Management of Deep Dentin Carious Lesions: A Contemporary Approach for Primary and Young Permanent Teeth

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Contents

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10.1   Introduction

Caries prevalence is still high worldwide, with the disease burden affecting all age groups [1]. When biofilm control and preventive measures fail, a carious lesion is expected to develop. Following its natural developmental history, the lesion will advance through the enamel and reach the dentin, leading to pulp infection and necrosis.

If appropriate control measures are implemented, such as biofilm control, adoption of a healthy diet, and rational use of fluoride, the lesion can be controlled, and remineralization of the dental tissue may occur. As discussed in Chap. 6, this is true for non-cavitated carious lesions and even for cavitated ones, as long as the patient is able to clean the lesion properly on a regular basis. However, in the presence of a cavity with no possibility of biofilm control inside it, the lesion is likely to progress, and the appropriate treatment is the placement of a restoration. Early management of deep carious lesions can arrest demineralization and reduce the need for pulp therapies. This chapter will guide the reader in the management of deep dentin carious lesions in the aim of improving patients' health.

10.2   Dental Caries Lesion Development and Pulp Reactions

Dental caries results from an interaction between the microbial biofilm and the mineralized dental tissues. Imbalance between the physiological processes of demineralization and remineralization with predominance of demineralization events will cause mineral loss from dental tissues with consequent formation of the carious lesion.

As thoroughly described in Chap. 6, the first stage of carious lesion development involves erosion and porosity of the enamel surface. A subsurface lesion is formed just below the enamel, and if no treatment is provided and the disease continues, increased porosity of the outer surface is established, with an increased subsurface demineralization. This can lead to the formation of a cavity, which first reaches the enamel and then the dentin on the coronal part of the tooth (or the cement and the dentin in the tooth root) and, finally, leads to the total destruction of the tooth.

Very early in the carious process, due to the porosity of the enamel, stimuli from the oral cavity pass through this tissue into the dentin. Dentin and pulp can be considered one entity, as odontoblast processes pass through the dentinal tubules. As bacterial acid, plaque metabolic sub-products, and bacteria wall components such as liposaccharides reach the dentin-pulp organ, different reactions can occur [2, 3]. The first alteration is the hypermineralization of the dentin just below the enamel lesion even before the demineralization reaches the dentin. First, a secretion of highly mineralized peritubular dentin is observed, which will reduce the diameter of the tubule. Intratubular deposits of mineral also take place [4]. Enamel demineralization takes place in the affected hypermineralized dentin. The first indications of cellular reactions are noted in active lesions involving about 1/4 of the enamel layer, but without discernible alterations in dentin mineralization [5].

Bacterial products diffuse through the dentinal tubules and may induce inflammatory response from the pulp even before it is exposed. The inflammatory process initiates already in the presence of non-cavitated enamel lesion [6] and intensifies as the demineralization reaches the dentin. During dentin demineralization, a series of products that have been trapped in the dentin during its mineralization are released. The pulp responds to the microbial product invasion, through the permeable tissue, liberating or activating mediators form polymorphonuclear and mononuclear leukocytes. Dentin-pulp complex permeability is likely to be reduced in carious teeth due to tubular sclerosis subjacent to the carious dentin.

Growth factors released during dentin demineralization could be related to tertiary dentin formation, which seems to occur when the demineralization reaches the dentin. Tertiary dentin formation is another form of pulp protection organized by pulp cells in response to caries' advance. The structure of tertiary dentin depends on lesion activity, i.e., on the severity of the irritating stimuli, and can be divided into two types: reactionary dentin, formed by the odontoblasts present in slow progression lesions (in mild irritation), and reparative dentin, formed by the odontoblast-like cells that differentiate from pulp stem/progenitor cells after the death of the odontoblasts (in severe irritation) [7]. The faster the progression of the lesion, the more irregular the structure of the newly formed tertiary dentin is, even with cellular inclusion. At this stage, lesion progression can be controlled, and the inflammation can subside, if the biofilm is regularly removed or the cavity is isolated from the oral environment by a restoration. However, when bacteria reach the tertiary dentin, the number of inflammatory cells is abundant. At this stage, usually severe pulp inflammation occurs, with decreased healing chances. Areas of necrosis in the pulp are not seen until the microorganisms reach the pulp [8]. The management of deep carious lesion is linked to the inflammatory pattern of the pulp.

