Etiology and diagnosis of dental caries

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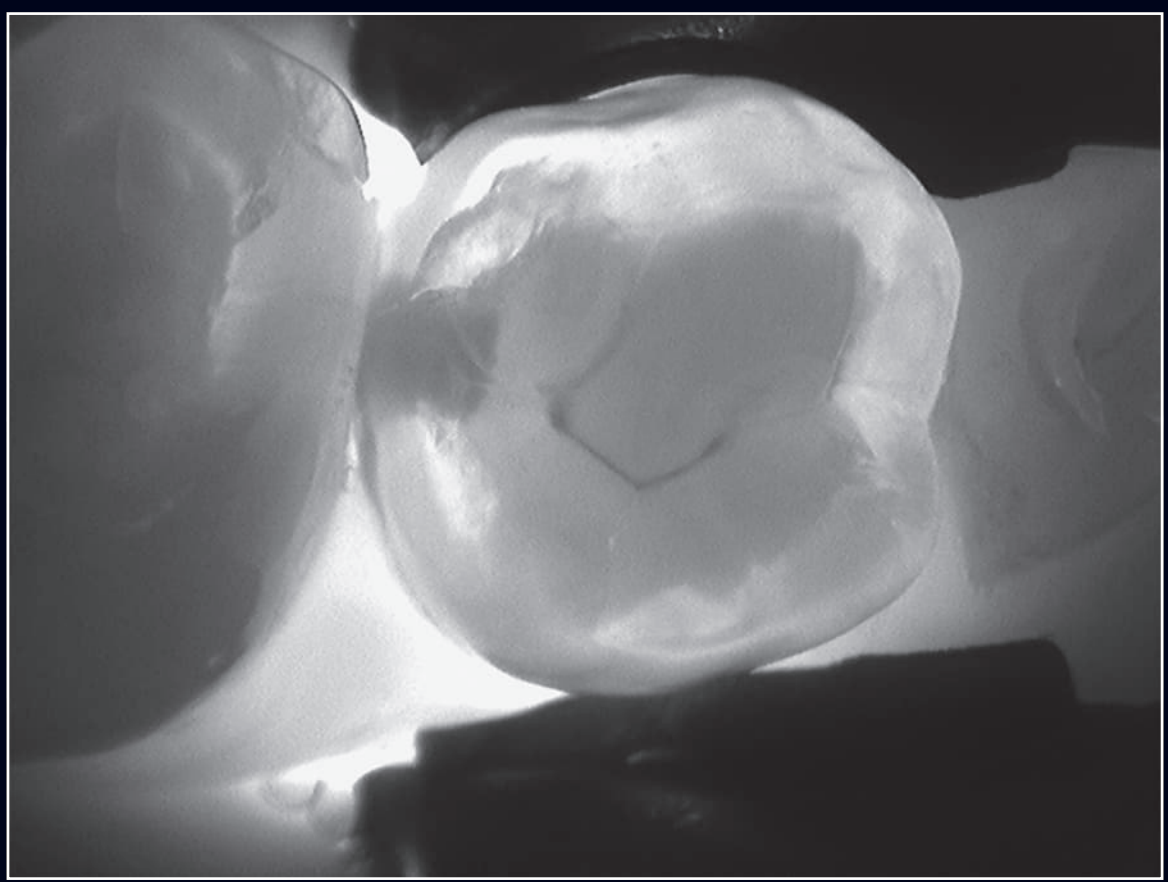
Federico Emiliani, Lorenzo Margarone, Roberto Turrini



Cariology: introduction

Before delving into the study of cariology, many dentists and dental hygienists, believed all was known about "caries," especially its etiology and its status as the primary disease. During our university studies and early postgraduate years, we were not as familiar with cariology, a branch of dentistry, as we are today. This is similar to other well-known branches, such as prosthodontics and periodontology. For us, dental caries was primarily linked to conservative, endodontic, or prosthetic treatments, and we were not accustomed to considering it as a separate field of dentistry.

Frequently interacting with dentists and dental hygienists, we have come to realize that the perspective on caries that we held years ago is still widely prevalent today. It is somewhat peculiar that we, as clinicians, use the term “caries” to refer to both the disease and its clinical manifestation, namely the formation of cavities in teeth that require our intervention. This distinction is not made in any other medical condition.

Patients also clearly experience this confusion. Frequently, patients come to our offices seeking treatment for a cavity, which involves restoring a tooth that exhibits varying degrees of decay. In most cases, according to the patients, the restoration itself is considered the treatment for the pathology. This statement is not entirely accurate because while restoration is an important part of the process required to control the disease, it alone does not constitute the "cure" for a cavity.

As dentists, our objective is to deliver top-quality conservative and prosthetic restorations while keeping abreast of advancements in modern adhesive procedures to provide the most effective rehabilitative therapies. In the past, our efforts were solely focused on developing less invasive, faster, and more aesthetically pleasing restorative techniques using the highest quality materials. Over time, we have learned one of the fundamental principles of cariology: **physically removing a carious lesion and filling it does not eliminate the bacteria, nor does it prevent the disease from progressing in the rest of the oral cavity and at the margins of the restoration.**[1] Caries is a transmissible bacterial disease. Therefore, relying solely on drilling to remove contaminated dental tissue without implementing additional strategies to eradicate the underlying cause is a mistake.

Have you ever diagnosed a recurrent carious lesion beneath a restoration and witnessed the patient's surprise upon learning that the tooth could still undergo a similar process? Have you ever wondered why some patients, despite regularly attending periodic clinical check-ups, continue to develop cavities, while others do not suffer from them at all? What strategies can we employ to effectively manage caries as a disease and prevent its recurrence, rather than just relying on conservative or prosthetic restorations? Is there a clinical protocol for defining the stages of severity of dental caries and implementing prevention measures?

Several years ago, we embarked on our exploration of cariology by posing important questions to ourselves. We soon realized that cariology is a rich field of study, filled with concepts and protocols that require daily application for our patients. We have underestimated its significance for far too long. We also discovered another crucial aspect: the fundamental role of dental hygienists, without whom it would be impossible to effectively provide education and prevent tooth decay. In this book, we will repeatedly emphasize the significance of collaboration between dental hygienists and dentists. We experience this daily in our clinical practice and have realized that teamwork is a crucial element that should never be absent in a contemporary dental practice.

In an effort to address the previous questions and explore modern protocols and methods for preventing tooth decay, we embarked on a journey to study and expand our understanding of cariology. For years, we have been following the guidelines recommended by scientific research, the World Health Organization (WHO), and the Italian Ministry of Health. We have successfully implemented a practical method based on existing literature that can be applied in any dental practice. The ultimate goal is to become expert in the treatment and prevention of caries, a disease in which dentists and dental hygienists are considered the foremost authorities, unless proven otherwise. It is, therefore, our duty to understand the causes of these conditions and to educate patients daily about adopting a healthy lifestyle and specific habits to combat them. As we will see later, these are crucial for winning the battle against this disease.

This text aims to serve as a practical clinical manual, enabling dentists and dental hygienists to improve their understanding of cariology and apply it in their professional practice on a daily basis. It is based on our experiences and internationally recognized guidelines found in the literature.

Etiology of dental caries

CARIES BALANCE

The 2015 Global Burden of Disease study identified dental caries as the most prevalent disease worldwide, despite being a preventable condition. It affects 60 to 90% of children and the majority of adults globally.[2]
CAMBRA, an acronym for "Caries Management By Risk Assessment," encompasses a set of interventions aimed at diagnosing an individual's risk level of developing caries disease and reducing its effects.
The CAMBRA method is based on the understanding that dental caries is a disease caused by a complex biofilm, rather than any single pathogen. This biofilm dynamically interacts with its environment, altering the local chemistry of teeth and saliva. This is in stark contrast to the traditional medical model, which focuses on a direct "pathogen-disease" relationship. Instead of solely focusing on eliminating a specific pathogen, caries management should be based on identifying the factors that cause the disease to manifest and to determine corrective and preventive actions.[3]
The CAMBRA method was the focus of two distinct consensus conferences organized in California by John Featherstone in 2003 and 2007, followed by subsequent updates. Featherstone proposed a "balanced" model to visually illustrate the multifactorial etiology of the disease **(Fig. 1.1)**.
Featherstone's "Caries Balance" aims to illustrate the determining factors of dental caries, based on the dynamic interaction of the biofilm with the oral environment. The local environment plays a crucial role in determining the behavior of the biofilm at a specific site and whether the disease is severe enough to cause demineralization or visible changes on the enamel surface.
Featherstone's proposed model considers the presence of caries disease on one side of the balance and the absence of caries on the other. According to this model, the highest likelihood of developing the disease is determined by the greater weight of risk factors compared to protective factors. This image effec-

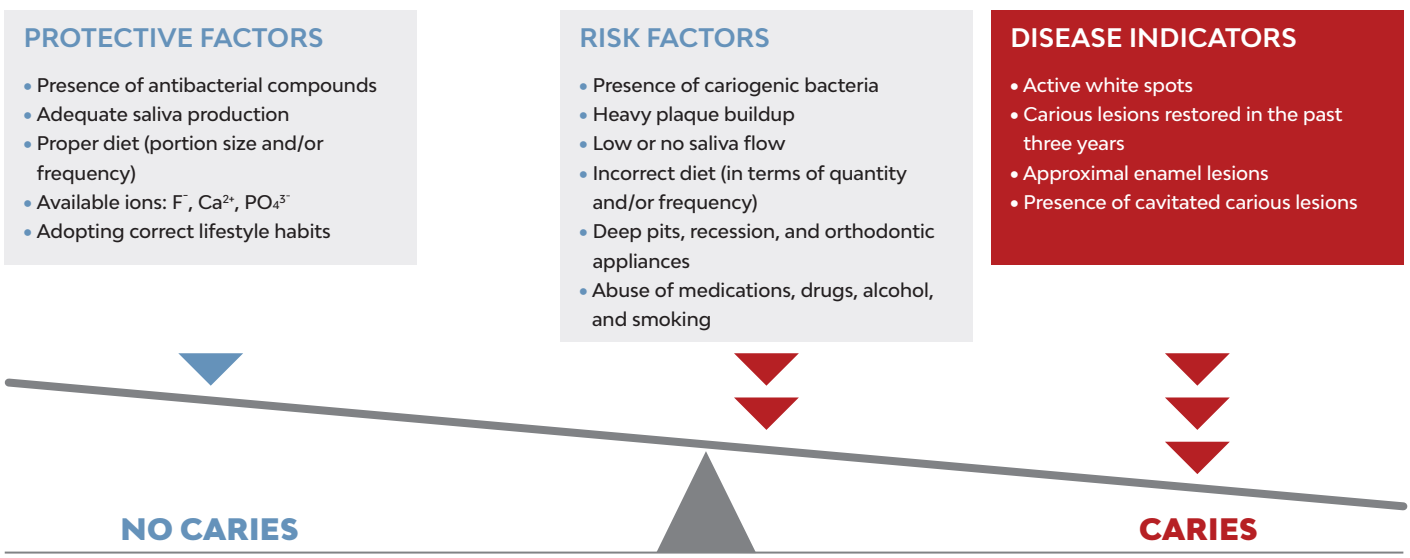


Figure 1.1 Caries balance. The graphical representation illustrates caries disease as a "balance," where various factors can influence the presence or absence of the disease, defining the individual caries balance of each patient. *J.D.B. Featherstone.*[1]

tively communicates with patients and serves as the first step in explaining that preventing caries disease requires consideration of various factors.
In cases where a patient presents with recurring carious lesions, it is the professional's responsibility to explain the importance of identifying the cause of this condition. It is essential to identify the presence of risk factors and disease indicators in the patient's personal "cariological balance" in order to counteract them by enhancing the protective factors.

DISEASE INDICATORS

The literature offers various definitions of caries disease indicators and risk factors. Disease indicators (Table 1.1) are clinical signs that indicate the current or past presence of caries. They are not the direct cause of disease, but they are strong indicators of future health issues.[4,5] Moreover, they are significant predictors of disease progression unless preventive measures are taken.
The presence of any one of these four indicators automatically places the patient at a high risk of tooth decay unless a therapeutic intervention is already underway. A carious lesion in a patient indicates the presence of high levels of cariogenic bacteria. Merely performing restorations does not significantly reduce the overall colonization of acidogenic bacteria in the mouth.[6,7] If there is also hyposalivation or xerostomia (dry mouth) along with one of these indicators, the risk of caries would be extreme.

Table 1.1 Disease indicators

■ White spots are visible on smooth enamel surfaces (known as white spot lesions)
■ Carious lesions have been restored in the last three years
■ Visible proximal enamel lesions are evident on the radiograph
■ Visible cavitation or radiolucent caries into the dentin can be observed on a radiograph

RISK FACTORS

Caries risk factors encompass biological and pathological elements that can contribute to the development of new carious lesions in the future or indicate the potential for existing lesions to progress. These pathological factors not only identify the imbalance but also provide guidance on how to correct it[4,5] (Table 1.2).
Whenever a patient with a history of caries or the presence of disease indicators seeks treatment, the clinician's objective should be to help them understand that restorative treatment, whether conservative or prosthetic, is necessary but not sufficient for resolving caries. Indeed, it should go hand in hand with the search for and elimination of the causes of the disease.
Caries is a disease and, as such, it should be diagnosed and treated. This concept can be explained to patients by drawing a parallel with another illness. Consider someone with the flu, exhibiting symptoms such as coughing,a sore throat, or a fever. Treating the flu involves more than just giving paracetamol to reduce fever; it requires identifying and eliminating the pathogen responsible for the illness. Paracetamol only provides symptomatic relief. Similarly, caries can be compared to the flu, and dental restoration to paracetamol.

Table 1.2 Risk factors

■ Presence of cariogenic bacteria such as *Streptococcus mutans* and Lattobacilli
■ Plaque on the surface of teeth
■ No or reduced saliva production
■ High consumption of fermentable carbohydrates (e.g., free sugars, carbonated drinks, etc.)
■ Frequent snacking between meals
■ Pits and deep fissures are present on the surface of the enamel
■ Receding gums
■ Orthodontic appliances
■ Abuse of medications, smoking, drugs and alcohol

Using an antibiotic to treat the flu is an example of causal therapy, similar to how addressing risk factors can help prevent caries.

PROTECTIVE FACTORS

Protective factors include biological or therapeutic elements that, together, can compensate for the pathological challenges posed by the caries risk factors discussed above. The more severe the risk factors for caries, the greater the intensity of the protective factors needed to maintain the patient's balance or to reverse the caries process (Table 1.3).

The industry is responding positively to the growing demand for products designed to mitigate these risk factors. Examples of protective factors include the consumption of xylitol, the proper use of fluoride toothpaste, and products containing calcium and phosphate.

The regular use of fluoride toothpaste has been shown to be one of the essential habits for maintaining oral hygiene at home and preventing the development of caries.

These protective factors can help maintain the patient's balance and enhance the remineralization processes, which are essential for the natural repair of early carious lesions.

Table 1.3 Protective factors

■ Adequate quantity and function of saliva
■ Presence of antibacterial compounds
■ Availability of fluoride and calcium/phosphate (as a complement to fluoride, not a replacement)[8]
■ Low consumption of fermentable carbohydrates
■ Effective lifestyle habits

Demineralization and remineralization cycles

Dental enamel is a highly mineralized, acellular tissue in which microscopic calcium phosphate crystals make up about 99% of its dry weight. The crystals form the mineral hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$, and are arranged in a repetitive pattern where calcium, phosphate, and hydroxide ions alternate in the crystalline structure. Dental tissues are not composed solely of hydroxyapatite; in fact, they contain other elements such as inorganic carbon (carbonate), magnesium, strontium, zinc, and fluoride. The hydroxyapatite structure is reportedly "doped" with other ions. These small quantities of ions can significantly alter the behavior of the structure. They can increase solubility, as in the case of carbonate, or enhance biocompatibility (strontium). Additionally, they can stabilize the molecule and intervene in the management of crystallization (magnesium), as well as confer antibacterial activity and resistance to demineralization (zinc and fluoride).[9]

The presence of carbonate increases the solubility of hydroxyapatite,[10] while the addition of fluoride has the opposite effect, reducing solubility.[11] However, there is a threshold for the amount of carbonate that can be incorporated into the enamel without compromising its structure. Some hydroxide ions are replaced by fluoride ions, and in this scenario, there is no limit to the extent of this process; if 100% substitution occurred, fluorapatite would be obtained.

Enamel apatite crystals are long and thin, approximately 50 nm wide across and up to 1,000 nm in length. They are tightly packed in a repetitive structure that forms the enamel prisms. Some crystals can penetrate the entire thickness of the enamel and connect with neighboring crystals at various points along their length.[12] The spaces between the crystals are filled with water (11% by volume) and organic material (2% by volume). Due to its high mineral content and minimal acellular matrix, the color, hardness, and other physical properties of enamel are similar to those of hydroxyapatite[13] (Fig. 1.2).

At different temperature levels, all minerals have a specific and well-known solubility in water at different

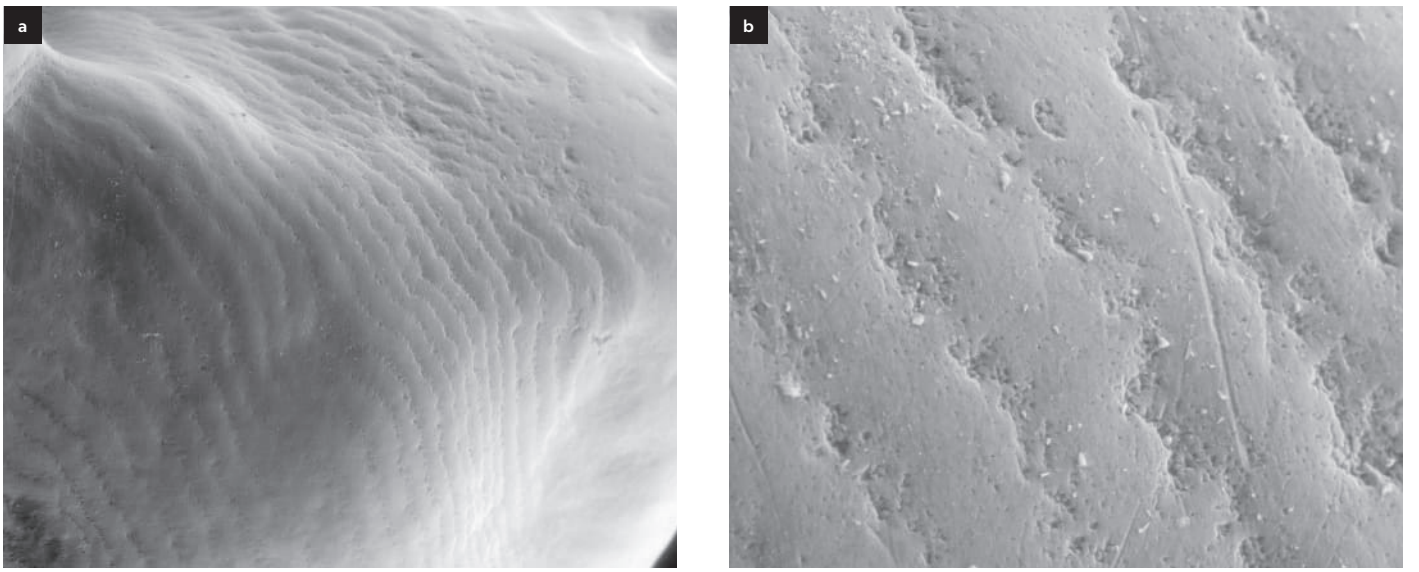


Figure 1.2 Dental enamel. In these two images, it is possible to observe the surface composition of the dental enamel. These are photographs taken using scanning electron microscopy of an intact crown from an extracted tooth. **(a)** 100x magnification. **(b)** 700x magnification. *Images were created in collaboration with the Department of Biomolecular Sciences at the "Enrico Mattei" Scientific Campus, University of Urbino "Carlo Bo."*

temperature levels. Dissolution in pure water occurs relatively quickly at first, then slows down as the concentration of ions in solution increases.[14] Eventually, dissolution stops as the solution becomes saturated with the mineral, although a gradual exchange of ions between the crystal and the solution persists. Under normal conditions, oral fluids are typically oversaturated with hydroxyapatite and fluorapatite, indicating a predisposition for the formation of these two minerals. This can lead to the development of apatite calculi and the remineralization of demineralized areas caused by carious lesions. When the pH of oral fluids (saliva and the liquid component of plaque) decreases, the supersaturation with hydroxyapatite also decreases, and is replaced by saturation below this pH value, with fluids becoming unsaturated towards the mineral. Since fluorapatite is less soluble than hydroxyapatite, the liquid component of plaque remains supersaturated with it. Under these conditions, demineralization processes begin, leading to the formation of carious lesions. The surface beneath hydroxyapatite dissolves, while fluorohydroxyapatite forms in the outermost layer of the enamel. In general, when the plaque fluid is more unsaturated with respect to hydroxyapatite, as is the case at low pH values, there is a greater tendency for the dissolution of enamel apatite.[15-17]

Dental remineralization, on the other hand, is the opposite process. It involves the movement of minerals from the surrounding environment, such as saliva and biofilm, into partially demineralized dental structures. This process can occur naturally or be induced by specific therapies.

