Adhesive dentistry meets restorative dentistry and endodontics – part one

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Abstract
Although adhesion to enamel has been a successful clinical procedure with good long-term results since the 1960s, the durability of the resin/dentine bond has been raised as an issue of concern during the past two decades. There are contradictory findings between experimental ex vivo and in vivo tests with the use of adhesive materials. Furthermore, the possible toxic effects of leached components from resin/based materials to the pulp, with in addition bacterial leakage and degradation of the interface adhesive resin/dentine are factors that greatly affect the long-term service of a restoration/endodontic treatment. Endodontics is not exempt from these factors and while the treatment is totally different, many adverse conditions can compromise the outcome of a root canal treated tooth. A thorough understanding of the physiopathology of the pulp/dentine complex and the physical properties and biocompatibility of the materials that are being used for treatment improves treatment outcomes and can result in long-term success. In that respect successful endodontic treatment is closely intertwined with adhesive restorative dentistry.

Key words: Dentine, dentine bonding, endodontics, hybrid layer, resin/dentine interface

Introduction

The restoration of vital teeth affected by caries involves the complete removal of the infected tissues, protection of the pulp with an appropriate material and replacement of the lost hard structures with a material that meets biocompatibility standards and can restore function and aesthetics. Dental amalgam, cements and liners have been used in the past with great success, however, direct adhesive restorations are now the preferred treatment of choice. The continued improvement of operative techniques and materials is in part driven by patients' demands for better aesthetics. The use of enamel adhesives started in the 1950s (Buonocore, 1955; Buonocore et al, 1966), however, the majority of the early materials revealed a poor clinical performance (Kramer and McLean, 1952). From the 1990s, a new generation of more reliable materials and techniques were introduced to the dental profession. These materials required as a first step dentine preparation with an acid gel, achieving demineralisation of the intertubular and peritubular dentine. Dentine is composed of a high percentage of water and organic material such as type I collagen together with noncollagenous proteins, proteoglycans, phosphoproteins and glycoproteins intertwined by a tubular network (Tjäderhane et al, 2012). Most of these dentinal tubules contain a cytoplasm extension of the odontoblasts. The odontoblastic processes establish a direct pathway of communication between a prepared cavity surface and pulp tissues. The odontoblastic processes are normally accompanied by the extension of pulp nerves (Figure 1) and the internal space of the tubules is filled with fluids. Consequently, the procedures for caries removal and cavity preparation as well as the direct application of restorative materials are always performed on vital pulp/dentine substrates. Therefore, clinicians should be aware of the various factors that regulate the response of the vital pulp and dentine structures to restorative materials. The biological evaluation of dental materials tested, according to the guidelines of the ANSI/ADA Spec # 41 (2015) [ISO 7405:2008 (E)], indicate that most of the contemporary adhesive materials and techniques are safe to the pulp/dentine complex.

In spite of this, there were many clinical reports of postoperative sensitivity and/or pulpal inflammation beneath dental restorations when tooth coloured resin-based

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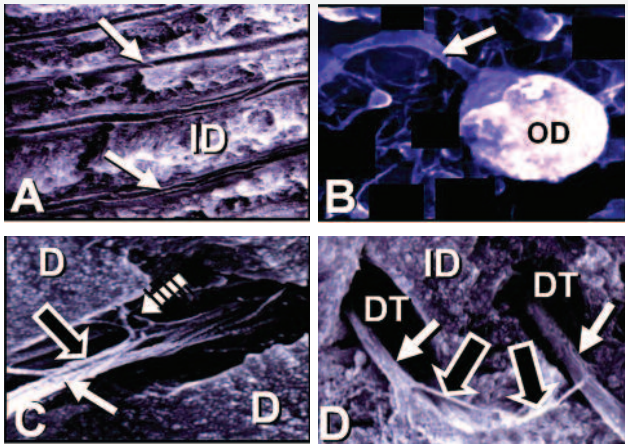