10.3 Different Stages of Dental Caries Lesions, Dental Tissue Bacterial Invasion, and Lesion Control

For many decades, the decision between a conservative treatment and an invasive treatment for dental caries lesions was related to the presence of microorganisms inside the dental tissue. Control of non-cavitated lesions without the necessity of restorations is well established since the 1980s [9], and it was also supported by the traditional understanding that non-cavitated lesions harbored no bacteria and therefore could be controlled with no tissue removal. On the other hand, once a cavity was formed and microorganism was present inside the dental tissue, this contaminated tissue had to be removed and a restoration be placed to control lesion progression. Notwithstanding, studies showed the presence of microorganisms even in non-cavitated lesions, in both enamel [10] and dentinal tubules [11], which does not prevent lesion arrestment. In addition, despite the great amount of microorganisms invading the dentin tissue once a cavity is formed, Anderson, already in 1938, showed that cavitated lesions of molars can be arrested once the biofilm is removed [12]. Another body of evidence that dentin caries can be controlled with no invasive

therapy is the root lesions. With the increased tooth retention currently observed in the adult and elderly population, a large number of arrested cavitated root lesions have been observed. In addition to this evidence derived from observational data, intervention studies also showed the possibility of arresting root carious lesions by adopting noninvasive therapies [13]. All this evidence shows that (1) the presence of microorganism within the dental tissue does not prevent lesion control and (2) carious lesions can be controlled once the external biofilm is regularly removed, independent of the microorganism invasion of the dental tissues.

Despite this knowledge, the elimination of carious dentin was considered essential to control the carious process for many decades. Traditionally, it was recommended to remove "all the carious tissue" prior to the placement of a restoration, until reaching dentin with clinical characteristics similar to those of healthy tissue in terms of hardness and staining. As early as 1908, when devising a logical sequence of procedures for the cavity preparation, Black [14] suggested that the cavity would be adequately clean and ready to receive the restorative material when it presented sufficient probing resistance to promote the "dentin scream," in addition to color similar to sound dentin.

Later studies from the 1950s and 1960s have already shown that the removal of carious tissue based on hardness criteria does not eliminate microbial contamination and that there is no relationship between tissue hardness and its level of contamination. Macgregor, Marsland, and Batty [15], when evaluating bacterial growth after total removal of softened dentin, showed that 51% of the evaluated teeth presented viable bacterial colonies. Whitehead, Macgregor, and Marsland [16], using the same methodology, demonstrated that the dentin, although hard, presented microbial growth in 75.5% of the primary teeth and 49.5% of the permanent teeth. Similarly, Shovelton [17] observed histologically that 36% of teeth with hard dentin had microbial contamination, while 39% of cases with leathery dentin and 28% of teeth with soft dentin were free of microorganisms. Using a molecular biology technique called in situ *hybridization*, Banerjee et al. [18] quantified the total population of viable bacteria in different dentin layers at different depths (superficial, medium, and advanced – the latter corresponding to the hard dentin usually kept after cavity preparation). Many microorganisms were found at all depths, including the dentin layer considered healthy. This reinforces the absence of association between the clinical aspect and level of contamination.

These results allow us to infer that the maintenance of bacteria under the restoration, which routinely occurs in dental practice, does not cause clinical failure. This knowledge gave rise to conservative treatment strategies for carious lesions of different depths, freeing the dentist from the obligation to remove all the carious dentin as a fundamental premise for treatment success.

Sealing carious lesion forms a physical barrier and cuts off the nutrients from the oral cavity to the cariogenic microorganisms. Studies on sealing carious tissue under sealants or restorations have shown reduction or complete elimination of the population of viable microorganisms [19–26], thus controlling caries progression. A clinical study compared the microbiological infection (in culture) immediately after conventional carious dentin removal and a conservative caries removal with

sealing of carious dentin for six months. Significantly less anaerobic bacteria, aerobic bacteria, and *Streptococcus mutans* growth was observed after sealing carious dentin than after traditional dentin caries removal [27].

Based on the abovementioned studies, it can be concluded that:

- Microorganisms invade the dental tissue already in non-cavitated lesions.
- Microorganisms within the dental tissue (either enamel or dentin) do not preclude lesion arrestment, as long as it is possible to control the external biofilm.
- When it is not possible to control lesion progression through biofilm control, sealing carious lesions or placing a restoration is necessary, depending on lesion depth.
- There is no relationship between dentin hardness and level of bacterial contamination.
- The traditional methods of carious dentin removal usually leave microorganisms within the hard dental tissue left at the bottom of the cavity preparation before restoration.
- It is not necessary to remove all carious dentin before the restoration is placed because, over time, sealing of carious dentin results in lower levels of infection than traditional dentin caries removal.

10.4 Treatment Options for Deep Dentin Carious Lesions

The traditional approach for the management of deep carious lesions was based on complete (or nonselective) caries removal, which results in a higher risk of pulp exposure, as widely demonstrated in the literature [20, 28–30]. Regardless of the dentition, whether primary or permanent, regardless of the study design, randomized trials or not, studies are unanimous in demonstrating that this strategy results in a higher risk of pulp exposure in comparison with less invasive techniques. Indeed, recent consensus reports have stated that complete or nonselective caries removal is no longer recommended; it is considered overtreatment according to current concepts for caries removal [31, 32]. The recent systematic review with meta-analysis by Schwendicke et al. [33] concluded that less failures were observed for conservative techniques based on selective caries removal or no caries removal in comparison with nonselective caries removal, for both primary and permanent dentition.