Saliva is a fluid that is particularly rich in ionic precursors of hydroxyapatite. It plays a dual role in reducing the extent of demineralization through its internal buffer systems (bicarbonate, phosphate, and protein) and providing the hard tissues with the necessary minerals to restore initial demineralization situations. Indeed, hydroxyapatite (HA) has a critical pH of about 5.5. This means that if the pH of a solution is below this value, hydroxyapatite begins to dissolve into its ionic components, and the dental tissues start to lose substance. In this context, it is important to consider that the metabolism of a cariogenic biofilm can lower the pH of saliva to values below 4.0. These values are likely even lower at the interface with tooth surfaces, where the flow is

reduced and the biofilm's ability to maintain homeostasis is diminished.

In reality, saliva's ability to buffer acidic metabolites produced by biofilms, while also ensuring nutrient clearance, allows a pH level above 5.5 to be restored in less than an hour.

If fluoride ions (F^-) are present in the solution, they can bind to apatite, replacing a hydroxyl ion (OH) in hydroxyapatite (HA), thereby initiating the formation of fluorapatite (FA). FA is more resistant to acid attacks than HA, as its critical pH is 4.5. When a crystal of carbonated fluorohydroxyapatite dissolves and new precipitation occurs, fluoride tends to be incorporated while carbonate is lost. The fluoride solution has an overall effect of significantly reducing the amount of calcium that can be released from enamel in an acidic solution **(Fig. 1.3)**. This forms the scientific basis for the current concept, highlighting how low concentrations of fluoride in the environment surrounding teeth have a greater beneficial effect in reducing caries than high concentrations of fluoride incorporated into enamel.[15,16]

Dentin, composed of a smaller amount of mineral substance and deposited in a different manner, has a higher critical pH of about 6.2. This indicates that dentin is generally less resistant to acid action than enamel.

Dentin is composed of 70% minerals by weight (50% by volume) and 20% organic matrix by weight (50% by volume). The mineral component is hydroxyapatite, similar to that of enamel, but the individual crystals are smaller than those of enamel. The organic matrix, unlike enamel, consists of collagen, which forms the support structure of dentin and holds together the apatite crystals.[13] Dentin is the calcified product of odontoblasts lining the inner surface of the dentin within the perimeter of the outer pulp tissue. Each odontoblast has an extension within a dentinal tubule. The tubules extend through the entire thickness of the dentin from the pulp to the amelodentinal junction.

The progression of tooth decay in dentin differs from that in the overlying enamel because of dentin's structural differences. Dentin contains much less mineral and has microscopic tubules that allow acids to enter and minerals to exit. The amelodentinal junction presents the lowest resistance to caries attack and enables rapid lateral spread once the lesion has penetrated the enamel. Due to these characteristics, dentinal caries have a "V" shaped cross-section, with a wide base at the amelodentinal junction and the apex directed toward the pulp. Compared to enamel, caries processes advance more rapidly in dentin because it offers less resistance to acid attack due to its lower mineral content.[17]

The alternation between events that cause deminer-

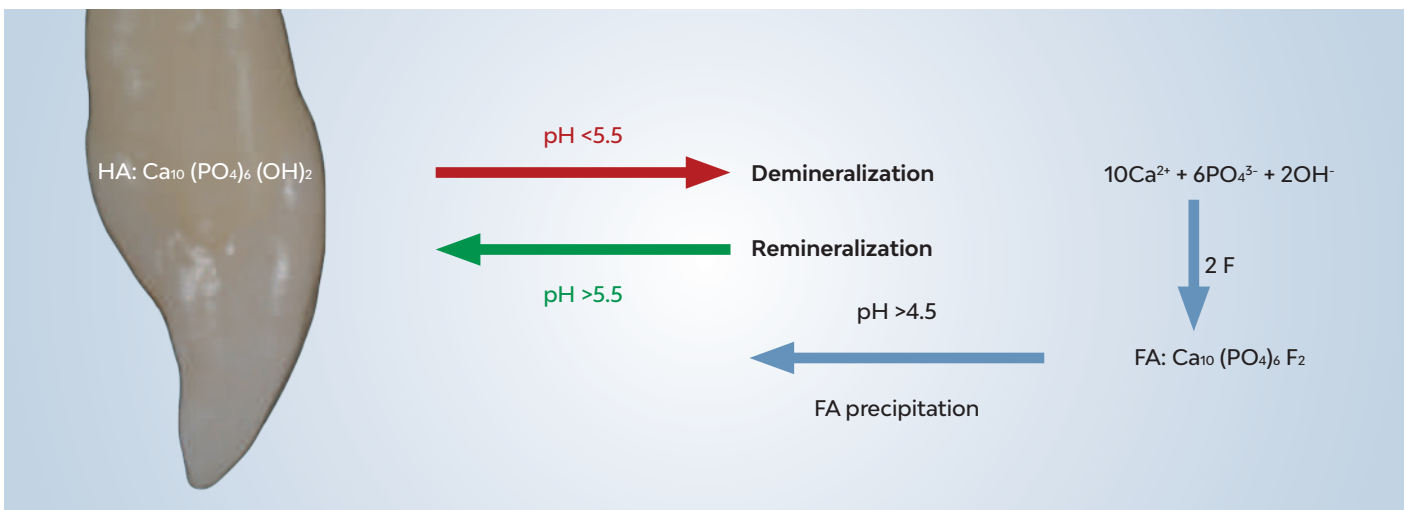


Figure 1.3 Demineralization and remineralization mechanisms. When the pH of the fluids surrounding the enamel (such as saliva and plaque) drops below the critical value of 5.5, hydroxyapatite (HA) is released into the solution, leading to demineralization. To facilitate the formation of new hydroxyapatite precipitation on the tooth surface, the pH of the surrounding fluids must rise above 5.5, thereby initiating the remineralization processes. In the presence of fluorapatite (FA), this process occurs when the pH is above 4.5.

alization and remineralization constitutes a cycle. This cycle is in dynamic equilibrium: under healthy conditions, there is no net negative balance, indicating that the hard tissues do not deplete their mineral content.
From this perspective, the duration of the saliva's "repair" action is crucial. If demineralization events occur frequently and intensely with equal mineral concentration, the tissue will undergo irreversible demineralization autonomously.[9]
Clinicians need to understand this mechanism of action and have the responsibility to effectively communicate it to patients, emphasizing the importance of maintaining a "non-cariogenic" lifestyle. This involves implementing a series of behaviors that are useful in ensuring a proper intake of fluoride and minerals, adequate saliva production, and controlling risk factors that may lead to more frequent and prolonged demineralization processes, thereby disrupting the dynamic equilibrium of the oral environment.

WHITE SPOT ETIOLOGY AND DIAGNOSIS

The presence of a visible white spot on the tooth surface is solely due to an enamel defect. In this clinical scenario, the dentin is never involved. Healthy enamel is the most mineralized tissue in the body. In the presence of a white spot, the mineral component is significantly reduced and replaced by organic fluids. For this reason, it is referred to as "enamel hypomineralization," as there is a change in the chemical composition of the substrate.
Optical laws state that when there is a difference in the refractive index between two phases, an interface will cause the incident light rays to deviate.
The refractive index (RI) of healthy enamel is equivalent to that of hydroxyapatite, which is the most significant component of the enamel (RI of healthy enamel = RI of hydroxyapatite = 1.62). In healthy enamel, there is no interface; the light ray passes through the substrate without altering its trajectory until it reflects at the enamel-dentin junction. In hypomineralized enamel, the light ray encounters multiple interfaces between organic fluids and the mineral phase, each with different refractive indices (1.33 and 1.62, respectively). At each interface, the light is diverted and reflected, creating a hyperluminous "optical labyrinth," which is perceived as white[18] (Fig. 1.4). The defect appears even whiter when dried with air because the organic fluids have been replaced by air, which has a lower refractive index (RI of air = 1 < RI of organic fluids = 1.33). So, increasing the difference in the refractive index compared to that of healthy enamel (RI hydroxyapatite = 1.62) makes the defect even more visible.
It is important to bear in mind that, unlike hypoplastic

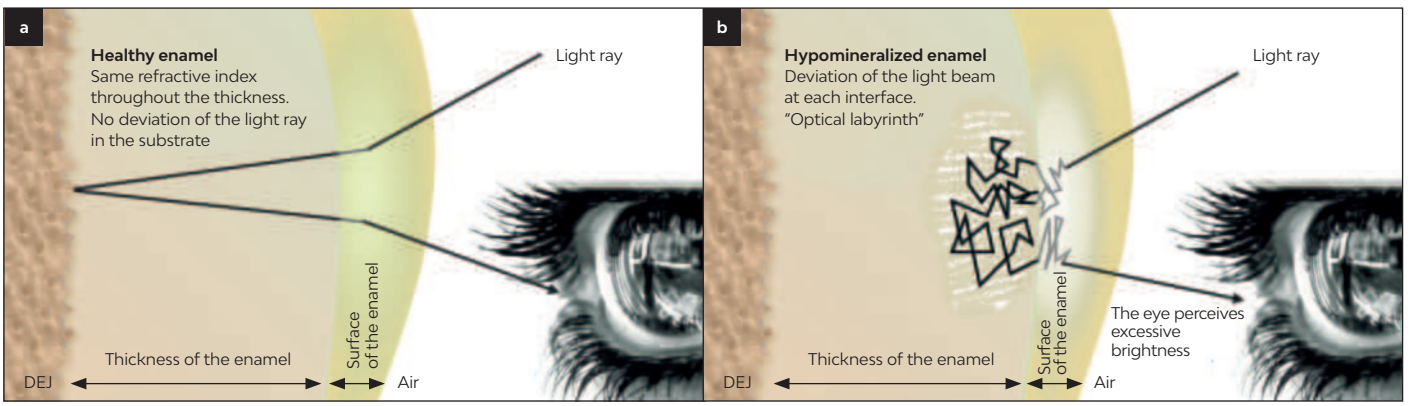


Figure 1.4 Influence of the laws of optics on the perception of color in dental materials. (a) A light ray is refracted at the tooth surface and then reflected by the dentin-enamel junction (DEJ), enabling the eye to perceive the color of the dental structure. (b) In hypomineralized enamel, the numerous variations in the refractive index caused by fluid/hydroxyapatite phase transitions create interfaces within the enamel substrate. At each interface, the ray is refracted and reflected. The hypomineralized lesion creates an "optical labyrinth" that appears white and opaque to the eye because of its excessive brightness. *Image from: Denis M, Atlan A, Vennat E et al. White defects on enamel: Diagnosis and Anatomopathology: Two essential factors for proper treatment (part 1). Int Orthod. 2013;11(2):139-65. Courtesy of Elsevier.*

enamel lesions, hypomineralization does not involve a loss of substrate volume. Thus, the presence of a white area indicates the presence of underlying hypomineralization.

The term "white spot" refers specifically to white lesions resulting from the demineralization processes that affect the enamel, and are therefore attributed to caries disease. There are also various other types of white lesions with extremely diverse causes, which will be discussed in **Chapter 12.**

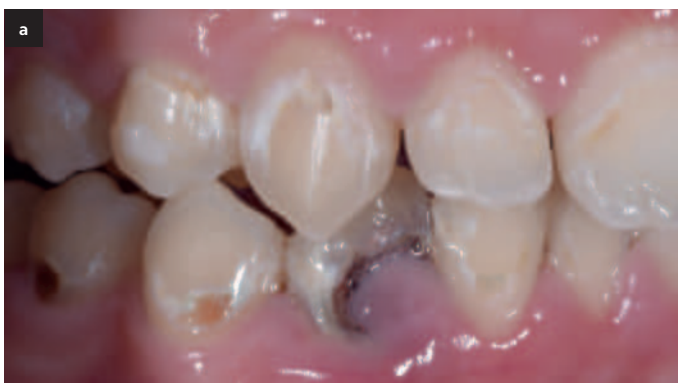
The initial stages of caries disease are characterized by hypomineralization without the formation of cavities. When this phenomenon becomes clinically visible, it is referred to as a "white spot" **(Fig. 1.5)**.

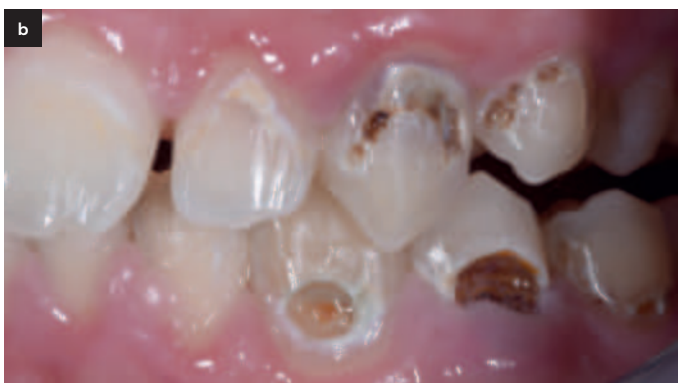
It is important to note that the white spot lesion is the main cause of white areas on the enamel surface, with a prevalence estimated at 24%.[19] This figure increases to 49.6% following orthodontic treatment, and the occurrence of at least one non-cavitated lesion before or after orthodontic treatment is 72.9%.[20] Treatment of these lesions is particularly important as the anterior teeth are more commonly affected **(Fig. 1.6)**.

Moreover, this prevalence is multifocal: it affects at least three teeth in 46% of patients not treated orthodontically, and rises to 77% in those who have received orthodontic treatment. These white lesions decrease by one-third at 3 months and by half at 6 months after the completion of orthodontic treatment. However, once they appear, they persist and are still visible 5 years after debonding.[21] The frequency and long-lasting nature of these lesions require dentists and dental hygienists to implement preventive measures and ensure early diagnosis, in order to minimize the necessary treatment.

The diagnosis of early carious lesions relies primarily on clinical observation. At this stage, the enamel is acellular, avascular, and nerve-free, and therefore, no associated symptoms are present. When these lesions reach the smooth surfaces of the anterior teeth, they are mainly found in sites where bacterial plaque preferentially accumulates: either in the cervical third or around brackets in the case of orthodontic treatment. They appear as opaque, rough, white chalky areas of enamel, with variable shapes and sizes and more or less defined contours.

During the initial caries process, alternating phases of demineralization and remineralization lead to the dis-





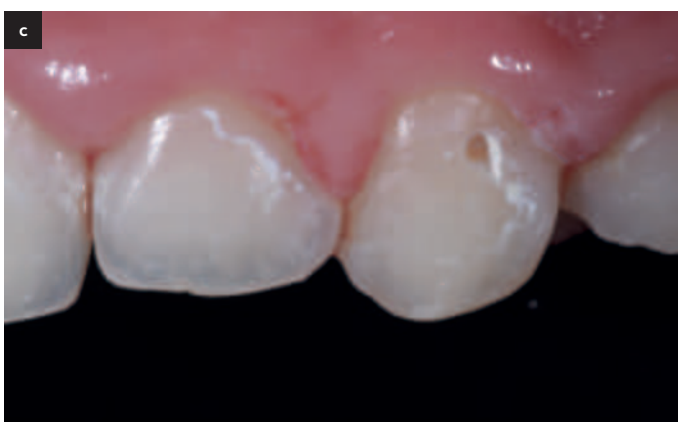


Figure 1.5 White spot lesions. In images (a) and (b), multiple white spot lesions can be observed on the patient's dental elements. In image (a), there are noticeable active white spots on nearly all tooth surfaces, along with accompanying brown stains on 1.4, 1.3, 1.1, and 4.2 and cavities with discoloration on 4.3, 4.4, and 4.5. Image (b) depicts a distribution of active white spots on nearly all teeth, indicating the simultaneous presence of brown stains on 2.1 and 2.2, as well as stains and cavitation on teeth 2.3, 2.4, 3.3, 3.4, and 3.5. In image (c) of another patient, an active white lesion is visible on the buccal surface of tooth 2.2 and a white spot on the buccal surface of 2.3, which has progressed to cavitation.

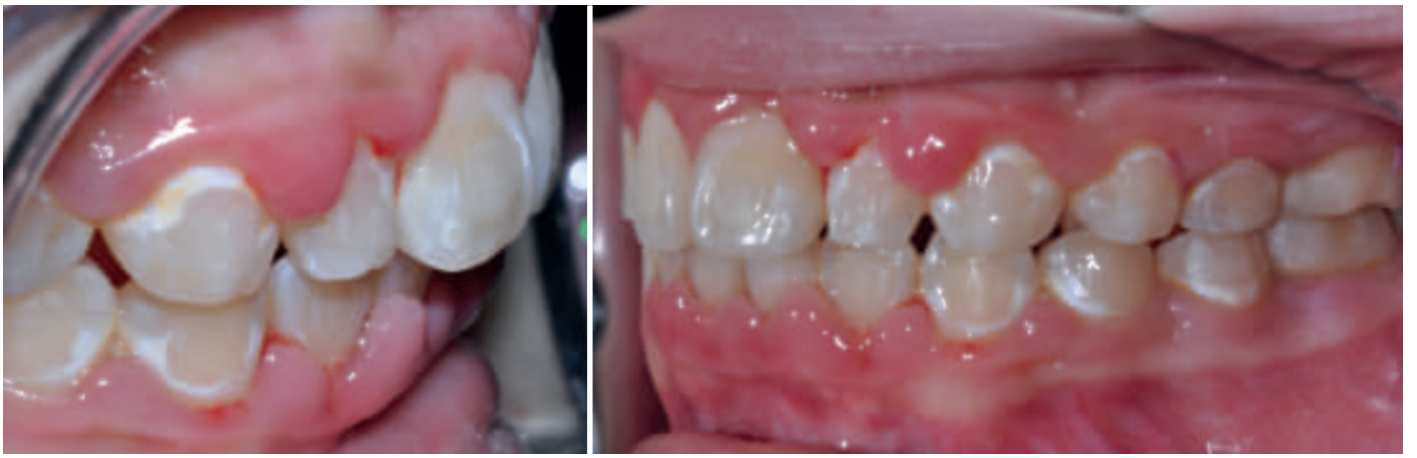


Figure 1.6 Post-orthodontic carious lesions. The images depict the condition of the tooth surfaces and the gingival inflammation at the conclusion of an orthodontic treatment that was halted due to the patient's lack of cooperation and inadequate oral hygiene. Numerous active white spots can be observed along the cervical margins of several teeth and near the areas where the orthodontic brackets were positioned. The lesions appear chalky white, opaque, and rough when probed, and the gingival tissue is inflamed. *Courtesy of Dr. Maria Moretti, Rome*.

solution and reprecipitation of minerals on the enamel surface. In these stages, a relatively intact "superficial layer" of minerals remains, beneath which the body of the carious lesion extends in a crescent or so-called "cone shape".[22] This histological arrangement has been thoroughly described by Silverstone[23] and reviewed by Kaqueler.[24]

Caries dissolves the crystalline structures at the weak points of the enamel,[25] longitudinally along the prisms and laterally along the Retzius lines, thus creating wide channels of communication that are directly accessible from the external environment. The hypomineralization beneath the white spot leads to the enlargement of the initial pores in the enamel,[26] resulting in a more sparse but still present crystalline structure. Its preservation, even when mineral dissolution has significantly progressed, is possible due to the binding of apatite crystals to an acid-tolerant protein sheath acquired during amelogenesis.[27] The lesion becomes clinically visible when the mineral deficit of the lesion body compared to healthy enamel reaches 10%. At an advanced stage, this can increase to over 40%.

