Therapeutic strategy for cracked teeth

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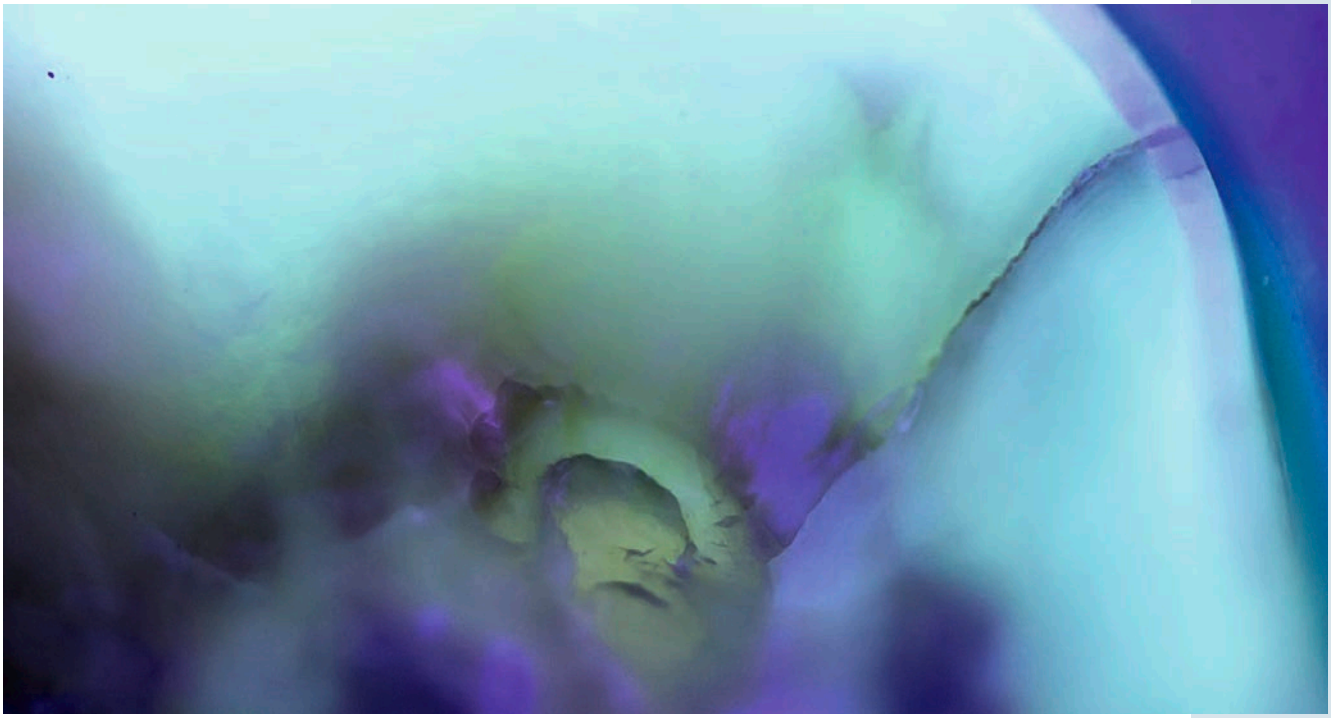

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Abstract

The management of cracked teeth represents a difficulty because their diagnosis is complex and there is no consensus concerning their treatment. The present article explains this pathology within enamel and dentin and also focuses on the clinical consequences of crack development in dental tissue. As cracks have both biologic and mechanical implications, a complete review of the literature on the subject has enabled the development of a comprehensive diagnostic approach to identify cracked teeth and optimize their management. The elements of diagnosis are the bite test, transillumination, the pulp sensitivity test, the periodontal test, radiologic examinations, removal of existing restorations, and the use of quantitative light-induced fluorescence. Finally, the management of biologic and mechanical imperatives relating to the treatment of cracked teeth has allowed the proposal of a reliable and reproducible therapeutic strategy based on two pillars: the arrest of bacterial infiltration using immediate dentin sealing, and the limitation of crack propagation using relative cuspal coverage. In this article, the proposed clinical protocol is explained through the use of a decision map and is illustrated by a clinical case example.

(Int J Esthet Dent 2022;17:336–351)

Introduction

Cameron introduced the 'cracked-tooth syndrome' in 1964.[1] The syndrome involves fractures that are not easily visible; however, the teeth exhibit a pain response to cold or to pressure applications and become necrotic, despite an apparently healthy pulp and periodontium. Then, in 2001, Ellis proposed a new definition, describing incomplete tooth fracture as "a fracture plane of unknown depth and direction passing through tooth structure that, if not already involved, may progress to communicate with the pulp and/or periodontal ligament."[2] Currently, the American Association of Endodontists defines amelodentinal coronal fractures as incomplete fractures (without visible separation or mobility of the fragments) that initiate at the coronal level and extend apically, most often in a mesiodistal direction. This differs from complete longitudinal dental fractures, where two fragments are mobile and separable.[3]

Cracks are a significant challenge for the practitioner because they are difficult to diagnose and treat and often have consequences that lead to the loss of the affected tooth.

Crack pathology

The tooth is a rather stiff organ that resists the spread of cracks throughout life fairly well, at least under normal conditions. Nevertheless, it is necessary to understand its tenacity in order to direct the therapeutic management of tooth cracks.

Enamel

Chai et al[4] described areas of enamel, called 'tufts,' located at the dentinoenamel junction (DEJ), which improve the mechanical properties of the enamel by allowing it to bear stresses.[4,5] However, tufts are, paradoxically, also weak areas because they are intrinsic defects of the microstructure that serve as a starting point for the development of cracks. Tufts are hypomineralized areas with high protein concentrations, located at the level of the DEJ.[6-8] Natural enamel cracks extend from the DEJ to the occlusal surface, but some are stopped along their path through the enamel.[5]

Mechanisms exist to increase the resistance to crack growth in the enamel:[9]

- *The deviation that takes place in the transition region between the internal and external enamel.* In the external enamel, the prisms are relatively straight, and the cracks extend along these prisms, while in the internal enamel, the cracks meet oblique prisms, which cause their deviation.[10,11] The microstructure change between the external and internal enamel causes a change in the direction of crack propagation and induces an increasing tenacity of the enamel toward the pulp.[12]
- *Microcracking, which prevents the propagation of cracks in the internal enamel.* Microcracking occurs in front of the growing crack and slows down the main crack by reducing the intensity of local stress by dividing the crack.[10,13]
- *Crack bridging, which also prevents the propagation of cracks in the internal enamel.* These bridges, induced by the organic matrix within the internal enamel, promote the closure of cracks (thanks to the maintenance of the protein frame) and stop crack growth.[7,11]