In an attempt to avoid pulp exposure, different treatment strategies have been proposed over the last decades for the clinical management of deep carious lesions. Several investigations in the last century have already demonstrated that decayed dentin could be retained so that pulpal exposure could be avoided during cavity preparation [34, 35]. The treatment, called indirect pulp capping (IPC), performed mainly on primary teeth, is based on excavating decayed dentin as much as possible without pulp exposure. In this technique, only a thin layer of carious dentin is left over the pulp. Microbiological studies showed that, although the deep layer of residual carious dentin left during IPC is almost always contaminated, this layer was either rendered sterile or the number of microorganisms was greatly reduced after

sealing [36]. More recently, however, the European Organization of Endodontology defined IPC as a therapy that leaves neither soft nor firm carious dentin behind [37].

The most conventional option for the conservative management of deep carious lesions is the stepwise excavation (SW) technique, currently seen as selective caries removal to firm dentin (SCR-FD) in two visits. In this approach, only the necrotic layer of carious dentin is removed from the pulpal/axial cavity walls at the first visit, during the acute phase of caries progression (this stage is currently called selective caries removal to soft dentin [SCR-SD]), followed by cavity lining with calcium hydroxide cement, glass ionomer cement, or hydraulic calcium silicate cement and temporary restoration [37]. In the second visit, SCR-FD is performed, and a restoration of long-lasting material is placed. The aim of the first stage is to change the cariogenic environment, replacing an active carious environment, clinically identified by a soft, discolored, and wet tissue, by an arrested carious environment, characterized by a darker, harder, and drier appearance [3, 37]. From a clinical/practical perspective, the reasoning behind this technique is that after a given time, the dentist will be able to reach firm dentin with no/lower risk of pulp exposure. Studies comparing SW with nonselective caries removal have shown that the two-step procedure significantly reduces the risk of pulp exposure [20, 28–30, 37]. The most robust evidence on this topic was generated by a 5-year randomized clinical trial that showed 60.2% of success for SW compared with 46.3% success for nonselective caries removal to hard dentin ($P = 0.03$) when pulp exposures per se were included as failures. Non-exposed pulp of teeth submitted to conventional dentin caries removal was 50% (95% CI = 1.01 to 2.2, $P = 0.04$) more likely to fail than the SW-treated teeth. In general, SW was superior to nonselective caries removal, with less pulpal exposure, less pain and more teeth with vital pulps in the SW group [38].

The clinical changes that occur in sealed carious dentin after the first session of SW led to questioning the need for reopening the cavity for final excavation and gave rise to a series of studies on this topic. Several studies using different methodologies have assessed the effect of sealing carious dentin, in both shallow and deep lesions. These studies may be categorized according to the outcome under investigation into:

