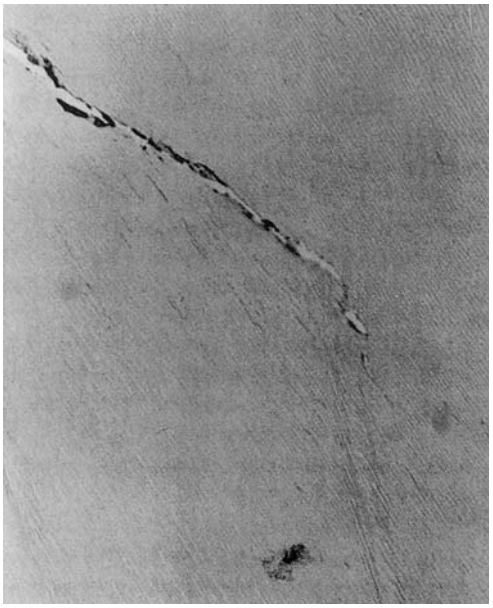


Fig. 1.30 Section from a tooth with an enamel–dentin crack filled with bacterial plaque. The crack runs at an angle to the dentinal tubules, and many tubules which are opened up by the crack contain bacteria as well (Brown–Brenn stain).

an opinion of its severity until the tooth gives a negative sensitivity response which suggests pulp necrosis. The therapeutic implications of this unclear diagnostic situation are discussed in Chapter 5.

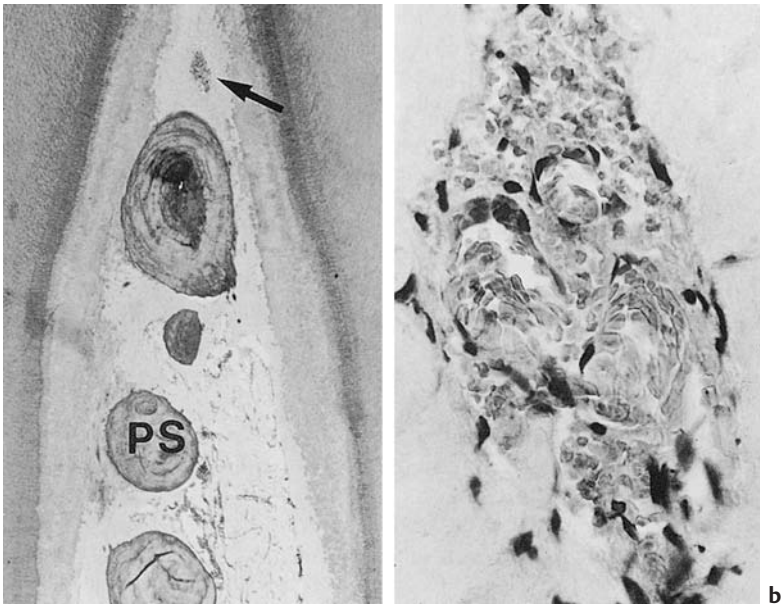
Dentin Exposed to Oral Cavity

Other etiological factors leading to an infectious pulpitis are conditions that contribute to the exposure of the dentin and dentinal tubules to the oral environment. With the refinement of foodstuffs, severe *attrition* is not as common as before. On the other hand, *abrasion*, particularly as a result of tooth-brushing, is becoming a more serious problem. *Erosion* leading to exposure of dentin was traditionally seen in individuals employed in certain chemical industries. Presently it has become a serious problem in the young generation from excessive intake of sweet and acid soft drinks. Nevertheless, it is periodontal disease and the extensive treatment of this disease that today particularly leads to exposure of dentin. Gingival recession is commonly seen in these patients and the root cementum and peripheral layers of the root dentin are being removed during scaling and root planing. It has also been shown that the so-called enamel cracks that are present in most teeth do not necessarily end at the enamel–dentin junction, but rather extend deep into the dentin (**Fig. 1.30**). These cracks are filled with plaque and microorganisms, and sometimes defects reminiscent of carious lesions are seen at the bottom of such cracks. Since the cracks usually run at an angle to the dentinal tubules, a large number of tubules may become exposed to the oral environment by a single crack.