Figure 1: A: SEM micrograph showing longitudinally fractured dentinal tubules (white arrows) and intertubular dentine (ID). (Original magnification x3000). B: SEM micrograph showing an odontoblast like-cell (OD) and the odontoblast process (arrow). (Original magnification x10.000). C: SEM micrograph of a longitudinally fractured dentine tubule showing the presence of an odontoblast process (white arrow) accompanied by a nerve fibre (black arrow) and a lateral branch (interrupted arrow). D: Dentine (Original magnification x5000). D: SEM micrograph of two dentinal tubules (DT) showing odontoblast process (white arrows) and two interconnected branches (black arrows). ID: Intertubular dentine. DN: Dentine. (Original magnification x5000)

materials were used (Council on Dental Materials, Instruments and Equipment, 1988; Costa et al, 2017). As a result of the sometimes contradictory findings between experimental ex vivo and in vivo tests and the clinical use of adhesive materials in humans (Wataha, 2012), the dental practitioner cannot be offered a guarantee concerning the lack of toxic effects and deleterious consequences of currently used materials and their reaction to the pulp. Bacterial microleakage as well as the stability of the bond at the interface is still a major concern. This text will discuss the biological relationship between contemporary adhesive materials and the pulp/dentine complex. Furthermore, factors influencing the long-term durability of adhesive restorations will be addressed for restorative dentistry and endodontics.

Biocompatibility tests of restorative materials: are they reliable?

After caries removal, the preparation of a cavity suitable for restoration involves enamel and dentine and on occasion part of the root cementum. Reaction of the pulp to operative procedures has been historically reported by Langeland (1959, 1961) in early subhuman primate studies. Copious water-cooling during high-speed cavity preparation appeared to be a crucial factor to cause the least amount of insult to the pulp. More recently Mjör (2001a) reported similar findings. Considering the fact that caries lesions, promotes the development of pulpal alterations (Langeland, 1987; Heyeraas et al, 2001), the clinician has to realise that the pulp will suffer subsequent additional injury from cavity preparation and placement of the final restoration. Thus, the pulp has to cope with a multifactorial attack. The impact of each individual factor on the long-term results of a dental restoration is difficult to predict.

Recommended pre-marketing tests start with the primary in vitro cytotoxicity tests using cell and tissue cultures [Dobie et al, 2002; Al-Sabek et al, 2005; Sun et al, 2016; ANSI/ADA Specification #41 (2015) (ISO-7405:2008 E)]. Many laboratory experiments use healthy dentine samples while clinicians usually deal with caries affected dentine. Although in vivo tests are more reliable procedures for evaluation of material biocompatibility, most manufacturers and researchers still rely on in vitro tests to assess the clinical performance of restorative materials (Wataha, 2012). The primary in vitro tests must be followed by secondary in vivo biocompatibility tests in connective and bone tissues of mammalian small laboratory animals. The secondary tests constitute an important safety pre-marketing evaluation of newly developed materials. They can show whether the components of, for instance, resin-based materials can elicit damage to the surrounding tissues when implanted in connective tissue or bone in small animals such as rats, rabbits or guinea pigs. Most materials generate mild to moderate inflammatory reactions of the surrounding tissues when evaluated over short-term periods of 10 and 30 days. But after 90 to 120 days they usually show acceptable biological responses. Nevertheless, there are reports of postoperative sensitivity ranging from minor to severe pain after caries excavation and restoration with adhesive resin-based materials (Council of Dental Materials, Instruments and Equipment 1988; Costa et al, 2017). More reliable pulp reactions can be obtained from tests in large mammalian animals, the tertiary tests; also called usage tests (Wataha, 2012). Ideally, subsequent well-controlled double-blind clinical trials in human subjects (gold standard) are recommended before marketing a new material (Van Dijken, 2004; Andersson-Wenckert et al, 2004; Accorinte et al, 2005; Nowicka et al, 2013; Koubi et al, 2013; Van Dijken et al, 2015). The usage tests mimic the in vivo clinical treatment in human teeth and can be done in rats (Higashi et al, 2000; Mori et al, 2014; Dammaschke et al, 2010), guinea pigs (Shayegan et al, 2012), cats (Olgart et al,