- Clinical evidence: replacement of softened and yellowish tissue by harder and darker dentin after cavity sealing, both in shallow [39–41] and deep [20, 24, 26, 42–45] carious lesions (Fig. 10.1)
- Laboratorial evidence: increased hardness perceived in tactile inspection of sealed carious dentin confirmed by microhardness analyses performed on exfoliated primary teeth [46, 47]
- Biochemical evidence: increased mineral content after sealing carious dentin, with higher levels of calcium [43] and phosphorus [48]
- Radiographic evidence: increased radiopacity in the radiolucent zone beneath the restoration [24, 49] (Fig. 10.2)
- Structural evidence: structural reorganization of sealed carious dentin, with total or partial obliteration of dentinal tubules observed by scanning electron microscopy analyses [43, 50] (Fig. 10.3)
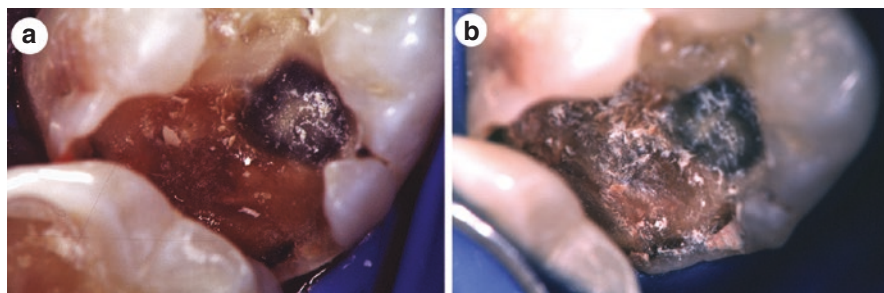
Fig. 10.1 Carious lesion immediately after SCR-SD, with a softened and yellowish tissue (**a**); clinical aspect after 6 months of cavity sealing, with a harder and darker tissue (**b**)
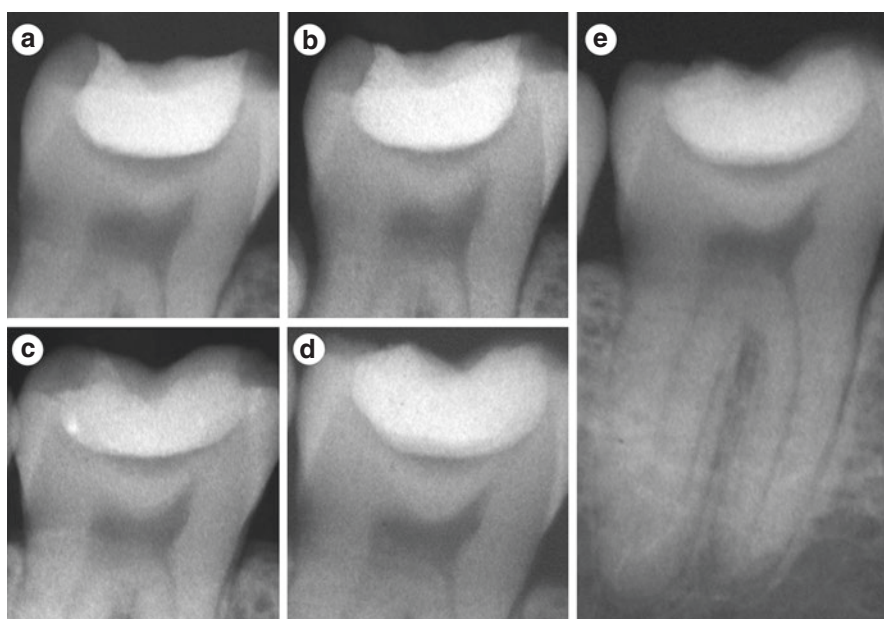
Fig. 10.2 Tooth treated with SCR-SD. Bitewing radiographs taken at baseline (**a**), after 6–7 months (**b**), 3 years (**c**), and 10 years (**d**). The 10-year follow-up periapical radiograph shows a normal periapical area (**e**). Increased radiopacity of the carious dentin left beneath the restoration can be observed [49]
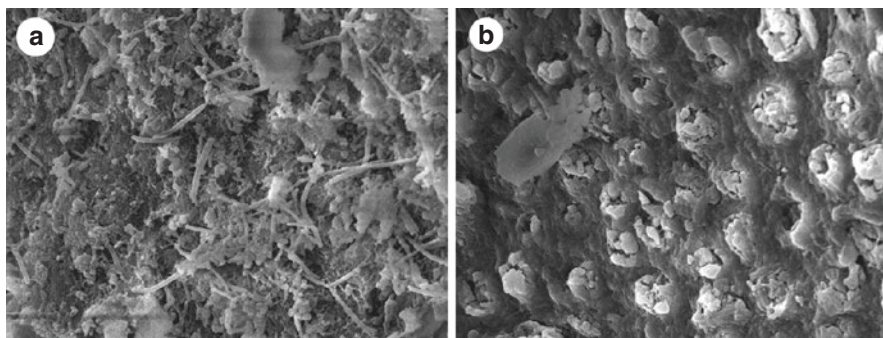
Fig. 10.3 Scanning electron microscopy photomicrographs (×3000) of dentin samples. Disorganized dentin structure with exposure of inter-tubular dentin collagen fiber and bacterial infection after SCR-SD (a); more organized dentin with total or partial obliteration of dentinal tubules and a reduction in bacterial infection after a 3-month sealing period with wax (**b**) [44]

- Microbiological evidence (quantitative assessment): reduction in the number of viable bacteria when the caries lesion was isolated from the external environment, either through the use of sealants or restorations [20–26, 36, 39–44, 50–52], achieving lower levels of infection than traditional dentin caries removal [27]
- Microbiological evidence (qualitative assessment): changes in the composition of the microflora, with less complex and less cariogenic microbiota after sealing [52, 53]

In addition to this body of evidence supporting the SCR-SD as a one-visit technique, some disadvantages of the two-visit procedure (associated with the need for a second visit to complete the treatment) also became evident:

- Additional cost [54–57], time, and discomfort to the patient.
- Possibility of pulp exposure during the final excavation, around 15–17.5% [28–30].
- Risk of patient absenteeism in the second visit, leading to fracture or loss of temporary filling and, consequently, lesion progression [58–60].

