Treatment Planning for TRAUMATIZED TEETH

Second Edition

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Contents

Dedication

This book is dedicated to Dr Jens O. Andreasen.

Upon this publication, I would like to thank Dr Jens O. Andreasen, the first president of the International Association of Dental Traumatology. He is truly the father of modern dental traumatology, and his numerous studies over the course of many years have made a profound contribution to the education and enlightenment of this science and treatment throughout the world. For that, I wholeheartedly respect and admire him. Without his research and publications, this book itself and the treatment presented in it would not exist.

I served as president of the International Association of Dental Traumatology for 2 years—2009 and 2010. I initially joined this association because of my strong admiration for Dr Andreasen, the first president. Thus, it is my greatest joy and a true honor to have had an opportunity to hold the same position as he.

Preface

Looking back on my professional development as a clinician, I first became interested in wound healing in periodontal disease and then encountered dental autotransplantation. This inevitably led me to dental traumatology, which is the foundation of the latter science. Feeling acutely aware of my lack of knowledge of treatment techniques in the field of dental trauma, I strove relentlessly to learn more about dental traumatology.

I believe that dental traumatology requires knowledge of the science and techniques of all areas of clinical dentistry, such as restorative dentistry, endodontics, periodontology, and dental implant treatment. I also believe that an understanding of dental trauma can bring a new perspective and valuable insights to dental treatment for clinicians who may not be interested or frequently involved in dental trauma cases. While there is a wealth of information and remarkable progress in the treatment and prevention of dental caries and periodontal disease, dental trauma appears to be left behind despite its frequent incidence. It is my sincere desire that this book may serve as a useful clinical guide for clinicians as well as dental students.

By publishing this revised edition, my intention is to make this book an entry point for minimal intervention in dental treatment. Dental trauma is, in contrast to dental caries and periodontal disease, categorized as an acute injury; in other words, it can be considered a dental disorder with little or no infection. In addition, because patients with dental trauma are relatively young in many cases, the body's healing ability is expected to be high. Therefore, more conservative, biologically tolerable treatment and predictable outcomes can be expected for the dental hard tissues, pulp, periodontal ligament, and alveolar bone. Minimal intervention (ie, avoiding restoration, pulpectomy, and extraction as much as possible) is an attainable and important treatment goal.

Eleven years have passed since the first edition of *Treatment Planning for Traumatized Teeth* was published. During the last decade, dental treatment and diagnosis have rapidly progressed. In particular, the recently developed dental cone beam computed tomography (CBCT) technology has dramatically improved diagnosis for traumatized teeth. I started using CBCT 8 years before the publication of this book, and that experience became a major driving force for this revised edition.

In addition, the book, originally published in Japanese, has been translated into English, German, Italian, French, Spanish, Korean, Bulgarian, Czech, Dutch, Turkish, and Chinese, which I could not have imagined at the time of publication. This is a great honor, but at the same time, quite surprising because it may indicate that there is still a lack of information regarding dental trauma throughout the world.

Books, knowledge, and technology are all eventually replaced over time. This book and the information herein will inevitably meet the same fate; however, I hope it holds a valuable place in the field of dentistry for some time to come and will have a role in the evolution of knowledge in the discipline.

Acknowledgments

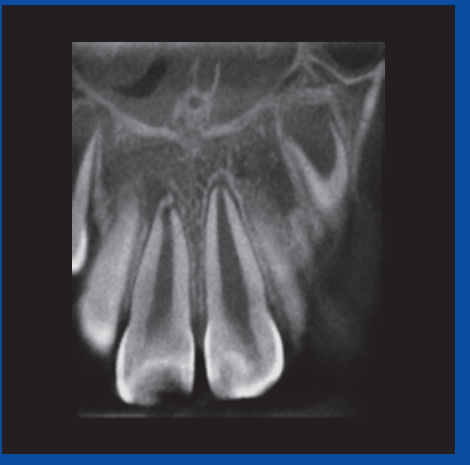
Special thanks to Dr Nozomu Yamauchi and Dr Shizuko Yamauchi for their collaboration and the many hours they spent translating this book into English. Biologic and pathologic descriptions were also reviewed and translated by Mitsuo Yamauchi. This edition would not be in English without their dedicated work.

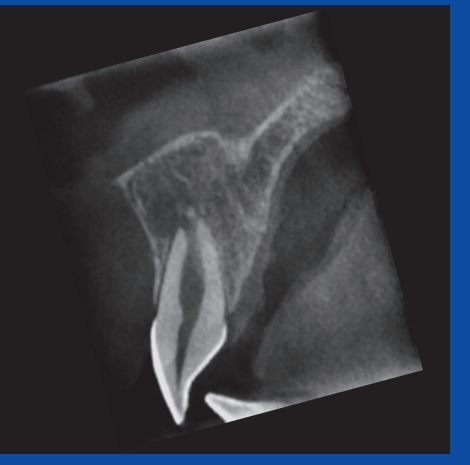
I would also like to thank Dr Leif K. Bakland for his assistance in reviewing and revising the English version of this second edition.