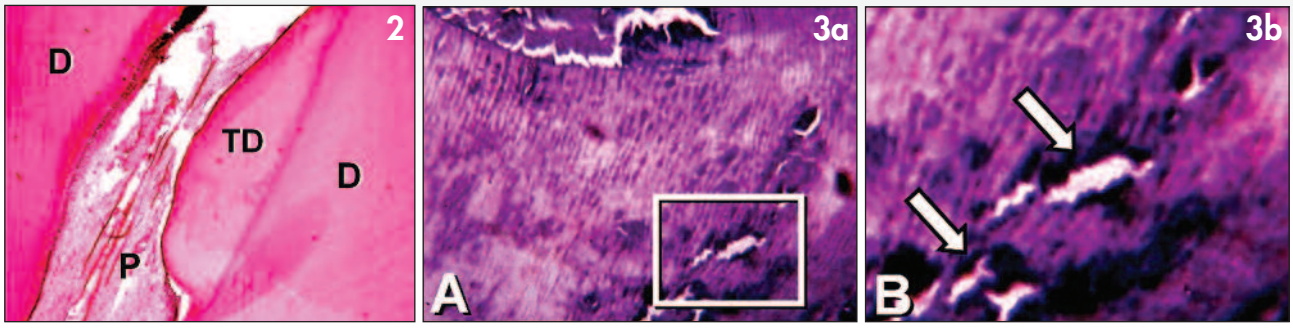

Figure 2: Microphotograph of a histology section from a subhuman primate tooth showing a 70-day pulp reaction to an indirect restoration with a test material. A considerable amount of tertiary (reparative) dentine (TD) that constricts part of the root canal space can be seen. D: Dentine; P: Pulp (H & E stain; Original magnification x100)
Figure 3: A: Beneath a deep caries lesion bacteria have invaded the dentinal tubules while the acidic environment has destroyed the surrounding mineralized tissue (square area). (H & E stain; Original magnification x100). B: Higher magnification of the square area in A showing numerous bacteria in the dentinal tubules. Arrows indicate destroyed dentine (H & E stain; Original magnification x1000)

1974), dogs (Faraco and Holland, 2001), swine (Koliniotou-Koumpia et al, 2014) monkeys (Mjör and Tronstad, 1974; Akimoto et al, 1998; Pameijer and Stanley, 1998, Cannon et al, 2014) and the teeth of goats (Zhang et al, 2008). These teeth must be free of caries and have healthy pulps. When human teeth are used, they should be from young patients, for instance premolars scheduled for extraction for orthodontic reasons. The recommended Class V cavity preparations should have as much as possible standardised depths and extend as much as possible to the mesial and distal proximal surfaces and placed on enamel and into dentine. However, there are some limitations pertinent to a reliable interpretation of the results as well with respect to the clinical significance. For instance we know that when practicing routine restorative dentistry there are a number of clinical variables that deviate from the ideal conditions of teeth employed in usage tests (Mjör, 2001b). Histological data from an experiment in subhuman primates using healthy teeth can generate information concerning the biocompatibility and reactivity of a test material. In the example of Figure 2 in a postoperative period of 70 days a considerable amount of reparative dentine was observed, which over time could have constricted the root canal space entirely. There is a fine line between a material that stimulates reparative dentine just so much to protect the pulp and a material that elutes components that continue to stimulate the odontoblasts to produce reparative dentine that is excessive and therefore counterproductive. Teeth that require adhesive restorations usually have decay that differs in depths and size, or have recurrent decay in failed restorations. As a result, the clinical and histological reactions of the pulp/dentine complex to operative procedures and restorative materials is quite different from what takes place in unaffected teeth. In teeth affected by caries bacteria have advanced through the dentinal tubules towards the pulp while the acidic environment caused by their by-products, together with collagenolytic enzymes activated by the low pH, have destroyed the surrounding mineralised tissues (Figure 3). Under these conditions, the pulp showed different degrees of degenerative or inflammatory changes (Langeland, 1959; Langeland 1961; Mjör, 2001a), while the dentine frequently revealed sclerotic alterations and/or dystrophic calcifications within the pulp tissues, including the apposition of tertiary dentine on the pulp chamber walls. The tertiary dentine can also be localised in the area of the pulp horns. Bacteria that invades tertiary dentine can cause slight to severe pulpal reactions (Warfvinge, 1986). On occasion the calcification of tertiary dentine can be so extensive that the pulp chamber is completely filled (Langeland, 1987), while the root canals are narrowed by mineralised tissue (Appleton and Williams, 1973; Vasiliadis et al, 1983a; Vasiliadis et al, 1983b; Björndal and Mjör, 2001). Therefore, in daily practice reactions of a compromised pulp will be very different from the reaction of healthy pulps used in experimental usage tests. Thus, the direct correlation of experimental results to the actual clinical outcome should be made with great caution.

