Preface to the Third Edition

The scope of the third edition of *Clinical Endodontics* is as before to be a simple, yet comprehensive textbook in endodontics that serves as an introductory text for dental students and as a suitable refresher source for general practitioners, postdoctoral students, and endodontists. With this concept in mind, *Clinical Endodontics* summarizes the biology of the endodontium and the apical periodontium and deals with the etiology and pathogenesis of endodontic diseases. Examination methods, diagnoses, treatment principles, and prognosis of endodontic treatment are discussed, and main endodontic techniques are described. The format of the previous editions has been kept, and new relevant information has been added to the text. The lists of references following each chapter have been updated. I again extend my thanks to friends and colleagues who have contributed illustrational material to the book.

Oslo, Summer 2008 *Leif Tronstad*

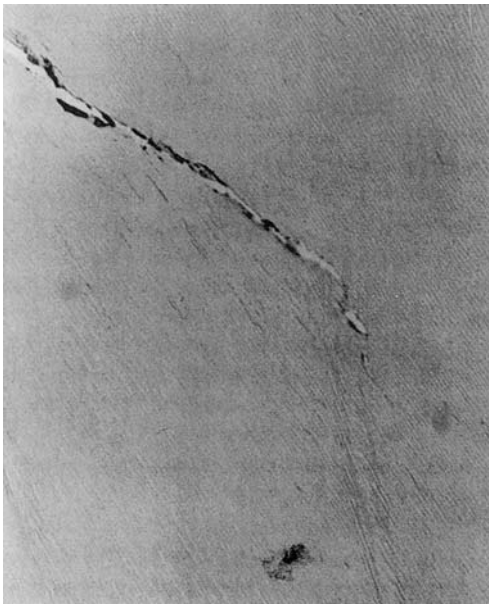


Fig. 1.30 Section from a tooth with an enamel–dentin crack filled with bacterial plaque. The crack runs at an angle to the dentinal tubules, and many tubules which are opened up by the crack contain bacteria as well (Brown–Brenn stain).

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.

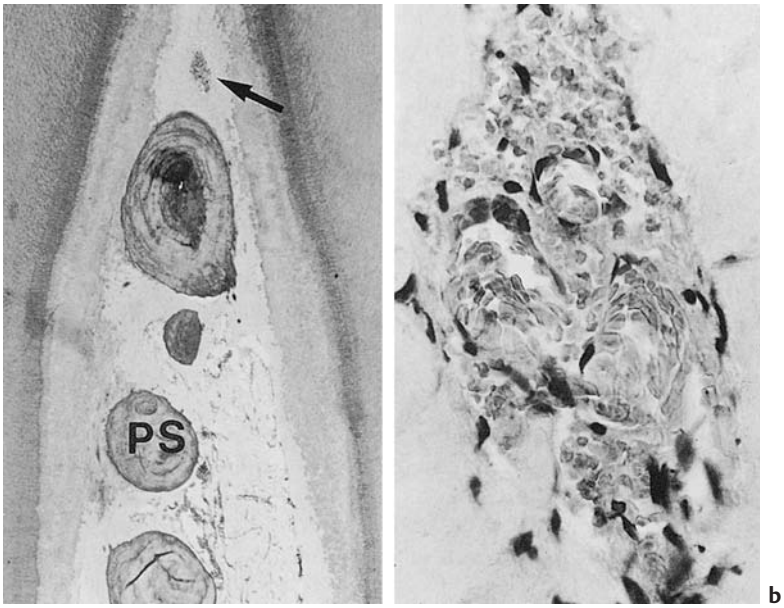
Dentin Exposed to Oral Cavity

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.

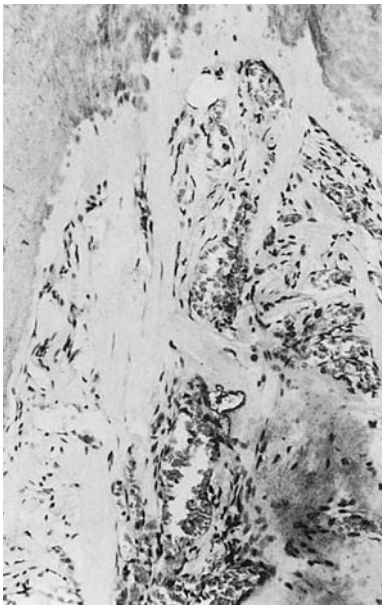


Fig. 1.32 Pulp horn from a tooth with exposed dentin. Large fiber bundles, few and dilated vessels as well as a calcified area are seen in the pulp tissue (hematoxylin-eosin).

