

Enamel cracks. The role of enamel lamellae in caries initiation

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Abstract

Lamellae or cracks are distributed throughout tooth enamel in both deciduous and permanent dentitions. While earlier authors postulated that lamellae may be pathways of entry for caries, no evidence was adduced and the theory appears to have been discounted. The present study seeks to show that, at least in some cases, lamellae are permeable to dyes, may be associated with caries initiated in the dentine, supporting the hypothesis of Hardwick and Manly of lamellae penetration by *Streptococcus mutans* and lactobacilli.

The enamel lamellae are shown to be a permeable pathway allowing caries-producing bacteria access to the dentine-enamel junction. Caries can thus be established within the tooth without visible evidence at the surface.

Key words: Enamel cracks, lamellae, caries, bacterial invasion.

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Introduction

The development of quartz-halogen light sources and fibre-optic delivery systems for dentistry have made transillumination a more useful and feasible tool for the examination of teeth in patients.¹ It has also revealed the high incidence of enamel cracks on the proximal surfaces of posterior teeth as these cracks diffuse the light passing through the proximal enamel. Some enamel cracks on proximal surfaces become visible in incident light through the absorption of stain but the great majority become visible only by transillumination. Even where accessible they are not detectable by dental probes. Similar cracks are found on the buccal and lingual surfaces of molars and premolars and on the labial and lingual surfaces of canines and incisors. Lamellae are

frequently found at the base of occlusal pits and fissures.

Bödecker in 1906 was the first to describe these developmental defects of enamel which he named 'lamellae'.² After an extensive study of lamellae, Gottlieb reached the conclusion that they are caused by 'imperfect calcification of enamel tissue' although he was undecided whether they consisted of 'inter-prismatic cementing substance' or of 'uncalcified enamel prisms'.³ Hyatt reported caries of dentine beneath an apparently intact occlusal surface revealed by radiography.⁴ The work of previous authors was reviewed by Thoma who published a radiograph said to illustrate a proximal enamel lamella leading to a large area of destruction in dentine.⁵ Although various authors speculated that lamellae might be the site of entry of caries, Thoma's radiograph was the only direct link between the two.

Manly states that 'the enamel lamellae . . . have long been regarded as possible portals of entry'.⁶ He suggested that lamellae may be considered normal parts of well-developed enamel but also that lamellae are due to 'failure of calcification'. Examination of ground sections and 'decalcified' sections led him to believe that a lamella is a mass of empty prism sheaths and thus lent support to the view of Fish that a lamella is formed by the 'sudden failure on the part of a group of ameloblasts to deposit calcium'.⁷

Boyle stated that the peripheral portion of some lamellae under high magnification exhibited the presence of bacteria;⁸ this was interpreted by Kronfeld as an instance of the earliest onset of caries, a view supported by Bevelander.⁹ Hodson conducted a very extensive study on the microscopic structure of enamel lamellae.¹⁰ He found 'the design of artefact cracks, true lamellae cracks, post eruption cracks and cracks caused by and contributing to

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the spread of caries to be very similar and difficult to distinguish'. As a result of his research, Hodson claimed that any unerupted tooth in which the crown is fully formed demonstrates lamellae similar in type and distribution to those in erupted teeth. He showed a lamella passing through the thickness of the enamel of a deciduous molar at birth; amelogenesis was incomplete. He expressed the opinion that lamellae probably occurred during the time of completion of the matrix and the progress of calcification. Orban postulates three types of lamellae:¹¹ type A composed of 'poorly calcified rod segments', type B composed of degenerated cells, and type C arising after eruption where the crack is filled with mucoproteins from the oral cavity, a situation, he suggested, more common than formerly believed. Orban also postulates that type A is confined to enamel while types B and C may extend into dentine and that all lamellae can constitute a path of entry for the bacteria that initiate caries. Pincus¹² suggested a rather different origin for lamellae – 'an occlusal fissure occurs at the junction of cusps. If then developing cusps fail to coalesce when forming a fissure lined throughout by intact enamel, a gap in the enamel occurs. Such a gap may vary in size from a crack or lamella upwards as shown by microscopy'. Although Pincus applied this hypothesis only to occlusal fissures, this failure to coalesce may well occur where enamel from any two centres of formation meet and thus result in not only proximal lamellae but also in buccal and lingual grooves of molars and on the labial and lingual surfaces of anterior teeth. A lamella at the base of an occlusal fissure provides an eminently suitable pathway for bacteria to gain access to the dentine and initiate caries beneath an apparently intact surface. The surface would appear to be intact if the superficial part of the lamella has become filled with mucoproteins from saliva.¹²