The appearance of white spots can vary. One of the primary features of white spots is their opaque, chalky appearance and a rough surface that can be detected by gently passing an explorer probe over them. Frequently, they are also linked to inflammation in the gingival tissues near the lesion, particularly when they are situated along the cervical margin of the dental crowns. The presence of these tactile and visual characteristics enables us to classify these white spots as **active lesions**, indicating areas of enamel that are experiencing mineral substance loss.

When white spots are consistently white in color, but the enamel is smooth, shiny, and accompanied by healthy gingival tissues, we classify them as **inactive lesions**.

In Figure 1.7, we can see an example of both types. These are intraoral photographs of an 18-year-old patient with a high DMFT score and inadequate dietary and oral hygiene habits. White spots can be observed along the cervical margins of many dental elements. Most of them exhibit the typical characteristics of active lesions: opacity, chalky appearance, and roughness. Element 3.6 also demonstrates cavitation within the context of the white cervical lesion. However, if we take a moment to observe the appearance of some of these lesions, we can notice differences. An example is the white spot on the cervical portion of tooth 3.4: the enamel of this tooth appears different compared to the adjacent lesions just described. Visually, we can observe a non-chalky appearance and a smooth enamel surface covering the lesion, giving it a "vitrified" appearance. This is the typical appearance of inactive lesions, in which a repair process has occurred. It consists of mineral precipitation phenom-

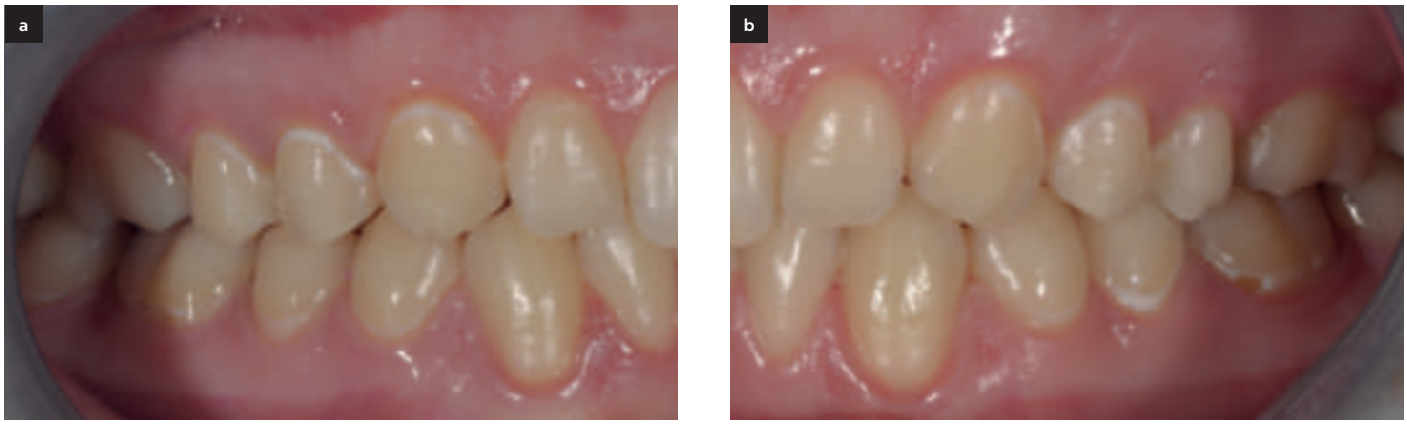


Figure 1.7 Cervical white spot lesions. (a) There are active white spots on the cervical margins of teeth 1.3, 1.4, 1.5, 4.4, 4.5, 4.6, and 4.7. Element 4.6 also exhibits simultaneous brown stains. (b) Active carious lesions are present on teeth 2.3, 2.4, and 3.5. Inactive carious lesion on tooth 3.4. Cavitated carious lesion on tooth 3.6.

ena that have facilitated the repair of the damage. The white body of the lesion is still visible, indicating that the repair processes have not affected the entire thickness of the enamel, but only the superficial layers. Figure 1.8 illustrates another example of the difference between an active and an inactive white spot.

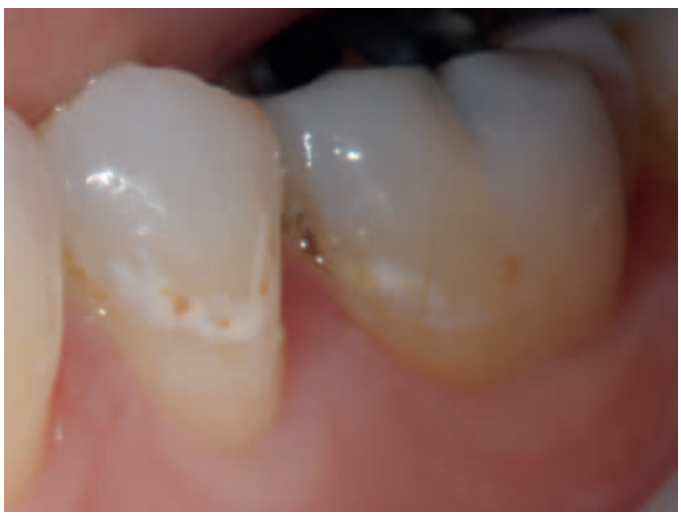


Figure 1.8 Active vs. inactive carious lesion. Both teeth exhibit white spots of caries on their buccal surface. Tooth 3.5 exhibits an active carious lesion. Tooth 3.6, on the other hand, exhibits an inactive white spot on its cervical-buccal surface. The remineralization process impacts the outer layers of the enamel. Therefore, despite the deeper lesion, the deposition of superficial minerals does not eliminate the lesion but does halt its progression.

WHITE SPOT TREATMENT STRATEGIES

Figure 1.9, extracted from Denis's 2013 research, depicts the distribution of hypomineralized tissue in the enamel of a dental sample, examined using three different types of equipment: an optical microscope (a), microradiography (b), and a transmission electron microscope (c).[18] It is evident that the white spot varies in depth, being superficial in some areas and deeper in others. However, its formation, induced by bacterial biofilm, still affects the outermost layers of the enamel. Given this localization, the recommended treatments include surface therapies with fluoride or casein phosphopeptides, or resin infiltration.

For minor lesions, which are barely visible after air drying of the tooth crown or show a limited area of opacity with only slight hypomineralization of the sub-superficial enamel layer, sufficient crystalline reprecipitation can be achieved to restore a normal mineral content of the enamel and an ideal aesthetic appearance. The recommended treatment protocol for early-stage buccal white spots (ICDAS 0-1) is as follows:

- Adequate instructions for home care
- Professional dental hygiene including the application of fluoridated compounds
- Use of toothpaste with 1,450 parts per million (ppm) fluoride 2-3 times a day
- Daily application (1/day) of calcium-phosphate compounds to lesions until they disappear

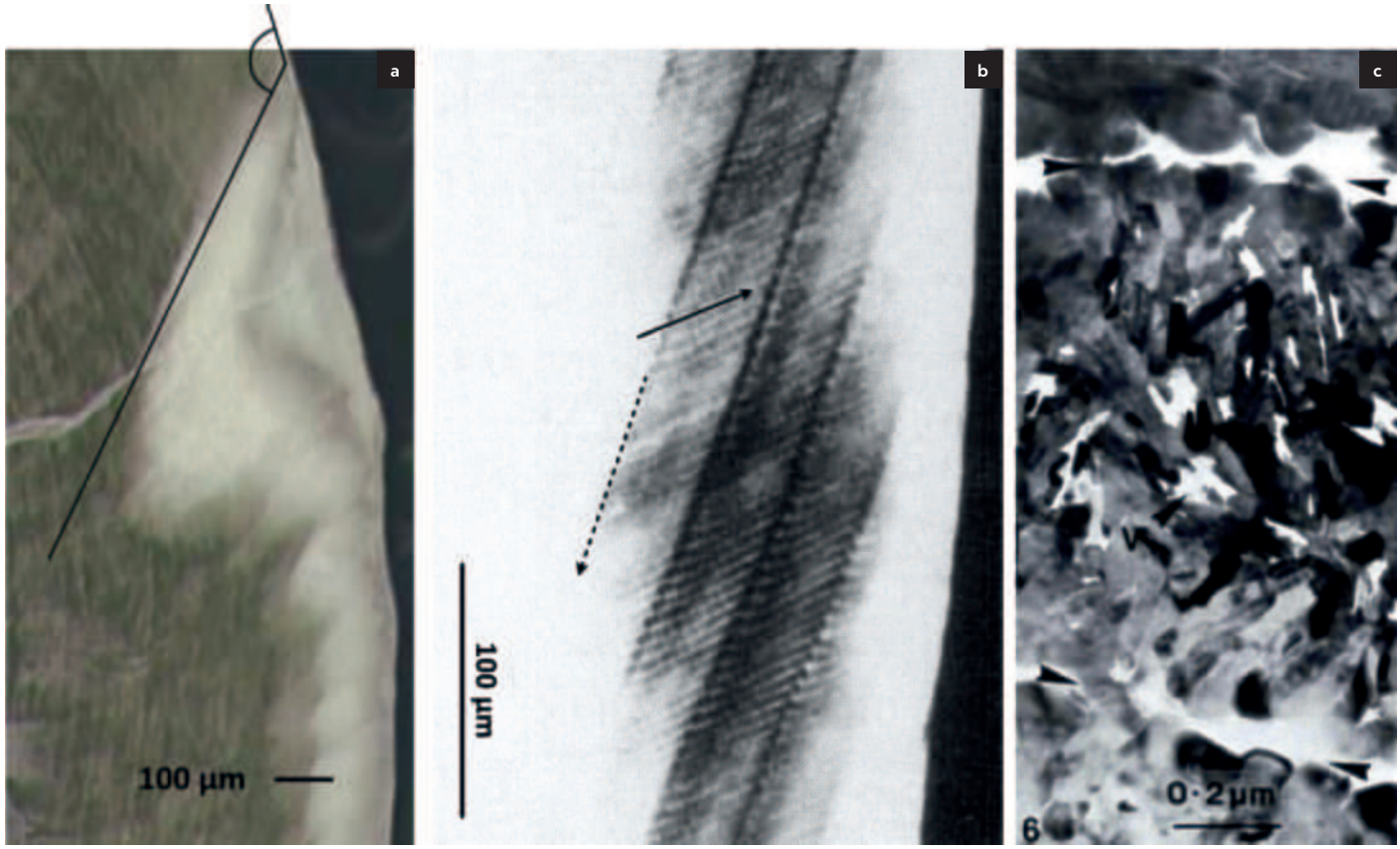


Figure 1.9 Anatomopathological properties of a white spot lesion. Cross-sectional view of a white spot lesion. (a) Observed under an optical microscope (Kielbassa, personal communication). The lesion extends from the surface of the tooth to the demineralization front with an obtuse angle. Hypomineralization of the white spot lesions remains in the sub-superficial layer without affecting the enamel volume. (b) Observed by microradiography (source: Pearce). The superficial layer remains relatively intact.
The dissolution of the crystals occurs along lines of weakness, such as enamel prisms (*solid arrow*) and Retzius lines (*dashed arrow*), which create wide communication channels. (c) Observed under a transmission electron microscope at 85,000x magnification (source: Palamara). The inter-prismatic micropores (*black arrows*) and intra-prismatic micropores are significantly enlarged. *Image from: Denis M, Atlan A, Vennat E et al. White defects on enamel: Diagnosis and Anatomopathology: Two essential factors for proper treatment (part 1). Int Orthod. 2013;11(2):139-65. Courtesy of Elsevier.*

- Control of diet, hydration, and risk factors
- Hygiene reminders and follow-up scheduled at regular intervals over time.

For deeper lesions with clearly visible outlines after drying with air and more intense opacity (ICDAS 2), remineralization treatments cannot guarantee the precipitation of minerals throughout the depth of the lesion. While these methods can guarantee bacterial control and strengthen the surface, the internal part of the lesion body, with its microporosity, may still be visible.
To create a smoother surface that is less conducive to bacterial adhesion and to remove the outer layer of hypomineralized tissue while preserving the enamel structure, it is recommended to polish the surface using fine-grain rubber polishers or burs, or by utilizing surface microabrasion products such as HCl-based pastes (see Chapter 12).
It is important to clearly understand the therapeutic, functional, and/or aesthetic objectives when treating white spots, depending on the tooth involved. For example, in the posterior areas, the primary objective is to halt the demineralization processes and promote remineralization in the affected zones. Conversely, in the anterior regions, a clinician should not only restore

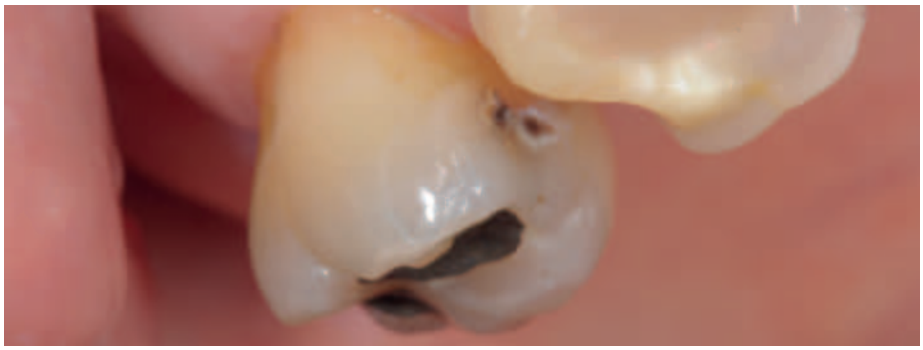
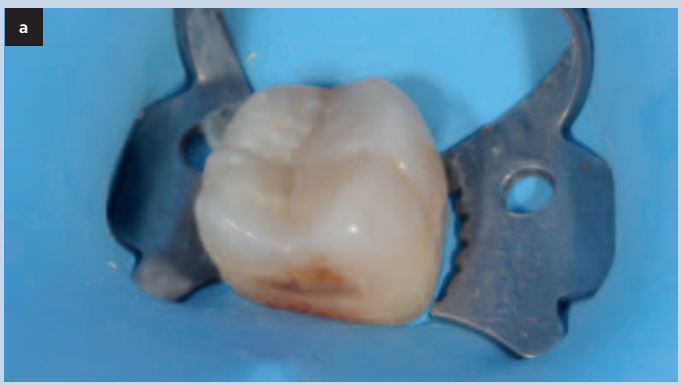


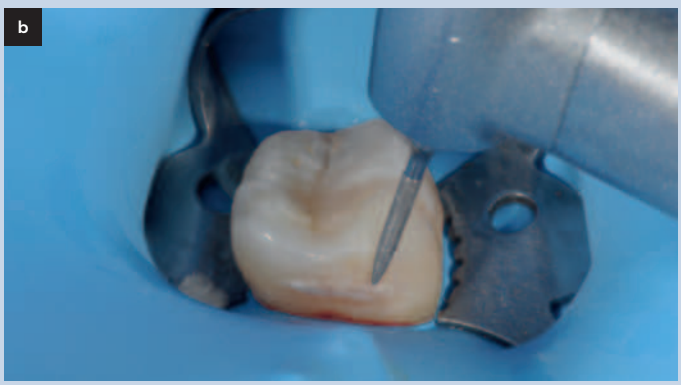
Figure 1.10 Approximal carious lesion. Active white spot lesion with concurrent brown stains. Opaque, chalky, and rough appearance.

proper mineral intake but also address the aesthetic aspect of the case. The progression and exacerbation of demineralization not only impacts the buccal areas but can also affect the approximal areas. White spots can persist over time regardless of perfect etiological control and may also be affected by concurrent brown staining due to progressive microinfiltration of extrinsic pigments, as shown in Figure 1.10. Today, the only truly "minimally invasive" treatment available on the market for these more advanced lesions, aimed at halting caries and restoring good aesthetics, is "erosion-infiltration." The only product currently on the market that follows this principle is Icon (DMG).[18]

In this **clinical case**, we present a scenario in which the management of risk factors is facilitated by the resin infiltration technique, used to treat an advanced stage, non-cavitated active lesion. The goal is to halt demineralization in an "artificial" and minimally invasive way, while maintaining the integrity of the enamel. The operational steps are illustrated in the caption.

CLINICAL CASE OF RESIN INFILTRATION TECHNIQUE



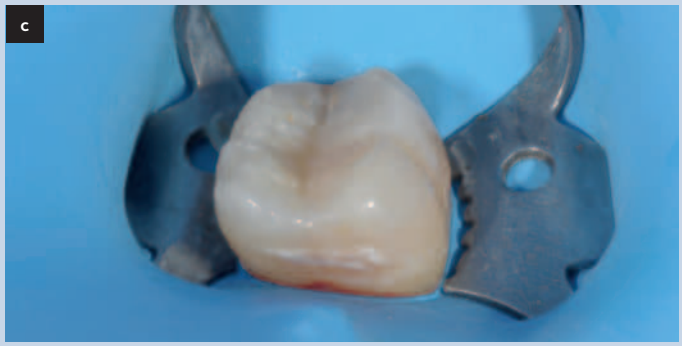


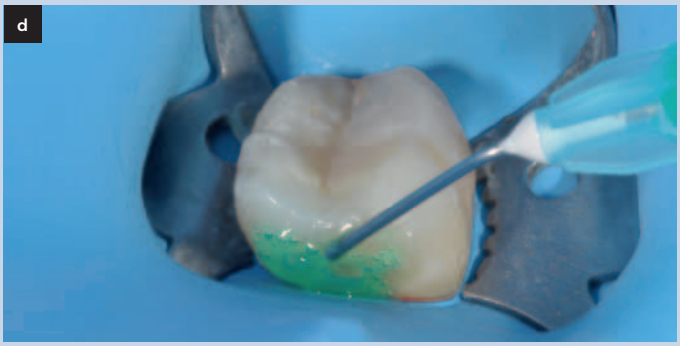
(a) Active carious lesion in the mesial portion of a mandibular molar. Prior to rehabilitating the edentulous space in front of the dental element, it was decided to protect this area from potential future deterioration by resin infiltration. The various operational steps are described below.

