layers are probably interconnected to form a three-dimensional structure (Fig. 20A). It is highly unlikely that a self-etching primer can etch through a discontinuous hypermineralised layer that is 10 - 15 μm thick. At a higher magnification, Fig. 20B shows the demineralisation
front represented by the scalloped electron-dense edge of the partially demineralised intermicrobial matrix. It is anticipated that bond failure occurs within the porous regions of this thick layer that is occupied by bacteria but not infiltrated with adhesive resin.

Summary of obstacles in bonding to sound vs. sclerotic dentine

Potential deterrents to resin-infiltration following total-etching or self-etching in sound and sclerotic dentine are summarised schematically in Fig. 21.27. The left side of the figure illustrates the response to bonding with a self-etching primer adhesive system alone, while the right side illustrates the response to pre-etching sclerotic dentine with 40% phosphoric acid prior to bonding with the same self-etching primer (Clearfil Liner Bond 2V). The thickness of the hybrid layer is fairly consistent both for self-etch and wet-bonded, acid-etched sound dentine, but is much thicker in the latter group. Conversely, application of the same adhesive strategy to sclerotic dentine results in substantial variation in the hybrid layer morphology in both treatment techniques. Absence of a hybrid layer in some parts of a lesion suggests that both treatment protocols are ineffective in completely overcoming the diffusion barriers in sclerotic dentine. This situation is comparable to the early generation dentine adhesives that were directly applied to the smear layers in sound dentine. Similar to the junction between the partially infiltrated smear layer and the underlying intact sound dentine, areas devoid of hybrid layer formation are potential weak links that may be responsible for the lower bond strengths observed when boning to sclerotic dentine.

Although reduction in hybrid layer thickness may not affect micromechanical retention, sporadic absence of the hybrid layer and resin tags indicate that both treatment techniques are

Figure 19  (A) Undemineralised TEM micrograph taken from the gingival aspect of a Clearfil Liner 2-treated sclerotic lesion. There was only partial demineralisation (pointer) of the hyperminerlised layer (HM), with the latter still attached to the underlying sclerotic dentine (SD). Dentinal tubules were heavily obliterated with sclerotic casts (arrow). A: filled adhesive. (B) A higher magnification of Fig. 19A, showing the crystallites (pointer) within the partially demineralised zone of the surface hypermineralised layer (HM). The ample porosities thus created within this layer for micromechanical retention of the adhesive is comparable to that of acid-etched aprismatic enamel. SD: sclerotic dentine. C. Undemineralised TEM micrograph taken from the gingival aspect of a Clearfil Liner 2V-treated sclerotic lesion. The self-etching primer diffused through the unstained bacteria layer (B) and partially demineralised (pointer) and infiltrated the superficial portion of the hypermineralised layer (HM). The basal portion of the hypermineralised layer appeared to be firmly integrated with the underlying sclerotic dentine (SD). A: filled adhesive.
inadequate in overcoming diffusion barriers in sclerotic dentine. Physical removal of the superficial obstacle layers with a bur may improve intertubular retention. In highly sclerotic lesions however, this may be offset by moving the bonding interface pulpward into an area where bonding requires increasing contribution from intratubular resin infiltration. Moreover, the formation of a smear layer that consists of acid-resistant hypermineralised dentine chips and whitlockite crystals derived from the sclerotic casts also creates additional diffusion barriers for both total-etch and self-etch adhesives.

Sclerotic dentine located at the apex of wedge-shaped natural lesions is derived from deep dentine. Consequently, resin tag formation should play an important role in achieving strong immediate bond strength in the sclerotic cervical lesion. However, resin tag formation is sporadic regardless of the conditioning methods. Absence of intratubular infiltration may even be observed in some of the occlusal parts of natural lesions that were etched with phosphoric acid, where the thickness of intertubular infiltration is comparable to that of the sound dentine.
present in sound dentine. Similar to the results of Ferrari et al.\textsuperscript{110} and Prati et al.,\textsuperscript{41} lateral branches of resin tags were rarely observed when bonded sclerotic dentine were examined by TEM.\textsuperscript{42,57} This is likely to be caused by the acid-resistant nature of the mineral-dense sclerotic casts that occlude the dentinal tubules. It has been suggested that 20% of the strength of an interfacial bond was contributed by resin-infiltration derived from resin tag formation and another 20% from hybridisation of the intertubular dentine.\textsuperscript{111,112} Regional tensile bond strength from cervical sclerotic root dentine was found to be 20–45% lower than those obtained from artificial lesions prepared in sound root dentine.\textsuperscript{40,42} This reduction may be due to the absence of resin tags and incomplete hybridisation in sclerotic dentine.