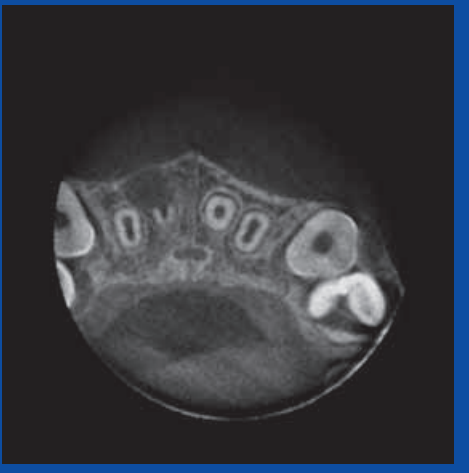
CHAPTER 2

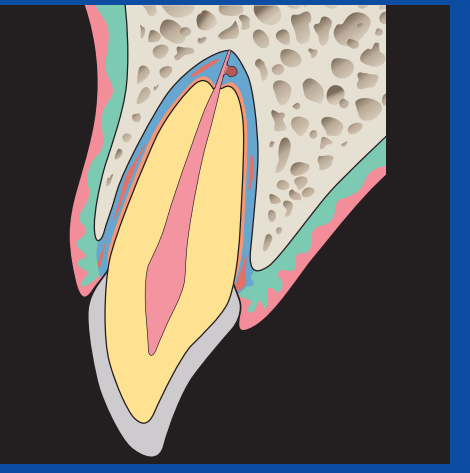
Examination and Diagnosis of Traumatic Dental Injuries

In this chapter, the initial examination, including information-gathering techniques for proper diagnosis, are discussed. Several clinical cases are used to illustrate these points.









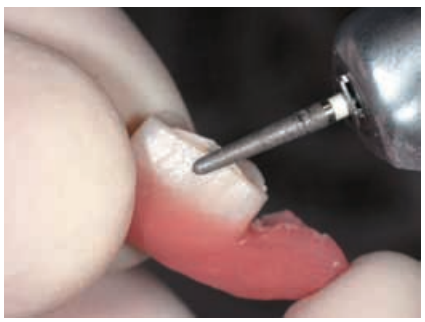


Fig 3-7p Beveling of the entire periphery of the fractured surface with a superfine diamond bur.

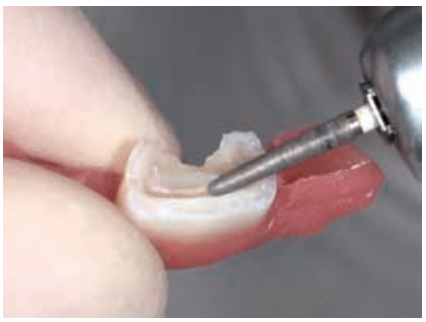


Fig 3-7q Removing any remaining soft tissue and some of the surrounding tooth structure that may cause discoloration of the tooth in future.

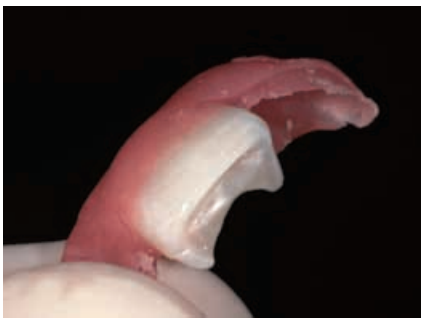


Fig 3-7r The tooth fragment after beveling and trimming.

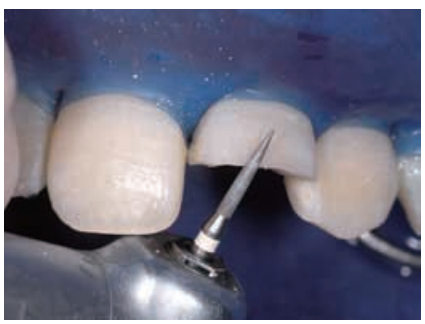


Fig 3-7s Beveling of the fractured surface of the remaining tooth.

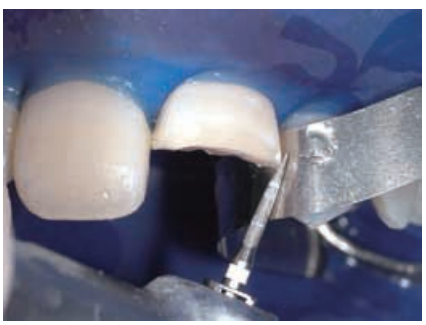


Fig 3-7t The adjacent tooth is protected with a metal matrix during beveling of the proximal surface.

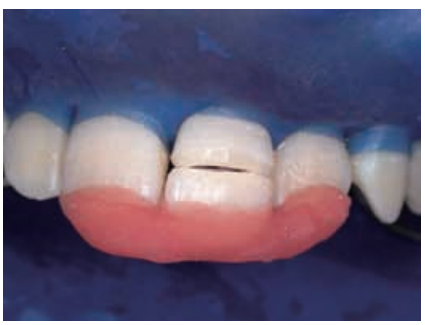


Fig 3-7u Repositioning of the fragment with the stent.