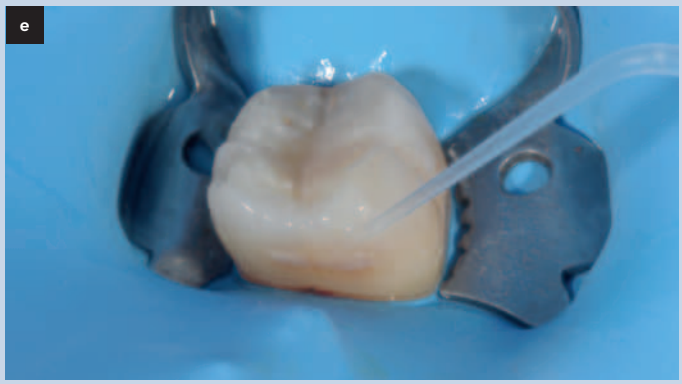
(b) Pass a fine-grain bur over the surface to polish it.

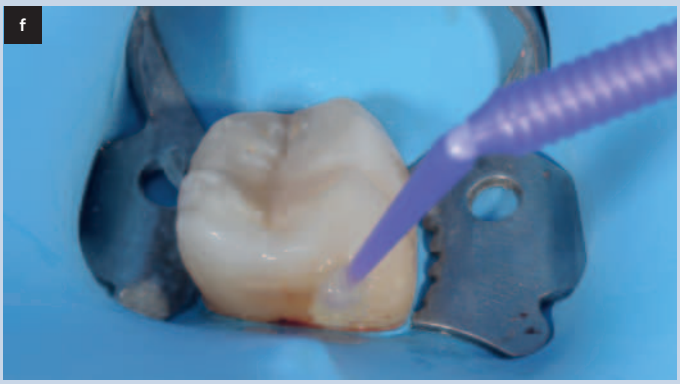
CONT'D ▶

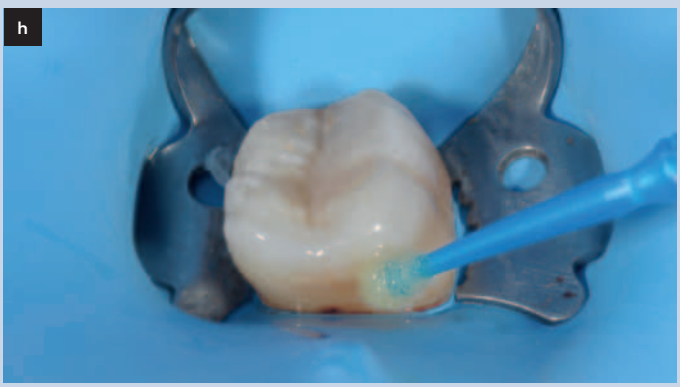
CONT'D ▶ Clinical case











(c) Appearance of the lesion after polishing.

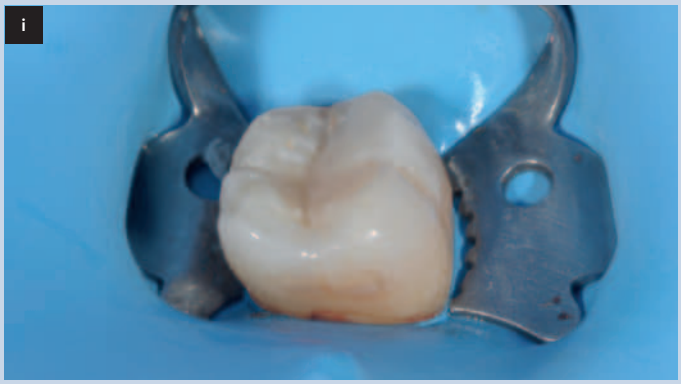
(d) Apply hydrochloric acid gel (HCl) for 2 minutes using Icon Etch from DMG. This step exposes the lesion. Suction and rinse Icon Etch with water for at least 30 seconds. Finally, dry with a jet of air without water.

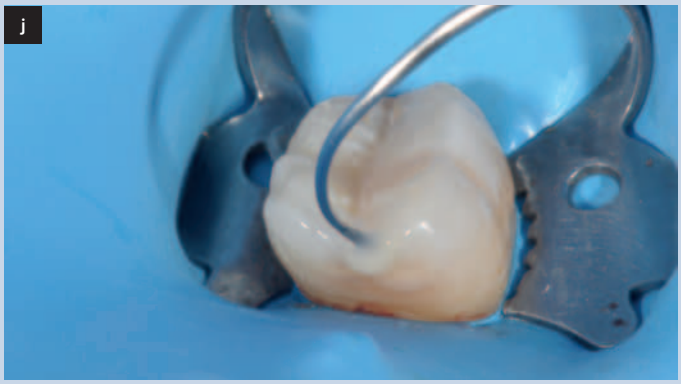
(e) Apply the second syringe of the Icon kit, Icon Dry (alcoholic solution), in a slightly greater quantity than necessary and leave for 30 seconds. Finally, dry with a jet of air without water.

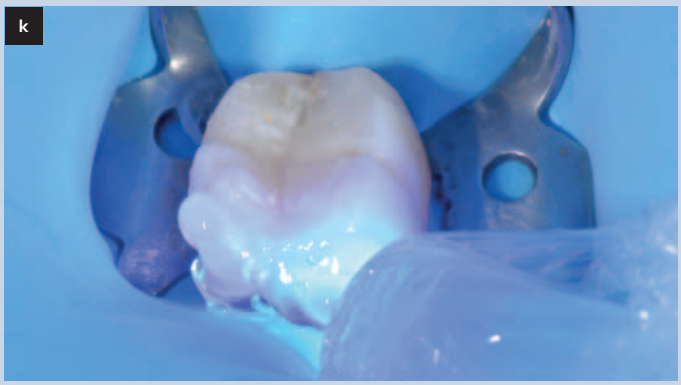
(f) Application of the infiltration resin. Leave Icon Infiltrant (resin) for 3 minutes. The material is activated by gently moving the applicator. In this case, a micro-brush is used. Please provide more details or content if needed. Before polymerizing, remove any excess material with dental floss or by suction, using clean cotton pellets or a clean microbrush.

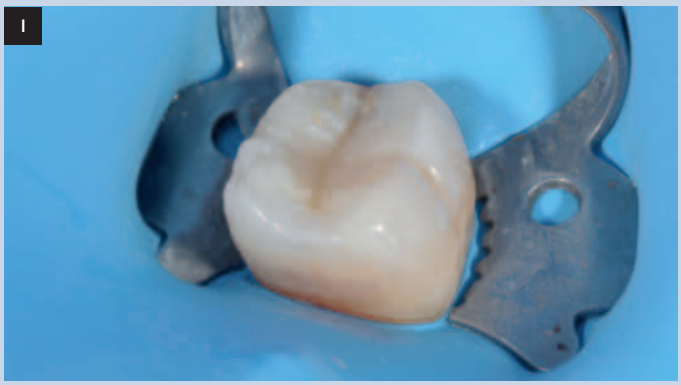
(g) Photopolymerize the infiltrating resin for at least 40 seconds.

(h) Subsequent application of Icon Infiltrant for 1 minute. Remove any excess and photopolymerize the resin for an additional 40 seconds.









(i) Appearance of the lesion after infiltration and photopolymerization.

(j) Application of a thin layer of flow composite using a blunt instrument to create a more protective layer over the infiltrated lesion. This step would be even more crucial in cases where, after the initial surface microabrasion with a bur or specialized pastes, further volumetric adjustment of a few microns of tissue was required. The flow layer adheres directly to the infiltrated surface without the need for additional adhesive and undergoes photopolymerization.

(k) The final photopolymerization is performed for 40 seconds under a glycerin gel to prevent oxygen from coming into contact with the treated surface and to ensure complete polymerization of the resin.

(l) The final appearance of the lesion after polishing. The tooth surface is now smooth, less susceptible to bacterial biofilm, and the layer of hypomineralized tissue has been protected by resin infiltration.

Diagnosing dental caries: methods and instruments

In the field of dentistry, there is not always a standardized diagnostic method for caries, and often different professionals interpret the same clinical signs differently. In some cases, there may be uncertainty about how to treat a carious lesion. The options include a conservative approach through remineralization treatments, resin infiltration, or using a drill and composite. In clinical practice, discrepancies in the information provided by the hygienist or dentist can lead to uncertainties in interpretation, which may affect the diagnosis.
When addressing this condition, diagnosing caries is the initial crucial step, along with conducting a risk analysis. Therefore, it is essential for all members of the dental team to be familiar with and consistently apply the same diagnostic interpretation criteria, as well as to know how to use the tools available to them in a uniform manner **(Fig. 1.11)**.

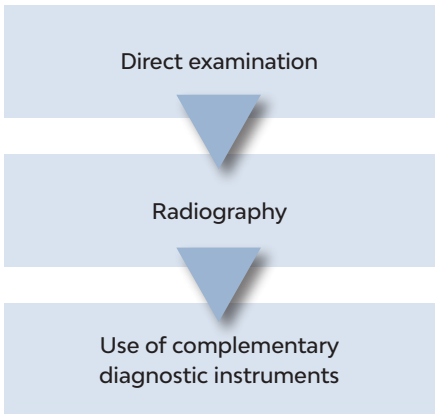


Figure 1.11 Diagnostic steps in the identification of carious lesions.

DIRECT EXAMINATION

A carious lesion can be evaluated after removing the biofilm that caused or is causing it. By using plaque disclosing substances, it is possible to measure the accumulation of biofilm on the tooth surface.
The biofilm can be gently removed during the diagnostic phase using a dental explorer or by delicately brushing the surfaces to achieve a dry and clean enamel surface, allowing for a correct visual interpretation of the clinical signs.[13]
Patients need to have their first visit with the dentist before undergoing a hygiene session. Patients are discharged with a diagnosis derived from visual and instrumental examinations. However, it is crucial to emphasize the importance of a follow-up appointment with a dental hygienist, which serves as a continuation of the initial visit. The hygienist's task is to verify or partially adjust the risk assessments made during the initial visit by completely removing biofilm.
To make an accurate assessment, it is important to thoroughly dry the affected area using the air syringe mounted on the dental unit. The same should be done when detecting other abnormalities.
Moreover, a direct examination cannot be conducted without the use of instruments that enhance the operator's vision. Both dentists and hygienists should use an optical magnification system during their clinical activities.

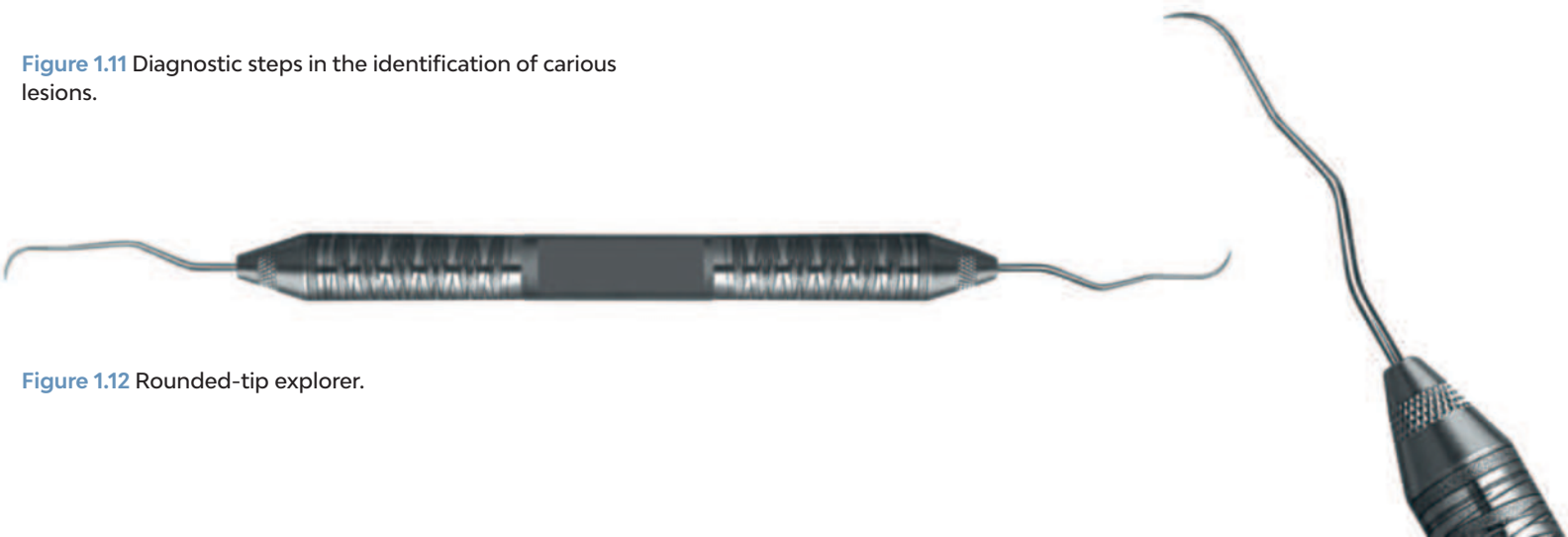


Figure 1.12 Rounded-tip explorer.

Dentists and dental hygiene students have been taught to use a sharp explorer to detect caries, especially in fissures – if it sticks or resists removal, caries has been found.[28] However, it has been emerged that this approach is not optimal, as it can cause traumatic defects in initial non-cavitated lesions. The pointed explorer could penetrate a relatively intact surface layer of the lesion, thereby transforming a subsurface defect into a cavity,[29] and may not enhance diagnostic precision.[30] Therefore, it is preferable to choose an explorer with a rounded tip **(Fig. 1.12)** and use it gently, without applying excessive force, as it still serves as a tool capable of providing valuable information on the consistency and surface structure of teeth.

The initial assessment involves determining the presence of a primary carious lesion in the enamel, as well as its extent and depth. If the cavity is filled with plaque and/or the dentin is soft, we can refer to it as an active cavitated lesion. Conversely, if the surface of the lesion is intact, it is important to determine whether this surface has a chalky consistency or a glossy appearance. A chalky surface indicates an active lesion that requires surface treatment without the use of drills. A shiny surface often indicates a lesion that has already stopped progressing **(Fig. 1.13)**. The color of the lesion can be useful in the assessment, but it should be observed cautiously. A brown color combined with a glossy surface often indicates that the lesion has stopped progressing. However, such a lesion can become active again under the appropriate conditions. Finally, sometimes the location of the lesion in relation to the gingival margin can be helpful. Lesions on the enamel with a smooth surface near a gingival recession are typically indicative of old, inactive lesions. This is a critical stage in the diagnosis because, when combined with subsequent examinations (such as radiographs and digital tools), it helps in formulating the treatment plan. It is essential to eliminate any areas that serve as "hotspots" for bacteria. A cavity in the enamel and dentin serves as a reservoir for the accumulation and growth of biofilm, leading to the expansion of the cavity. When dealing with a non-cavitated lesion, the preferred approach is non-invasive treatment, such as remineralization or surface infiltration, along with monitoring risk factors.

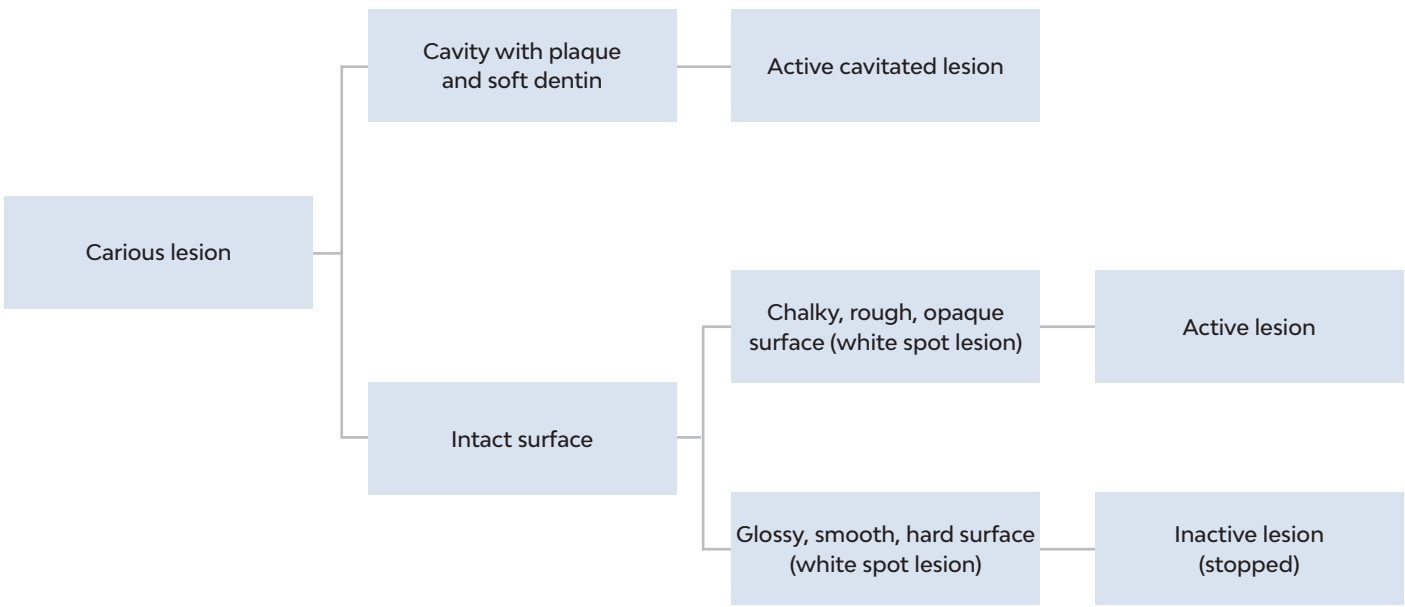
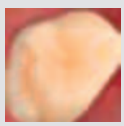
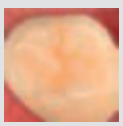
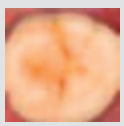
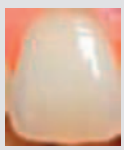
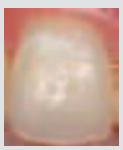
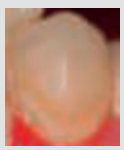
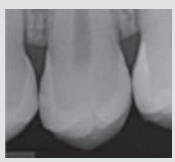
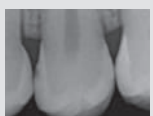
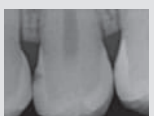
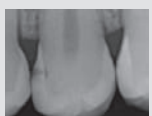


Figure 1.13 Visual assessment of a carious lesion. Possible clinical signs that can be detected during a direct examination.