These mechanisms seem to exist only in the internal enamel, particularly in the zone of decussation, and contribute to making the enamel an extremely tenacious tissue against the propagation of cracks.[9] Cracks that only affect the enamel and are stopped at the DEJ are called 'craze lines.' They do not cause any pain, are fortuitously discovered most of the time, and rarely progress to a cracked tooth.[14]

Dentinoenamel junction

The DEJ microanatomy promotes the interruption mechanism of cracks due to its lower mineralization and higher collagen content, which prevents stress concentration.[15] Crack arrest is explained by a differential modulus of elasticity between the enamel and the dentin; indeed, enamel and dentin have very different moduli of elasticity. The scalloped and microscalloped DEJ design allows optimal cohesion between the enamel and the dentin and forms a complex area capable of plastic deformation.[16] This plastic deformation capacity allows it to resist crack propagation. A deflection of the crack occurs near the DEJ, in the same way as between the internal and external enamel.[10,17] It has been found that the crack arrest mechanism only appears if the cracks approach the DEJ on the enamel side. The enamel acts as a compression dome – the enamel compression loads are transferred via the DEJ to the dentin. In this transfer, a stress concentration occurs at the DEJ as it converts the enamel vertical load into a horizontal load in the dentin.[18]

Dentin

Crack growth in the dentin is influenced by its microstructure: the 'density of the diameter' of the dentinal tubules and their geometry. The crack will extend from tubule to tubule. The resistance to cracking decreases from the DEJ to the pulp due to the increase of dentinal tubules and the decrease of hydroxyapatite crystals. This allows easier crack propagation.[19] Mantle dentin, which is the dentin right below the DEJ, participates in stopping cracks, thanks to its tenacity (its mineral content is lower) and because it contains few dentinal tubules.

The mechanisms at the dentin level are much the same as those at the enamel level. These mechanisms contribute to the dentin rupture strength[20] – firstly, as regards the deviation of the cracks;[10] secondly, as regards crack branching, which occurs from an initial crack and leads to multiple microcracks that protect each other;[19] and thirdly, with the bridging of cracks, when the non-cracked protein frame and collagen fibers fill the cracks and prevent them from opening further.[21] Resistance to crack growth due to fatigue decreases in dentin with age and tissue dehydration.[22,23]

Risk factors

In general, the posterior teeth are the most affected by cracks. Molars can withstand heavy loads. However, if a crack occurs, the load capacity will be reduced and the enamel will transfer the loads to the internal components of the tooth.[6] Due to their orientation and certain constraints (lateral forces, training movements), the guide cusps are more affected by cracks.[24]

Risk factors include the following: Firstly, deep restorations and cavities; indeed, occlusal preparations decrease the resistance of the teeth in proportion to the width of the preparation.[24-29] Secondly, endodontic treatment, which decreases the fracture resistance of a tooth by 5%. Also, a devitalized tooth suffers a loss of proprioception, thus increasing the mechanical stress.[29,12] Thirdly, the wedge effect caused by the occlusal relation between cusp and fossa during chewing induces cyclic fatigue. Fourthly, materials, including setting contraction for composites or expansion for amalgam; the interface between the restoration and the tooth is a region of high-stress concentration, which serves to initiate cracks.[5,30,31] Finally, repeated thermal or mechanical shocks (lingual or labial piercing) are also contributing risk factors.

Consequences of cracks

The presence of a crack on a dental structure will have consequences as much on the biologic as on the mechanical level.

Biologic impact

Cracks are colonized by bacteria that create a biofilm.[32,33] Without treatment, bacteria invade the dentinal tubules. On the other hand, the fluid movements in the dentinal tubules cause pressure on the odontoblastic process and generate pain.[33,34]

An inflammatory cell accumulation is present in the pulp area underlying the tubules involved in the crack. In the event of the crack extending to the pulp, this causes reactions ranging from acute inflammation to pulp necrosis.[32,35] The crack allows the outside surface of the tooth to communicate with the pulp chamber, which causes an inflammatory degeneration state in the pulp tissue. The defense reaction to bacterial infiltration within the crack causes pulpal hyperemia.[34] However, not all cracks systematically lead to pulp or periapical pathologies.

Mechanical impact

Cracks can cause changes to tooth strength, for example, if a crack reaches a marginal ridge, the quantity of healthy tooth structure is reduced.[36] A crack on the marginal ridge leads to significant weakening, and the loss of a marginal ridge leads to a reduction of 46% of the tooth strength, and 63% in the event of the loss of two marginal ridges.[37] Lateral chewing forces as well as cavity preparations create internal shear and tensile stresses that can lead to a complete or incomplete fracture of the root.[36]

Crack growth in the pulp direction may require endodontic treatment and therefore lead to a significant weakening of the tooth. In extreme cases, a crack can lead to a longitudinal fracture and extraction.[34]

Diagnostic procedures

It is very difficult to diagnose dental cracks.[36] They can go unnoticed until a reconstruction removal or a periodontal defect detection, but sometimes warning signs are reported such as temperature variations, pain or intermittent chewing pain.[38]

Bite test

The bite test is the most common test in the diagnosis of cracks.[39] In the presence of a crack, the response to this test is an absence of pain when loading and severe pain when unloading.[40] If the pain stops quickly after bite relaxation, a coronal crack can be suspected. If the pain persists or is present without overload, a radicular fracture with a desmodontal inflammation can be suspected. The bite test sets the microfragments in motion on both sides of the crack, which stimulates the odontoblasts via fluid movements within the dentinal tubules.[34] Pain during this test is a pathognomonic sign of a cracked tooth. However, the absence of a reaction to this test does not exclude the possibility of a crack.[38,30]

Transillumination

Transillumination consists of placing a light beam perpendicular to the supposed crack plane. If the tooth is deeply cracked, the light is blocked and only part of the tooth is illuminated (Fig 1). If the tooth is microcracked, the light continues to spread and illuminates the whole tooth.[41] Depending on the wavelength, near-infrared transillumination can be utilized through devices such as the DIAGNOcam or the DEXIS CariVu (KaVo Dental).[42]

Pulp sensitivity test

Hypersensitivity to cold is the most common symptom of dental cracking. According to a study by Hilton et al,[38] 81% of patients with a dental crack experienced pain in response to cold.