4
Endodontic Examination and Diagnosis

As was mentioned above, as many as 90% of patients with oral or maxillofacial pain should be examined for endodontic diseases. A correct diagnosis is arrived at by combining information from the patient and sometimes the patient's physician with the actual clinical findings. Although the diagnosis may often appear obvious and straightforward, the clinical examination should always be thorough and systematic. Only then can the dentist's knowledge and clinical experience be fully utilized. A good chart system is invaluable in this regard.

History

Taking the history is an important part of the clinical examination, often giving information that immediately points to the patient's illness. Since there are virtually no systemic contraindications to endodontic treatment, the medical history can normally be short and of a summary nature. However, it is necessary to form a picture of the general health of the patient, and not infrequently this information will contribute directly to the dental diagnosis. The following questions must be included in a medical history:
Do you have heart problems?
Are you allergic to anything?
Are you diabetic?
Have you been infected with hepatitis or HIV?
Are you taking any medication at the moment?
What is your physician's name, address, and phone number?

Depending on the patient's answers, the history will develop in as great a depth as is deemed necessary. Do not hesitate to call the patient's physician for information, and with obviously sick patients it is advisable to treat them with the knowledge of and in cooperation with their physicians.

The *dental history* should identify the reason why the patient is seeking dental care (*chief complaint*). First the patient must be allowed to describe this reason *in his or her own words*. Afterward the dentist asks necessary leading questions to expand on the information given by the patient. During the interview the patient usually remembers additional information and a rather complete picture of the patient's problem will develop.

Clinical Examination

The clinical examination begins with an extraoral inspection while taking the patient's history. The examination is then extended to the area the patient has referred to in the history and to adjacent and contralateral areas. One quick glance may be enough to arrive at the diagnosis and to decide on appropriate therapy. For example, a tooth may be so damaged by caries that a remaining root rest will have to be extracted. Other times visual findings like caries, discolorations, swellings, fistulae, etc. may lead to other considerations. The clinical examination then continues with the aid of a mirror, explorer, periodontal probe, and other instruments and devices as found to be practical or necessary for a general oral/dental examination.

Percussion and Palpation Tests

The percussion test is a simple but extremely useful examination method which is used to ascertain an inflammatory condition in the apical periodontium of a tooth. The handle of a hand instrument, usually a mirror, is used to tap on the teeth in a *vertical direction.* A tooth with symptomatic apical periodontitis will then be more sensi-

tive to the percussion than the contralateral or neighboring teeth. Only remember, healthy teeth may be somewhat sensitive to percussion as well and if the symptoms from an apical periodontitis are weak or uncertain, it is important to test several teeth repeatedly in no special order to ensure consistency in the observations. Pulpal diseases cannot be revealed by means of the percussion test until the apical periodontium is involved. On the other hand, a pe riodontal abscess or even asymptomatic periodontal disease with severe loss of marginal bone may render teeth tender to percussion. However, such teeth will react mostly to *horizontal percussion*, i.e., percussion perpendicular to the long axis of the tooth and less to vertical percussion like endodontically involved teeth. The percussion test is the examination method that will first give *clinical indications* of an apical periodontitis.

Palpation is performed to ascertain tenderness, swelling, fluctuation, hardness, and crepitation in underlying tissues. Here again it is important to make comparative examinations of neighboring and contralateral areas. Intraorally the palpation test preferably is carried out with the tip of an index finger. Sometimes and in special areas like the floor of the mouth it may be practical to use the index finger on both hands. During extraoral palpation of lymph nodes, swellings, sinus, and tempero-mandibular joint areas, etc., two to three fingers on one or both hands are used. The palpation test may be very useful during an endodontic examination and its usefulness will only increase with increasing skill and clinical experience.

Sensitivity Tests

Sensitivity tests are performed in an effort to determine whether the pulp of a tooth is vital or not. This test is often of crucial importance. The sensitivity of a tooth can be tested in many ways. Normally heat, cold, or electrical stimulation are used. With all test methods it is imperative that the tooth to be tested be clean and completely dry so that the possibilities for conduction of the stimulus to nerves of the gingiva and the periodontal ligament are minimized. For a heat test, temporary stopping (gutta-percha) may be used. It is heated over an alcohol flame and with a plastic instrument applied with slight pressure to assure good contact to the incisal edge or to the buccal surface of the tooth away from the gingiva. It is immediately removed if the patient shows a reaction, otherwise it is left in place until it cools off.