Pulp reactions to a material may depend not only on the toxicity of material components but also on the remaining dentine thickness (RDT), the distance between restorative material and the pulp (Langeland, 1967; Qvist and Stoltze, 1982; Murray et al, 2000). It is generally accepted that a RDT of 2 mm or more is safe to avoid possible harmful effects of a restorative material to the pulp. However, in order to

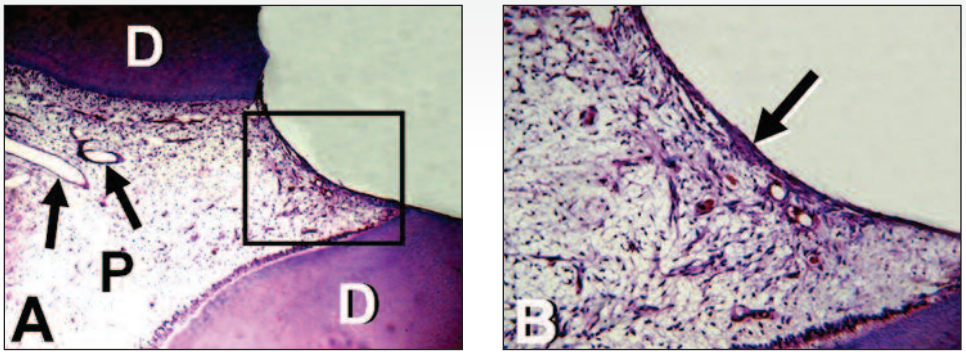

Figure 4: A 15-day postoperative microphotograph of an exposed vital rat pulp treated with 37% phosphoric acid for 15 seconds followed by rinsing with sterile twice-distilled water. Note the presence of a thin fibrous capsule at the exposed pulp surface and some dilated blood vessels (arrows). D: Dentine; P: Pulp (H & E stain; original magnification x100). B: Higher magnification from the square area in A. Note the presence of the fibrous capsule (arrow) and dilated vessels in the adjacent areas (H & E stain; original magnification x450).

obtain accurate data of the biological properties of an experimental material, the ANSI/ADA Spec, #41 [ISO 7405-2008 (E) standards] recommends that the RDT must be approximately 1 mm or less. In adult teeth physiological aging causes dentine tubule occlusion, resulting in a reduction of dentine permeability (Pashley, 1985; Pashley, 1996) thus lessening the potential toxic action of materials. Note that the scenario for pulp capping is quite different. RDT is irrelevant since the test material is in direct contact with the exposed pulp. In a clinical situation pulp reactions depend on several factors, i.e. toxicity of the capping material, location of the exposure, dimension of the exposure, presence of infected or affected dentine, and maturation of the tooth. Under these conditions the ability of the pulp to react to different injuries is significantly compromised, especially when a pulp exposure was caused by a caries lesion. Understandably pulp reactions are very different from the usage tests conditions, in which healthy pulps are exposed and essentially only the toxicity of the capping materials is tested.

Another difference between the experimental usage tests and the actual clinical situation is the location of the preparation. According to the ANSI/ADA Spec. #41(ISO 7405 Standards) placement of the cavity has to be in dentine. In clinical practice however, caries lesions are frequently occurring in root cementum, especially in the older population. Consequently, there are two factors to consider. First, after removal of caries the RDT is usually small. However, dystrophic calcification is a mitigating factor reducing the reaction of the pulp (Amir et al, 2001). Secondly, root cementum is very thin and easily destroyed during cavity preparation resulting in postoperative immediate or late dentine sensitivity (Murray et al, 2000; Murray et al, 2001).