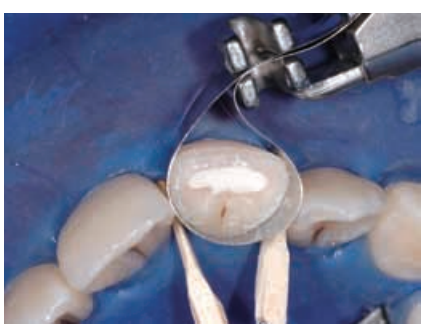


Fig 3-7v Fitting the Tofflemire retainer and metal matrix band. The matrix band is placed loosely, then wedges are placed on both proximal sides to provide better adjustment and stability of the matrix band.

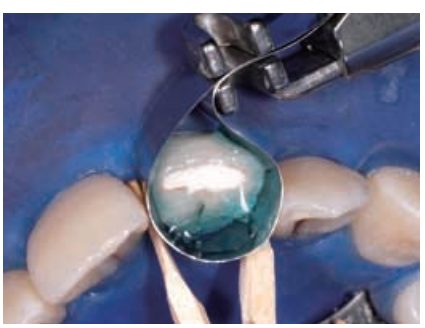


Fig 3-7w Etching enamel with 37% phosphoric acid for 15 seconds, which is followed by rinsing with water and air drying.

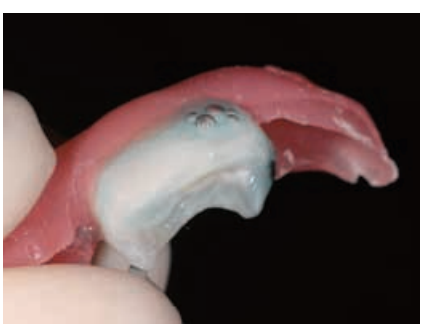


Fig 3-7x The tooth is etched extraorally with 37% phosphoric acid for 15 seconds, which is followed by bonding agent application and light curing.

a polished surface in the interproximal areas, which are difficult to polish. The author often uses a Tofflemire retainer (Waterpik) and metallic matrix. At this point, it is better to place the matrix band loosely (Fig 3-7v). After the matrix band is placed, the enamel is etched with 37% phosphoric acid for 15 seconds (Fig 3-7w). The fragment is also etched (extraorally) in the same manner (Fig 3-7x). Although all-in-one bonding systems are well accepted and thought to be ideal for dentin bonding, it is clinically questionable whether the material adheres to enamel. Therefore, the author believes that etching the enamel surface with phosphoric acid is an important step. After etching, the tooth surfaces are thoroughly rinsed and dried, followed by an application of the bonding agent (eg, AQ Bond, Sun Medical; Bond Force, Tokuyama Dental) and light curing.

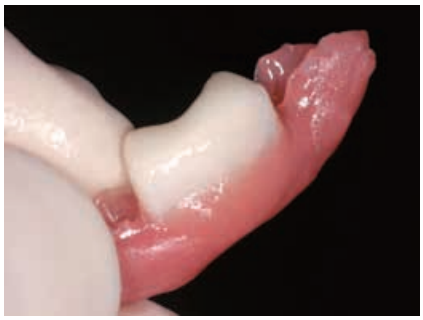


Fig 3-7y Following bonding, composite resin is placed on the fragment. Note that either flowable or regular composite may be used; however, flowable composite with too much viscosity may be difficult to manage.

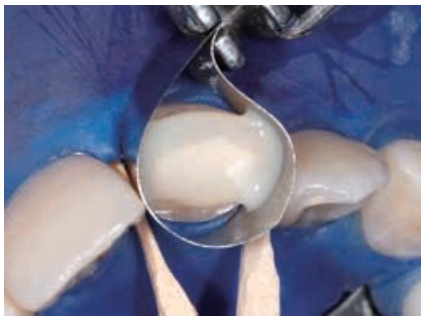


Fig 3-7z Application of an excess amount of composite resin on the remaining tooth structure. Again, either flowable or regular composite can be used.

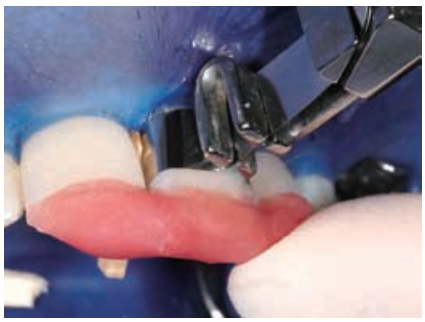


Fig 3-7aa Placing the fragment back into position using the stent. The matrix band should be tightened while the fingers hold the stent.

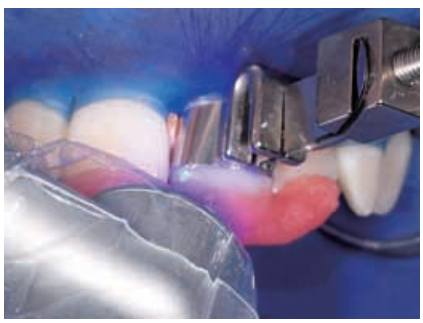


Fig 3-7bb The curing light is directed from the incisal, palatal, and labial aspects to properly cure the composite.

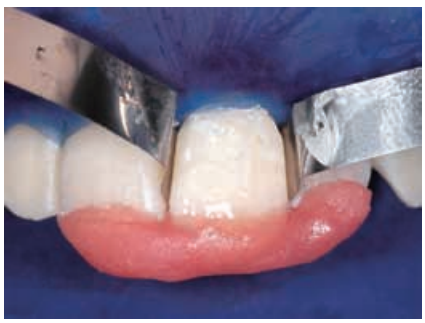


Fig 3-7cc Before removal of the entire matrix band, light curing should be performed again.