A *cold test* can be performed with ice, but more effective are frozen sticks of carbon dioxide (–78 °C). Effective tests can also be carried out with a cotton pellet sprayed with difluorodichloromethane (DDM) (–50 °C), which is *immediately* placed in good contact with the incisal edge or buccal surface of the tooth. The cotton pellet is removed if there is a reaction, or it is left in place until it has lost its effect.

For *electric sensitivity testing* a special apparatus, an electric pulp tester, is required. The electric pulp tester is equipped with an electrode that is placed in contact with the tooth to be tested as described for the thermal tests (**Fig. 4.1**). An electric current can then be supplied to the tooth through the electrode, and the current (or the voltage) is increased manually or automatically until the patient shows a reaction. The electrode is then immediately removed. Otherwise the test

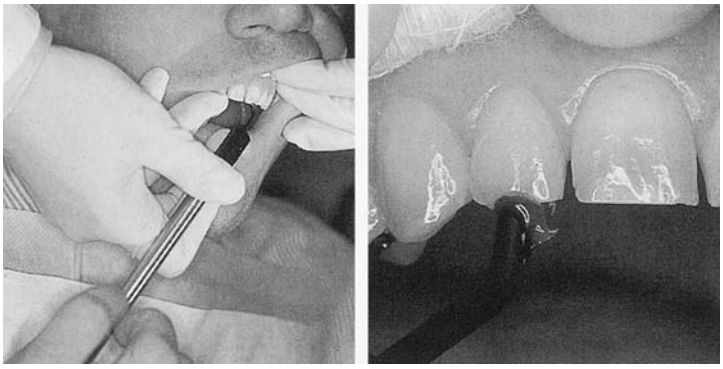


Fig. 4.1
a Electric pulp tester in use. Since the dentist wears rubber gloves, the patient needs to touch the handpiece of the testing device to complete the electric circuit.
b Test electrode in contact with the incisal edge of a tooth. Toothpaste is used to ensure good contact.

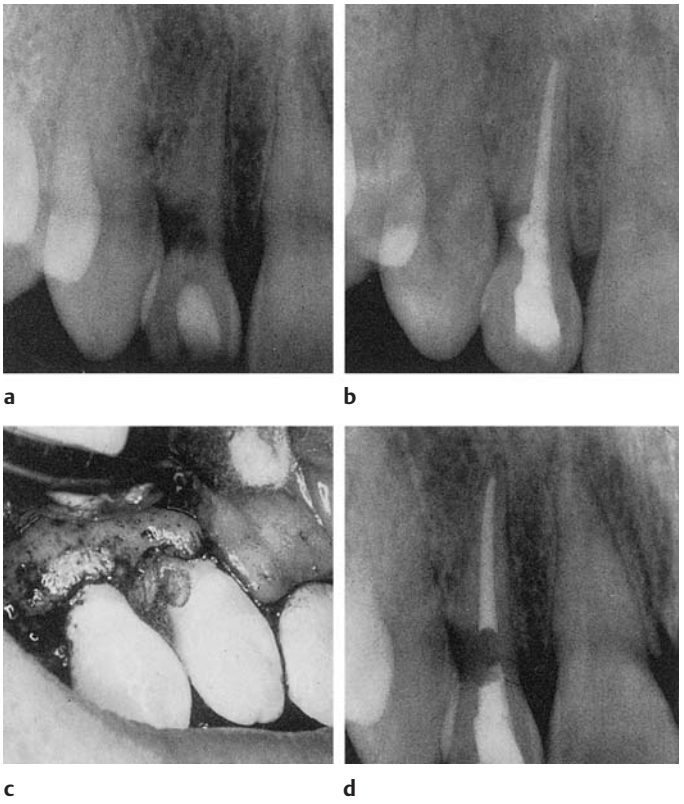


Fig. 8.13
a Radiograph of a maxillary lateral incisor with cervical root resorption. The patient presented with severe symptomatic pulpitis because of exposure and infection of the pulp through the undiagnosed resorption cavity.
b Pulpectomy with root canal filling is performed.
c The cervical resorption lacuna is exposed, cleaned, and restored with the acid-etch resin technique.
d Postoperative radiograph (radiolucent resin was used for cervical restoration).