Periodontal test

In the absence of treatment, cracks lead to pulp necrosis and periodontal inflammation. Bacteria grow along the endodontium and/or periodontium. The tests to be performed are then percussion, palpation, and periodontal probing. In the presence of local attachment loss, the presence of a crack should be considered.[43] However, the absence of attachment loss does not exclude the presence of a crack.

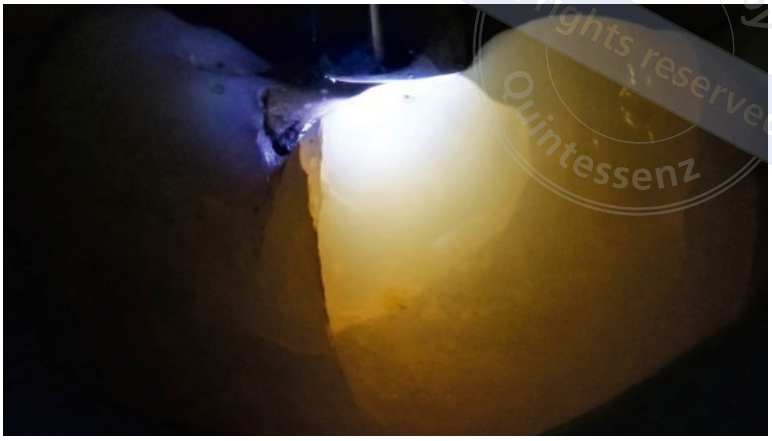

Fig 1 Light blocked by crack during transillumination.

Radiologic examinations

It is impossible to visualize cracks on radiographic images. Only fractures (the next stage of cracking) oriented in the radiograph axis will be detectable. The most frequent radiographic features of the fracture appear to be a 'halo' around the root, which is a periapical and periradicular radiolucency combined on one or both sides of the root.[44] CBCT can aid diagnosis.[45]

Coronal restoration removal

The presence of an unbounded restoration (amalgam, failed composite, gold, etc) is a risk factor because the enamel compression dome is broken, so the distribution of stresses through the dentin does not occur due to the blocking effect of the cavity. This causes a concentration of stresses between the peripheral rim enamel and the cavity wall.[18] The diagnosis is made by a careful visual examination of the cavity floor and the marginal ridges. Thereafter, the crack can be highlighted with methylene blue.[37]

Quantitative light-induced fluorescence

Quantitative light-induced fluorescence (QLF) is carried out with a digital camera that can not only detect enamel cracks but also quantify their depth. The images obtained are then analyzed by the QLF software. This method measures the loss of fluorescence of the enamel in an area in relation to the surrounding enamel[46] (Figs 2 and 3).

Quantitative percussion diagnostics

Quantitative percussion diagnostics (QPD) is based on the use of a percussion probe diagnostic instrument (Periometer; Perimetrics) that records and analyzes the percussion response of teeth.[47,48] This method achieved 96% specificity and 100%

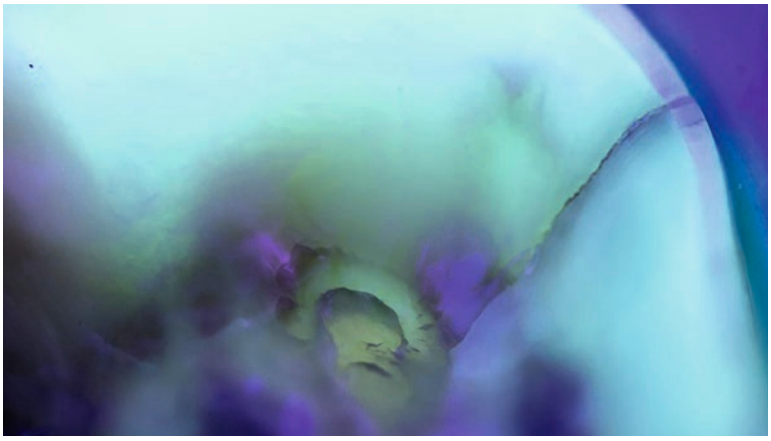

Fig 2 Fluorescence image of a cracked tooth.

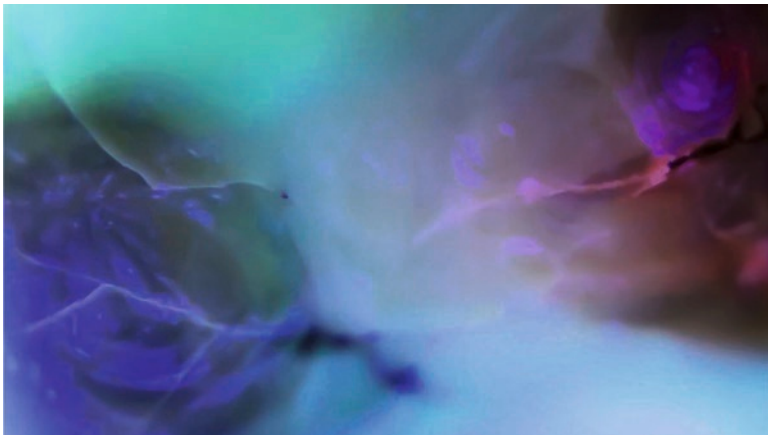

Fig 3 Fluorescence image of a cracked tooth.

sensitivity for detecting cracks and fractures in natural teeth.[49] It can be used either in the presence of a restoration or with no restoration; however, this instrument is not currently marketed.[50]

Therapeutic strategy

Limit bacterial infiltration

Biologically, pulp–dentin and periodontal biocompatibility are necessary. Pulp–dentin biocompatibility aims at protecting the pulpo-dentinal complex and ensuring sealing using adhesive systems to avoid bacterial contamination, percolation, and pulp irritation. Firstly, it involves removing the line of cracks to prevent further bacterial penetration.[3] Nevertheless, it may be practically impossible and also damaging to attempt to chase cracks extending to the pulp or along the root surface. Thus, crack opening and cleaning is mostly indicated at the peripheral aspect of the cavity and must be limited depending on the clinician's ability to restore it. Thereafter, sealing is ensured through immediate dentin sealing (IDS),[51] and eventually some small composite buildup and/or cervical margin elevation.[52] The hybrid layer inhibits the propagation of cracks due to its capacity for deformation and absorption of stresses such as at the DEJ.[53,54] Composite resin has a shock absorption effect by increasing cusp stiffness and redistributing occlusal loads away from the crack toward the axial walls and along the long tooth axis.[55,56] In addition, optimal bonding of restorations improves the strength of the ceramic and the prepared tissue and helps to stabilize weakened cusps.[57]

Limit crack propagation

One of the goals of treatment is to immobilize the fragments of the cracked tooth that move during loading. This can be achieved by splinting and covering the cusps. Splinting corresponds to cracked cusp girdling and cuspal coverage corresponds to cracked cusp overlap.