ADA CCS CLASSIFICATION

Although modern dentistry is increasingly focusing on interceptive measures to prevent, treat, and reverse caries processes, especially in the early stages, until a few years ago, the most commonly used system to describe lesions was based on the classification by Dr. Greene Vardiman Black. This classification, coined by the author after whom it is named in the late 19th century, is still occasionally used in clinical practice to indicate the location and severity of carious lesions. However, Black's classification only pertains to cavitated lesions, categorizing it as an 'interventional' rather than a 'preventive' approach. Dr. Black's system does not address non-cavitated lesions, such as the initial enamel lesions, like white or brown spots. In 1896, Dr. Black wrote, *"The day is surely coming [...] when we will be engaged in practicing preventive rather than reparative dentistry."*[31] More than a century later, Black's vision has become a reality, leading to the development of more modern classifications for carious lesions. One of the most comprehensive and up-to-date classifications is the American Dental Association's Caries Classification System (CCS). The ADA CCS was developed to update and integrate other classifications that have supplemented and expanded Black's outdated classification. In 2008, the ADA assembled a panel of experts to initiate the development of the current classification, with the goal of integrating

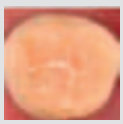
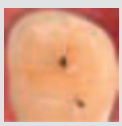
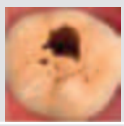
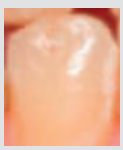
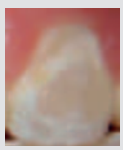
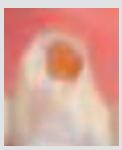
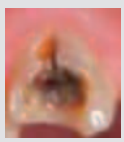
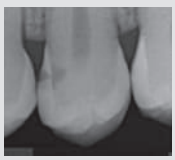
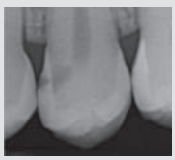
Table 1.4 American Dental Association Caries Classification System

	Sound	Initial		
Clinical Presentation	No clinically detectable lesion. Dental hard tissue appears normal in color, translucency, and gloss.	Earliest clinically detectable lesion compatible with mild demineralization. Lesion limited to enamel or to shallow demineralization of cementum/dentin. Mildest forms are detectable only after drying. When established and active, lesions may be white or brown and enamel has lost its normal gloss.		
Other Labels	No surface change or adequately restored	Visually noncavitated		
Infected Dentin	None	Unlikely		
Appearance of Occlusal Surfaces (Pit and Fissure)*†	ICDAS 0 	ICDAS 1 	ICDAS 2 	
Accessible Smooth Surfaces, Including Cervical and Root‡				
Radiographic Presentation of the Approximal Surface§	 EO¶ or RO# No radiolucency	 E1¶ or RA1#	 E2¶ or RA2#	 D1¶ or RA3#
		Radiolucency may extend to the dentinoenamel junction or outer one-third of the dentin. Note: radiographs are not reliable for mild occlusal lesions		

* Photographs of extracted teeth illustrate examples of pit-and-fissure caries.
† The ICDAS notation system links the clinical visual appearance of occlusal carious lesions with the histologically determined degree of dentinal penetration using the evidence collated and published by the ICDAS Foundation over the last decade; ICDAS also has a menu of options, including 3 levels of carious lesion classification, radiographic scoring and an integrated, risk-based caries management system ICCMS. (Pitts NB, Ekstrand KR. International Caries Detection and Assessment System [ICDAS] and its International Caries Classification and Management System (ICCMS): Methods for staging of the caries process and enabling dentists to manage caries. *Community Dent Oral Epidemiol* 2013;41(1):e41-e52. Pitts NB, Ismail AI, Martignon S, Ekstrand K, Douglas GAV, Longbottom C. ICCMS Guide for Practitioners and Educators. Available at: https://www.icdas.org/uploads/ICCMS-Guide_Full_Guide_US.pdf. Accessed April 13, 2015.)

clinical and operational aspects. The work underwent several revisions and was officially published in 2015. Born from the reworking and further expansion of previous classifications, it shares several common points with them. The DMF, ICDAS, CAST, and PUMA are the main references that were used.[32] In 2015, the ADA established this classification as an essential tool. Caries can manifest in various clinical presentations throughout the disease process, and clinicians require a classification system that can facilitate appropriate therapeutic decisions using both surgical and non-surgical approaches.[32] Defining the location of a lesion, its site of origin, extent, and, if feasible, its activity level, should be a standard component of every direct examination. This approach can help establish the level of risk and determine the necessary procedures to be implemented.[3,33,34]

The ADA CCS was developed to encompass both non-cavitated and cavitated carious lesions and to describe them based on their clinical presentation, without specifying a particular treatment approach. Furthermore, the ADA CCS, unlike some caries classification systems, connects the clinical presentation of the lesion to radiographic findings and offers an approach to identify, when feasible, the activity of the carious lesion over time. In accordance with the Journal of the American Dental Association (JADA), the carious lesion classification system published in 2015, along with a description and its clinical advantages, can be found in **Table 1.4**.

Moderate		**Advanced**	
Visible signs of enamel breakdown or signs the dentin is moderately demineralized.		Enamel is fully cavitated and dentin is exposed. Dentin lesion is deeply/severely demineralized.	
Established, early cavitated, shallow cavitation, microcavitation		Spread/disseminated, late cavitated, deep cavitation	
Possible		Present	
ICDAS 3	ICDAS 4	ICDAS 5	ICDAS 6
			
			
			
D2¶ or RB4# Radiolucency extends into the middle one-third of the dentin		D3¶ or RC5# Radiolucency extends into the inner one-third of the dentin	

‡ "Cervical and root" includes any smooth surface lesion above or below the anatomical crown that is accessible through direct visual/tactile examination.
§ Simulated radiographic images.
¶ EO-E2, D1-D3 notation system.[33]
# RO, RAI-RA3, RB4, and RC5-RC6 ICCMS radiographic scoring system (RC6 = into pulp). (Pitts NB, Ismail AI, Martignon S, Ekstrand K, Douglas GAV, Longbottom C. ICCMS Guide for Practitioners and Educators. Available at: https://www.icdas.org/uploads/ICCMS-Guide_Full_Guide_US.pdf. Accessed April 13, 2015.)

The ADA CCS assesses tooth surfaces using the following criteria: tooth surface, presence or absence of a carious lesion, anatomic site of origin, severity of change, and estimation of lesion activity. The clinical application of the ADA CCS relies on examinations conducted on a clean tooth with compressed air, adequate lighting, and the use of a rounded explorer or ball-end probe. Indicated radiographs should also be available.
The criteria for detecting the tooth surface sites of origin are defined in Table 1.5 as follows:

- Pit and fissure
- Approximal
- Cervical and smooth surface
- Root.

In the ADA CCS system, smooth, cervical, and root surfaces are given similar considerations because they share many common characteristics and are accessible for visible and tactile clinical examination. Classifying the site of origin of a carious lesion is essential in a caries management system for assessing the cause of the lesion and determining the available treatment options.

SOUND SURFACE

In a healthy state, the surface is intact, and there are no clinically detectable lesions. The dental tissue appears normal in color, translucency, and glossiness, or the tooth has an adequate restoration or sealant with no signs of a carious lesion.

INITIAL CARIOUS LESION

These are the earliest detectable lesions consistent with a net loss of minerals. They are limited to the enamel or cementum, or the very outermost layer of dentin on the root surface, and in the mildest forms, they are detectable only after drying. The clinical presentation includes changes in color to white or brown (e.g., cervical demineralization along the gingival area) or well-defined areas (e.g., white spot lesions on smooth surfaces). In pits and fissures, there is a noticeable change in color to brown, but there are no signs of significant demineralization in the dentin (i.e., no underlying dark gray shadow). These early lesions are non-cavitated and can be reversed through remineralization. Most of these lesions would be categorized as "healthy" in epidemiologic studies.

MODERATE CARIOUS LESION

Moderate mineral loss leads to deeper demineralization, with the potential for microcavitation on the enamel surface,early shallow cavities, and/or visible dentin shadowing through the enamel, indicating possible dentin involvement (e.g.,microcavitation with noticeable dentin staining). These lesions exhibit visible signs of enamel loss in pits and fissures, on smooth surfaces, or visible signs of cementum/dentin loss on the root surface. Although the pits and fissures may appear intact (albeit brown), dentin involvement (demineralization) can often be detected by the presence of a dark gray shadow or translucency visible through the enamel. Dentinal involvement in moderate lesions

Table 1.5 Detection criteria for the origin sites on teeth surfaces, as used in the ADA CCS Classification

SITE	Definition
Pit and fissure	Referring to the anatomical pits or fissures of teeth, such as those found on the occlusal, facial, or lingual surfaces of posterior teeth, or on the lingual surfaces of maxillary incisors or canines
Approximal	Referring to the immediate proximity to the contact area of an adjacent tooth surface
Cervical and smooth surface	Referring to the cervical area or any other smooth enamel surface of the anatomical crown adjacent to an edentulous space
Root	Referring to the root surface below the anatomic crown

in proximal areas can be detected in a similar manner by examining the marginal ridges over the suspected lesion site, which may exhibit gray discoloration or appear translucent. If the suspected location of an approximal lesion cannot be directly inspected, which is often the case, the presence and extent of lesion cavitation cannot be assessed without the use of radiographs,[35] tooth separation,[36,37] or both, in combination with an assessment of lesion activity, where possible **(Fig. 1.14)**.

ADVANCED CARIOUS LESION

Advanced carious lesions have fully cavitated through the enamel, and the dentin is clinically exposed. In the ADA CCS, any clearly visible cavitated lesions showing dentin on any surface of the tooth are classified as "advanced." In epidemiologic studies, these lesions are classified as "decayed."

Note that any carious lesion described above may also be associated with an existing restoration or sealant.

This classification is undoubtedly a useful tool for integrating multiple evaluation criteria, as mentioned earlier, while considering the stages of the carious lesion from the initial surface changes to cavity formation. A comprehensive clinical case examination for decision-making purposes should include an assessment of the lesion's clinical activity in addition to examining its appearance. In the presence of an active lesion that does not show cavitation, nonsurgical procedures can be implemented to reverse the mineral loss at a specific site before it progresses to the next stage of cavitation.

Therefore, during the direct examination, the ADA CCS classification can help professionals define the appearance of a carious lesion and classify the surface based on its clinical appearance: sound, initial carious lesion, moderate, or advanced. In the patient's clinical record, this description specifies whether the lesion is "active" or "inactive," and, if necessary, its radiographic extent, particularly for approximal lesions. By recording these characteristics, it will be possible to determine whether to pursue an interventional approach in advanced cases or to initiate remineralization and prevention processes. Subsequently, the data can be compared over time during follow-up sessions

ICDAS CLASSIFICATION

The well-known International Caries Detection and Assessment System (ICDAS) is a globally recognized classification system, also referenced within the ADA CCS.

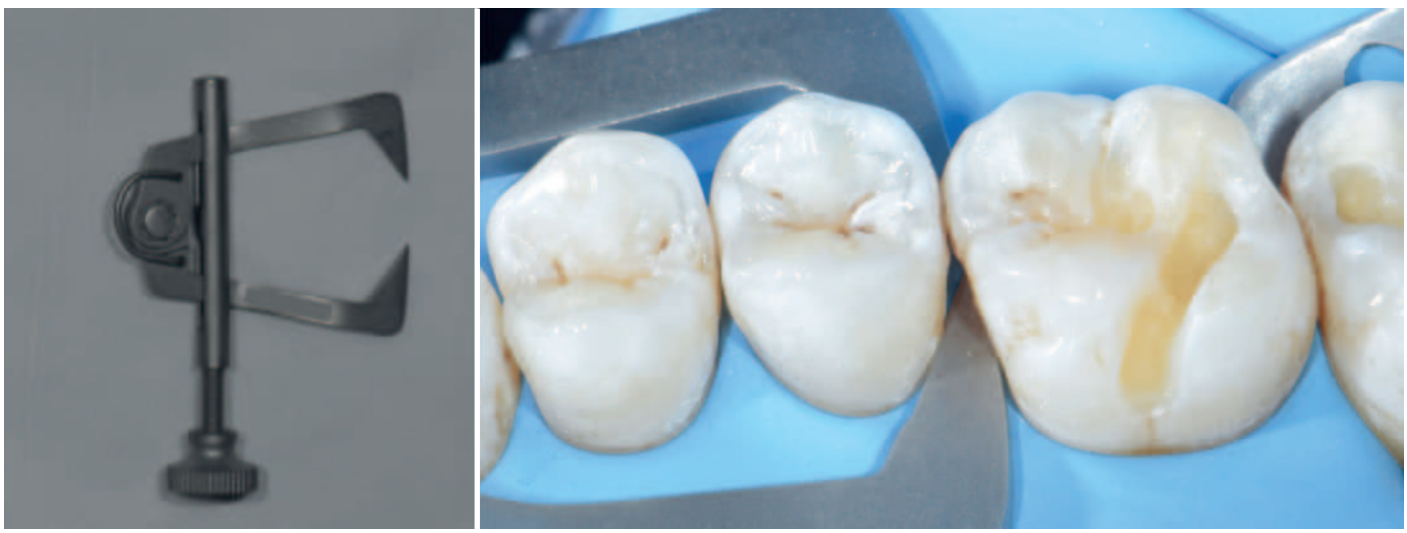


Figure 1.14 An instrument that can help clinicians in exploring the interdental spaces is the separator. This image shows an Elliot separator in action.

As shown in Table 1.4, the ICDAS classification includes different stages of carious lesions, which are defined as codes numbered progressively from 0 to 6. These levels are integrated into the ADA classification and are used internationally as a common language to describe the appearance of tooth surfaces during direct examination. Below are the ICDAS codes.[38]

ICDAS 0: SOUND TOOTH SURFACE

This code indicates the absence of caries, meaning there is either no change or a questionable change in enamel translucency after prolonged air drying for 5 seconds. Surfaces with developmental defects, such as enamel hypoplasia, fluorosis, tooth wear (attrition, abrasion, and erosion), and extrinsic or intrinsic stains, will be recorded as healthy.

ICDAS 1: FIRST VISUAL CHANGE IN ENAMEL

Pits and fissures

At this stage, when observed while wet, there is no evidence of any color change indicative of carious activity. However, after prolonged air drying, a carious opacity or discoloration (white or brown spot lesion) becomes visible, which does not align with the clinical appearance of healthy enamel. It is recommended to thoroughly dry a carious enamel lesion for about 5 seconds.

Smooth surface (mesial and distal)

When wet, there is no evidence of any color change due to carious activity. However, after prolonged air drying, an opacity (white or brown spot lesion) becomes visible, which does not align with the clinical appearance of healthy enamel.

ICDAS 2: DISTINCT VISUAL CHANGE IN ENAMEL

The tooth must be examined while wet. When wet, there is a carious opacity (white spot lesion) and/or brown carious discoloration (brown spot lesion) that is wider than the natural fissure or fossa, which is not consistent with the clinical appearance of sound enamel. (*Note*: the lesion must still be visible when dry).

ICDAS 3: LOCALIZED ENAMEL BREAKDOWN DUE TO CARIES WITH NO VISIBLE DENTIN OR UNDERLYING SHADOW

The wet tooth may exhibit a clear carious opacity (white spot lesion) and/or brown carious discoloration (brown spot lesion) that is wider than the natural fissure or fossa, which does not align with the clinical appearance of sound enamel. Once dried for about 5 seconds, there is carious loss of tooth structure at the entrance to or within the pit or fissure. This will be visually evident as demineralization (opaque/white, brown, or dark brown walls) at the entrance to or within the fissure or pit. Although the pit or fissure may appear significantly wider than normal, the dentin is not visible in the walls or base of the cavity. If you are unsure or need to confirm the visual assessment, the rounded probe can be gently used across a tooth surface to verify the presence of a cavity apparently confined to the enamel.

ICDAS 4: UNDERLYING DARK SHADOW FROM DENTIN WITH OR WITHOUT LOCALIZED ENAMEL BREAKDOWN

This lesion appears as a discolored shadow of dentin visible through an apparently intact enamel surface, which may or may not show signs of localized breakdown (loss of continuity of the surface that is not revealing the dentin). The shadow's appearance is often more easily seen when the tooth is wet. The darkened area is an intrinsic shadow that may appear as grey, blue, or brown in color. The shadow must clearly represent decay that originated on the tooth surface being evaluated. If in the opinion of the examiner, the carious lesion originated on an adjacent surface and there is no evidence of any decay on the surface being assessed, then the surface should be assigned a code of "0".

ICDAS 5: DISTINCT CAVITY WITH VISIBLE DENTIN

The surface exhibits distinct cavitation in opaque or discolored enamel, exposing the dentin beneath. The wet tooth may appear darkened due to the dentin being visible through the enamel. Once dried for 5 seconds, there is visible evidence of tooth structure

loss at the entrance to or within the pit or fissure. There is visual evidence of demineralization (opaque/white, brown, or dark brown walls) at the entrance to or within the pit or fissure, and the examiner judges that dentin is exposed. A dental explorer may be used to confirm the presence of a cavity in the dentin.

ICDAS 6: EXTENSIVE DISTINCT CAVITY WITH VISIBLE DENTIN

In this case, there is an obvious loss of tooth structure, with the cavity being both deep and wide, and dentin is clearly visible on the walls and at the base. An extensive cavity affects at least half of a tooth's surface or may extend to the pulp.

RADIOGRAPHS

The use of radiographs is essential for monitoring a patient's oral health during the initial visit, periodic follow-ups, and as a diagnostic tool. There are various dental radiological examinations available to dentists, and it is crucial to interpret them correctly.

ORTHOPANTOMOGRAPHY

Orthopantomography is a commonly available examination that patients often already have, as it is frequently used in dental practices as a fundamental radiological diagnostic tool during the initial visit. However, it is the least reliable diagnostic tool for caries, especially for assessing initial enamel lesions. Orthopantomography is a commonly prescribed and performed basic radiology examination that offers a comprehensive view of the dental structures. It is a valuable screening tool for detecting abnormalities in the hard oral and perioral tissues, skeletal and dental structures, and for assessing the temporomandibular joints and maxillary sinuses. As a two-dimensional representation of three-dimensional structures, radiograms can suffer from distortions or overlapping of anatomical structures during image acquisition, potentially resulting in the formation of artifacts.[39] This makes it unsuitable for caries diagnosis. Hence, the use of intraoral radiographs with centering devices is emphasized and suggested as the preferred logical diagnostic tool for caries diagnosis, especially bite-wing radiographs.

INTRAORAL RADIOGRAPHS

Intraoral radiographs are semi-rigid films of various sizes that can be placed inside the oral cavity. They are available in both analog and digital formats. The latter option is considered better in terms of minimizing the patient's biological exposure, as it has the potential to reduce X-ray exposure time. The reader can enhance the image using a luminescence system, which reduces the radiation dose to the patient. A significant advantage of digital exams over analog ones is the ability to post-process the image using specialized software. The clinician can perform on-computer magnifications, measurements, contrast adjustments, and gray scale modifications, enabling the detection of more details and simplifying data storage. The communicative aspect of digital exams is also important. It allows for the projection of high-definition images on screens, providing a clearer illustration of radiograms to the patient during the diagnostic phase and facilitating the explanation of the treatment plan.

Traditional radiographs are produced in the same manner as digital ones, yielding comparable image quality, but the development process is different. They require a dedicated darkroom for processing. The chemical process involves several steps. First, the light and moisture-proof envelope containing the film is removed. Then, the film is immersed in the developing solution, followed by an intermediate washing in water. Next, it is passed through the fixing liquid and further washed until it is ready for reading on a backlit screen. Common issues include errors in the development phase, whether accidental or due to incorrect timing, instability of film quality over time, storage challenges, and the requirement for a dedicated space in the clinic for the darkroom. However, this can be avoided by using self-developing films.

Digital radiographs, on the other hand, have a faster development process. The most common types are memory phosphor, which are digitally processed and "developed" using a scanner in a few seconds. They can be post-processed and easily stored in dedicated

software, making them quick to search and reference (Fig. 1.15).
Digital films, unlike analog ones, are not disposable. After being processed by the reader, the cards are "reset" and ready for reuse. Protective devices are available to prevent the film from coming into contact with oral fluids and patient tissues. These are disposable, waterproof envelopes that protect the sensor during image acquisition.
Radiovisiography (RVG) is another digital imaging system that involves placing a specialized sensor, rather than a film, into the oral cavity. This sensor is connected to a monitor via a cable. When exposed to X-rays, it converts the energy into an electrical signal and transfers the image to the monitor in real time (Fig. 1.16). It offers the same benefits of speed, post-processing, and ease of storage as phosphor digital systems, but it also has limitations. Unlike a phosphor film, which is practically identical in size and maneuverability to an analog film, the RVG sensor is thicker and bulkier, making it challenging to acquire data easily. The presence of the connected cable can also pose an obstacle. The film, whether phosphor or analog, is adaptable to all situations due to its flexibility and thinness, and is available in various sizes. The RVG sensor, on the other hand, has a fixed size, is much thicker and more rigid. Even with equal image quality, this presents a significant limitation.

