Advances in Restorative Dentistry

Edited by
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Preface

Dentistry has undergone a major transformation over recent years and decades. New technologies have been developed and a better understanding of biological principles and processes has been gained. This book sheds light on these new aspects in preventive dentistry and restorative dentistry.

Advances in Restorative Dentistry gives an overview of current trends in this diverse and important specialist field for dental practitioners. The broad scope of restorative and preventive dentistry is examined in 25 chapters. The following subjects are discussed:

- Structure and pathology of the tooth
- Aspects of prevention
- Caries
- Magnification aids in restorative dentistry
- Damage to adjacent teeth and minimally invasive preparation
- Yesterday retention – today adhesion?
- Bleaching
- Dental erosion
- Endodontology
- Halitosis

The wealth of illustrations and highlighted key sentences make it easy to incorporate current knowledge into daily practice as well as into teaching and study activities.

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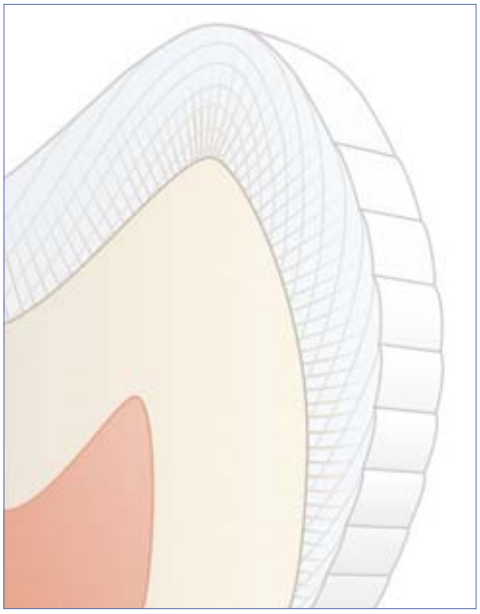


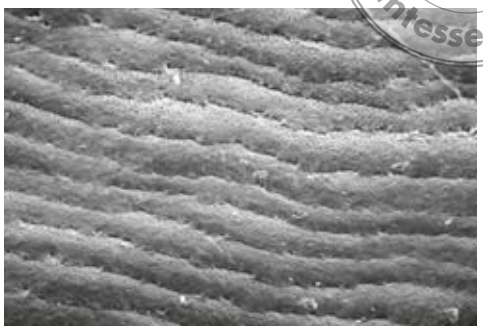


Fig 1-7 **Structural characteristics – enamel**
The periodic laying down of enamel is expressed in the lines of Retzius. Where these lines reach the surface, the perikymata are visible. Viewing the longitudinal and transverse sections of enamel by light microscopy reveals light and dark striae in the inner two-thirds. These Hunter-Schreger bands are caused by the wavelike path of the enamel prisms.

Fig 1-8 **Perikymata under scanning electron microscope (SEM)**
The magnification clearly shows not only the perikymata but also the lines of imbrication running between them.