Regional microtensile bond strength evaluation

Microtensile bond strength measurements comparing resin bonds to the occlusal, gingival and the apex or deepest part of natural lesions and artificially wedge-shaped defects created in sound cervical dentine were reported by Kwong et al.\textsuperscript{42} using the microtensile bond test.\textsuperscript{113} Lesions were restored with Protect Liner (Kuraray) and Clearfil AP-X resin composite (Kuraray) following treatment with Clearfil Liner Bond 2V, with or without phosphoric acid pre-conditioning of the lesions. Using the nontrimming technique developed by Shono et al.\textsuperscript{114} beams with a mean area of $0.46 \pm 0.03 \text{mm}^2$ were prepared and stressed to failure. The use of a nontrimming technique (Fig. 22) facilitated preparation of a series of slabs, thus allowing more than one beam to be harvested from each lesion.\textsuperscript{113}

The mean tensile bond strengths of bonds produced by the self-etching primer alone to natural lesions (48.7 MPa) were 26% lower (Fig. 23) than those from artificial lesions (65.8 MPa) when all of the bonds were pooled, and the result was statistically significant ($p < 0.001$). Similarly pooled data on bonds made using the self-etching primer with adjunctive phosphoric acid pre-conditioning to natural lesions (53.1 MPa) were 24% lower ($p < 0.005$) than those produced from artificial lesions (69.8 MPa). Pooled data, however, showed no significant difference among the bonds made by self-etching or total-etching to either sound dentine ($p = 0.415$) or sclerotic dentine ($p = 0.314$). Of the three factors (substrate, conditioning method and location) tested, only the difference in the type of substrate (i.e. sound dentine vs. sclerotic dentine) was found to have a significant influence on bond strength ($p < 0.05$). Multiple comparison tests showed that there was no difference in self-etching or total-etching sclerotic dentine except for the gingival aspect of the lesions, in which higher bond strengths were obtained for total-etching (Fig. 23).

These results are comparable to those of Yoshiyama et al.\textsuperscript{40} in that lower bond strengths were found in natural sclerotic lesions, and to the work of Phrukkanon et al.\textsuperscript{115} showing that bonding of a self-etching primer to sound dentine is independent of the tubular orientation. The orientation of dentinal tubules in the occlusal or upper wall of wedge-shaped lesions is approximately parallel to the surface, while their orientation in the gingival wall is perpendicular to the prepared surface.\textsuperscript{116,117} Many believed that resin tag formation would be more prominent in surfaces where
the tubules are oriented perpendicular to the surface rather than parallel. However, measurement of microtensile bond strengths of self-etching primers and total-etch adhesives to these walls in wedge-shaped cavities prepared in normal dentine revealed significantly higher bond strengths to dentine in which the bonded surfaces were oriented parallel to the tubules (i.e. occlusal walls).\textsuperscript{116,117} Double-etching of dentin by phosphoric acid followed by a self-etching primer adhesive has been shown to increase bond strengths to enamel but to lower bond strengths in dentine,\textsuperscript{118} that may be caused by incomplete infiltration of the adhesive into the phosphoric acid-etched dentine.\textsuperscript{119} Using Clearfil Liner Bond 2, Ogata et al.\textsuperscript{120} found that multiple applications of the primer to wedge-shaped lesions increased bond strength due to the weak acidity of the primer. Although this has not been tested in sclerotic dentine, the same result would be expected. That is, multiple applications of weakly acidic agents using constant agitation, should improve bonding.

TEM examination of the failed bonds exhibited by bonds created in sclerotic dentine revealed a wide variation in the mode of failure that included all of the different structural components that are present within the resin-sclerotic dentine interfaces.\textsuperscript{42} The complexity of failure modes indicates that reduced bond strength in sclerotic dentine is not related to any single factor. Similar to other biological variations, it is possible that each factor contributes to a variable degree in different lesions. The summation of all these factors, however, leads to an overall reduction in bond strength.

The presence of a partially mineralised bacterial zone in sclerotic lesions is analogous to the presence of a smear layer on sound, abraded dentine. This zone is porous, allowing easy penetration of acids and primers to form a zone of hybridised intermicrobial matrix. The presence of a hybridised intermicrobial matrix may not affect bonding, at least in the short term, providing that the self-etching primer can effectively etch through this layer into the underlying bonding substrate. This is analogous to the hybridised smear layers in sound dentine. It remains to be seen whether the eventual degradation of the bacteria in this layer would lead to decrease in bond strength with time. The large standard deviation in bond strength results in natural sclerotic lesions may simply reflect the large biological variation in the thickness of such a layer.

The presence of a hybridised hypermineralised layer together with an underlying zone of hybridised dentine does not necessarily result in low bond strength. This is comparable to the infiltration of a self-etching primer through smear layer-covered sound dentine. Provided that the acids can penetrate the overlying diffusion barriers to engage the underlying substrate with even a very thin hybrid layer, strong initial bonds may still be achieved. However, erratic bonds may be expected when the hypermineralised layer in sclerotic dentin is too thick for acids to etch through. Resin attachment to the partially demineralised surface of this layer is still strong and may be comparable with bonding to unground, aprismatic enamel.\textsuperscript{121} However, since a layer of hybridised dentine is not produced, the strength of the bond will be highly dependent upon the strength of the hypermineralised layer to the intact sclerotic dentine.