Clinically undetected occlusal caries has been reported by a number of authors.^{13,14} With the decrease in caries incidence in some geographical areas, occlusal caries beneath pits and fissures now forms a higher proportion of the total caries experience so that 'hidden caries' beneath intact surfaces has come into prominence, attracting the names fluoride syndrome and fluoride bomb.¹⁵⁻¹⁸ Weerheijm *et al.* found in a group of 6-18 year olds that 15 per cent of the first and second molars judged clinically sound exhibited bite-wing radiolucencies and that these hidden lesions occurred in children with low DMF scores.¹⁹ Weerheijm *et al.* also drew a bacteriological profile of the hidden lesions which were found to contain *Streptococcus mutans* and lactobacilli in soft, light-coloured dentine, thus classed as active carious lesions.²⁰

Hardwick and Manly showed a photomicrograph of a sound caries-free premolar exhibiting a lamella with an associated lesion in dentine.²¹ They stated further that 'in a clinically caries-free tooth a lesion can be produced in the dentine by micro-organisms which invade and pass down the lamella'.

Despite the volume of research suggesting a connection between enamel lamellae and caries this line of inquiry was not pursued. Ten Cate states quite categorically that tufts and lamellae are of no significance and do not appear to be sites of increased vulnerability to caries attack.²²

The present study was designed to show the significance of the enamel lamellae as sites associated with caries, apparently initiated in the dentine, leading to the supposition that the lamella, in this small incidence of cases, is the pathway for cariogenic bacteria.

Materials and methods

From a collection of wet-stored, extracted human teeth, 50 were selected by examination with a quartz halogen lamp in subdued ambient light as exhibiting proximal enamel lamellae. Radiographs were taken. The teeth were sealed with varnish except in the vicinity of the lamellae and immersed in a variety of stains over various time periods up to 24 hours. The teeth were then washed, mounted in resin and sectioned for optical microscopy.

A further 200 teeth in all were selected as showing by means of transillumination proximal lamellae, labial or lingual lamellae in anterior teeth, or selected for possible lamellae beneath clinically sound occlusal fissures.

All teeth were photomicrographed under incident light and transillumination.‡ The teeth were immersed in saturated Orange-G stain for 24 hours, then washed and the crowns mounted in mounting resin. During these and subsequent procedures the specimens were stored in water at all times to prevent preparation shrinkage cracks from dehydration.

The mounted crowns were progressively sectioned using a diamond saw and those showing stain penetration were examined and photographed. All specimens exhibiting carious lesions patent to the surface were discarded from the study since sections cut at the periphery of an open lesion would present a similar appearance. Serial sections of retained specimens ensured that the enamel was intact superficial to the lesions. For examination under polarized light a selection of sections showing stain absorption were reduced to approximately 100 µm thickness. These sections were then dehydrated in methanol, immersed in quinoline, and examined under polarized

‡Photomacroskope M400. Wild-Leitz, Heerbrugg, Switzerland.