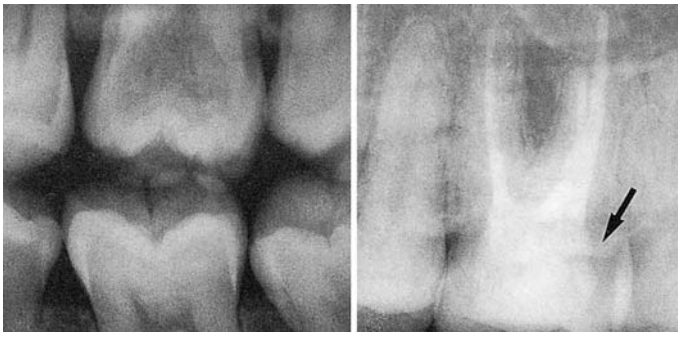


Fig. 8.14
a Radiograph of a maxillary first molar with a cervical resorption lacuna encompassing the greater part of the coronal dentin.
b Following pulpectomy and root canal filling, the pulp chamber, including a minute distolingual opening to the periodontium (arrow), was restored with the acid-etch resin technique

External Inflammatory Root Resorption

The condition referred to as external inflammatory root resorption is a commonly occurring complication following displacement of the teeth, i.e., after luxation and avulsion injuries. The extrusion or intrusion of the tooth as well as subsequent repositioning or replantation procedures will inevitably cause damage to the root, resulting in denuded areas on the root surface which will be chemotactic to phagocytes. Transient root resorption will then ensue.

In addition, displacement of the teeth leads to a disruption of the pulpal blood vessels at the api-

cal foramina and to ischemic pulp necrosis (see p. 25). Microorganisms may then reach the root canal through enamel–dentin cracks and exposed dentinal tubules, and establish an infection, usually after a few days. The transient root resorption induced by the denuded areas of the root surface may now have exposed the tubular root dentin. Bacterial products from the infected root canal can then reach the resorptive areas on the root surface through the dentinal tubules and sustain the resorption of the root (**Fig. 8.15**).

Thus, external inflammatory root resorption is initiated by mechanical trauma, resulting in the removal of cementoblasts, precementum, and sometimes cementum in areas of the root surface. The resorptive process is then maintained by bacterial products from the infected root canal which provide the necessary continuous stimulation of the resorbing cells. *The condition can be recognized radiographically after a few weeks as periradicular radiolucent areas encompassing areas of the root and the adjacent alveolar bone* (**Fig. 8.16**). If allowed to progress, the resorptive process may destroy the tooth completely in a few months. However, by means of endodontic treatment, i. e., removal of the irritants from the root canal, the external inflammatory resorption can be arrested.

Any adequate endodontic treatment method will have an effect on the resorptive process. However, there is considerable clinical evidence that long-term treatment with calcium hydroxide provides the most predictable results. When calcium hydroxide is placed in the root canal, it will effectively kill the bacteria and, in addition, it will influence the local environment at the resorption sites on the root surface through the dentinal tubules (**Fig. 8.17**). Because of its high pH, calcium hydroxide will neutralize the lactic acid from the osteoclasts, thus preventing a dissolution of the mineral component of the root. Moreover, an alkaline pH at the resorption site will be unfavorable for the collagenase and acid hydrolase activity of the resorbing cells, and may also stimulate alka-