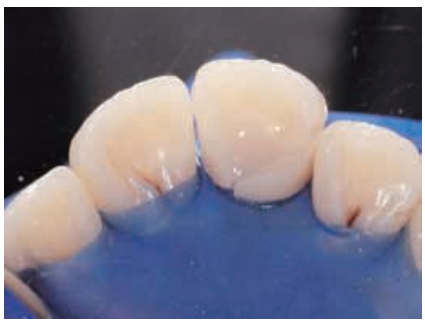


Fig 3-7dd Immediately after removal of the matrix band, light curing should be performed on the labial and palatal sides once more.

Bonding and polishing of fragments

After the bonding agent has been cured, a light-curable composite resin (eg, Estelite, Tokuyama Dental) is applied in excess to both the fragment and the remaining tooth (Figs 3-7y and 3-7z). The stent with the fragment is then placed firmly back into position in the mouth (Fig 3-7aa). While keeping pressure on the stent, the matrix band is tightened. This allows the composite resin to adapt and flow into the spaces. The curing light is directed from the incisal as well as the labial and palatal aspects to properly cure the composite (Figs 3-7bb and 3-7cc). After removal of the matrix band, the composite should be cured once more from the labial and palatal directions to ensure that the composite has set and cured (Fig 3-7dd).

After the composite is cured, there will be excess composite and bonding materials that are not cured. This material should be trimmed, and then the surface should be polished (Figs 3-7ee to 3-7kk). The author uses a superfine diamond bur (eg, Mary Dia, Hinatawada Seimitsu) for trimming and a silicone point (eg, CeraMaster or CompoMaster, Shofu) to finish.

Note that the above was a detailed description of treatment in a case in which a stent could be used. However, in cases in which a stent cannot be used, the treatment should be the same as described above but without the use of the stent. The fragment may tend to be misaligned or repositioned incorrectly, so it is important to pay careful attention during the reattachment and bonding of the fragment.

Fig 6-5 Clinical example of subluxation injury with TAB and pulp canal obliteration.

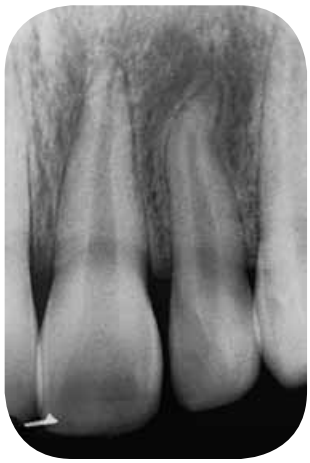


Fig 6-5a Initial visit of a 14-year-old adolescent boy with subluxation of the maxillary left central and lateral incisors. Both teeth are EPT negative.

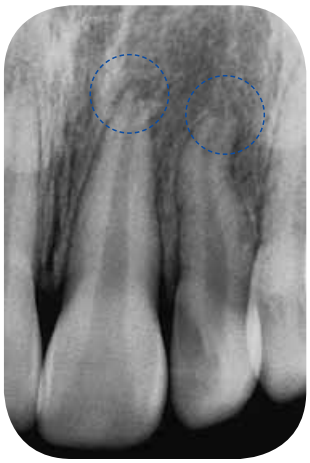


Fig 6-5b At 1 month after the trauma, the apices of the maxillary left central and lateral incisors now appear open *(circles)*. Both teeth remain EPT negative.

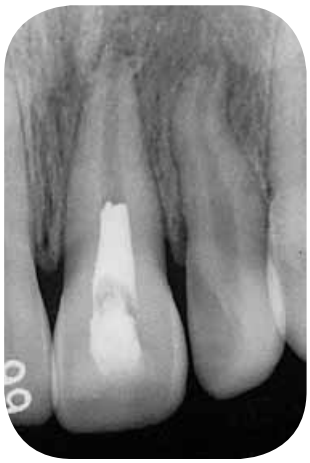


Fig 6-5c Radiograph taken the same day as Fig 6-5b. Discoloration of the maxillary left central incisor was evident, thus root canal treatment was initiated. However, there was pulp tissue present and sensitivity at the midroot level, so calcium hydroxide was placed to that point.

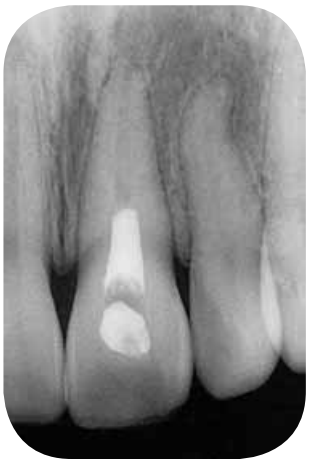


Fig 6-5d At 8 months after the trauma, the maxillary left central and lateral incisors show pulp canal obliteration. Details of the treatment of this patient are shown in chapter 10, Fig 10-1.