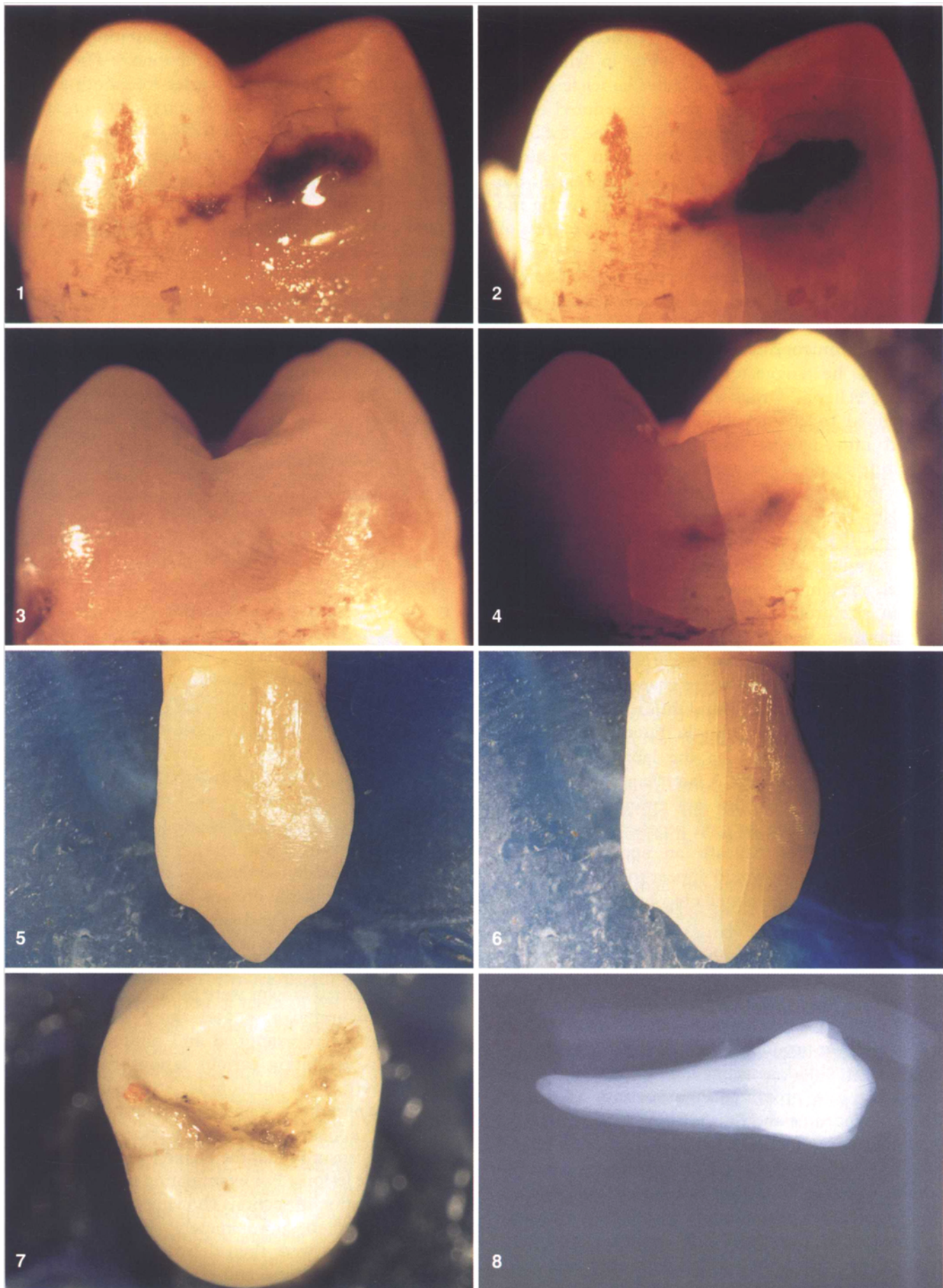


Fig. 1.—Molar photographed in incident light. **Fig. 2.**—Tooth in Fig. 1 photographed with transillumination. **Fig. 3.**—Bicuspid photographed in incident light. **Fig. 4.**—Tooth in Fig. 3 photographed with transillumination. **Fig. 5.**—Cuspid photographed in incident light. **Fig. 6.**—Tooth in Fig. 5 photographed with transillumination. **Fig. 7.**—Occlusal surface of bicuspid tooth. **Fig. 8.**—Radiograph of tooth in Fig. 7.