Based on all this evidence, it became clear that once carious dentin is isolated from the nutrient supply, deep carious lesions can be managed by SCR-SD. Considering
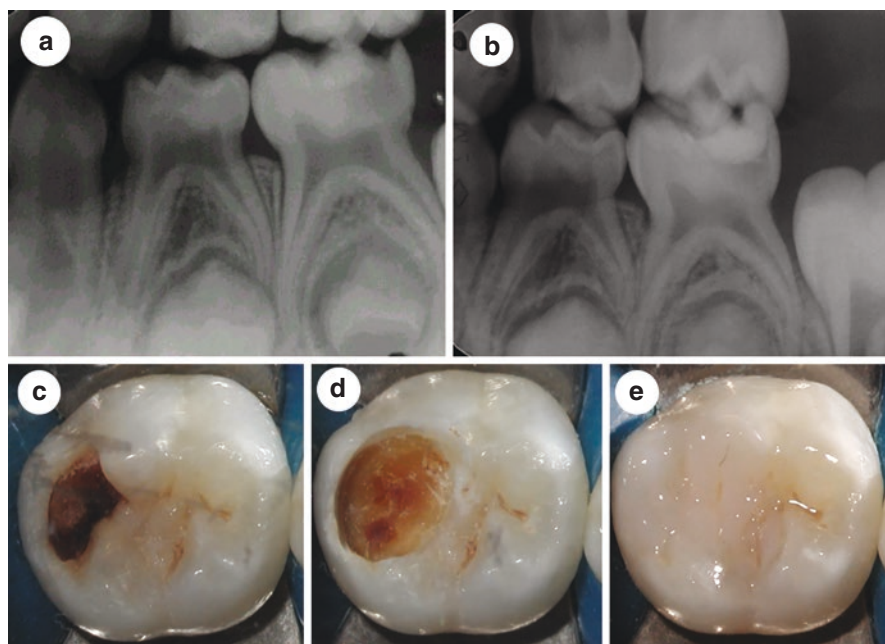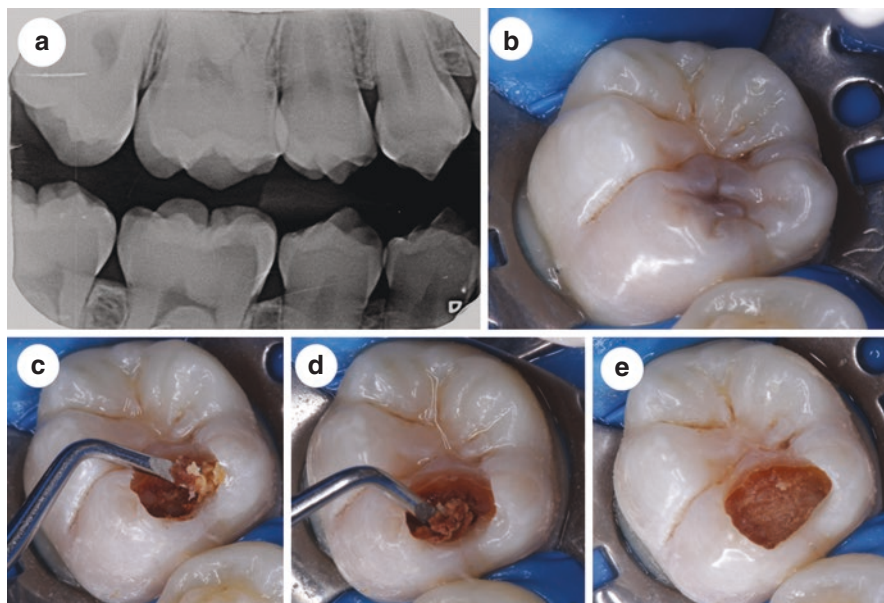
Fig. 10.4 Clinical and radiographic images of SCR-SD in a primary tooth. Periapical radiograph of tooth 75 with radiolucent image extending to the inner half of dentin (**a**). Radiographic aspect after cavity restoration (**b**). Clinically, the lesion presents as a large cavitated lesion (**c**). Clinical aspects of the cavity after nonselective removal of carious tissue from the surrounding walls and SCR-SD at the pulpal wall (**d**). Composite resin restoration (**e**)

that an effective isolation from the nutrient supply is only achieved by adequate marginal sealing of the cavity, it is worth mentioning that the peripheral/lateral walls of the cavity must be excavated to hard dentin. Therefore, SCR-SD should be restricted to the pulpal/axial walls of the cavity (Figs. 10.4 and 10.5).

Before the publication of recent consensus reports [31, 32] this technique was also known as partial, incomplete, minimally invasive, or ultraconservative caries removal. For standardization purposes, the current terminology used in the literature for caries removal techniques was adopted in the present chapter.

Fig. 10.5 Clinical and radiographic images of SCR-SD in a permanent tooth. Bitewing radiograph of tooth 46 with radiolucent image extending to the inner half of the dentin (**a**). Clinically, the lesion appeared as an underlying dentin shadow on the mesial portion of the tooth (**b**). Complete removal of carious tissue from the surrounding walls (**c**) and SCR-SD at the pulp wall using a hand excavator (**d**). Final appearance of the cavity after SCR-SD (**e**). Images are courtesy of MSc Rafael Schultz de Azambuja

10.5   Clinical Evidence for Primary Teeth

Clinical studies assessing the effectiveness of SCR-SD are usually focused on two different outcomes: maintenance of pulp vitality and restoration longevity. Regarding the maintenance of pulp vitality, similar success rates were found for SCR-SD (92%) and nonselective caries removal (96%) [61] after 24 months of monitoring. This study also showed that the operative time was significantly higher for nonselective excavation, which is an important aspect to be considered – mainly during the treatment of children. This study was recently corroborated by a multicenter randomized clinical trial that also compared the SCR-SD technique with nonselective caries removal and found high (≥96.5%) success rates for both strategies, with no difference between them [62]. In a series of studies performed at the Federal University of Rio Grande do Sul, the success rate of SCR-SD in primary molars ranged from 79% to 96% after follow-up periods from 2 to 5 years [63–68].