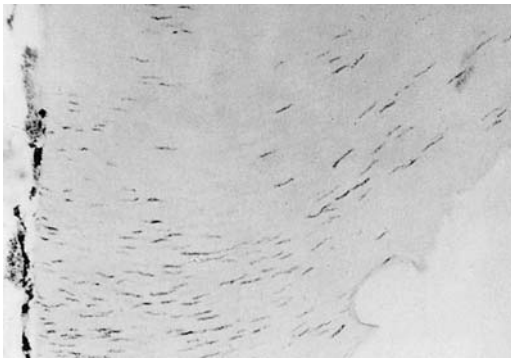
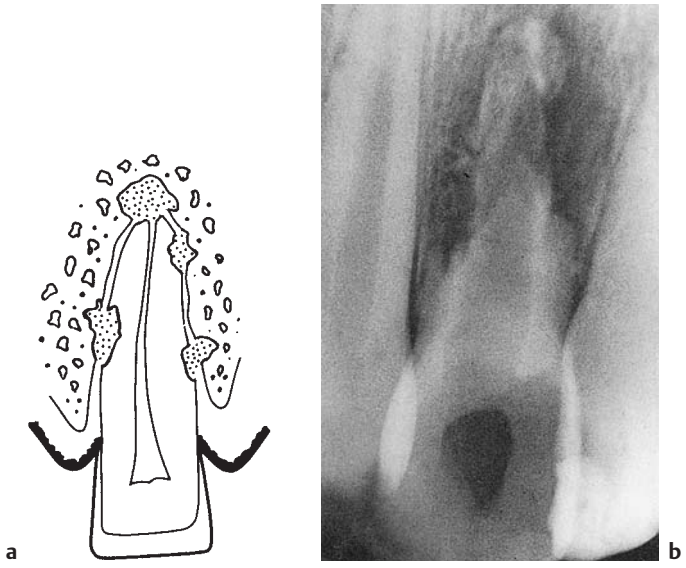


Fig. 8.15 Section of the root end of a nonvital tooth. Microorganisms are seen in necrotic tissue in the root canal (to the left) and in the dentinal tubules leading to external resorption lacunae (Brown–Brenn stain).

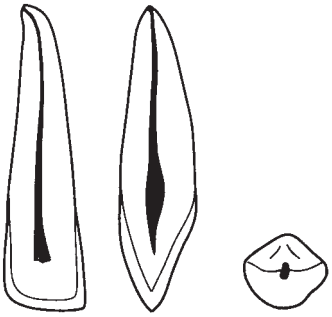


a **b**

Fig. 8.16
a **Diagram of a tooth with external inflammatory resorption.** The resorption of the root is associated with resorption of the adjacent bone.
b Radiographically, radiolucent lesions are seen in the periodontium adjacent to external resorption lacunae.

the pulp chamber (see p.179). It is very important to include the pulp horns in the cavity so that all tissue and discolored dentin are removed to prevent discoloration of the tooth. A round bur no. 2 is used for this purpose. Also, the root canal has a definite cervical constriction that should be removed before the actual root canal instrumentation begins. This is done with a long, tapered, diamond-coated bur, a Gates-Glidden, or similar bur. The apical part of the root canal is circular in shape and a circular apical box may be readily prepared in this tooth.

Maxillary Lateral Incisor



Average length of the tooth:		22 mm
Number of roots:		1 (99.9 %)
Number of root canals:		1 (99.9 %)
Lateral root canals:		Occasional
Apical root canal delta:		Frequent
Apical foramen	0 – 1 mm from apex:	90 %
	1 – 2 mm from apex:	10 %
Diameter of the root canal	1 mm from apex:	0.3 – 0.6 mm
	2 mm from apex:	0.35 – 0.8 mm
	3 mm from apex:	0.4 – 1 mm
	5 mm from apex:	0.4 – 1 mm
Recommended apical enlargement, straight canal:		60 – 80

Fig. 12.3 Diagram of a maxillary lateral incisor: Buccal, proximal, and incisal view.

Treatment Guidelines

The access cavity is prepared with long-shank, round burs as described for the maxillary central incisor. *The lateral incisor typically has a wide root canal in a narrow root.* Thus, the diameter of the canal in the apical 5 mm of the root is generally wider than both in the maxillary central incisor and the maxillary canine. However, the most apical part of the canal has a fairly round shape, so that a circular apical box may be prepared in lateral incisors. *Still, it must be understood that the canal of these teeth should be enlarged considerably more in the apical area than would normally be expected judging by the size of their roots.*

The root of the maxillary lateral incisor is frequently curved apically, *often in a palatal direction* so that the curve may not be apparent in a radiograph immediately. Although the curved canals are generally narrower than the canals in lateral incisors with straight roots, they are still wide and may be difficult to instrument adequately. *As a result, historically speaking, endodontic treatment of the maxillary lateral incisor has failed more than in any other tooth.* However, the use of flexible nickel-titanium instruments has made it easier to instrument wide, curved canals without perforations, canal transportation, or other mishaps.