The fact that higher bond strength was observed along the gingival site of total-etched natural lesions (Fig. 23) merits further discussion. This suggests that the inability of the adhesive to form resin tags in tubule lumina that are blocked by mineral deposits is an important parameter that leads to the reduction in bond strength. If the above hypothesis is correct, then grinding of the surface hypemineralised layer of these cervical wedge-shaped defects prior to bonding\textsuperscript{122} should not result in an increase in bond strength, since the underlying sclerotic dentine still contains dentinal tubules that are blocked by whitlockite crystallites. Application
of stronger phosphoric acid to these defects could have resulted in partial dissolution of the sclerotic casts and/or complete removal of the surrounding peritubular dentine, allowing resin infiltration into the dentinal tubules. This may result in higher bond strength along the gingival site of phosphoric acid-etched, sclerotic dentine.

Restoring the class V sclerotic lesion

Maintaining the marginal integrity and retention of Class V resin composite restorations without the use of additional retention has always been a challenge for clinicians. One major factor, already analysed is the difficulty in bonding to sclerotic dentine. Removing the hypermineralised surface layers by grinding or by using stronger acids (Fig. 23) are possible strategies to improve micromechanical retention in sclerotic dentine. While it is possible to produce hybrid layers in sclerotic dentine with thin diffusion barriers, these hybrid layers become erratic or even nonexistent in the presence of thick barriers. As clinicians have no way of discerning these differences at a clinical level, removal of the surface layer of sclerotic dentin prior to bonding should be adopted.42,122 Although such a recommendation may not result in an increase in bond strength to sclerotic dentin, it does remove one potential source of inconsistency that leads to bond failure. Based on the results of a two-year clinical trial, it has been suggested that micromechanical retention by acid etching of the enamel margin is still indispensable for the clinical success of cervical Class V composite restorations.123 Such a concept, however, was recently challenged by two Japanese studies, in which the authors maintained that sclerotic dentine, being a part of the body’s natural defence mechanism, should be preserved as much as possible and that acid-etching should be avoided to promote the marginal integrity of resin composites that are bonded to these lesions.45,124

While one may remove bacteria overgrowths from the surface hypermineralised layer, it is not possible to remove bacteria entirely from dentinal tubules. This is analogous to the application of fissure sealants to stained enamel fissures,125,126 or the bonding of resins to the inner layer of carious dentine.127,128 The use of bactericidal solutions (i.e. chlorhexidine) or adhesive resins with antibacterial activity129,130 would be helpful. However, the longevity of bonds that contain dead, degradable bacteria should be further investigated. This is particularly applicable to adhesives that absorb water. Recent studies showed that both hydrophilic resins131,132 and collagen fibrils within the hybrid layer133–136 degrade upon long term water storage.137

Conclusions

The structural complexity of noncarious sclerotic cervical dentine is remarkable. The common presence of adherent bacteria on such surfaces and their incorporation into the bonded restorations is disconcerting. This raises issues such as whether these bacteria are dormant, and whether their confinement by adhesives will create any long term liability. These questions have also recently been raised in reports that bacteria are present in resin-bonded caries-affected dentine.127,128 The presence of bacteria on these surfaces justifies the use of 2% chlorhexidine disinfectant treatment or the use of antibacterial adhesives to disinfect the substrate prior to bonding.

Microtensile bond strengths of self-etching primers to sclerotic dentine were comparable with those made to phosphoric acid-etched sclerotic dentine, although they were lower than those attained with sound dentine. These bond strengths are probably high enough to retain class V restorations even under heavy loads, if is bonding is performed with etching of the enamel to create additional micromechanical retention. Although there are clinical studies that showed encouraging results with the use of dentine adhesives on noncarious cervical lesions,138–140 the failure rates of some specific adhesives have been reported to be high in other studies. For example, the retention rate for One-Step, a total-etch single-bottle adhesive, in noncarious cervical lesions involving sclerotic dentine was only half of that of nonsclerotic dentine.141 The retention rate of the same adhesive in noncarious cervical lesions was reduced from the original 100% at 6 months to 75% after three years.142 There are also other clinical studies that reported more favourable retention rates when glass-ionomer based restorative materials were compared with dentine adhesives/resin composites in restoring these lesions.143–145 Unfortunately, there are no TEM studies that examine the bonding of glass-ionomer cements and resin-modified glass-ionomer cements to sclerotic dentine. Admittedly, TEM work on these types of restorative materials that are susceptible to dehydration is difficult to perform. However, this should be done to
complete our understanding of this alternative type of chemical/micromechanical interaction with sclerotic dentine.

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