Execution technique

Regardless of whether an analog or digital film is used, the process of executing the radiogram is the same. There are two techniques. The first technique, known as the "bisecting angle" technique, requires positioning the X-rays perpendicularly to the bisector of the angle formed between the film and the longitudinal axis of the teeth. However, this technique is challenging to execute and susceptible to projection errors.
The other image acquisition technique is the "parallel technique" (Fig. 1.17) using the Rinn aiming device, which is more widely used and easier to perform. Rinn aiming devices (Fig. 1.18) include a terminal support for the film, an intermediate support for stability, and an extraoral ring for positioning and centering the radiographic tube. This positions the film parallel to the axis of the teeth and perpendicular to the incidence of the X-rays, producing an intraoral periapical or bitewing radiographic image that closely represents the

Figure 1.15 Digital intraoral radiographs. Phosphor system. The carious lesions are well detectable, as well as the bone defects secondary to infections.

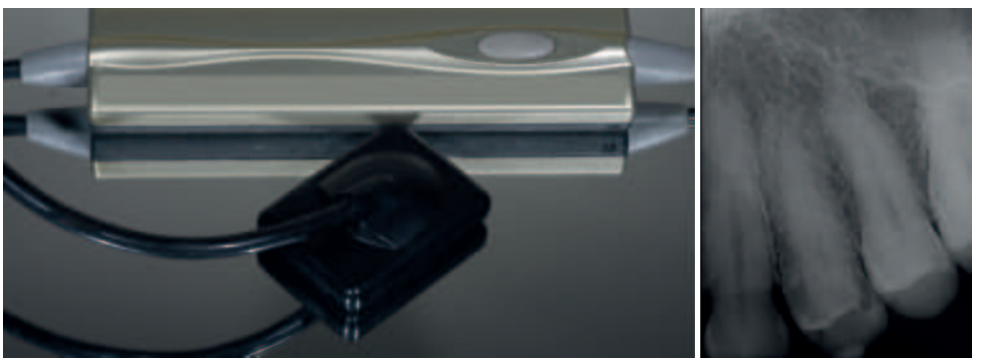


Figure 1.16 Digital intraoral radiographs. RVG system. The RVG system is more cumbersome for image acquisition due to its bulk, the presence of the cable, and its stiffness.

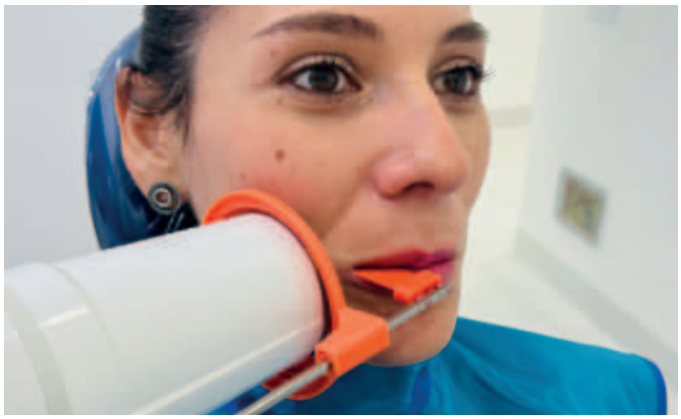


Figure 1.17 Correct positioning of the centering device and the X-ray tube.

Figure 1.18 Rinn Aiming Devices. There are four different Rinn aiming devices, each distinguished by color: blue for the anterior sectors, yellow for the lateral/posterior elements, green for taking an X-ray under a dam, and red for the bite-wing.

actual situation, without significant magnification, distortion, or under sizing of the examined elements. This achieves an image of the studied dental elements and adjacent periodontium with a 1:1 ratio.[40]

Intraoral radiographic examination is considered a fundamental diagnostic tool for identifying carious lesions, determining their extent, monitoring previous restorations, and assessing the integrity of the approximal areas of the teeth. For these assessments, **bite-wing radiographs** are most suitable. When correctly executed, they allow visualization of all coronal portions of the posterior teeth on one film, reducing biological exposure to X-rays and avoiding overlapping of the marginal ridges. This provides a more accurate assessment of the approximal areas[41] (Fig. 1.19).

Interpreting the images

Radiographic interpretation is essential for identifying and evaluating the normal appearance of the structures being examined. In the process of image formation, structures that are not penetrated by X-ray photons appear radiopaque, tending towards white, with varying shades of gray depending on their density and the degree of X-ray penetration through the tissue.

Tissues that are less dense and allow X-rays to pass through are defined as radiolucent, appearing grayer and darker in the image.

Enamel, being highly mineralized, appears radiopaque, appearing whiter than dentin, which is slightly less radiopaque. Dentin is nearly indistinguishable from cementum because they have a similar level of mineralization.

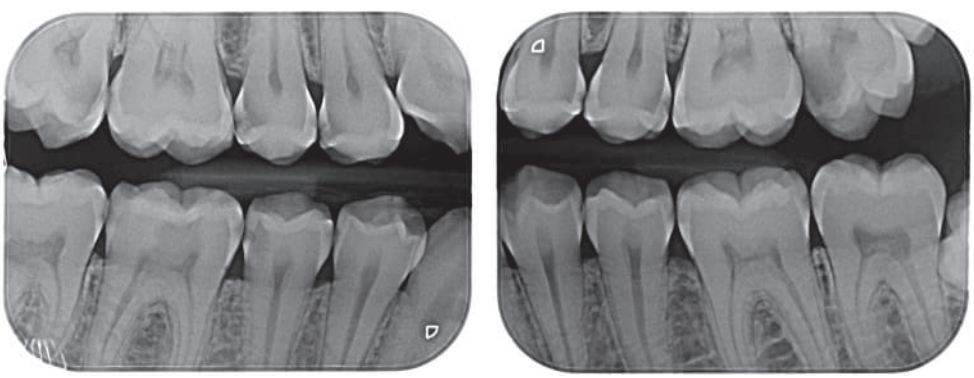


Figure 1.19 Bite-wing radiography. It is the most effective radiographic examination for assessing caries and allows for the visualization of both upper and lower teeth in a single radiograph.

The pulp chamber, along with its extensions and the root canals, are visible as radiolucent, darker structures, as they contain non-mineralized soft tissue.
The dental perimeter, within the alveolar bone complex, is accompanied by a thin radiolucent line due to the presence of the periodontal ligament. This line is usually wider in young subjects, becomes narrower with age, and may be absent in particular situations.[42]
The visualization of carious lesions in radiographs is attributed to the interaction of X-rays with less dense areas, which are caused by the demineralization induced by the caries process on the hard dental tissue, making it more radiolucent.
Carious lesions, particularly in the initial stages, may not be detected during direct clinical examination but are often visible in radiographs.[43] Early detection of caries is crucial for initiating preventive measures and/or minimally invasive treatments to manage the disease promptly.
The examination of intraoral radiographs has also resulted in the development of specific classifications related to the severity of approximal carious lesions, enabling clinicians to make preventive or interventional decisions in each case. This also enables a shared vision and consistent diagnostic interpretation among different operators, particularly within the hygienist-dentist team. The most widespread radiological classification is the E-D classification.

E-D CLASSIFICATION

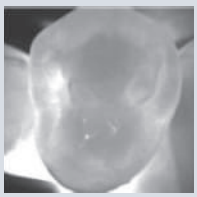
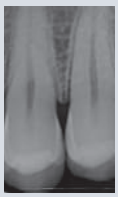
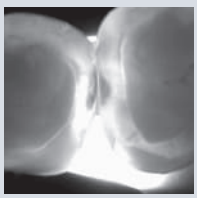
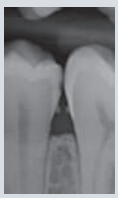
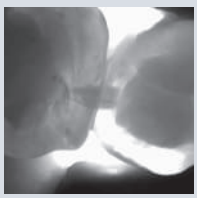
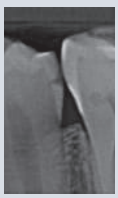
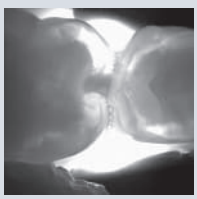
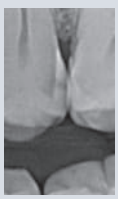
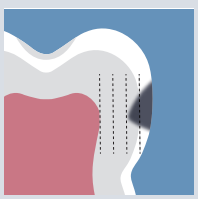
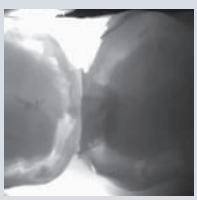
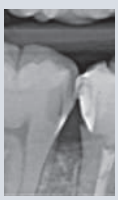
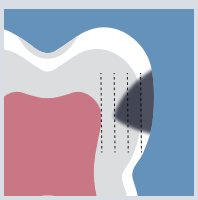
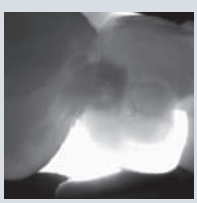
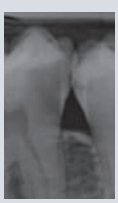
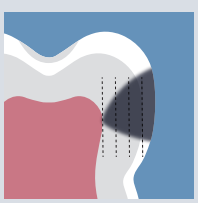
A radiological classification of approximal carious lesions was developed based on the observation of bite-wing radiographs, taking into account the extent of radiolucency in the enamel and dentin.
The classification, known as E-D, is divided into five levels, two for enamel (E) and three for dentin (D).
As shown in **Figure 1.20**, the classification comprises the following levels:

- E1: Carious lesion confined to the outer half of the enamel
- E2: Carious lesion also affecting the distal half of the enamel
- D1: Carious lesion involving the outer third of the dentin
- D2: Carious lesion extending into the inner one-third of the dentin
- D3: Carious lesion affecting the entire thickness of the dentin.

This classification plays a crucial role not only in facilitating communication among team members, allowing them to attribute a specific significance to approximal lesions observed in radiographs and/or transillumination exams, but also in the operational and decision-making aspects related to the prognosis of these lesions.
The minimally invasive dental approach is based on assessing the risk of caries. Therefore, the decision to remove compromised dental tissue for restoration should be postponed until it is evident that the dental surfaces have cavities or are at risk of developing cavities despite all practical efforts of prevention and remineralization.[44]
According to the study by Pitts and Rimmer, enamel lesions should not be restored, as only 10.5% of permanent elements are likely to be cavitated when proximal lesions are in the first half of the enamel.[35] Therefore, classifying the severity of the lesion is important as decisions on whether to perform a restoration can be made on a more standardized basis. **Figure 1.21** shows the likelihood in percent of finding cavitation for each type of lesion. From the interpretation of statistical data, a more conservative or less conservative approach can be adopted depending on the case.
In the presence of non-cavitated approximal lesions, our clinical approach should always involve controlling risk factors and aiming for the remineralization of the lesions. However, if the patient presents a high or extreme risk of caries, or shows low compliance, or if the lesions already affect the entire thickness of the enamel and it is deemed appropriate to perform a more advanced procedure, we can resort to the resin infiltration technique to "artificially" stop the lesion from progressing (see Clinical Case). This procedure is certainly less invasive compared to the execution of a restoration and can be helpful in managing more advanced or higher-risk approximal lesions. By scanning the QR code at the end of the chapter, you can access the online section of this text to view examples of the technique's application, using the Icon DMG Approximal kit.

Figure 1.20

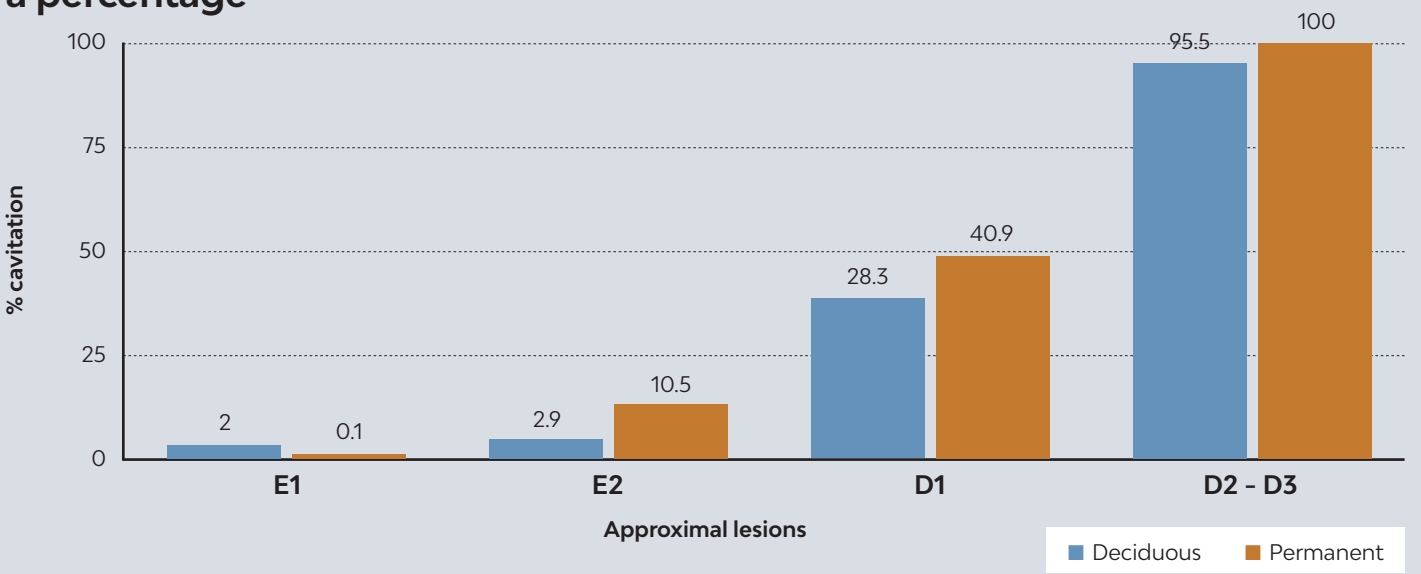
E-D Classification

Description	DIAGNOcam	X-ray	Clinical setting	Therapeutic recommendations
Healthy tooth surface				Monitoring and prevention
E1 First signs of approximal caries affecting the outer half of the enamel				Monitoring and caries prevention. Remineralization or resin infiltration
E2 Approximal caries affecting the entire thickness of the enamel				Monitoring and caries prevention. Remineralization or resin infiltration
D1 Carious lesion involving the outer third of the dentin				Monitoring and caries prevention. Remineralization or resin infiltration. In the event of cavitation, provide restoration
D2 Carious lesion extending to the inner one-third of the dentin				Provide restoration
D3 Carious lesion affecting the entire dentin thickness				Provide restoration

The image schematically illustrates the types of approximal carious lesions that affect only the enamel (E1-E2), or also the dentin (D1-D2-D3).

Figure 1.21

Likelihood of finding cavitation in approximal lesions, expressed as a percentage



The graph, based on data published by Pitz and Rimmer, illustrates that E1 lesions have a 0.1% chance of developing cavities in permanent teeth and a 2% chance in deciduous teeth.

E2 lesions have a slightly higher likelihood of cavitation: 10.5% in permanent teeth and 2.9% in deciduous teeth. This means that the approach can be very conservative in most cases, as there is no need for restoration without cavitation.

Therefore, when E1 or E2 lesions are present and visualized by radiography or transillumination, the clinician should inform the patient about the presence of caries, which can still be reversed by promoting remineralization.
The remineralization of initial E1-E2 lesions is achievable through a series of measures: enhancing at-home oral hygiene, possibly by improving dental flossing techniques, using fluoride toothpastes and mouthwashes, replenishing minerals in saliva by staying hydrated (and if needed, using calcium phosphate compounds), managing dietary habits and lifestyle, and scheduling regular follow-up appointments.
In cases where it is believed that the patient has limited cooperation, or the overall risk level is high, resin infiltration can be used to halt the lesions and further decrease the likelihood of cavitation in the future. We recommend using the Icon Kit (DMG) for approximal infiltration. Please refer to the dedicated in-depth analysis for more information.
Approximal lesions require continuous monitoring. For this reason, it is crucial to record them in the patient's clinical record and to confirm their unchanged state or any worsening over time during follow-up hygiene sessions. This may lead us to consider a different therapeutic approach compared to the initial decisions. Periodic use of bite-wing radiographs or transillumination tools, such as DIAGNOcam, based on the risk level, are the ideal instruments for long-term control of approximal carious lesions.

D1 lesions, on the other hand, have a 40.9% probability of cavitation in permanent teeth and 28.3% in deciduous teeth. This means that the likelihood of choosing restorative solutions increases, but it may not be applicable to all cases. The decisive factor is always determined by the direct examination and by verifying whether cavitation is present or not. In the absence of cavitation, the therapeutic approach can be the same as described for E1-E2 lesions, with a preference for resin infiltration procedures.
If cavitation is present or the general risk level is high or extreme, we will opt for a restorative approach.

The likelihood of finding cavitation in D2 and D3 lesions is 100% in permanent teeth and 96.5% in deciduous teeth. This data clearly indicates a preference for an immediate restorative approach.

Guided enamel regeneration for the treatment of initial carious lesions

THE SELF-ASSEMBLING BIOMIMETIC PEPTIDE P11-4 (CURODONT™ REPAIR)

In recent years, a new treatment has emerged in the field of remineralization procedures, specifically concerning the management of non-cavitated carious lesions. The application of fluoride varnishes (FV) is the standard for preventing and treating initial carious lesions. It is effective in protecting healthy enamel but less so when a carious lesion has developed.[45] Although carious lesions can be arrested, fluoride's ability to remineralize enamel is limited to approximately 40 microns in depth and is not visible on radiographs.[46,47]

However, non-cavitated carious lesions can be 1 mm and larger in the hard tooth tissue (enamel or dentin) before cavitation,[48,49] necessitating a reparative action in deeper areas as well as the preventive action of fluoride to make the caries process reversible.

Curodont™ Repair is the first biomimetic technology designed to treat initial caries through enamel regeneration. Curodont™ Repair offers a simple, painless and conservative solution for patients of all ages. The product is based on the revolutionary technology of the *self-assembling peptide P11-4*,[48] a simple and intelligent peptide composed of amino acids that are found in nature.

After application in the dental office by a dentist or dental hygienist, the peptide penetrates deep inside the initial carious lesions within 5 minutes where it organizes into a biomatrix similar to the natural matrix of enamel.[50]

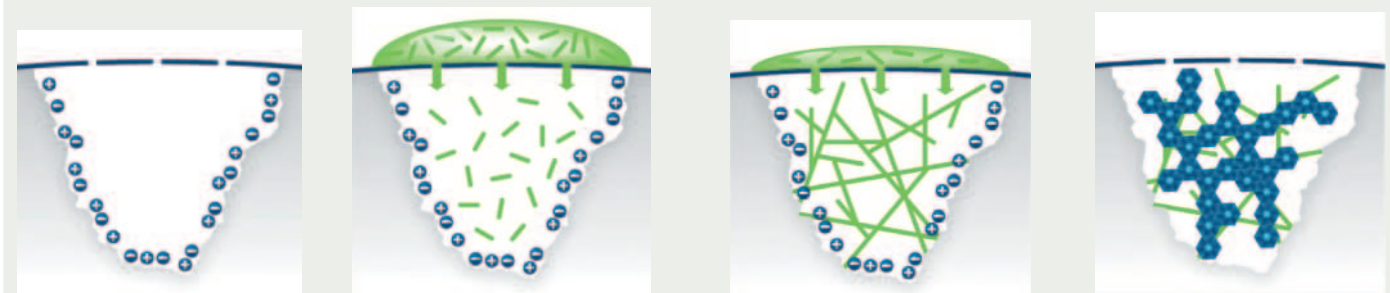
The biomatrix attracts calcium and phosphate ions from saliva, forming new hydroxyapatite crystals over the following weeks and months. This process enables guided enamel regeneration within the lesion.