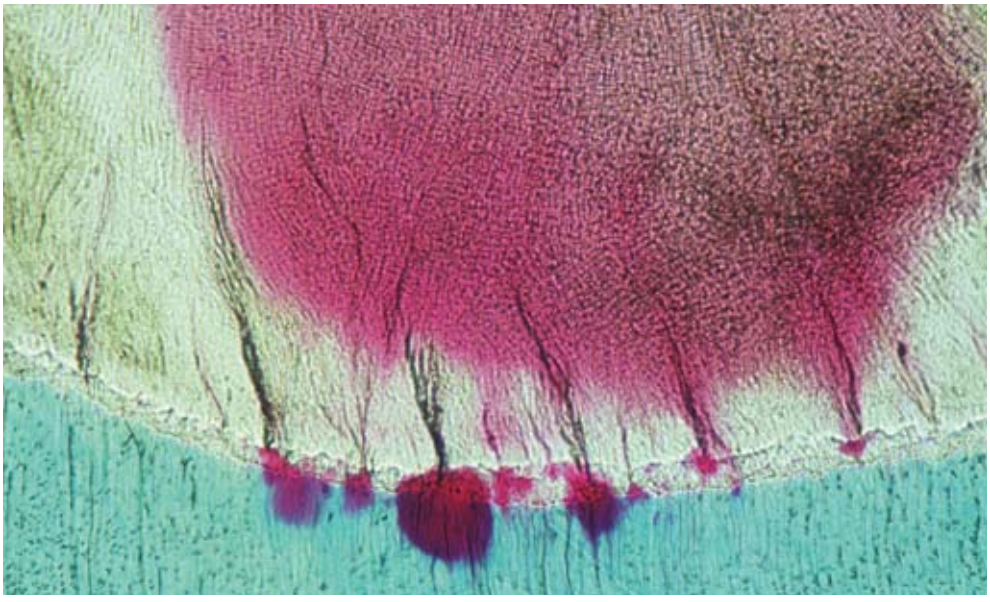


Fig 1-9 **Enamel tuft**
Enamel tufts are hypomineralized areas of enamel which look like tufts of grass under light microscopy. Enamel tufts can provide a location favourable to bacteria in the event of carious attack. Caries is clearly visible in the histologic image.

Fig 1-10 **Enamel pearl**
Left: radiograph of an enamel pearl in the interproximal area of a maxillary molar.
Right: enamel pearl in the bifurcation of a molar.

Structural defects and paraplasias of the enamel

In most teeth, enamel structural defects can be identified by light microscopy. A large proportion of these defects arise during amelogenesis. These include enamel tufts (Fig 1-9) and enamel lamellae. Enamel tufts and lamellae can prove to be the line of least resistance in respect of the spread of caries.

The enamel pearl is a paraplasia of the enamel. This means the formation of enamel in an atypical localization. Enamel pearls can cause isolated periodontitis in the area of the furcation (Fig 1-10).

Dysplasias of the enamel (and dentin)

Dysplasia of enamel and/or dentin can be caused by defects of genes that are responsible for odontogenesis. However, traumatic, inflammatory, and chemical processes as well as metabolic disorders and systemic diseases can also cause malformations of the enamel and/or dentin.

In enamel and/or dentin dysplasias of genetic origin, all the teeth of one or both dentitions are usually affected to a varying degree. They can be inherited from generation to generation, so that similar disorders of odontogenesis can be found in siblings, parents, and grandparents (Figs 1-11 to 1-13, see also Fig 1-19).

Fig 1-11 **Amelogenesis imperfecta, hypoplastic form (pitting type)**
Deposition of exogenous dyes makes the enamel pits in the area of the vestibular surface clearly visible.

Fig 1-12 **Amelogenesis imperfecta, hypomatured form**
Left and right: the enamel is incompletely mineralized. White, opaque enamel areas are visible in the area of the cusp tips and incisal margins.

Fig 8-1 Smooth surface caries with intact surface.

Fig 8-2 Smooth surface caries with local surface breakdown. Left: initial finding. Centre: after 10 years. Right: after 20 years.

Fig 8-3 Smooth surface caries with pronounced breakdown of surfaces.

Pit and fissure caries

Studies have shown that the occlusal surfaces of the permanent molars in children and adolescents are most commonly affected by caries. The proportion of pit and fissure caries in children with minimal caries is between 75 % and 92 % depending on age. Thus pit and fissure caries is bound to be a common diagnosis. There are various possible reasons for the high caries prevalence in fissures:

- Until final occlusion-finding, an increased accumulation of plaque can be seen in the fissures.
- The enamel is prone to caries in the first few years following eruption. Maturation of enamel involves remineralization and demineralization cycles. The reduced susceptibility of mature enamel to caries is not fissure-specific, but makes a greater impact there.
- The unfavorable fissure morphology prevents adequate cleaning of the fissure base and impedes saliva access (Figs 8-4 to 8-7).