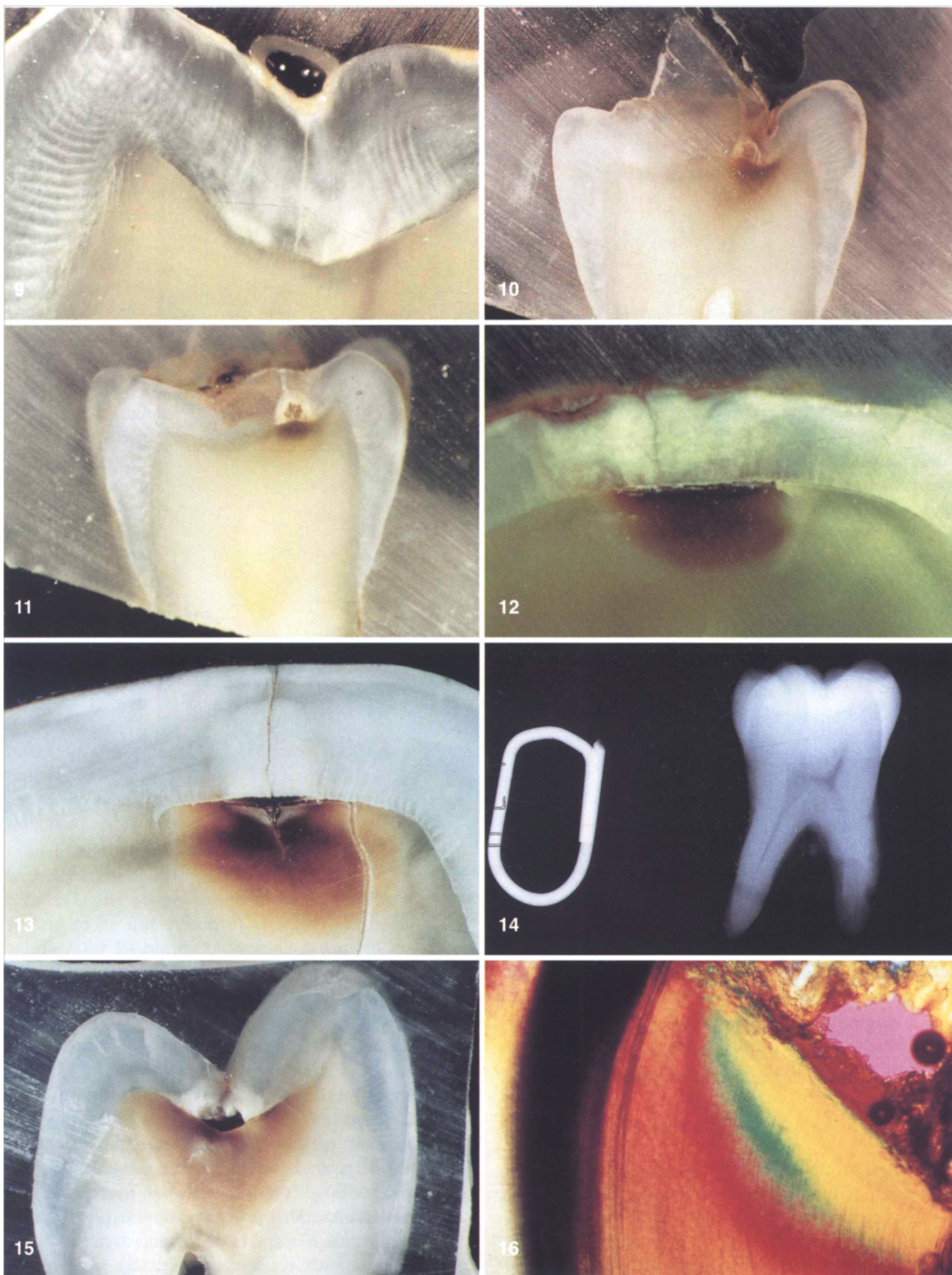


Fig. 9.—Section of tooth in Fig. 7 with lamella transversing intact enamel. **Fig. 10.**—Section of tooth in Fig. 7 with lesion at dentino-enamel junction. **Fig. 11.**—Section of tooth in Fig. 7 with advanced lesion of dentine. (Note that as in Fig. 10, lesion lies beneath intact enamel.) **Fig. 12.**—Cross-section of bicuspid tooth. (This displays both a lesion associated with an enamel lamella and a surface initiated lesion.) **Fig. 13.**—Cross-section of molar stained with Orange-G. **Fig. 14.**—Radiograph of molar in Fig. 13. **Fig. 15.**—Dentine lesion associated with enamel lamella. **Fig. 16.**—Section in Fig. 15 examined under polarized light.