Realising the differences between the results of the usage tests and the actual clinical situation, some researchers have induced inflammatory pulp reactions prior to pulp exposures and placement of the capping material (Mjör and Tronstad, 1972; Mjör and Tronstad, 1974; Lervik and Mjör, 1977). Most of these experiments revealed that under these conditions the reparative ability of the pulp was similar in all samples depending on the type of material tested. From a clinical point of view the experiments performed in humans revealed that it is not possible to analyse the pulpal cellular changes that take place based on signs, symptoms and vitality tests. This has lead to the conclusion that pulpal responses to a restorative material or operative technique are best analysed by histopathological and histomorphometric methods (Browne et al, 1980; Warfvinge, 1987; Pameijer, 1992; Accorinte et al, 2005; Nowicka et al, 2013). Unfortunately, the cellular events are not easily standardised and measured, especially when teeth of young individuals are used. A greater capacity to respond to different type of insults has been observed (Björndal et al, 1998).

In summary, results of experiments on healthy teeth with intact pulps in animals or humans are mostly reliable but should nevertheless be interpreted with caution as these conditions may be more favorable than the actual clinical situation. To date, there are no standardised studies on the healing of pulps in teeth with inflamed pulps when adhesive restorative techniques are used. In teeth showing some degree of pulpal inflammation beneath a deep caries lesion, the adaptation and penetration of resin-based materials after acid etching may be prevented by the early mineralisation of peritubular dentine and precipitation of dissolved mineral salts within the lumen of dentinal tubules. In addition, the

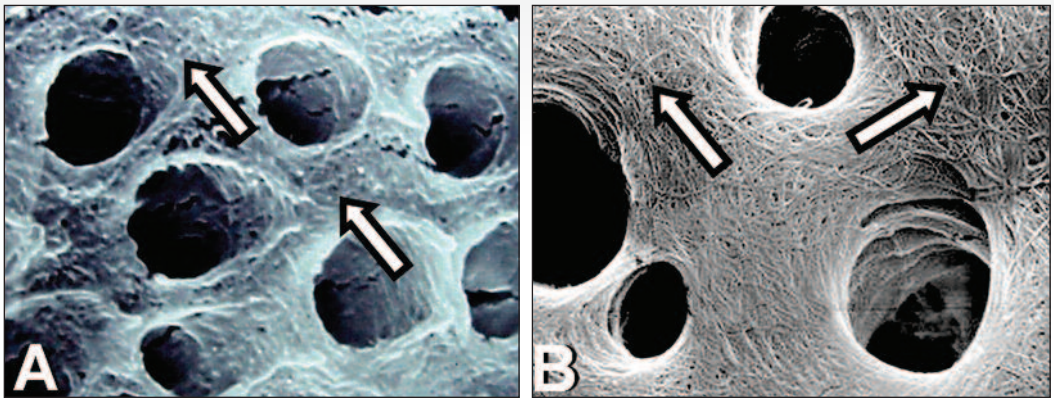

Figure 5: A: SEM microphotograph of a dentine surface treated with 37% phosphoric acid for 20 seconds followed by 5.25% EDTA solution and finally rinsed with sterile twice-distilled water. Note the empty dentinal tubules and the microporosity of the intertubular dentine (arrows), which was caused by the acid (original magnification x4000). B: SEM microphotograph showing the unprotected dentine collagen fibers (arrows) after acid-etch treatment (Original magnification x5000). (Courtesy of Dr Jorge Olmos)

augmented pressure exerted by fluids in the dentinal tubules of an inflamed pulp may also prevent the penetration of resins. Vasoconstrictors in anaesthetic solutions may decrease the internal pressure of an inflamed pulp (Ciucchi et al, 1995) causing another variable. What should be emphasised is that defence against pulpal inflammation is not unlike what occurs in other connective tissues in the body (Holland et al, 2001; De Rossi et al, 2014). With one exception, the dental pulp is confined within hard tissues causing a very different reaction resulting in pain. The many studies on the pulp/dentine reaction to adhesive dental materials are unfortunately characterised by important differences in the tests methods. In our opinion, it is necessary to use one single and universally accepted standardised method to allow comparison of the results between different researchers. The ANSI/ADA Spec. #41 or ISO 7405 (the guidelines have most recently been synchronised) offer this possibility but the guidelines are more often not followed carefully, misinterpreted or ignored. Heyeraas et al (2001) have suggested that the use of new experimental technologies in conjunction with the present knowledge of morphogenetic factors and tissue engineering will allow researchers to find improved experimental methods in order to get a more reliable evaluation of the pulp/dentine complex to resin-based restorative materials and techniques.