Another possible concern related to the SCR-SD technique that has motivated some studies in this research field is the risk of reduced restoration longevity due to the maintenance of decayed tissue beneath the restoration. A systematic review with meta-analysis on this topic that included four studies found that SCR-SD increased the risk of experiencing restoration failure [69]. The risk of bias of the studies

included was classified as high, and the quality of evidence was low, which led the authors to conclude that the evidence level was insufficient for definitive conclusions. In fact, taking a closer look at the four studies included in this systematic review, two studies assessing glass ionomer restorations have not found difference between the study groups [70, 71], while another one detected no failure over the study period in any of the comparison groups [72]. Therefore, there is only one study in the literature showing lower success rates for resin restorations after SCR-SD than after nonselective caries removal in deep carious lesions of primary molars [61, 73]. After 24 months of follow-up, the authors found 66% of success rates for restoration in primary molars after SCR-SD and 86% after nonselective caries removal ($p = 0.03$) [61], with these rates decreasing to 57% and 81%, respectively, after 36 months of monitoring ($p = 0.004$) [73].

Another recent randomized clinical trial compared SCR-SD and SW for the management of deep carious lesions in primary molars [57, 74]. Similar success rates were observed after 12 [74] and 24 months [57] regarding the primary outcome of the study [57]. The authors concluded that the significantly higher costs for performing the two-step procedure instead of the one-step treatment may not be justified. It is important to mention that the International Caries Consensus Collaboration organized by the European Organization for Caries Research has not recommended SW for the management of deep carious lesions in primary teeth [31].

10.6   Clinical Evidence for Permanent Teeth

Studies including permanent teeth are less abundant in the literature. The pioneering study coordinated by Maltz et al. followed patients with deep carious lesions in permanent posterior teeth who underwent SCR-SD and resin restoration for 6–7 months [24], 14–18 months [75], 3 years [76], and 10 years [49, 77]. After 5 and 10 years of monitoring, success rates were 82% and 63%, respectively, when all reasons for failure were combined (restoration fractures and pulp necrosis). When the failures caused by fracture were subtracted out, success rates concerning pulp vitality increased to 93% and 80% at 5- and 10-year recalls, respectively. This was the first long-term evidence on SCR-SD in permanent teeth available in the literature; however, it was a single-arm clinical trial, with no comparison group.

Bearing in mind that randomized controlled clinical trials are the study design of choice to assess the effectiveness of a given therapy, this evidence was generated more recently. A multicenter, randomized, controlled clinical trial was conducted to compare the effectiveness of SCR-SD and SW in individuals with deep carious lesions ($\geq 1/2$ dentin thickness) in permanent molars [58, 78]. It is worth highlighting that this study compared a technique that leaves carious tissue (SCR-SD) and a technique that completely removes carious dentin at the second visit (SW). After 5 years of follow-up [59], success rates in terms of maintenance of pulp vitality were 80% for SCR-SD and restoration in a single visit and 56% for SW ($p < 0.05$). This low success rate observed in the SW group was mainly related to incomplete treatments, in which the patients have not attended the second visit to receive the

final restoration. After a given period, the temporary fillings tend to fail, leading to lesion progression and pulp damage. The comparison of SCR-SD and complete cases of SW showed similar success rates (80% vs. 75%, $p > 0.05$). When it comes to restoration longevity, the 5-year survival analysis showed similar success rates for SCR-SD (79%) and the SW procedure (76%) [79]. These findings confirm that maintaining decayed dentin in the pulpal/axial wall of deep cavities by adopting the SCR-SD technique can preserve pulp vitality without damaging restoration survival in permanent teeth during a 5-year period.

Another study comparing SCR-SD and SW for the management of deep carious lesions (>2/3 of dentin thickness) in permanent teeth was conducted in Egypt, but only the 1-year results have been published so far [56]. During this short-term follow-up period, the authors found similar success rates for both techniques (89.4% for SCR-SD and 84.9% for SW). The estimated mean time free of complications was 23 months for SCR-SD and 18 months for the SW procedure, without significant differences between them ($p > 0.05$). This study also assessed the total cost of both strategies and found a significantly higher total cost for SW than for SCR-SD ($p > 0.05$).

Additional evidence on this topic was provided by a retrospective study that evaluated the longevity of adhesive restorations performed in deep carious lesions in young permanent molars after nonselective caries removal or SCR-SD in a university setting [80]. The survival of restorations reached 57.9% up to the 36-month follow-up, with no difference between the two caries removal techniques ($p > 0.05$).