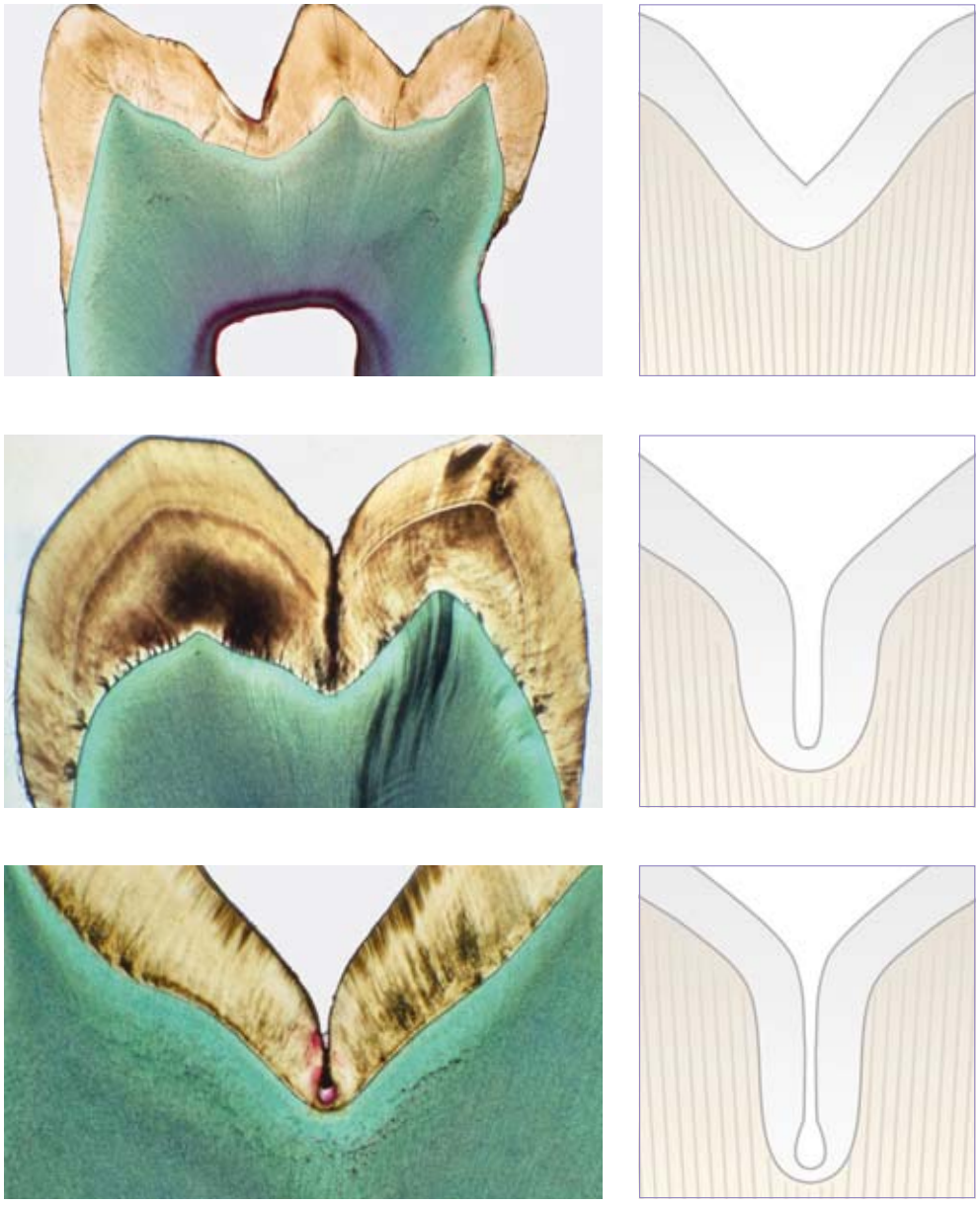


Fig 8-4 to 8-6 *Different types of fissure morphology.*

It is important for the teeth to be cleaned before diagnosis so that white spots at the fissure entrance can be identified (Fig 8-8). If a white spot is already visible before drying, it is reasonable to assume that the caries is more advanced than in a white spot which needs to be dried before it can be detected. This long-known fact was recently systematized with the ICDAS system, one of the aims being to publicize comparable diagnostic criteria in all countries.[5]