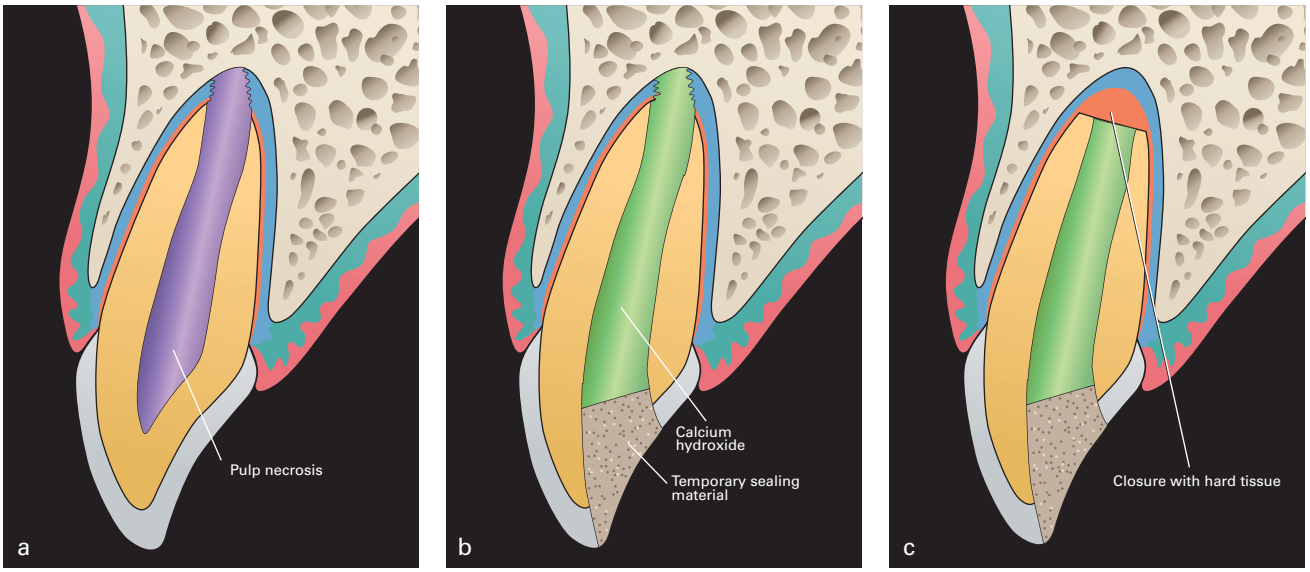
Root canal treatment in case of pulp necrosis

When TAB does not occur or is not expected (eg, if the patient is more than 20 years old), the presence of pulp necrosis is confirmed by continuation of crown discoloration, pain on percussion, apical lesion, and negative EPT result. In cases with pulp necrosis, root canal treatment is recommended. In adults, there are advantages to performing proper canal enlargement with cleaning, shaping, and filling in the same day. In young adults, because the apex is still slightly open, it is recommended to perform apexification (see the next section). Upon completion of root canal treatment, internal bleaching and composite resin restoration are performed, and proper follow-up and maintenance are continued.

Apexification

Apexification is the process by which the apex of an immature tooth with pulp necrosis is closed with hard tissue deposition (ie, cementum-like tissue)[1–4] (Figs 6-6 to 6-8). This is achieved by removing necrotic tissue to the apex, preparing and irrigating the canal, and filling with calcium hydroxide. Generally, after the apex is closed by hard tissue (after approximately 6 months, based on clinical experience), the root filling is performed with sealer and gutta-percha (see Fig 6-8g). The mechanism by which the apex is closed with calcium hydroxide is shown in Fig 6-7.

Fig 6-6 Apexification treatment.



Figs 6-6a to 6-6c *Apexification* is the process by which the open apex of a tooth with pulp necrosis and an incompletely formed root can be closed by deposition of hard tissue (ie, cementum-like tissue).

Fig 6-7 Healing mechanism of apexification according to Shinagawa.[4]