Irritants from the plaque and saliva may reach the pulp through the exposed dentinal tubules. This is shown by the fact that mineralized deposits are laid down in the exposed tubules. The intratubular deposits are characterized by the large, needle-like hydroxyapatite crystals and the rhomboidal whitlockite crystals seen peripherially in the tubules of carious lesions (**Fig. 1.28**). However, only occasionally are exposed tubules fully occluded as seen in age-changed root dentin and in the sclerotic dentin of carious teeth (see **Fig. 1.6**). Secondary dentin forms as a result of external irritation in all teeth with exposed dentin. The secondary dentin is usually more regular than in carious teeth, but is still characterized by morphological irregularities with a varying number of tubules and with inclusions of functioning blood vessels and strings of soft tissue (**Fig. 1.31**). Pulp stones may be observed in the coronal pulp of these teeth as well, which have possibly developed as a result of external irritation.

In spite of the mineral deposits in the tubules of the primary dentin and formation of often large amounts of secondary dentin, the exposed dentin remains as a rule partially open to the mouth and does not fully protect the pulp from exogenous irritants. In the pulp of these teeth the odontoblastic layer is reduced or missing (**Fig. 1.31**). Circulatory disturbances are evidenced by hemorrhages and disintegrating erythrocytes, and the blood vessels are few and prominent. The most conspicuous finding in teeth with long-standing exposed dentin is a fibrosis of the pulp (**Fig. 1.32**). Large bundles of collagenous fibers are seen in the affected area of the pulp, often in continuity with the secondary dentin. Frequently, fiber bundles may be mineralized.

Connective tissue cells, and sometimes lymphocytes, macrophages, and plasma cells are seen between the fiber bundles. However, a possible in-



a b

Fig. 1.31
a Pulp horn from a tooth with exposed dentin. Large amounts of secondary dentin as well as pulp stones (PS) are present. Functioning blood vessels are seen deep inside the secondary dentin (arrow).
b Higher magnification of blood vessels in secondary dentin (hematoxylin-eosin).

flammatory reaction is mild and will not lead to pulp necrosis. The exception lies in teeth with severe periodontal disease and pocket formation to the foraminal areas of the roots. In such teeth a retrograde pulpitis may develop (**Fig. 1.33**). In a single-rooted tooth this condition will lead to disturbances in the blood supply to the pulp relatively quickly, resulting in total pulp necrosis. In teeth with multiple roots, a retrograde pulpitis in one root will spread slowly in a coronal direction, and it may take a long time before the entire pulp becomes necrotic.

Traumatic Injuries

Traumatic injuries to the teeth and jaws will often result in pulpitis and pulp necrosis. A *complicated crown fracture* with pulp exposure will result in an infectious inflammation of the pulp. The actual crown fracture invariably leads to a hemorrhage in the pulp subjacent to the exposure. The blood clot is an excellent substrate for bacterial growth, and the microorganisms of the plaque accumulating on the fractured surface will readily invade the pulp. A local inflammation is seen in the tissue near the exposure after 2 – 3 days, and total pulp necrosis in such teeth has been observed as early as 7 days after the injury (**Fig. 1.34**). However, in some instances it may take weeks before total necrosis is seen.

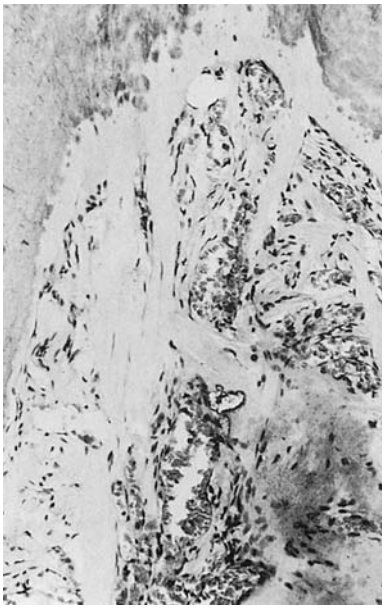


Fig. 1.32 Pulp horn from a tooth with exposed dentin. Large fiber bundles, few and dilated vessels as well as a calcified area are seen in the pulp tissue (hematoxylin-eosin).