The residual walls can become too thin to support the stresses and can benefit from cuspal coverage. Cuspal overlap reinforces the tooth and prevents further potential decaying fractures.[58,59] This allows, in vitro, the maximum fracture stress to increase compared with a tooth reconstituted with an inlay, which would not have benefited from cuspal coverage.[60] The recommended care

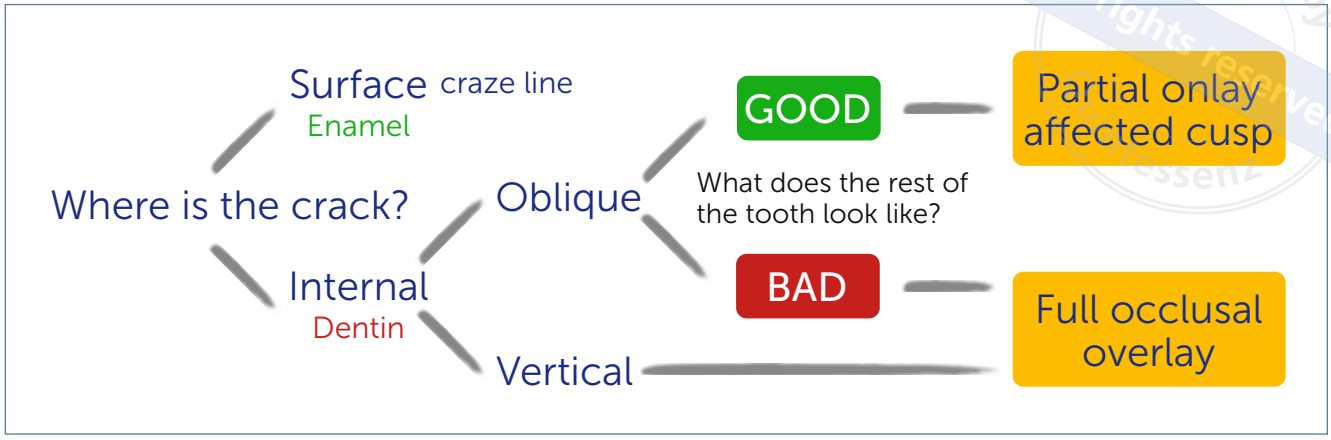

Fig 4 Decision map for cracked teeth.

in the case of cracked teeth includes cusp protection by cuspal coverage.[61,62]

The reduction and covering of cusps allows:

1. Reduction of flexion during loading and therefore fewer symptoms.[55,56,58,62]
2. Distribution of the occlusal load over the entire prepared tooth, which minimizes stress.[55]
3. Protection against crack propagation.[62,63]
4. Reduction of fracture risk.[64,65]
5. Protection of cusps from stresses toward the outside.[62]
6. Increase of fracture toughness of a restored tooth to the level of an intact tooth.[66]

In the absence of cuspal coverage, repeated loading of the restoration or the residual dental tissue may stress the adhesive layer, causing it to break.[55,67]

It is important to note that cuspal overlap reinforces the tooth but may also increase the risk of catastrophic failure.[68] Cusp overlapping also involves eliminating a significant amount of healthy enamel and dentin, which is why the diagnosis is imperative in order to minimize this as much as possible.

Splinting is based on the ferrule effect of the cracked cusp. As it minimizes the flexion of the compromised cusp, it not only alleviates pain symptoms during biting but also prevents crack growth, therefore reducing bacterial infiltration.[55] Splinting allows the prevention of the micromovement of the cracked parts during occlusal loading and thus avoids liquid movement in the tubules, which causes pain.[66] Splinting is provided by the ferrule effect. It allows strapping and improves the concentration of forces toward the inside of the tooth, thus reducing the spread of the crack. This ferrule effect may be provided by a chamfer or a bevel around the preparation. Splinting can be used first as a temporary treatment to assess whether or not it results in the disappearance of the symptoms.[67]

Splinting, associated with cuspal coverage, allows the orientation of stresses in the axis of the tooth and the axis of the crack

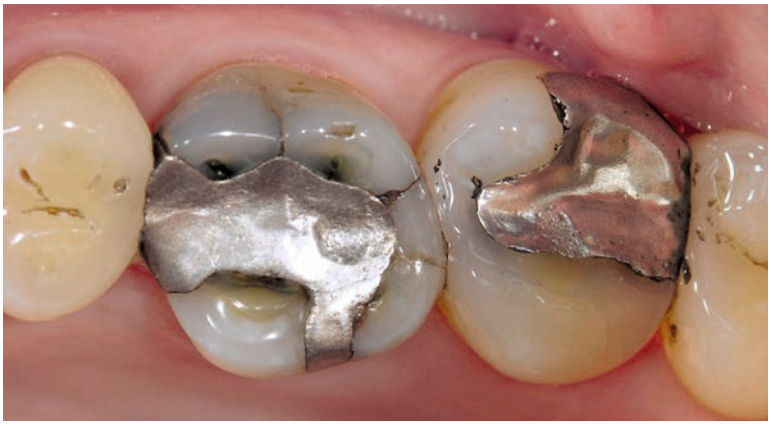

Fig 5 Initial situation.

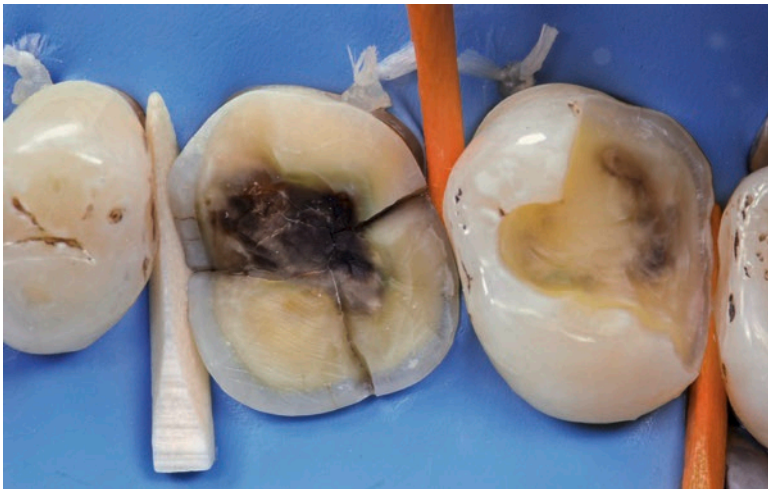

Fig 6 Situation after amalgam restoration removal and preparation for the cuspal coverage.