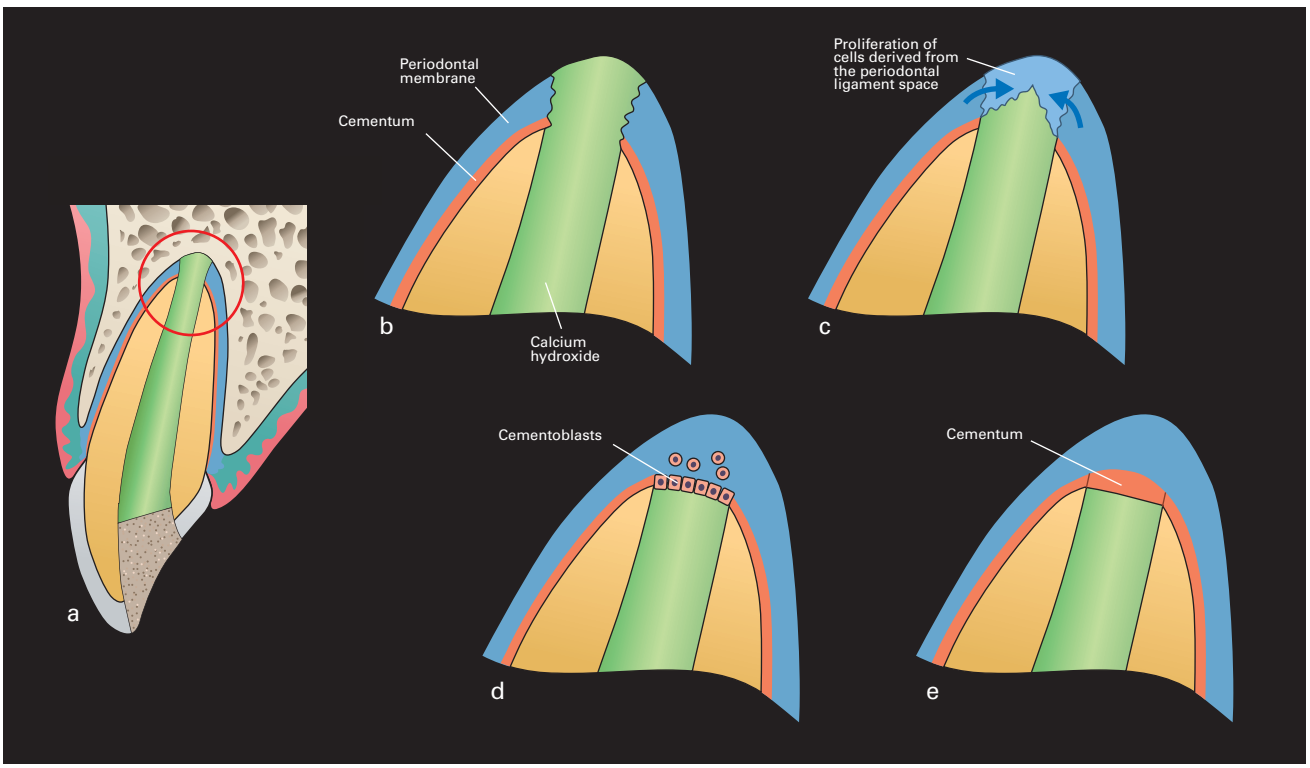
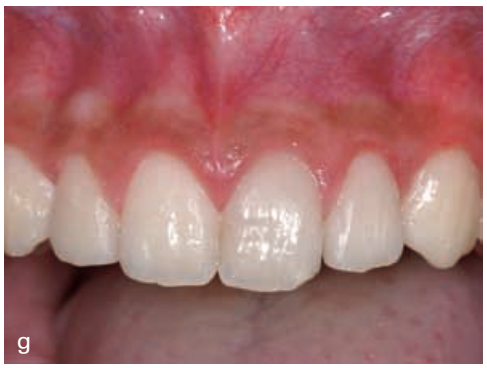
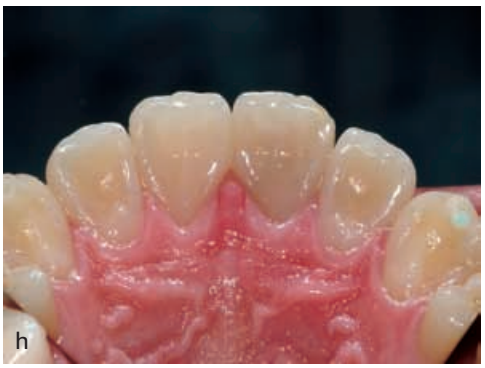
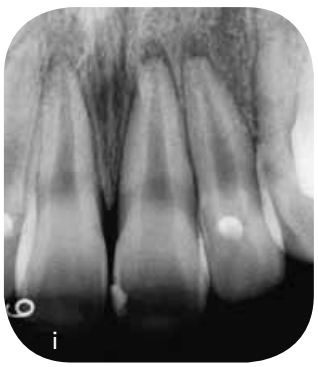
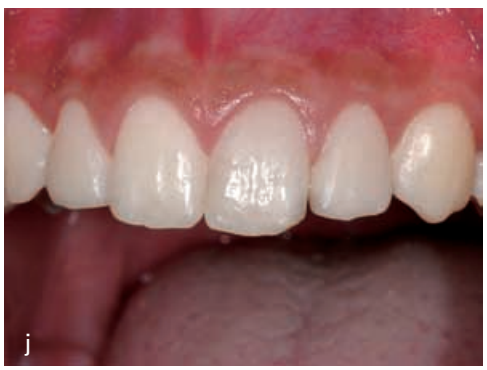
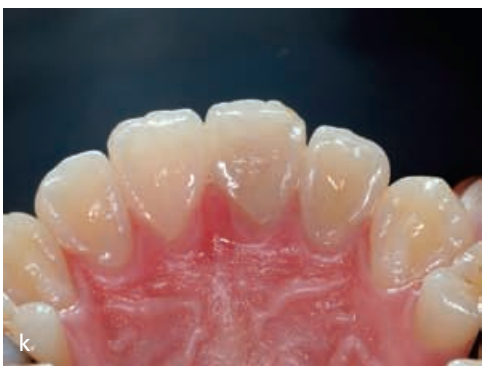
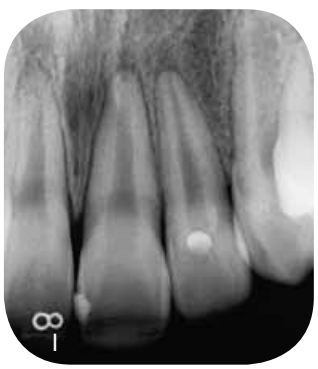


Fig 6-7 *(a and b)* Immediately after treatment. Calcium hydroxide extruded through the apex causes degeneration or necrosis of the periodontal membrane and osseous tissue. There is calcific deposition near the border of the necrotic layer and healthy tissues. *(c)* Approximately 1 month later. The necrotic layer and calcific deposit dissipate. Note the immature fiber and periodontal membrane tissue with an abundance of blood vessels around the apex. *(d)* Approximately 2 months later. Because cells have differentiated from the periodontal membrane (cementoblasts), there is hard tissue (cementum) apposition. *(e)* Approximately 3 to 6 months later. The apex is closed by hard tissue deposition and is surrounded by periodontal membrane tissues.