Concluding remarks

The use of intact teeth in animals or humans to determine pulpal responses to different operative techniques and/or materials does not accurately mimic the typical clinical situation since healthy teeth without caries or inflamed pulps are used for testing of experimental materials. However, the biocompatibility of these materials and the degree of pulp reaction it produces provide good evidence as to the suitability for commercial use. The ultimate test, however, is the performance in humans.

Pulp/dentine complex reaction to resin-based materials

The advent of bonding in restorative dentistry by means of the acid etch technique was introduced in the mid 1950s by Buonocore (1955) and based on resin technology developed by Hagger (1951). Early strong resistance slowly gave way to general acceptance and bonding materials and techniques completely changed the way dentistry is being practiced today. Initially only hydrophobic resins were available. However, over time these were supplanted by hydrophilic resins and about 30 years of research resulted in a change from using 85% phosphoric acid liquid for 60s to etch only enamel to 35-37% phosphoric acid gels for 15s–20s to etch both dentine and enamel. The biologic and clinical effects of the early restorative materials were dramatic for dentine and pulp tissues. Most of the treated teeth reacted with severe pulpal inflammation and pulp necrosis (Langeland, 1966; Heyeraas et al, 2001). Consequently, the majority of these teeth required endodontic therapy or were extracted by emergency dental services. More recently, restorative materials and techniques have improved by gaining knowledge of enamel and dentine bonding procedures. Currently tooth-colored resins are the material of choice by most clinicians. These improved resins are based on dimethacrylate monomers comprising a matrix of resins and inorganic fillers. The total-etch technique using

phosphoric to etch enamel and dentine as part of adhesive restorative treatment has been an issue of concern. However, a study by Lee et al as early as 1973 demonstrated that phosphoric acid gels at a concentration of 35%-37% for 15s–20s did not notably increase the permeability of dentine. When higher concentrations (50%-72% for five minutes) were used the acid only penetrated a few micrometers into dentine and did not reach the pulp chamber. The same was observed in deep cavities (Lee at al, 1973). Aida et al (1980) used different concentrations of phosphoric acid (10%, 30%, 50% and 70%) to investigate clinically and histopathologically the effect of the total etch technique. Pulp reactions were studied in 120 class I cavities of vital human permanent teeth in patients ranging from 10 to 42 years in age. None of the phosphoric acid concentrations elicited pulpal inflammation. Similar results were obtained by Retief et al (1974). Why this does not happen is a legitimate question to ask. Perdigão and Lopes (1999) offered two explanations. Because of the high concentration of the phosphoric acid, crystals of calcium phosphate are formed, which block the deeper penetration of the acid. Secondly, the interaction of the acid with dentine is limited by the buffering action of the hydroxyapatite and protein contents of dentine. These observations suggest that the limited penetration of phosphoric acid into dentine and the subsequent removal by vigorous rinsing do not have a deleterious effect on the pulp. Furthermore, Zmener and Kokubu (2003; unpublished data) observed that 35% or 37% phosphoric acid for 15 seconds in direct contact with vital rat pulps, followed by vigorous rinsing with sterile twice-distilled water, did not have a harmful effect. In this experiment the pulps were protected with a silicone film followed by a glass ionomer cement. After 15 days, histological analysis of the pulps showed a thin fibrous layer with normal pulp tissue (Figure 4). The main difference of opinion among researchers revolves around the question as to whether the total etch technique caused pulpal inflammation or does it make the dentinal tubules more permeable to the toxic components of resin-based materials. Yet, many practitioners still prefer to use cement bases or liners for pulp protection, in spite of scientific data that has demonstrated that treatment of dentine by acid etching constitutes a biologically acceptable procedure (Tay et al, 1997; Perdigão and Lopes, 1999; Armstrong et al, 2003; Toledano et al, 2007). From a clinical point of view the preventive use of bases or liners to protect the pulp cannot be criticised, however, it can significantly reduce the dentine surface available for bonding.