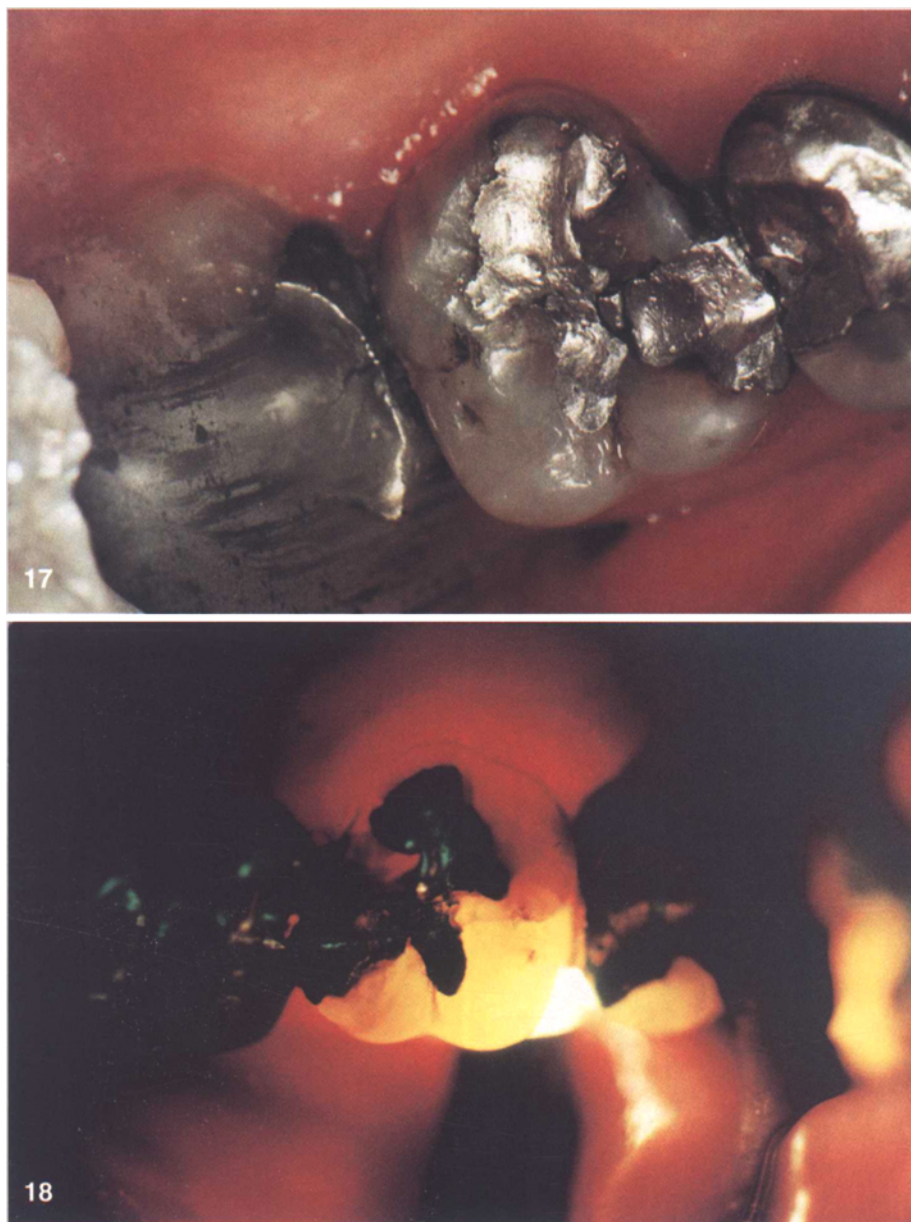


Fig. 17.—Intra-oral view of maxillary molars. (Note lamella transversing distal marginal ridge of first molar.) **Fig. 18.**—Teeth in Fig. 17 photographed with transillumination.

light. Sections from the same teeth were mounted and carbon-coated for examination under the SEM. The changes in enamel and dentine were too subtle to be discernible at the levels used so recourse was made to microanalysis of calcium and phosphorus atomic intensities.

Results

The first group of 50 teeth with proximal lamellae were examined radiographically but the lamellae were not detectable and radiography was discontinued. Transillumination, while an excellent tool for detecting proximal lamellae, did not disclose occlusal lamellae.