Figs 10-3g to 10-3i Two months after the initial visit. The radiolucencies around the apices of the maxillary left central and lateral incisors have increased in size. Both teeth are EPT negative.

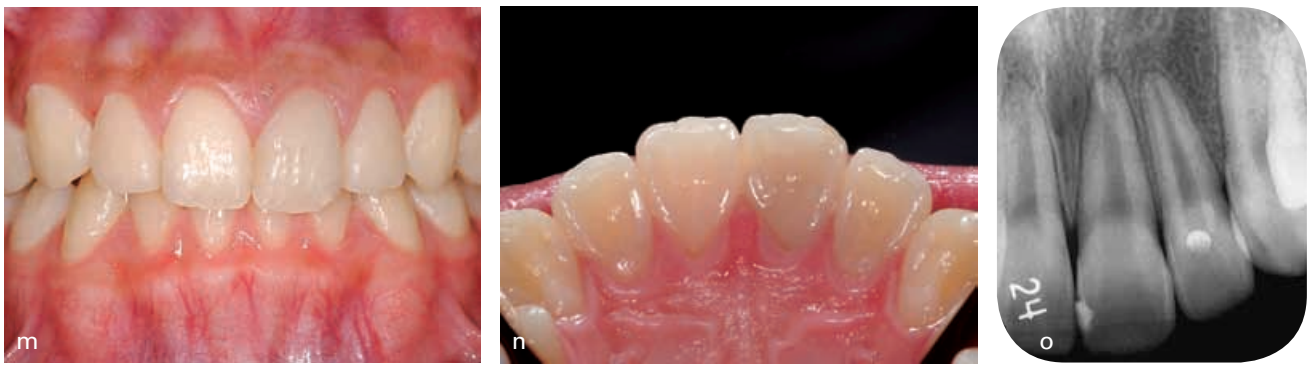
Figs 10-3j to 10-3l Three months after the initial visit. There are no changes in discoloration. The radiolucencies around the apices of the maxillary left central and lateral incisors appear to have decreased in size.

The TAB phenomenon was clearly seen radiographically 2 and 3 months after the initial visit (see Figs 10-3i and Fig 10-3l). The intraoral photographs show that the left central incisor has slight crown discoloration (see Figs 10-3g, 10-3h, 10-3j, and 10-3k). Continued healing and improvement of TAB is seen at the 3-month follow-up (see Fig 10-3l).

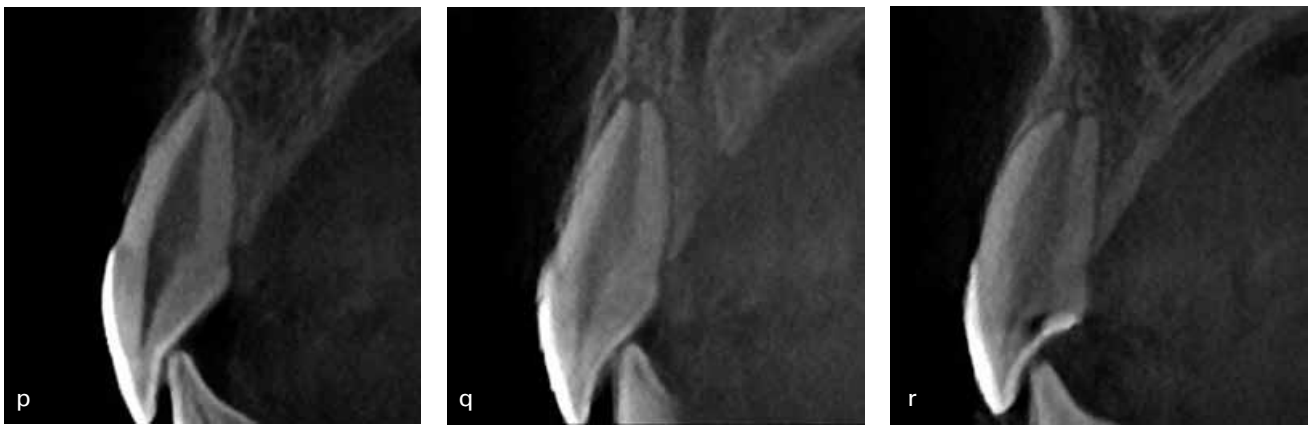
The 6-month radiograph (see Fig 10-3o) shows that bone resorption at the apex was completely gone, but the apical foramen appears wide open. The CBCT images at 6 months (see Figs 10-3p to 10-3r) show enlargement of the apical foramen of the maxillary left central and lateral incisors compared with the first visit. The periapical area of the maxillary left central incisor shows clear bone resorption (see Fig 10-3q). Clinical photographs (see Figs 10-3m and 10-3n) show no improvement of the crown discoloration of the central incisor. Both teeth are still EPT negative.