Several clinical studies have demonstrated that this unique and patented formula achieves significantly greater regression and inactivation of caries compared to the sole use of the current gold standard, fluoride varnishes, with a demonstrated success rate of 93% compared to approximately 35% for fluoride varnishes[51,52] **(Fig. 1.22).**

The application of the self-assembling biomimetic peptide P11-4 (SAP P11-4) has been the subject of numerous clinical trials.[53-61]

The first human study indicated that it is safe.[53]

Schlee and colleagues[54] confirmed the *in vitro* results,[50,52] demonstrating that SAP P11-4 can remineralize caries throughout the lesion body, even in depth,



t = 0 min

Active carious lesion with a pseudo-intact enamel surface

t = 5 min

The monomer peptide 10^4 technology diffuses within 5 minutes to the depth of carious lesion

t = 5 min

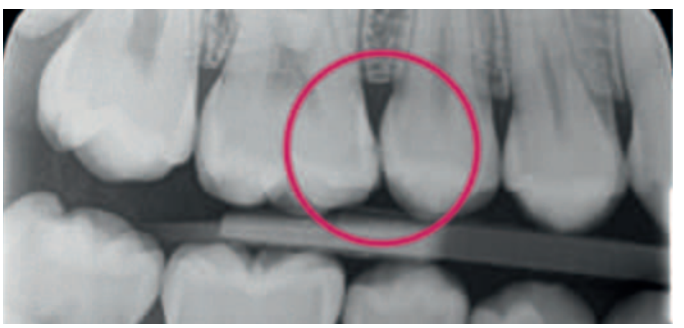
The peptide self-assembles within the carious lesion, forming a biomatrix

t = 5 months

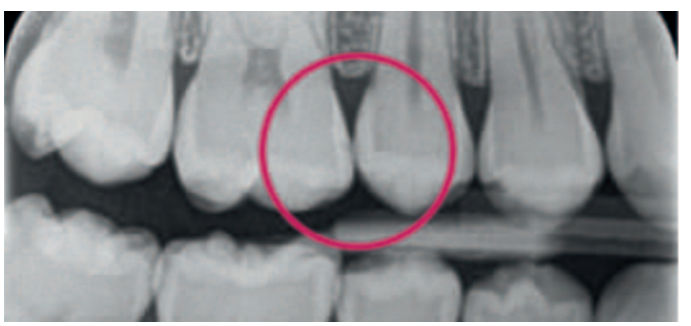
The biomatrix attracts calcium and phosphate ions from the saliva, forming new hidroxyapatite crystals, thus leading to remineralization

Figure 1.22 Curodont™ Repair technology is formulated as a low-viscosity liquid with a high affinity for hydroxyapatite. The unique conditions within carious lesions allow the organization of peptide monomers into a biomatrix. This matrix guides enamel regeneration by acting as a "platform" for the formation of new hydroxyapatite crystals. *Courtesy of vVardis.*

as evidenced by clinical radiographs (Fig. 1.23).
Sedlakova Kondelova and colleagues[61] reported a greater reduction in lesion size with SAP P11-4 compared to a placebo and fluoride varnish (FV), without interfering with the beneficial effects of fluoride. Researchers from two studies evaluating SAP P11-4 in combination with FV for initial caries in children's permanent teeth observed no safety issues. They noted better remineralization, caries inactivation, and stage regression compared to the use of FV alone.[48,59] Several meta-analyses on SAP P11-4 have been published.[62-65]
In a 2023 meta-analysis, researchers reported a decrease in lesion size, improved caries arrest, and positive trends for preventing cavitation compared to controls.[65]
A retrospective study by Godenzi and colleagues[51] on 219 children and adolescents aged 10 to 19 demonstrated that SAP P11-4 can be a safe and effective treatment for initial carious lesions in children, remineralizing to the depth of the defect. This treatment presents no risks, only potential benefits, and can lead to regeneration to a state of health. The introduction of SAP P11-4 into routine dental practice has proven feasible.



Dec 2015
D2/E2/RA2 (Caries extending to the inner half of enamel)



October 2016
D1/E1/RA1 (Regression of caries to the half of enamel)

Figure 1.23 *Courtesy of vVardis.*

PROTOCOL FOR USE

The application of SAP P11-4 can be performed on all non-cavitated carious lesions, regardless of their location: buccal (Fig. 1.24), lingual, occlusal, and approximal.

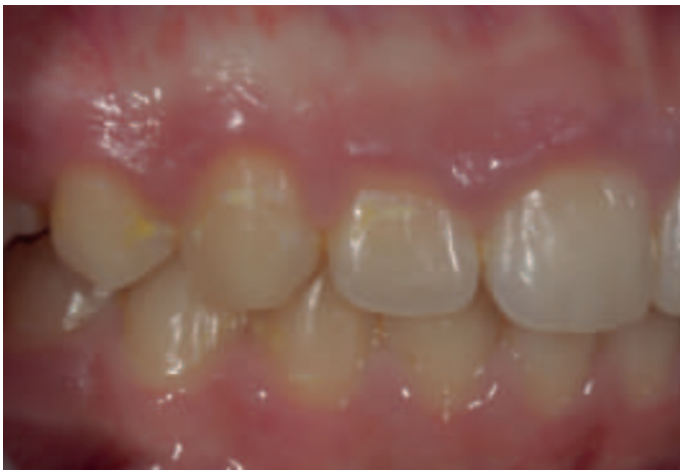


Figure 1.24 Post-orthodontic treatment white spot lesions on teeth 1.2, 1.3, and 1.4.

The entire Curodont™ Repair application process generally takes about 10 minutes. The following are the procedural steps.

1. Perform oral prophylaxis in the area to be treated to remove calculus, plaque and materia alba. Isolate the area by using cheek retractors and cotton rolls. The use of a rubber dam is optional (Fig. 1.25).
2. Clean the tooth surface for 20 seconds with a cotton swab soaked in 2-3% sodium hypochlorite to remove the organic pellicle (Fig. 1.26); then rinse with water.
3. Etch the surface of the initial carious lesion with 35-37% orthophosphoric acid for 20 seconds (Fig. 1.27). For proximal surfaces, an unwaxed dental floss can be used to distribute the etchant in the interdental space. Rinse and dab the tooth dry. Prolonged drying is not necessary.
4. Use a Curodont™ Repair applicator to treat one to three lesions. After extracting the application sponge, press it onto the surface of the lesion to release all the liquid (Fig. 1.28). For proximal caries on adjacent teeth, one applicator can be used to treat two lesions.

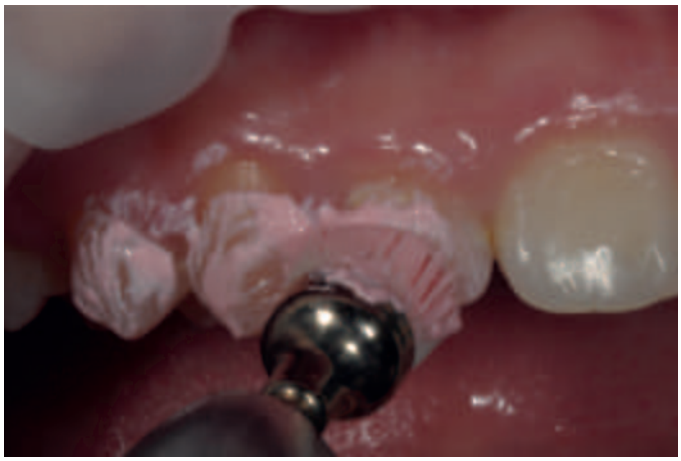


Figure 1.25

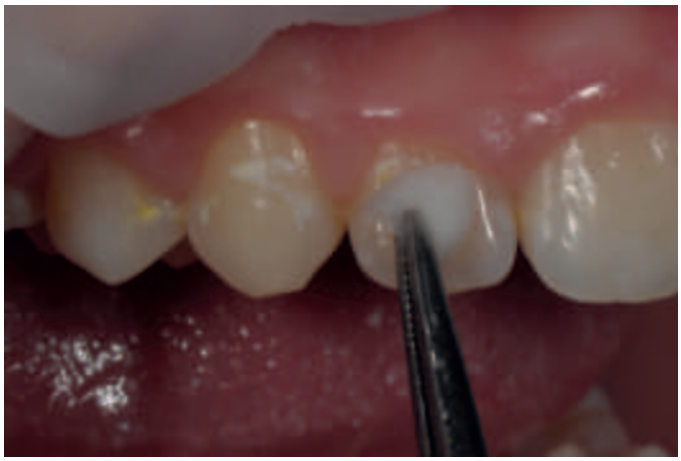


Figure 1.26

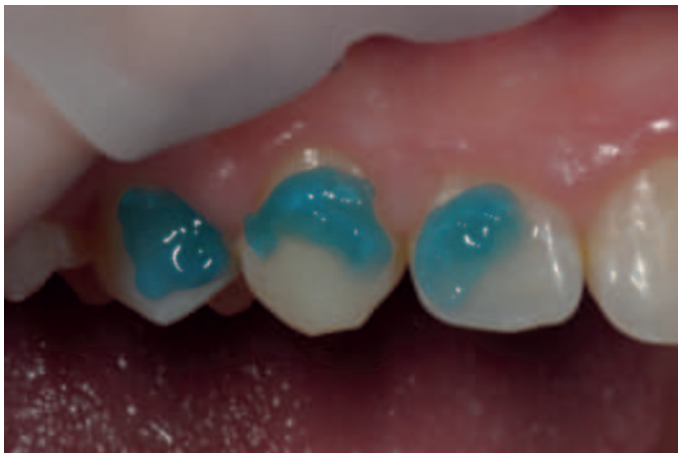


Figure 1.27

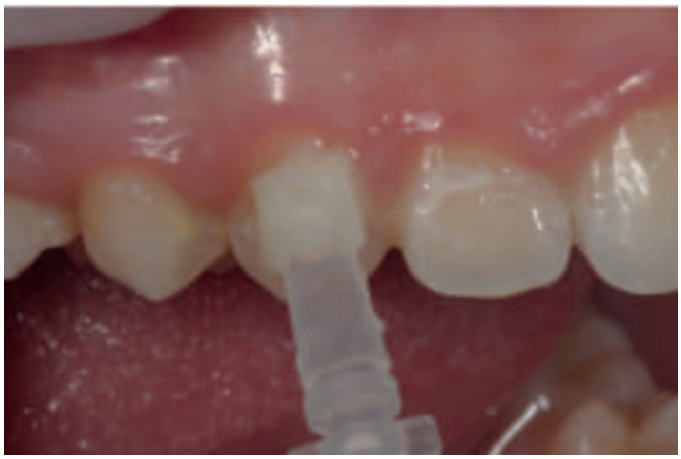


Figure 1.28

5. Wait 5 minutes. Do not rinse or ask the patient to spit. Discharge the patient with routine instructions and emphasize the importance of maintaining good oral hygiene.

A single application of Curodont™ Repair has been shown to promote the arrest and regeneration of enamel within the lesion. However, in high-risk caries patients, such as those with rampant caries, the application may be repeated if deemed necessary by the clinician. Natural enamel formation in primary and permanent teeth takes several months or years, respectively. Since Curodont™ Repair utilizes biomimetic P11-4 technology, which mimics nature to regenerate enamel, the process continues over time. Regeneration progressively increases based on the patient, their habits, and saliva quality. When Curodont™ Repair regenerates enamel within initial carious lesions, the newly formed hydroxyapatite crystals deposit in a fan-like pattern on the biomatrix, which differs from the prismatic structure of natural enamel. This leads to slightly different optical properties compared to those of natural enamel. Therefore, the white appearance of initial caries treated with Curodont™ Repair may significantly diminish but may not disappear completely, especially in particularly deep lesions **(Fig. 1.29)**.
Curodont™ Repair can be used in combination with other remineralizing agents, such as fluoride varnish, to enhance enamel regeneration. The concurrent use of Curodont™ Repair and fluoride varnish has been successfully utilized to achieve greater caries inactivation compared to fluoride varnish alone. However, all treatments should be performed only after an interval of at least 5 minutes from the application of Curodont™ Repair and not before.

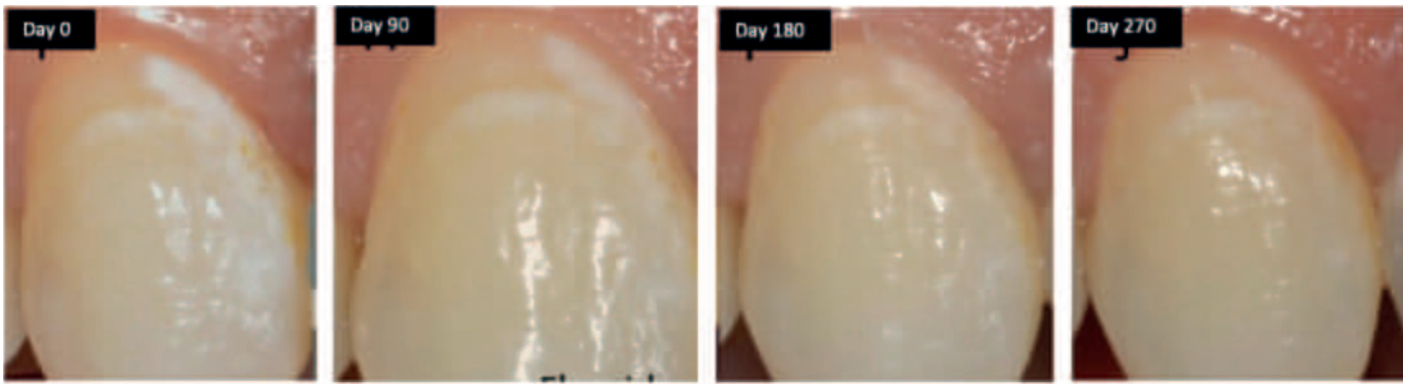


Figure 1.29 Application of Curodont™ Repair on day 0 and subsequent follow-ups over time Although the lesion does not appear to completely vanish, it is evident that its appearance improves, and healthy tissue regeneration occurs, with the inactivation and remineralization of the white spot lesion. *Courtesy of Sci Rep. 2020; 10(1):20211.*

CURODONT™ PROTECT

It is important to emphasize that the success of enamel regeneration protocols is directly related to maintaining good oral hygiene, a proper diet, and an adequate supply of minerals (calcium and phosphate) in the patient's saliva. Along with providing home oral hygiene instructions, the practitioner may choose to enhance the effects of Curodont™ Repair by recommending the use of at-home agents containing calcium and phosphate compounds. These include Curodont™ Protect dental gel. This product is based on the P11-4 peptide technology, presented as a biomimetic matrix that has been further enriched with calcium, phosphate, and fluoride.
Thanks to its high affinity with hydroxyapatite, the peptide matrix adheres to the tooth surface, creating a stable and protective layer. This mineral-rich peptide layer:

1. Protects enamel from acid-induced demineralization, defending against caries and erosion.
2. Attracts ions from saliva, promoting the formation of new hydroxyapatite crystals, which reinforce the tooth by strengthening the natural enamel.
3. Provides the teeth with a noticeably smoother and shinier surface, more resistant to plaque deposition and staining.

Curodont™ Protect is an easy-to-use regenerative gel that can be applied both by dental professionals and by patients themselves.

IN THE DENTAL OFFICE

Curodont™ Protect can be applied by dentists or dental hygienists after a professional dental cleaning or bleaching session. Additionally, it can be applied at the end of each orthodontic check-up visit.

1. Apply Curodont™ Protect to all tooth surfaces using a rubber polishing cup, a microbrush, or an interdental brush.
2. Wait 1-2 minutes.
3. Ask patients to spit out any excess gel if necessary. Rinsing immediately after application is not recommended to maintain the gel's contact with the teeth for as long as possible.

AT HOME

Curodont™ Protect can be used by patients at home once or twice a week after brushing before bedtime, to enhance their oral hygiene routine.

1. Apply Curodont™ Protect with a finger on all tooth surfaces.
2. Wait 1-2 minutes.
3. Spit out any excess gel if necessary. Rinsing immediately after application is not recommended.

COMPLEMENTARY DIAGNOSTIC TOOLS: TRANSILLUMINATION AND FLUORESCENCE

Diagnosing approximal lesions can be complex, sometimes leading to uncertainty among clinicians. Given their location and often poor visibility, especially in the initial stages, it is preferable to supplement the direct examination with instrumental examinations. The use of a bite-wing radiograph is the gold standard for diagnosing approximal carious lesions. Overall, the radiographic clinical examination ensures high specificity but lower sensitivity in the early diagnosis of approximal carious lesions, as indicated by Schwendicke's systematic review. However, good results are shown when clinical examination is coupled with the use of bite-wing intraoral radiographs, which are specifically indicated for diagnosing these types of lesions.[66]
However, it is not always possible to expose the patient to radiographic examinations. Common situations, such as pregnancy, uncooperative patients requiring centering devices, or patients who have already undergone multiple radiographic exams and wish to limit further exposure, need to be taken into consideration.
A valid and highly effective complementary diagnostic tool in these cases is transillumination. Numerous *in vitro* and *in vivo* studies have investigated the effectiveness of transillumination using light beams with wavelengths ranging from 700 to 1,700 nm for detecting primary occlusal and proximal lesions. These studies suggest that transillumination is suitable for the early detection of carious lesions.[67–71] Additionally, Simone et al. concluded that transillumination at a wavelength of 1,310 nm is suitable for detecting secondary carious lesions around composite restorations.[72]

FOTI (FIBER-OPTIC TRANSILLUMINATION)

This is a simple and widely used method that involves exposing hard tissues to a light ray with a specific wavelength. Demineralized areas appear dark due to alterations in light absorption and refraction. Fiber-optic transillumination is especially valuable in the front sections for assessing both approximal carious lesions and the thickness of white enamel lesions **(Fig. 1.30)**.

DIFOTI (DIGITAL IMAGING FIBER-OPTIC TRANSILLUMINATION)

This is an evolution of the previously described approach, incorporating the use of a CCD sensor, which is also utilized in digital radiology. However, in this case, it is coupled with electromagnetic radiation within the visible spectrum, as opposed to X-rays. The sensor captures real-time images from the occlusal, lingual, or buccal aspects of the tooth. The DIAGNOcam (KaVo®) utilizes this technology.