Acidic agents

Phosphoric acid as well as other acidic agents on dentine surfaces will remove the smear layer caused by cavity preparation, demineralise the dentine and widen the dentinal tubules. This has been conclusively demonstrated by Scanning Electron Microscopy (SEM) and Transmission Electron Microscopy (TEM), which showed resin tags penetrating the dentinal tubules (Perdigão and Lopes, 1999; Breschi et al, 2008; Tjäderhane et al, 2013) giving the restorative material "micromechanical retention" which is frequently erroneously interpreted as an "adhesive mechanism". The acidic agents also increase the microporosity of the intertubular dentine leaving the collagen fibres unprotected (Figure 5). In a study using transmission electron microscopy (TEM), Tay et al (1997) demonstrated that acid treatment of dentine in deep cavities does not cause harmful effects on the pulp. Their data has been confirmed by Ferracane and Condon (1990) who showed that the actual cause of pulp damage comes from leaching of residual unreacted monomers and the elution of other leachable components from the resins. The toxic effects produced on cell cultures and connective tissues, which were in direct contact with adhesive resins were largely investigated by several authors (Hanks et al, 1991; Rathbun et al, 1991; Costa et al, 2000). The unreacted monomers persist in polymerised resins and undergo a rapid elution from the bulk of the material. Costa et al (2000) demonstrated that the leaching of unbound molecules along with other components from resin-based materials caused severe inflammatory reactions on the subcutaneous connective tissues of the rat. On many occasions the severity of the inflammation persisted during long-term periods. These findings agreed with those of Rathbun et al (1991) who observed loss of components from BIS-GMA dental composites in organic water-based mediums.

Most of the present adhesive systems are composed of light polymerisable hydrophilic monomers which micromechanically attach to the collagen fibres that are exposed after acid-etching of dentine. This structure is composed of a mixture of residual hydroxyapatite crystals and resin impregnated collagen fibers, commonly referred to as the hybrid layer (Erickson, 1992). The remaining hydroxyapatite crystals tend to stabilise the collagen and prevent its denaturation and collapse (Perdigão and Lopes, 1999). Clinical steps to avoid collagen collapse after etching involve lightly drying the dentine with filtered compressed air leaving the surface slightly moist. Too much drying will result in desiccation of dentine causing collapse

of the collagen which will prevent the penetration of the adhesive resin (Gwinnett, 1992).

The final step of the restorative treatment is to placement of a tooth-colored resin composite to restore form, function and aesthetics. According to Van Meerbeck et al (1994) the hybridisation of collagen is a very important step. The authors reported that of a total of 1,117 class V cavity preparations in 346 patients evaluated after six months and three years, the group in which the smear layer was removed and hybridised behaved clinically better compared to the group without that treatment. However, many studies demonstrated that the hybridised areas are subjected to further degradation processes which in turn significantly reduced the long-term durability of the restorations. Over time the mineralised dentine matrix may be destroyed by the acidic environment produced by the microbial products and the activation of endogenous matrix metalloproteinases (MMPs) and cysteine cathepsins, a group of fossilised mammalian collagenolytic enzymes expressed by odontoblasts that are present in the dentine matrix and capable of degrading extracellular matrix components (van Strip et al, 2003; Sorza et al, 2004; Carrilho et al, 2007; Sulkala et al, 2007; Tersariol et al, 2010; Liu et al, 2011). These events occur because after acid etching adhesive monomers are unable to fully cover the exposed collagen fibrils, which remain vulnerable to the time-dependent MMPs and cysteine cathepsins attack. Based on the knowledge of the role of these enzymes in the hybrid layer degradation process researchers have proposed different strategies to prevent the collagenolytic degradation of the resin/dentine interface. Among these strategies the MMPs and cysteine cathepsins inhibition by specific agents such as galardin (Breschi et al, 2010), or non-specific ones such as chlorhexidine digluconate (CHX) (Gendron et al, 1999; Hebling et al, 2005; Bracket et al, 2007; Tjäderhane et al, 2013) and ethylene diaminetetracetic acid (EDTA) (Osorio et al, 2005; Thompson et al, 2012) have been reported to be effective to protect the resin/dentine interface. CHX appears to be the most accepted method as in addition it has disinfection properties.