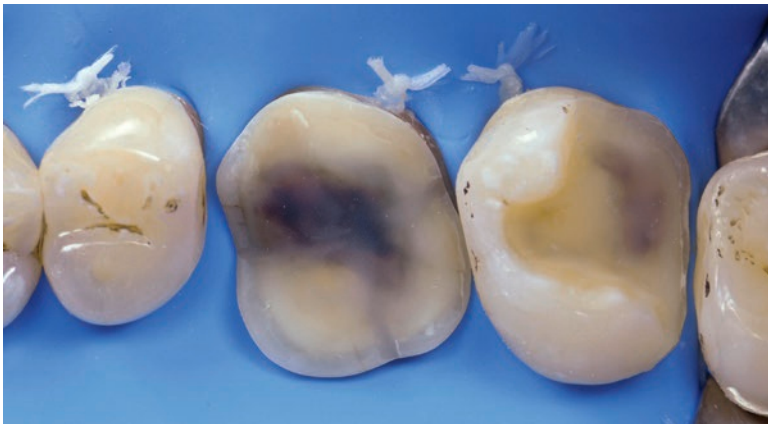

Fig 7 Preparations after the immediate dentin sealing (IDS) procedure.

'closure,' with an optimized load distribution. This is because it restores an adhesive compression dome, which allows the transferral of vertical occlusal loads to horizontal stress in the dentin.[69]

In view of the above information, a decision map can be devised (Fig 4). Bonding is mandatory to limit bacterial infiltration, and cuspal coverage is mandatory to limit crack propagation and change the range and direction of the occlusal load. The extent of this cuspal coverage will be decided by the depth and direction of the crack: a vertical crack increases the risk of nonrestorable fracture due to its depth, while an oblique crack can be completely removed. Cuspal coverage is also dependent on the quantity of residual dental tissue, particularly regarding the thickness and quality of the residual walls and the depth of the main cavity, the loss of one or both marginal ridges, the position of the residual cusps (because guide cusps are more at risk of fracture) or the presence or not of peripheral enamel.

A clinical example

As an example, the clinical photographs on the left illustrate the clinical approach. The initial situation (Fig 5) showed two teeth reconstituted by amalgam fillings that needed to be replaced. The amalgam on tooth 17 encompassed the distal marginal ridge and distobuccal cusp, while the amalgam on tooth 16 encompassed the mesial marginal ridge, presenting a palatal extension that did not overlap a cusp. On the other hand, tooth 17 did not show cracks in this view, while tooth 16 showed a large distal crack as well as a vestibular crack.

After restoration removal (Fig 6), tooth 17 showed no cracks, and cuspal coverage was deemed unnecessary on any cusps other than the distobuccal one, while tooth 16 presented three deep vertical cracks associated with the loss of two marginal

ridges and low residual tissue thickness. All this indicated a complete cuspal coverage on tooth 16. Bonding (Fig 7) using IDS was performed with a view to stopping bacterial infiltration and reducing postoperative sensitivity prior to restoration bonding.

Clinical protocol

The clinical case presented here consists of amalgam replacements on teeth 35 and 36 in the context of cracked tooth syndrome on tooth 36. Registration of occlusal contacts and rubber dam placement are prerequisites for therapeutic management (Fig 8). A quick analysis of the occlusal wear surfaces exhibited the different directions of stress that had been loaded on the two teeth over the years. Figure 9 shows the compressive (red) and tensile (blue) stresses. On closer examination, multiple occlusal cracks related to the occlusal load were revealed (Fig 10). The presence of a mesiodistal crack on tooth 36 was particularly evident. Tensile forces during chewing serve to open such cracks. This confirmed our diagnosis of cracked tooth syndrome. The initial radiograph (Fig 11) confirmed the small size of the initial cavities and their restorations. Fissures and cracks are usually undetectable on radiographs. The challenge was to minimize tissue loss by allowing previous measures to limit bacterial infiltration and crack propagation.

After amalgam removal, secondary decays were discovered, increasing the volume of the initial cavities and further undermining the mechanical resistance of the remaining structures (Fig 12). Also, a large part of the enamel was no longer supported, and the peripheral enamel walls were very thin. Fissures extended on the floor and walls of the cavities. Besides the tissue loss, occlusal cavities in a posterior tooth had caused it to flex under compressive and tensile loads on the external and

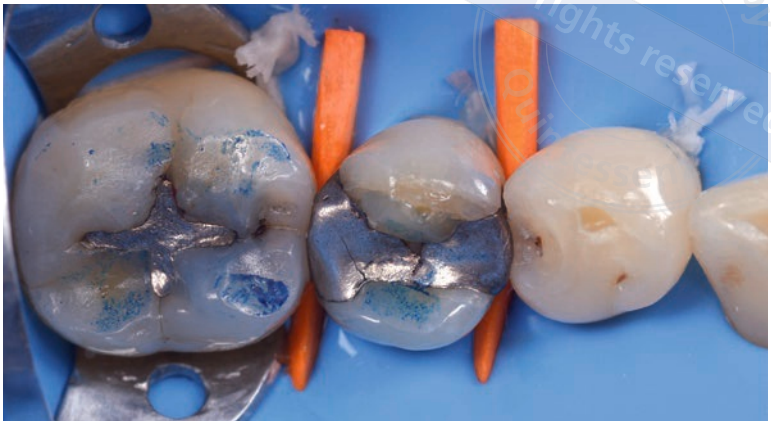

Fig 8 Initial situation.