The systematic review with meta-analysis by Schwendicke et al. [33] showed that for deep carious lesions in permanent teeth, the odds of failure were higher for SW than for SCR-SD (OR 2.25, 95% CI 1.33 to 3.82; 3 studies, 371 teeth; moderate-certainty evidence). However, no difference was observed between these two strategies in the network meta-analysis on the relative effects of different interventions for treating deep lesions.

Based on all the literature discussed in this chapter, it seems clear that there is no evidence to support the need for cavity reopening for further excavation and that SCR-SD may be adopted for the treatment of deep carious lesions in permanent teeth as a single-visit approach.

10.7   Pulp Response to SCR-SD

In addition to clinical studies assessing outcomes such as pulp vitality and restoration longevity, histological studies have been performed to investigate pulp and dentin response to nonselective and selective caries removal [8, 81]. The control (sound) teeth of these studies showed (1) no changes in the dentin/predentin/odontoblast complex; (2) dentinal tubules and odontoblasts layers with normal anatomy and no tertiary dentin or other calcifications; (3) no presence of inflammatory cell accumulations or dilated vessels in the pulp tissues; and (4) no bacteria in the circumpulpal dentin. On the other hand, teeth with deep carious lesion restored after nonselective caries removal presented no pulp inflammation in 28/59, mild

inflammation in 14/59, and irreversible inflammation in 17/59. Regarding the teeth treated with SCR, although all teeth presented inflammation (8/8 [81] and 12/12 [8]), the authors found that: (1) teeth presented no symptomatology and responded within normal limits to thermal and electric tests [8, 81]; (2) teeth presented mild inflammation, with no case of moderate or severe inflammation [8]; (3) tertiary dentin was present in all teeth [8]; (4) bacteria were present in dentin adjacent to the cavity, in the transition between the secondary and the tertiary dentin and, in some cases, in the superficial tertiary dentin [8, 81]. Based on these results, it is possible to conclude that the risk of pulp inflammation is observed with both non-selective and selective caries removal techniques. In addition, it is important to highlight that while almost 30% of teeth treated with nonselective excavation underwent irreversible inflammation and consequently the need for more invasive treatments, no similar cases occurred after SCR. Furthermore, the above-described clinical studies show that less invasive approaches such as SCR-SD may improve patient-relevant clinical outcomes.

10.8   The Role of Cavity Liners After SCR-SD

Cavity liners have been traditionally proposed during the management of deep cari-ous lesions due to several reasons: to reduce the level of contamination, induce ter-tiary dentin deposition, remineralize demineralized tissues, act as thermal or electric insulator, and protect pulpal cells against irritation stimuli derived from adhesives [82, 83]. Among the used materials, calcium hydroxide cement (CHC), glass iono-mer cement (GIC), and hydraulic calcium silicate cements have been used in the clinical practice when the pulpal/axial cavity walls are close to the pulp.

During the decades when decayed tissue was excavated as much as possible and only a thin layer of carious dentin was left over the pulp to avoid its exposure, cavity lining was seen as an essential clinical step during the management of deep carious lesions. However, more recently, the literature has investigated whether the use of cavity liners plays a vital role on pulpal outcomes when applied to deep lesions after conservative caries excavation techniques.

Although the European Society of Endodontology (2019) [37] recommends the use of hydraulic calcium silicate or glass-ionomer cement over the deep dentin in both treatment strategies advocated for the management of deep lesions (SCR-SD and SW), the need for cavity lining in maintaining pulpal vitality has been ques-tioned. A systematic review with meta-analysis by da Rosa et al. [84] investigated whether the use of CHC liner improves the clinical success in the treatment of deep carious lesions. A total of 17 studies were included, of which 15 were conducted in primary dentition and 2 in permanent dentition. Meta-analyses were performed to compare CHC with GIC (data derived from two studies in primary teeth) and CHC with adhesive (data derived from four studies in primary teeth), and no significant differences were observed. The authors concluded that the use of CHC as a cavity liner did not influence the clinical success of treatment for deep lesions; however, the evidence was of moderate to very low quality.

Regarding permanent teeth, the only two studies included in this systematic review [44, 85] reported short-term outcomes (3–4 months of follow-up), and no meta-analysis was performed. Corralo et al. [44] compared lining with CHC, GIC, and an inert material (wax) and found no difference among the tested materials regarding dentin hardening, obliteration of dentinal tubules, decreasing bacterial numbers, and dentin reorganization after 3–4 months of sealing. Pereira et al. [85] compared teeth with and without CHC lining in conjunction with resin-modified GIC restorations. Irrespective of CHC liner use, no difference was found regarding tooth vitality, dentin darkening, hardening, and decreasing contamination after 3 months. Another short-term study not included in the cited systematic review compared the lining with GIC and an inert material for 2 months [86]. The authors found enhanced calcium and phosphorus levels and a better-organized tissue with a more compact intertubular dentin in both groups. They concluded that caries arrestment with dentin reorganization occurs regardless of the lining material placed in contact with the infected dentin.