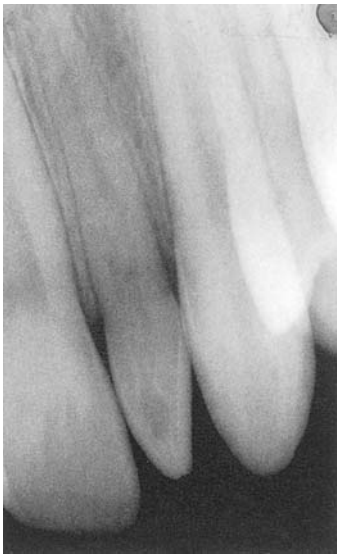
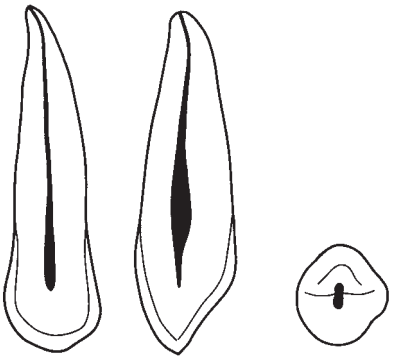


Fig. 12.4 Radiograph of a maxillary lateral incisor with enamel invagination in a 16-year-old.

Enamel invagination and dens in dente occur most often in the maxillary lateral incisor (**Fig. 12.4**). These malformations may allow penetration of bacteria to the pulp and endodontic

treatment of these teeth will frequently be necessary. The practical approach will depend on the clinical and radiographic findings and the degree of irregularities of the tooth. Often a surgical intervention with retrograde fillings to support the best possible orthograde obturation of the root canals will be necessary.

Maxillary Canine



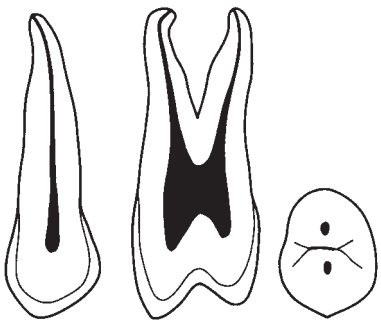
Average length of the tooth:		26.5 mm
Number of roots:		1 (99.9 %)
Number of root canals:		1 (99.9 %)
Lateral root canals:		Infrequent
Apical root canal delta:		Occasional
Apical foramen	0 – 1 mm from apex:	70 %
	1 – 2 mm from apex:	30 %
Diameter of the root canal	1 mm from apex:	0.2 – 0.45 mm
	2 mm from apex:	0.2 – 0.55 mm
	3 mm from apex:	0.3 – 0.7 mm
	5 mm from apex:	0.3 – 0.7 mm
Recommended apical enlargement:		50 – 70

Fig. 12.5 Diagram of a maxillary canine: Buccal, proximal, and incisal view.

Treatment Guidelines

The access cavity is prepared with long-shank, round burs as described for the maxillary central incisor. The maxillary canine is the longest tooth in the dentition and teeth 30 mm and longer are occasionally seen. The root canal is straight and circular in shape and only a very slight apical curvature of the root is sometimes seen. A circular apical box may be readily prepared, and this tooth lends itself exceptionally well to treatment with the standardized technique. On extremely rare occasions, the maxillary canine may have two root canals.

Maxillary First Premolar



Average length of the tooth:		20.6 mm
Number of roots:		1 (19 %); 2 (80 %); 3 (1 %)
Number of root canals:		1 (4 %); 2 (95 %); 3 (1 %)
Lateral root canals:		Infrequent
Apical root canal delta:		Infrequent
Apical foramen	0 – 1 mm from apex:	95 %
	1 – 2 mm from apex:	5 %

Diameter of the canal in teeth with

	3 canals	2 canals	1 canal
1 mm from apex:	0.15 – 0.2 mm	0.15 – 0.7 mm	0.5 mm
2 mm from apex:	0.15 – 0.2 mm	0.2 – 1 mm	0.7 – 1.2 mm
3 mm from apex:	0.15 – 0.35 mm	0.2 – 0.9 mm	0.7 – 1.2 mm
5 mm from apex:	0.25 – 0.35 mm	0.25 – 1 mm	0.8 – 1.2 mm

Recommended apical enlargement in teeth with 1 canal: 50 – 70
Recommended apical enlargement in teeth with 2 canals: 35 – 50
Recommended apical enlargement in teeth with 3 canals: 35

Fig. 12.6 Diagram of a maxillary first premolar: Buccal, proximal, and occlusal view.

Index

Page numbers in *italics* refer to illustrations

B

C

D

E

F

J

K

L

M

N

O

P

Q

R

S

T

U

V

W

Z