At the 9-month follow-up, radiographic and clinical examinations show no changes (see Figs 10-3s to 10-3u). However, at this point, both the maxillary left central and lateral incisors are EPT positive for the first time.

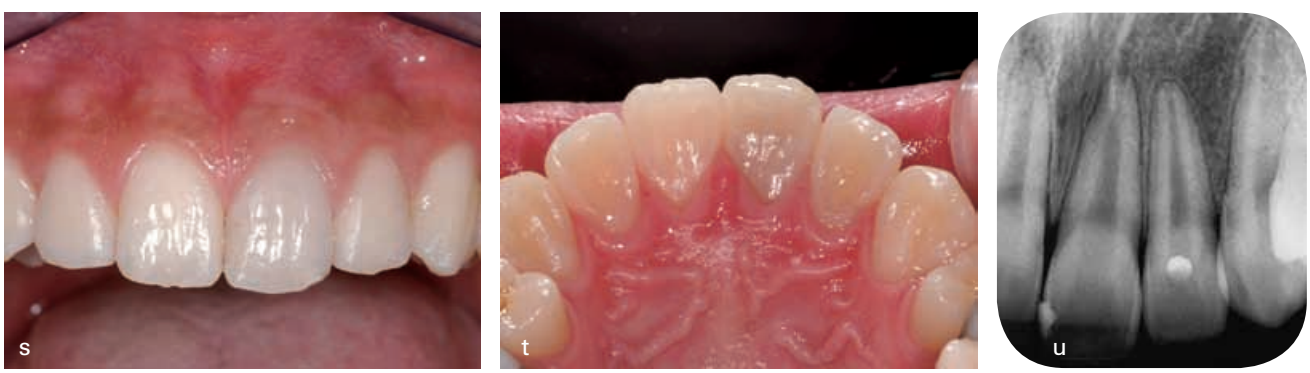
At the 2-year follow-up, pulp canal obliteration of the maxillary left lateral incisor has progressed. Slight obliteration at the apical area of the maxillary left central incisor is seen. There is slight improvement in the discoloration of the central incisor. Both teeth are EPT positive (see Figs 10-3v to 10-3x). Based on CBCT images at the 2-year follow-up, the apex of the central incisor became slightly rounded and shortened as a result of surface resorption and remodeling. There is normal lamina dura present (see Fig 10-3z). In the case of the lateral incisor, canal obliteration has progressed, and calcification can be seen throughout the pulp space (see Fig 10-3aa). No pathologic bone radiolucencies are seen around the roots of either tooth.



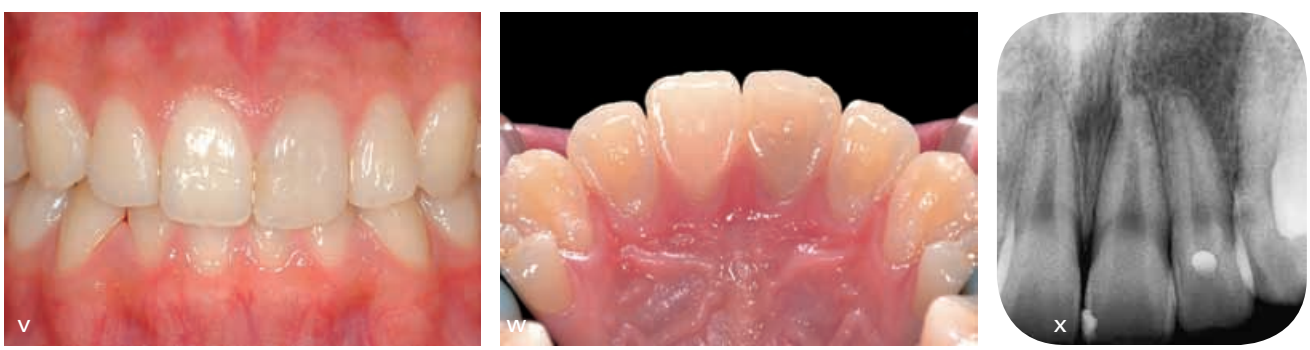
Figs 10-3m to 10-3o Six months after the initial visit. There are almost no signs of radiolucencies around the apices of the maxillary left central and lateral incisors. Both teeth are EPT negative.



Figs 10-3p to 10-3r Sagittal CBCT images taken 6 months after the initial visit. *(p)* The maxillary right central incisor, which sustained no trauma. *(q)* The maxillary left central incisor shows a radiolucency at the apex. There is evidence of apical root resorption with rounding and shortening of the apex. There is resorption of the internal wall of the apex area, which gives the appearance of an open apex. *(r)* The maxillary left lateral incisor shows no sign of bone resorption, but there is root resorption of the apex and widening of the foramen.



Figs 10-3s to 10-3u Nine months later. The maxillary left central incisor shows crown discoloration. The radiograph shows no significant changes. However, both teeth are now EPT positive.



Figs 10-3v to 10-3x Two years after the trauma. The maxillary left lateral incisor shows progressing canal obliteration. Both teeth are EPT positive.

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