No long-term study has been performed on permanent teeth. A randomized clinical trial comparing the use of CHC, resin-modified GIC, and adhesive system after SCR-SD reported the 12-month results [87]. Success rates regarding the maintenance of pulp vitality were 96.8%, 96.5%, and 94.6% for CHC, resin-modified GIC, and adhesive, respectively, with no significant difference among groups ($P = 0.81$). A randomized clinical trial, performed by our research group at the Federal University of Rio Grande do Sul, evaluated the effectiveness of indirect pulp protection with CHC or universal adhesive in deep carious lesions after SCR-SD or SW. Over 18 months of monitoring, similar success rates regarding the maintenance of pulp vitality were found (95.5% for CHC and 99.1% for adhesive) (unpublished data).

Lining with CHC has been also compared with mineral trioxide aggregate (MTA) in a randomized clinical trial [88, 89]. Similar success rates were found regarding the maintenance of pulp vitality at the 2-year follow-up (91.7% for CHC and 96.0% for MTA) and at the 4-year follow-up (82.9% for CHC and 86% for MTA).

Based on the available literature on this topic, the adequate cavity sealing is responsible for bacterial reduction and lesion control after SCR-SD. The remaining carious dentin has been seen, itself, as a protective factor for pulp vitality in deep carious lesions. Therefore, using cavity liners does not seem to be important when using conservative caries removal techniques [84, 90, 91].

10.9 Future Perspectives for the Management of Deep Carious Lesions

Current understanding of pulp biology and the pulp response to the release of dentin-bound bioactive growth factors demonstrate that the pulp has a greater regenerative capacity than previously thought [92]. Preserving all or part of the pulp is beneficial, resulting in a less invasive treatment than conventional endodontic

treatment [93], which, in addition to being specialized and costly, removes tooth structure, weakening the tooth.

Pulp preservation or the choice between invasive or more conservative treatment for deep carious lesions depends on the pulpal diagnosis of reversible and irreversible pulpitis, i.e., on the inflammatory status of the pulp. Correlations between histological findings and clinical signs and symptoms have been used to differentiate between stages of reversible and irreversible pulpitis despite limitations [3]. Traditionally, a reversible pulpitis should be treated with a conservative treatment, whereas an irreversible pulpitis should be treated with pulpectomy or tooth extraction [3, 81]. In the same line, Wolters et al. [94] proposed a classification of pulp inflammatory processes with treatment indications:

1. Mild/reversible pulpitis: positive response to the thermal sensitivity test (−20 °C), lasting up to 20 s, with possible sensitivity to vertical and horizontal percussion, but there is no previous history of spontaneous pain. The proposed treatment for these cases is indirect pulp therapy, which provides SCR-SD as a counterpoint to conventional pulpotomy therapy or root canal treatment.
2. Moderate/irreversible pulpitis: symptoms are prolonged response to the thermal test (−20 °C), which can last for minutes, sensitivity to percussion, and presence of spontaneous pain that can be suppressed with pain medication. This may or may not present with thickening of the space of the periodontal ligament and is described as extensive local inflammation confined to the coronary pulp. The therapy indicated for these cases is coronal pulpotomy.

Despite these traditional treatment indications, preliminary investigations have suggested that pulp preservation is possible when vital pulp therapies (such as SCR-SD, IPC, or pulpotomy) are performed in symptomatic teeth [95–98]. Clinical case reports have shown that in some situations of moderate/irreversible pulpitis, where the pulp is conventionally diagnosed as having irreversible inflammation, it can be maintained with clinical and radiographic success [95, 96, 99]. Results of a clinical study by Asgary et al. [93] indicated that vital teeth can be treated with SCR-SD irrespective of the presence of signs and symptoms of pulpitis (mild or moderate). After 1 year of follow-up, the success rates for SCR-SD, direct pulp capping, and partial and total pulpotomy were 95.6%, 88.5%, 85.6%, and 88.7%, respectively, with no statistical difference among groups.

It is important to point out that SCR-SD is a well-established technique for the management of deep carious lesions in teeth with signs and symptoms indicatives of pulp vitality, such as the absence of spontaneous pain, positive response to the cold test, and negative response to percussion, in addition to a periapical radiograph suggesting no pulpal involvement. However, these recent studies including teeth with signs of irreversible pulpal inflammation shed light on this research field. Further studies, mainly long-term randomized clinical trials may broaden the indications of the SCR-SD in the future.

10.10 Concluding Remarks

Based on the aspects discussed along this chapter, it is possible to summarize the contemporary approach for the management of deep dentin carious lesions in primary and young permanent teeth as follows:

- Nonselective caries removal results in a high risk of pulp exposure and is considered overtreatment according to current concepts for caries removal.
- Selective caries removal to soft dentin as a single-visit approach is recommended for the management of deep carious lesions in both primary and permanent dentition.
- There is no reason to reopen the cavity to excavate further, thus eliminating the disadvantages of the stepwise excavation technique related to the need for a second visit.
- The use of a cavity liner after selective caries removal, while not harmful, seems to be an unnecessary clinical step.

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