The illustrations characterize the experimental results obtained. Figures 1, 3 and 5 show (respectively) molar, bicuspid and cuspid teeth

photographed under incident light, while in Fig. 2, 4 and 6 enamel lamellae in the same teeth are revealed by transillumination. Figure 7 is of the occlusal surface of the bicuspid tooth shown in the radiograph in Fig. 8. Figures 9, 10 and 11 are representative of serial sections of the tooth in Fig. 7 and 8. Together they show a lesion at the dentino-enamel junction associated with a lamella in intact enamel. Figure 12 depicts a cross-section of a bicuspid tooth with a dentine lesion associated with enamel lamella. Also present is a separate surface-initiated lesion. Figure 13 is a stained cross-section of the molar shown radiographically in Fig. 14. Neither the dentine lesion nor the effect on the enamel are detectable in the radiograph. Figure 15 shows a dentine lesion associated with an enamel lamella; Figure 16 was photographed from the same

section under polarized light. Figure 17 is an intra-oral photograph of maxillary molars in a woman aged 73 years, the area of interest being the lamella traversing the distal marginal ridge of the first molar and shown under transillumination in Fig. 18.

No structural changes were detected by the SEM at the level of magnification employed. The specimens were then subjected to microanalysis on the same machine employing the EDAX DxAuto Quantitative Analysis software. With respect to both calcium and phosphate ions, consistently significant results were not obtained; this suggests that the changes in atomic intensities were too subtle to record at such an early stage of the caries process.

Discussion

Clinical examination by transillumination discloses three types of enamel cracks. Firstly the surface crazing of enamel thought to be due to thermocycling and typically seen on incisor teeth. Secondly the transverse or oblique cracks of enamel as a result of trauma, typically seen in incisors and cracked cusps. Thirdly the developmental lamellae which extend the full length of the axial surface. Earlier authors postulated that lamellae may penetrate the dentine-enamel junction and run into dentine.²⁻⁸ In the 300 lamellae studied in this research, in no case did the lamella enter dentine. Indeed, it would be quite unexpected that a developmental fault in enamel formed by ameloblasts should be continuous with a developmental fault in dentine formed by odontoblasts unless caused by a persistent discrete local cause; in no case was this seen in this sample.

In a pilot study, dye penetration was observed with Orange-G, Procion Red and Methyl Blue; the most effective technique to disclose dentine lesions proved to be immersion in Orange-G concentrated solution for 24 hours. Orange-G is a mono-azo dye of molecular number 460.

The specimen in Fig. 13 was inadvertently allowed to dehydrate during the examination phase with the development of the artefact cracks exhibited in the photograph. Since earlier studies were performed on extracted teeth or ground sections, it is suggested that many of the cracks observed were due to dehydration or flexure during extraction. In this study, all specimens were wet-stored except those prepared for SEM studies and that in Fig. 13 and only in these did artefact cracks subsequently appear. It is suggested that the term 'lamella' should be reserved for the developmental fault of enamel which extends the full length and thickness of enamel.

It is argued that at least some lamellae are prone to bacterial invasion by caries-causing bacteria. The

mechanism of lamella invasion on an intact surface followed by proliferation at the dentino-enamel junction explains the clinical features of hidden caries.^{19,20} A similar situation is seen by the clinician in proximal preparations in posterior teeth where caries has been diagnosed by radiography beneath an apparently intact surface.

Because of the unknown histories of the teeth available for this study it was judged that statistical analysis was not appropriate. Proximal lamellae can be detected usually by transillumination but beneath occlusal fissures only sectioning of extracted teeth will disclose lamellae. Further the presence of an open carious lesion or a previously placed restoration precludes the determination of the possible occurrence of a lamella in either proximal or occlusal surfaces.

The quandary for the clinician lies not only in detecting lamellae but also in deciding which lamellae need treatment and how best to treat them. It is suggested that three different stages or types of lamella can be distinguished, each with its own treatment needs.

Firstly, it is reasonable to assume that lamellae in proximal enamel which are without stain have not been invaded by bacteria. The clinician may elect to not treat, to administer local fluoride, or at most to use a smooth surface sealant treatment as described by Pitts and Longbottom.²³

Secondly, stained lamella revealed by transillumination can be considered permeable or permeated. It is suggested that these teeth be treated by tunnel or the internal cavity preparations to place fluoride-releasing glass ionomer at the dentine enamel junction in order to block the ingress of cariogenic bacteria through the lamellae.

The third type of lamellae is that where a 'shadow' is cast with transillumination. This is caused by carious changes at the dentino-enamel interface. This third stage of lamellae requires treatment as the caries process is already established at the dentino-enamel junction. It may be treated by the tunnel preparation, by the 'internal cavity' of Hunt, or by a classical class II amalgam filling.²⁴

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