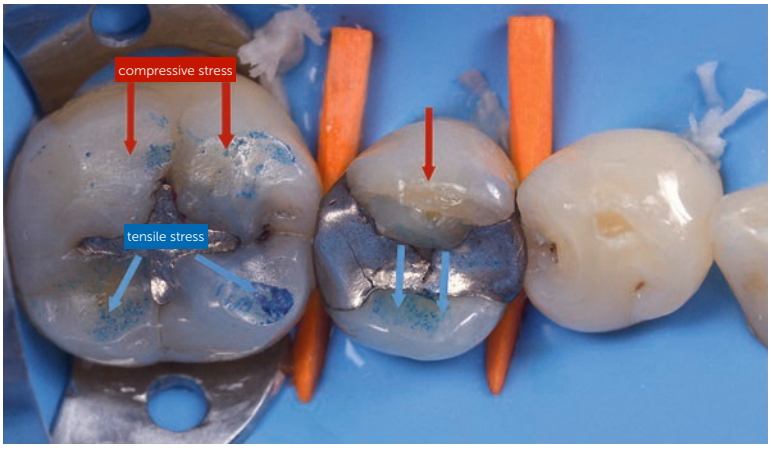

Fig 9 Analysis of the occlusal wear surfaces.

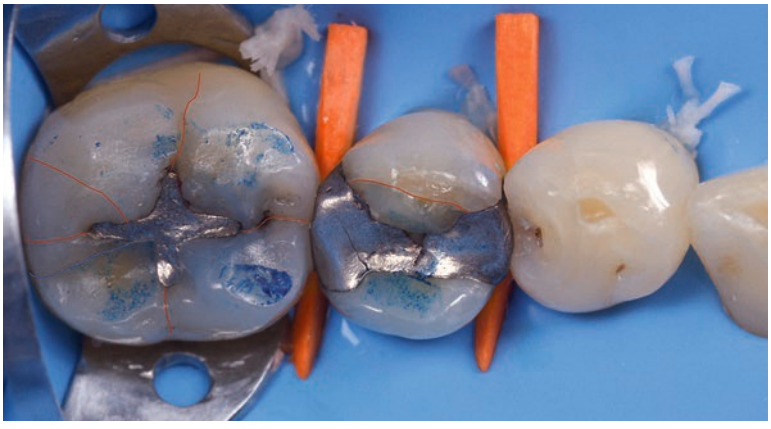

Fig 10 Highlighting of occlusal cracks related to the occlusal load.

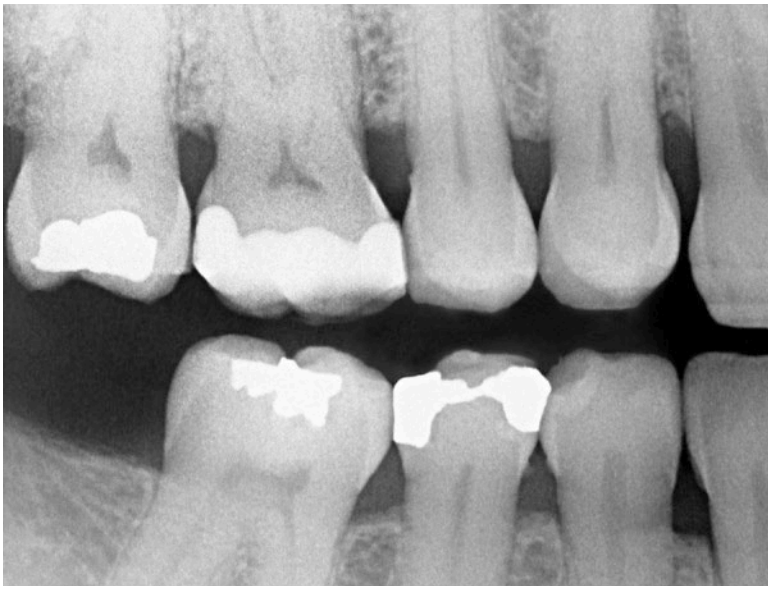

Fig 11 Initial radiograph.

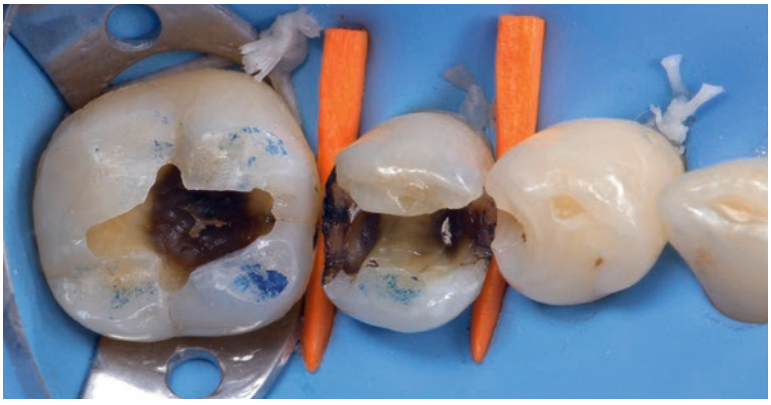

Fig 12 Situation after amalgam removal.

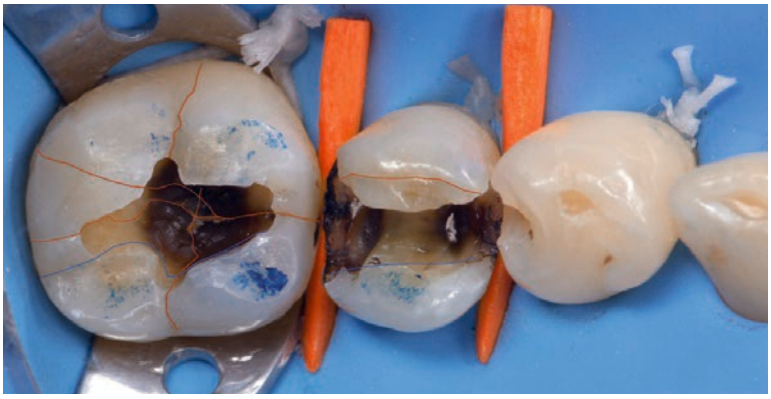

Fig 13 Highlighting of the crack extensions on the floor and walls of the cavities related to the occlusal load.

internal cusp planes, to the extent that the distortion in the tooth resulted in structural failure of the peripheral rim (Fig 13).