Diagnosis is difficult because dentin caries can exist underneath an apparently intact surface. In most cases, however, drying and close inspection will reveal an area of decalcification at the fissure entrance. The frequency of the so called "hidden caries" in molars varies between 10 % and 50 %. It appears to be a direct consequence of suboptimal technique in clinical diagnostics.

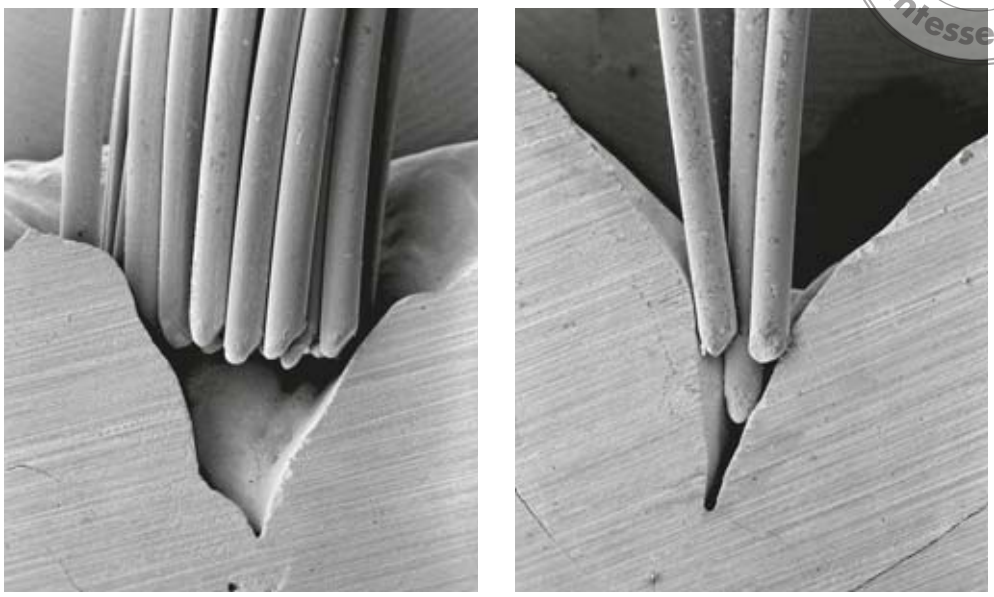


Fig 8-7 *It is not possible to clean the base of the fissure with a toothbrush and individual bristles.*

Fig 8-8 *White spots before and after drying of the fissure.*

The use of a probe does not improve the diagnostic investigation of pit and fissure caries. Furthermore, a disadvantage of probing with pressure is that enamel areas decalcified at the surface are destroyed and this can accelerate the progression of caries. Drying the surface will reveal an area of decalcification that is a definite sign of caries.

Occlusal caries that has penetrated into the dentin can be diagnosed by bitewing radiographs. Dentin caries that is visible on a radiograph but which has an intact surface is generally treated by minimally invasive treatment and restoration (Figs 8-9 and 8-10).

Fluorescence measurement

Tools enabling caries to be detected early, even when the surface is apparently intact, have been sought for a number of years. The systems now available on the market and suitable for daily use take advantage of the fluorescence of dental hard substance that has been altered by caries.