The hydrolytic degradation of the resin/dentine bond is another issue of concern that clinicians should be aware of. Water sorption of the resin/dentine interface is considered responsible for adhesion degradation (Feitosa et al, 2012). In an ex vivo experiment Van Landuyt et al (2010) reported considerable degradation of the resin/dentine interface after a relatively short six months storage in water. In a clinical situation, however, the resin/dentine interface is usually protected by the surface resin/enamel bond (Torkabadi et al, 2009; Reis et al, 2008). Howver, hydrolytic degradation of the resin/dentine bond is of concern. A vital pulp has a permanent hydrostatic water pressure provided by blood and lymphatic circulation (Heyeraas, 1989). Therefore, under in vivo conditions, the main source of water uptake by the resin/dentine interface is through the dentinal tubules due to pulpal pressure. (Feitosa et al, 2012). This pressure together with MMPs and cysteine cathepsins allow for degradation of the hybrid layer, which negatively affects the long-term durability of the restoration. A worse scenario caused by enzymatic and hydrolytic degradation is the subsequent bacterial penetration into the dentinal tubules and diffusion of their endotoxins often resulting in pulpal inflammation and eventual periapical pathosis (Tay et al, 1994; Bergenholtz, 2000; Mjör and Ferrari, 2002; Zmener, 2014). Beneath resin restorations or in carious affected teeth with permeable dentine, bacteria can reach the pulp chamber causing irreversible inflammation. When dentine is highly calcified, however, bacteria appear to be less dangerous (Björndal and Mjör, 2001). Clinicians must be aware that in deep caries lesions, the leathery dentine is invaded by bacteria. Below it, bacteria and their toxic by-products have advanced towards the pulp or have reached the pulp tissues already. In a histological study in human patients between 1990 and 2000 at the endodontic department of a private clinic (Zmener and Domingues; unpublished data), 852 teeth presenting with asymptomatic deep carious lesions without pulp exposures endodontic treatment was indicated. After the pulps were extirpated they were fixed in 10% buffered formalin and prepared for histological evaluation. Microscopic findings of Hematoxylin and Eosin (H&E) stained sections revealed that 13.14% presented with acute inflammation, 76.76% were chronically inflamed, while 10.09% were necrotic. In both acute and chronically inflamed pulps the pathology was mainly observed in the coronal pulp. Other common histopathological findings in the chronically inflamed pulps were the presence of a great number of lymphocytes, macrophages and multinucleated giant cells. It has been long recognised that macrophages and lymphocytes are specific target cells in recognising antigenic products (Jontell et al, 1998). Macrophages are essential cells to phagocyte foreign particles, dead cells (mainly polymorphonuclear and red cells) or foreign bodies (Metzger, 2000) to be further processed by lymphocytes. As is the case in other inflamed tissues of the body, this mechanism is produced in the pulp via intercellular connections. These intercellular connections are not detectable in stained paraffin sections, but easily observed with the SEM (Zmener and Pameijer, 2012).

Recovery of the pulp is the ultimate goal after caries removal and cavity restoration with adhesive resin-based materials. Even in the presence of a mild or moderate inflammatory reaction the pulp can heal, providing atraumatic clinical procedures are practiced for cavity preparation and restoration. Tertiary dentine (reparative dentine) has been deposited on the pulp chamber walls, especially under caries affected areas. Tertiary dentine is formed by surviving odontoblasts or by newly developed odontoblast-like cells and is an indication of pulp healing. However, in teeth with deep caries lesions that have affected the pulps there is almost no chance for healing. A case in point is an early assay by Mjör and Tronstad (1974), who induced severe pulp pathologies in healthy monkey teeth by filling deep cavities with soft caries dentine. After different time periods the cavities were excavated and restored. Eighty two and 90 days postoperatively, the teeth were extracted and prepared for histological evaluation. The results showed that the pulps had healed by apposition of tertiary dentine. It should be emphasised, however, that essential differences exist between the results from the above described simulated caries attacks in teeth with healthy pulps and progressive caries affected teeth in a clinical situation with subsequent restorative treatment.

Summary and concluding remarks

In cases of deep caries lesions endodontic treatment is not necessarily always indicated. With proper restorative materials and techniques the pulp, even when inflamed, has the capacity to heal, thus maintaining the vitality of the tooth. Adhesive dentistry involving acid-etch-techniques and resin-based materials can be used successfully as long as practitioners have an understanding of the biocompatibility and physical properties of the materials they use.

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