For tooth 35, the loss of the two marginal ridges, the thinness of the enamel walls, the presence of cracks on the vestibular cusp, and the risk factor of leaving a guiding cusp alone determined a full cuspal coverage. The therapeutic choice was more complex for tooth 36, but the thinness of the residual enamel walls associated with the presence of a mesiodistal crack indicated the loss of mechanical capacity of the two marginal ridges and the resultant considerably increased fracture risk. Also, the occlusal analysis confirmed the presence of significant mechanical stress on the guide cusps, which is a cyclic fatigue risk factor. Finally, the presence of symptoms encouraged the achievement of a complete cuspal coverage as minimally invasively as possible. As cracks cannot get remineralized or repaired, cuspal coverage is then indicated to change the direction of the occlusal forces so as to avoid the extension of the cracks. The extension of the cuspal coverage is indicated by the presence of oblique and vertical cracks, especially here, at the level of the mesiodistal cracks (Fig 14). The etiology of the cracks is always established based on assumption. Nevertheless, the clearer the fracture lines appear clinically, the more vertical the cracks are, and the more likely they are due to compressive stress, which tends to ovalize the whole structure of the tooth (roots included). On the other hand, the shallower the fracture lines appear, the more oblique the cracks will be, and the more likely they are related to tensile stress, which tends to deflect the cusps outside of the occlusal perimeter of the tooth (Fig 15).

After final cavity cleaning, the entire dentin surface was immediately sealed using a conventional adhesive procedure and composite buildup (Fig 16). The final views

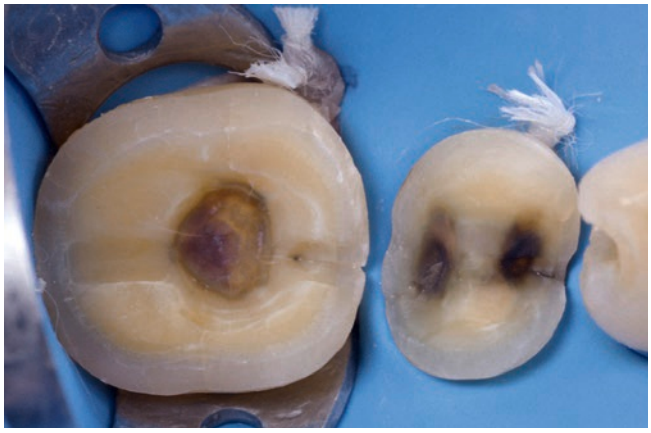

Fig 14 Situation after preparation for the cuspal coverage.

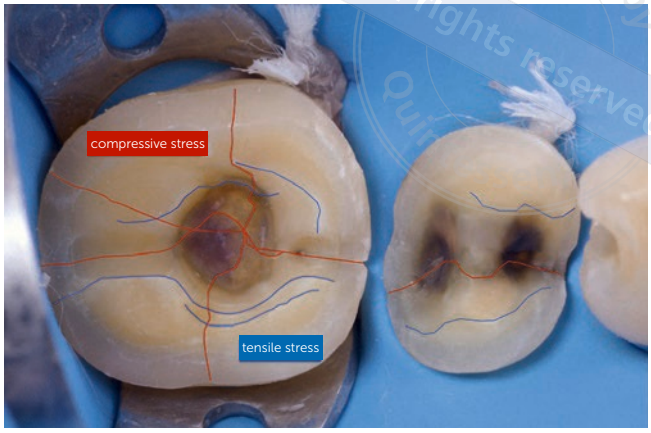

Fig 15 Highlighting of the different cracks in various colors according to their supposed etiologies.

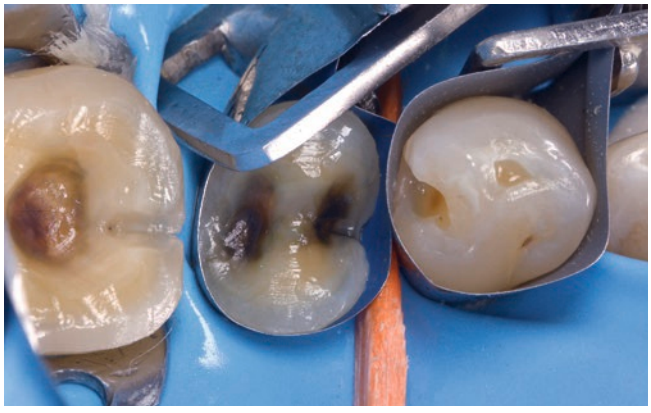

Fig 16 Situation during the IDS sealing procedure.

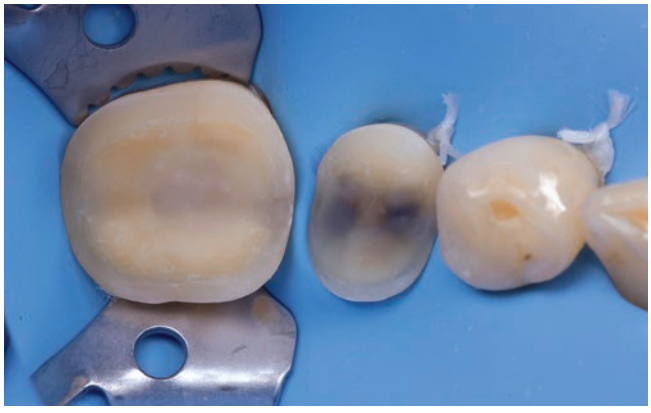

Fig 17 Final view of the preparations after the IDS procedure.

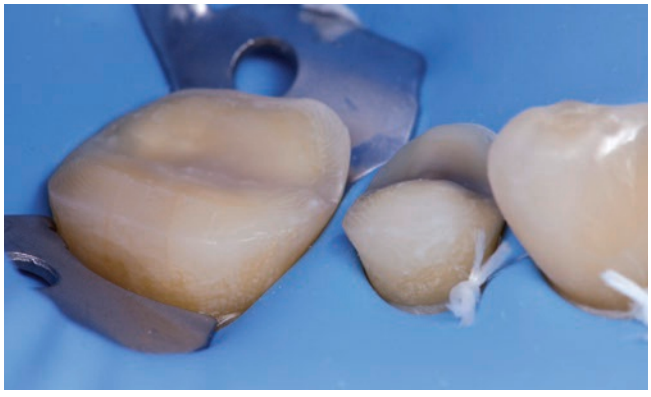

Fig 18 Marginal limit of the preparations.