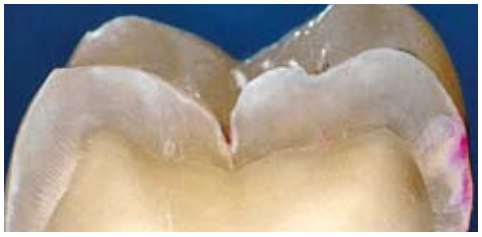


Fig 8-9 *Left: intact fissure surface with discoloration. Right: no caries despite pronounced fissure discoloration.*

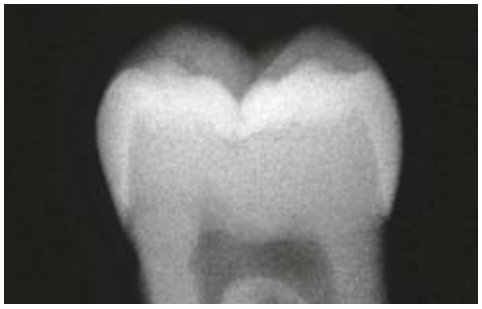


Fig 8-10 *Top left: intact fissure surface. Top right: seemingly, the radiograph shows early radiolucency in the dentin. Bottom: pronounced enamel and dentin caries.*

When radiant energy is applied to a tooth it causes a temporary transition of certain molecules into an excited state. That energy is then released as the molecules return to their initial state; part of the energy is released into the surrounding tissue as heat, while another part is lost as an emission of light, namely fluorescent radiation. The fluorescent light emitted has a longer wavelength (> 680 nm) than the light causing the excitation (655 nm).

This principle was developed into a practical device for caries detection in the form of the DIAGNOdent® (DD) and DIAGNOdent® pen (DD pen) (KaVo, Biberach) (Fig 8-11). Unwanted light is retained by a filter system. An acoustic signal that changes in pitch as the tip of the device is rotated enables the operator to locate the point of highest fluorescence at a specific site without having to look at the display on the device (Fig 8-12). The maximum value is read off after the measuring process. Existing studies prove that the DIAGNOdent based on laser fluorescence has good sensitivity for detecting dentin caries. As previously mentioned, clinical inspection achieves good specificity levels. Therefore, the advantages of the higher specificity and speed of clinical diagnostic examination can usefully be combined with the advantages of this device.

Table 24-2 *Special features of the clinical examination*

Caution is advisable during clinical examination because of particular aspects of some children's behavior. The following points should be looked for.
Is there any swelling of the face and/or lymph nodes?
Are there any intraoral swellings? Do these swellings fluctuate on palpation?
Can fistulous tracts be probed? Does any pus exude?
Is the painful tooth sensitive to percussion? What is the tap note like?
Are there any sore mucosal areas on palpation?
Is there mobility of the aching tooth?
If caries is present, the consistency and color of the carious material are of interest.
If pulp is opened, the size and location of the opened site as well as the extent and nature of the bleeding can provide valuable information about inflammation of the pulp.
The informative value of the CO_2 test is limited in small children because of the lack of cooperation and, for psychological reasons, it should therefore be used sparingly.

Reversible and irreversible pulpitis due to caries

Owing to the large pulp cavity and the minimal thickness of enamel and dentin, caries reaches the dentin close to the pulp after only a short time. Initial signs of inflammation in primary tooth pulp can be observed histologically soon after first contact of caries with dentin. At the beginning, this process is still reversible (reversible pulpitis). However, if the caries advances further, it will result in irreversible spread of the inflammation (irreversible pulpitis). These changes do not always involve severe pain. However, if a primary tooth causes persistent pain and/or pain in response to heat, this means the inflammation has spread to the entire pulp of the primary tooth. Sensitivity to percussion means the inflammation has reached the apical or interradicular periodontium. Clinically, it is often very difficult to distinguish between reversible and irreversible pulpitis, especially because the sensitivity test with cold is not very informative in children. In the same primary tooth, healthy, vital areas of pulp can be observed alongside severely inflamed to necrotic pulp segments (Fig 24-1).