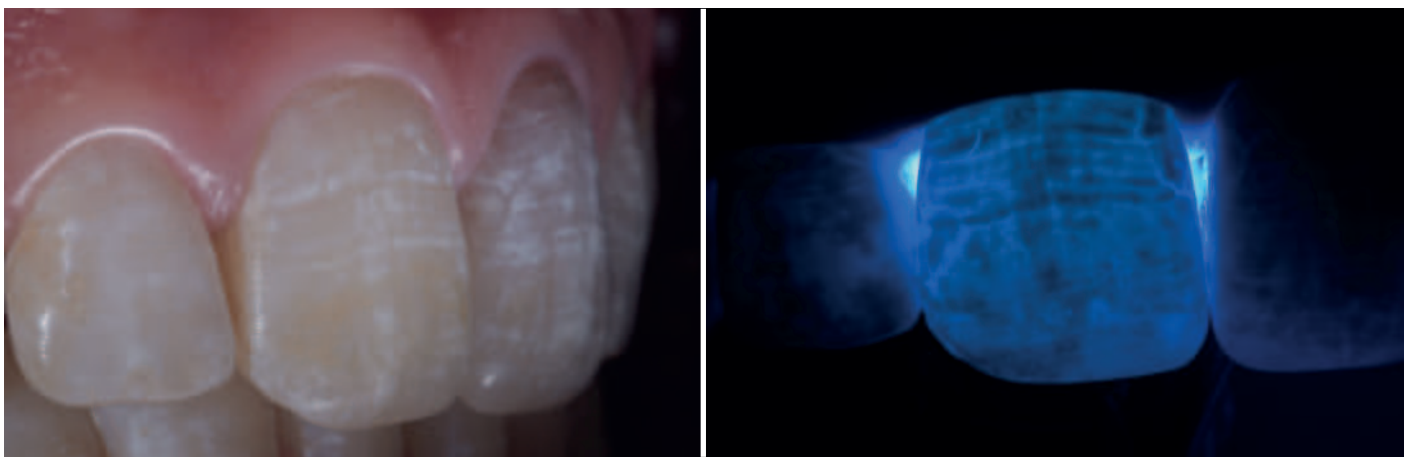
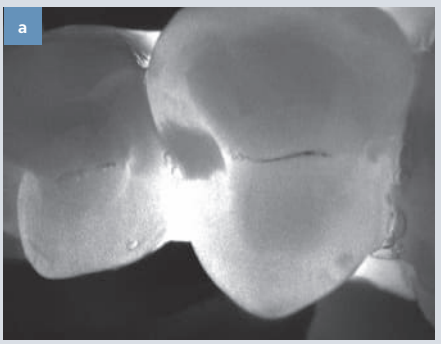
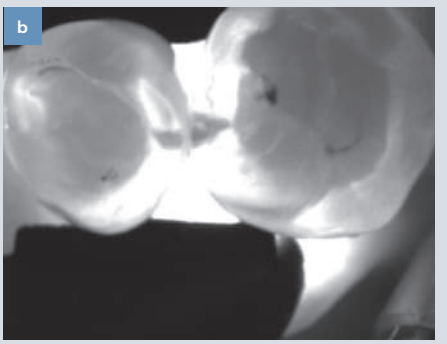


Figure 1.30 Fiber-Optic Transillumination (FOTI). The light source used in the image is a dental curing light. The purpose is to backlight the enamel with a light source, which will transmit through the enamel and highlight any hypomineralized areas, if present. In this particular case, the examination helps to identify the location and extent of fluoride white spots, providing an indication of their thickness. *Courtesy of Dr. Niccolò Rizzi, Florence.*

Figure 1.31

DIFOTI (Digital Imaging Fiber-Optic Transillumination)

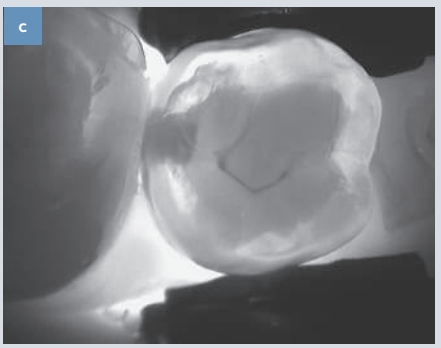


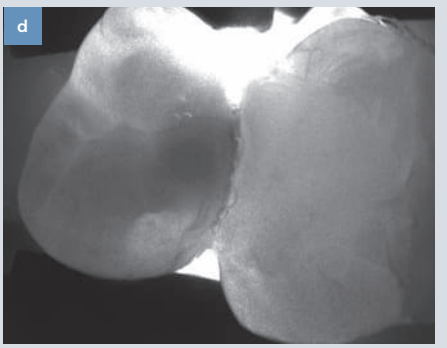


Examples of transillumination photographs taken during the clinical use of the DIAGNOcam (KaVo®).

(a) Highlighting of an approximal carious lesion of type E2.

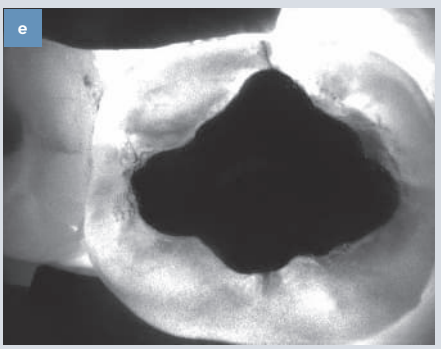
(b) Presence of two E2 lesions affecting the approximal surfaces of two adjacent teeth.

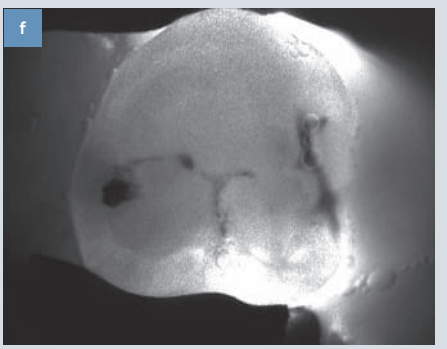




(c) Approximal D1 lesion.

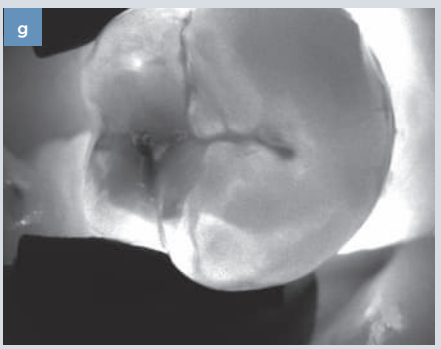
(d) D3 lesion on an upper premolar of a pregnant woman. In this case, it was possible to make a diagnosis without the use of X-rays.

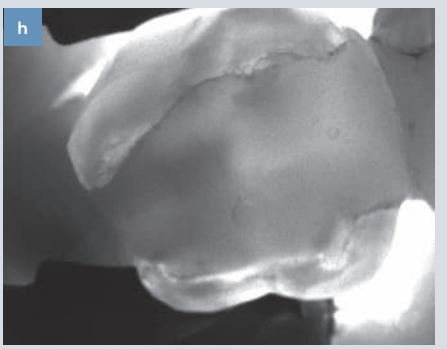




(e) Inspection of the condition of an amalgam restoration.

(f) Image depicting the condition of the pits and fissures on the occlusal surface of a molar.





(g) Highlighting an occlusal cavitated carious lesion and the surrounding demineralized area.

(h) Inspection of the condition of the margins of an old composite restoration.

The principle is straight forward. It is similar to "fiber optic lamps," which enable a light source to transmit light through fibers that illuminate at the ends. Dental components behave in the same manner. Here, there are no glass fibers to transport light; the enamel takes on this function. To utilize this phenomenon more effectively for caries diagnosis, two laser light beams at a wavelength of 780 nm are used to illuminate the inside of the tooth and convey the corresponding transillumination to the sensor. The digital camera captures the image and displays it on a computer screen. Healthy dental tissue simply fluoresces. Conversely, areas that do not transmit light, such as carious lesions, appear as dark spots **(Fig. 1.31)**. Without exposure to radiation, images can be repeated frequently as part of a follow-up protocol. This is especially beneficial for children, pregnant women, disabled patients, and all other individuals for whom radiographs are not feasible. A 2019 study on pediatric patients demonstrated the accuracy of DIAGNOcam in diagnosing caries, showing high sensitivity and accuracy comparable to, if not superior in some cases, to a bite-wing radiograph. This highlights the benefit of using this method as an alternative to administering ionizing radiation in children.[73]

FLUORESCENCE

In addition to transillumination methods, other complementary examinations include fluorescence. The device used is a camera that employs fluorescence technology, with very bright LEDs emitting high-energy violet light onto the tooth surface. Light at this wavelength stimulates porphyrins, which are special metabolites of cariogenic bacteria, to emit red light that contains less energy. Healthy enamel, on the other hand, emits green light. The light signals are captured by high-sensitivity optics and then analyzed by specialized software. In the fluorescent image on the monitor, the porphyrins appear red, making cariogenic bacteria easily identifiable. The denser the colonization, the more intense the red fluorescent signal, indicating a higher presence of caries (tooth decay).[74] There are systems available that rely solely on fluorescence imaging and systems that integrate multiple technologies, such as the KaVo DIAGNOcam Vision Full HD. The innovative 3-in-1 concept of this instrument enables the capture of intraoral, transillumination, and fluorescence images in Full HD quality. With a simple click, three clinically significant images are generated. Thanks to this feature, DIAGNOcam Vision Full HD optimally supports a straightforward, dependable, and patient-focused diagnostic process. The operator can choose between a single-photo mode or a combination of two or three modes, in order to optimize and personalize the workflow according to the treatment process **(Fig. 1.32)**.

Another system based on the principle of laser-induced fluorescence is the KaVo® DIAGNOdent pen. This instrument provides numerical values on a dedicated display rather than images, as in the case of the previously described cameras. The laser light with a wavelength of 655 nm is directed onto the occlusal or proximal surface of a dental structure using a fiber optic probe. The probe reflects the fluorescent light back to the device through specific fibers, and the intensity of the light is measured and converted into a numerical value displayed on a monitor.[75,76] This score correlates with the intensity of the fluorescent light and is converted into a value ranging from 0 to 99 **(Fig. 1.33)**.

The light beam is very narrow, making it easier to read in confined spaces and less accessible areas, as the laser light is reflected by the smallest openings.

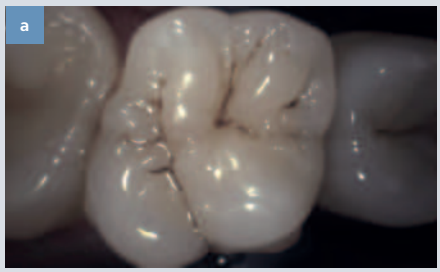
Numerical values ranging from 0 to 7 indicate the absence or low level of bacterial activity, suggesting that no therapeutic interventions are needed other than normal prophylactic measures, such as using fluoride toothpaste. Values between 8 and 15 indicate more intense bacterial activity, which may not necessarily indicate greater invasiveness, but certainly warrant the clinician's attention to intensify prophylactic and/or remineralization processes.

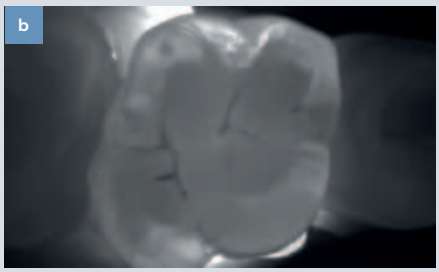
When the instrument shows numerical values above 16, it is necessary to conduct a more thorough assessment to determine the potential presence of cavitation, and conservative procedures should be considered. The absence of a visual image linked to this instrument may be a limitation, but its clinical utility remains essential because of its compact size and ease of use during diagnostic or follow-up procedures.

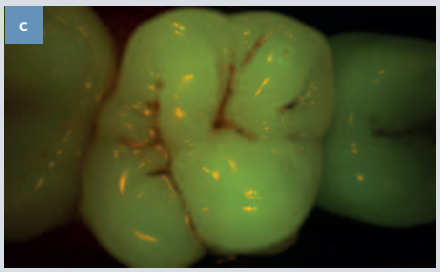
Figure 1.32

Transillumination + Fluorescence

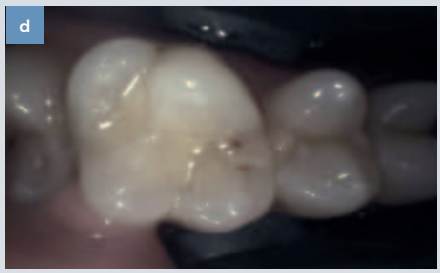
The KaVo DIAGNOcam Vision Full HD enables the simultaneous capture of intraoral transillumination and fluorescence images in Full HD quality. The device's handpiece can capture three different types of images simultaneously with just one click, and it also offers the option to select individual acquisition modes.

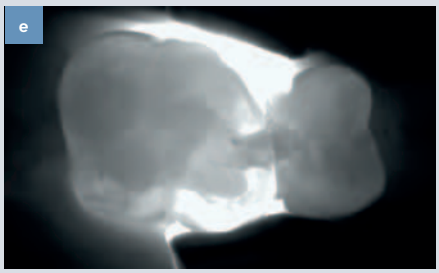


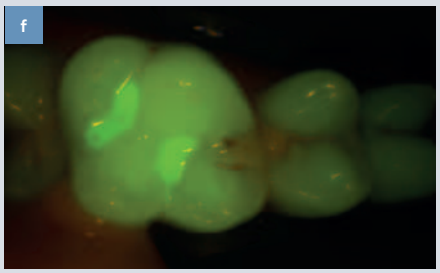




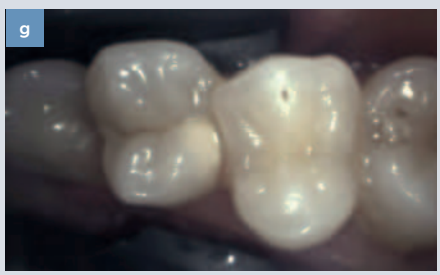
The images demonstrate a sequence of three photos in different modes: normal (a), transillumination (b), and fluorescence (c).

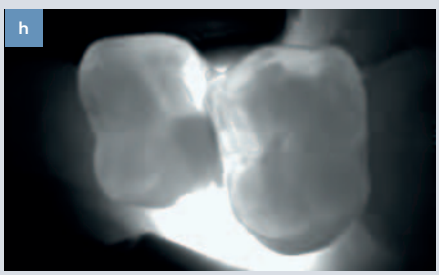


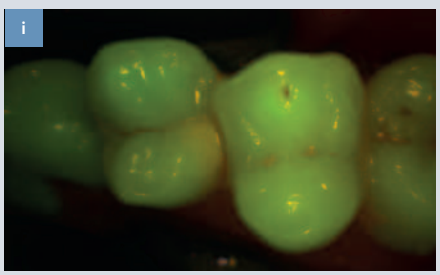




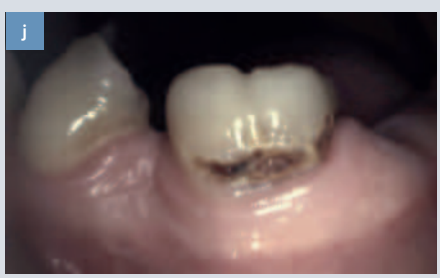
Dark shadows beneath the enamel were highlighted during direct examination (d), D1 approximal lesions were highlighted through transillumination (e), and fluorescence (f).

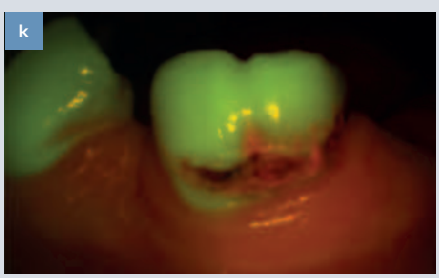




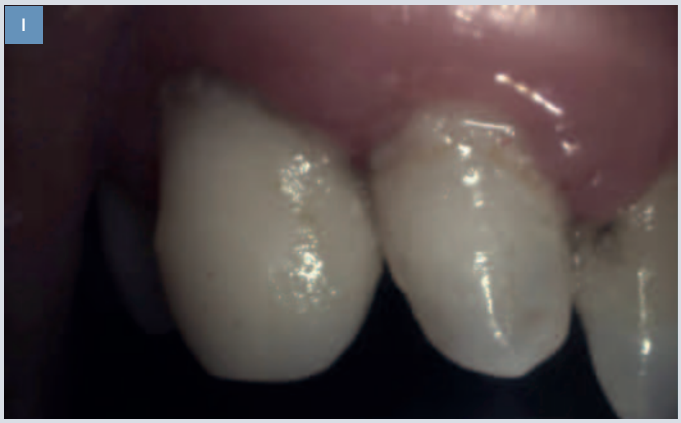


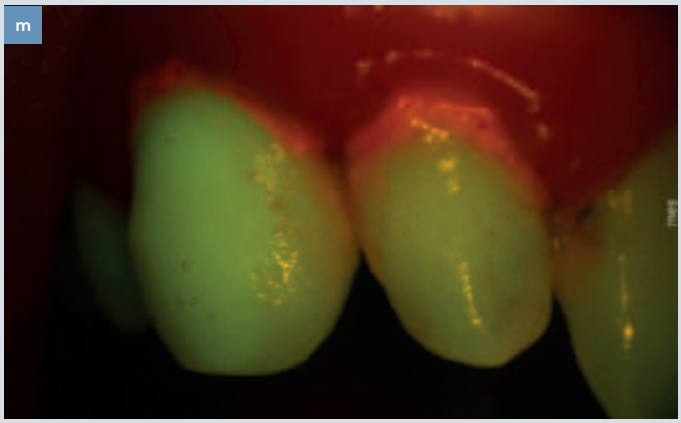
Presence of a significant opacity between teeth on physical examination (g). The transillumination examination highlights a large D2 lesion (h), which is also visible in the fluorescence photo (i).



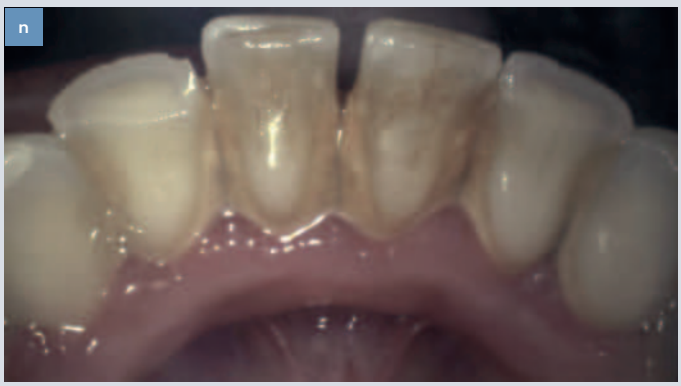


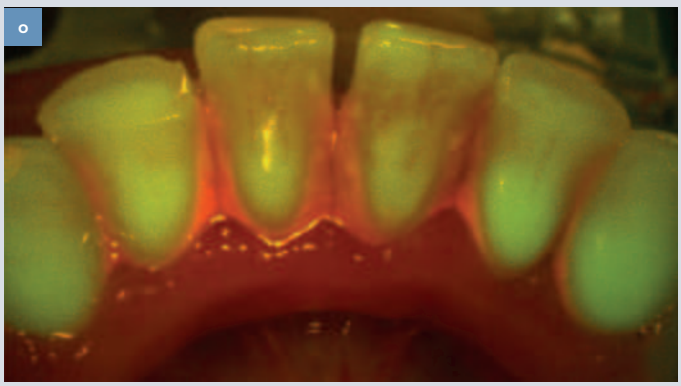
Highlighting a large cervical carious lesion on tooth 3.7 (j) for communication purposes and demonstrating the ongoing bacterial activity in the affected area of the caries process (k).





The DIAGNOcam Vision Full HD is used to display to the patient the areas of biofilm build-up (l) and the bacterial activity highlighted by the fluorescence image (m) during the first visit.





Photographs were taken in intraoral camera mode (n) and fluorescence mode (o) to illustrate biofilm accumulation and ongoing bacterial activity during a session with the dental hygienist.

Figure 1.33
KaVo DIAGNOdent pen. It is a tool that processes only numerical values, not images. 0-7: Indicates no or low bacterial activity; 8-15: Suggests more intense bacterial activity; >16: Indicates possible presence of cavitation.

ONLINE

Scan the QR code to access additional content about the use of the Approximal Icon kit (DMG).