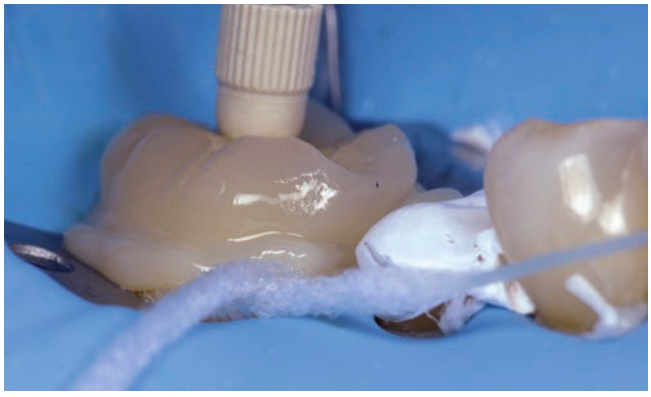

Fig 19 Adhesive cementation of a monolithic ceramic overlay on tooth 36.

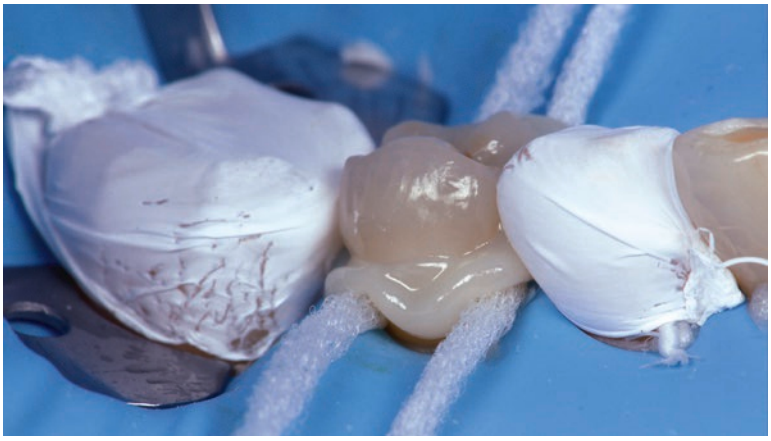

Fig 20 Adhesive cementation of a monolithic ceramic overlay on tooth 35.

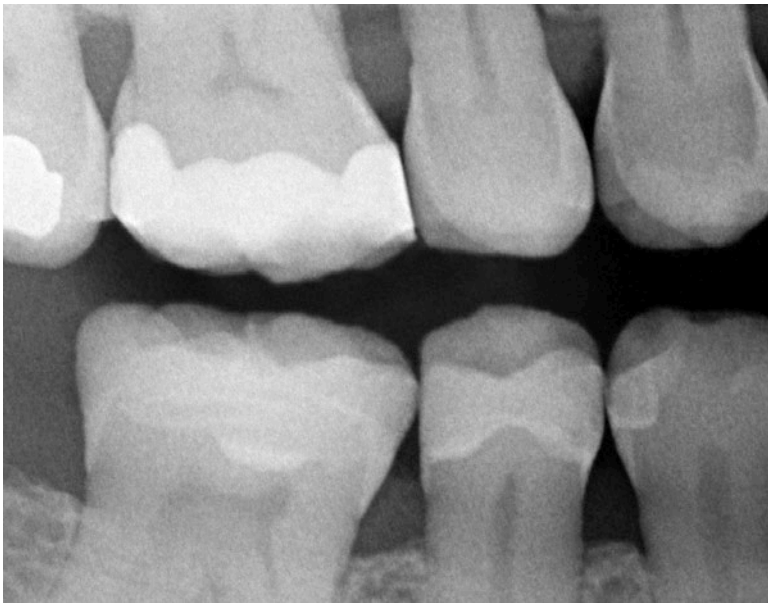

Fig 21 Immediate postoperative control radiograph.

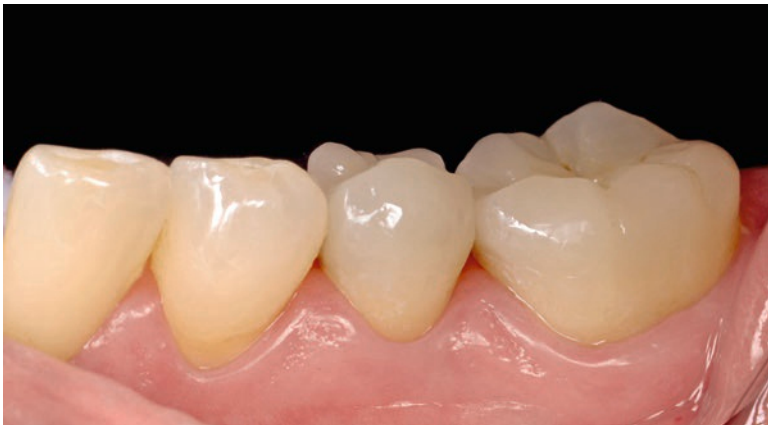

Fig 22 3-year follow-up.

of the preparations after the IDS procedure assume the redistribution of the occlusal loads (Fig 17). Furthermore, buccal and lingual cuspal coverage was performed with the design of a 15- to 30-degree bevel at the enamel margin to orientate the occlusal load to the central tooth area (Fig 18). Adhesive cementation was performed using warm composite (Estelite Sigma Quick; Tokuyama Dental) and the overlays were realized in monolithic lithium disilicate-enriched glass-ceramic (IPS e.max Press; Ivoclar Vivadent) (Figs 19 and 20).

The immediate postoperative control radiograph (Fig 21) confirmed the quality of the bonded joint and the absence of excess adhesive cement. Besides, it is interesting to note the very thinness of the overlay on tooth 36 to be the least invasive possible, despite the deterioration caused by the cuspal coverage. Regular follow-up (Fig 22) allowed for the maintenance and absence of aging of the restorations and confirmed the disappearance of the symptoms.

Conclusion

Treating cracked teeth still remains very challenging in daily practice. Therefore, a careful diagnosis associated with a clear understanding of the biomechanical behavior of the natural teeth is the best basis from which to draw guidelines for cracked tooth restorations.

Nevertheless, rebuilding and reinforcing the remaining structure of cracked teeth often leads to certain clinical frustrations because mineralized dental tissue cannot heal by itself and, to date, be remineralized or repaired *ad integrum*. Thus, changing the range and direction of occlusal loads is often the best treatment option to prevent the extension of cracks, which could possibly lead to the loss of the tooth. In such cases, adhesive dentistry is not necessarily minimally invasive, but at least it provides an

opportunity to preserve as much sound cervical tissue as possible and avoid the further tooth preparation that would be required for other treatment options, for instance, full crowns.

Ongoing investigations in the field of biomaterials as well as future research into the biologic repair processes will, without doubt, increase the therapeutic range available to treat and repair cracked teeth.

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