Treatments for reversible pulpitis

Incomplete (stepwise) caries excavation

In the case of a vital, symptom-free primary tooth with profound caries, pulp opening can be prevented by incomplete caries excavation. Preparation and thorough excavation of caries close to the pulp are first performed. The carious residual dentin close to the pulp is left in place. The dentin wound is then cleaned and disinfected (eg, with Tubulicid or chlorhexidine). If disinfecting the cavity with hydrogen peroxide, it is important to make sure that polymerization of acrylic resin can be inhibited. After the carious residual dentin has been

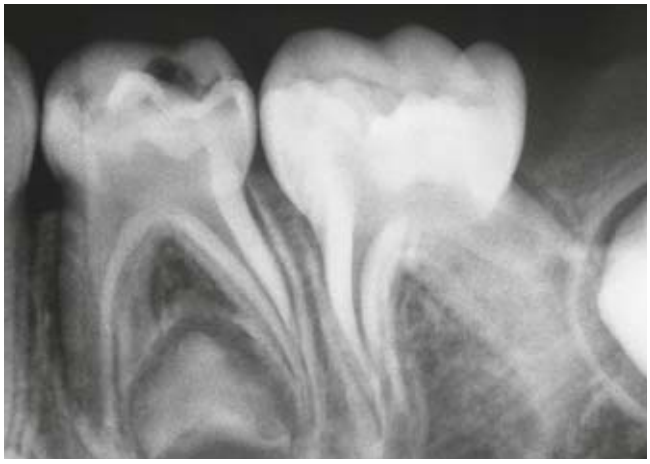
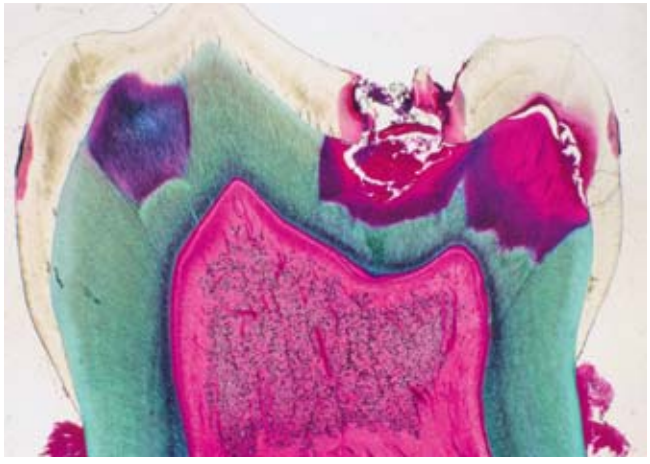


Fig 24-1 *Reversible versus irreversible pulpitis*

Left: coronal radiolucencies, but no interradicular or periapical radiolucencies, can be seen on the radiograph of the primary left mandibular first molar.

Right: in the histological section through the mesial area of the primary left mandibular first molar, apart from widening of the odontoblast border below the carious lesion and slight proliferation of the blood vessels, no inflammatory changes can be seen. Given appropriate treatment, these are reversible.



Left: in the histological section through the middle of the primary first molar, bacterial infiltrates and enlarged blood vessels are visible in the pulp. The entire pulp cavity in the area of the section shows pulpitis changes which are only partly reversible.

Right: the histological section through the distal area of the primary first molar shows massive pulp necrosis with opening of the pulp due to the carious process. The pulpal changes are irreversible.

covered with a glass ionomer cement cavity liner (eg, Vitrebond™), a tight seal is created using an adhesive system and composite resin. Various studies have shown that complete removal of caries is not necessary in deep carious lesions in order to prevent progression of the caries.[17] However, a tight restoration that isolates any bacteria remaining in the cavity is a prerequisite. Individual authors dispense with coverage of the carious residual dentin with a cavity liner.[13] Reopening of the cavity, as has been propounded for stepwise caries excavation, is therefore unnecessary for a symptom-free primary tooth where the restoration is intact.

Direct pulp capping

If the pulp is opened at points during caries removal from a symptom-free, vital primary tooth, direct pulp capping can be carried out. The opened pulp is covered with a calcium hydroxide material. This is followed by the application of a liner, then tight closure with a composite resin restoration. The use of mineral trioxide aggregate (MTA) is another option (though not very economical).

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