Treatment Planning for TRAUMATIZED TEETH
Second Edition

Mitsuhiro Tsukiboshi, DDS, PhD
Private Practice
Amagun, Aichi
Japan

Translated by
Nozomu Yamauchi, DDS
Private Practice in Endodontics
Honolulu, HI

Shizuko Yamauchi, DDS, MS
Assistant Professor
Department of Endodontics
School of Dentistry
University of North Carolina at Chapel Hill
Chapel Hill, NC

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Contents

Dedication vi
Preface vii

1 Anatomical Considerations and Classification of Dental Trauma 1

2 Examination and Diagnosis of Traumatic Dental Injuries 9

3 Crown Fracture 25

4 Crown-Root Fracture 49

5 Root Fracture 71

6 Subluxation 89

7 Extrusive Luxation 109

8 Lateral Luxation 119

9 Intrusive Luxation 129

10 Transient Apical Breakdown 143

11 Avulsion 163

12 Trauma to the Primary Dentition 197

13 Trauma to the Supporting Structures 221

Index 229
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.
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.
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.
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 entire 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.
Root canal treatment in case of pulp necrosis

When TAB does not occur or is not expected (e.g., 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 (i.e., cementum-like tissue) (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.
Figs 6-6 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 (cemento-blasts), 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.
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.
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 10-3m to 10-3o

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 10-3s to 10-3u

Two years after the trauma. The maxillary left lateral incisor shows progressing canal obliteration. Both teeth are EPT positive.

Figs 10-3v to 10-3x

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.
Abrasion, 226–227
Alveolar bone fracture, 7, 14
  in avulsion, 190
  in intrusive luxation, 130
  in lateral luxation, 120, 121
  in maxillary/mandibular trauma, 222, 223
  treatment of, 224
Anatomy, 2
Ankylosis
  in avulsion, 170, 172, 179, 186, 187, 189, 190
  in intrusive luxation, 134, 139, 141
  on examination, 10
Apexification, 100–102
Apexogenesis, 39, 40
Avulsion, 6, 7
  classification, 164
  definition of, 164
  delayed replantation for, 165, 177–192
  diagnosis, 164–165
  education about, 193
  examination, 164–165
  immediate replantation for, 165, 166–177
  periodontal ligament in, 164, 170–171, 175
  preservation in, 164, 165, 175–177
  in primary teeth, 216–219
  root maturity and, 164
  treatment, 165–192
Biologic width
  in crown-root fracture, 51
  definition of, 2
Bleaching
  in immediate replantation of avulsed tooth, 168
  internal, 103–106
Calcium hydroxide, 41
Cementoblasts, 3
Cementoenamel junction (CEJ), in intrusive luxation, 131
Cementum, 3
  necrosis, 144
Concussion
  definition of, 6, 7
  primary teeth, 209
Cone beam computed tomography (CBCT)
  advantages of, 14
  examination on, 14–19
  intrusive luxation on, 132–133
  principles of, 15
  radiography vs, 14
  transient apical breakdown on, 149–152
Contusion, 226–227
Crown fracture, 4–5, 16
  anesthesia in, 31
  bevel placement, 33
  bonding, 33–34
  classification, 26
  complicated, 29
  diagnosis, 27
  etching, 33–34
  examination, 27
  follow-up visits, 36–38
  fragment bonding, 35
  fragment polishing, 35
  with luxation, 29
  matrix placement, 33–34
  in primary teeth, 209
  pulp capping material selection, 41–44
  pulpotomy, 31–32
  stent fabrication, 31
  tooth fragment missing, 44–45
  treatment, 27–38
  wound healing, 39–40
Crown hypoplasia, 201
Crown-root fracture
  biologic width reestablishment, 51
  complicated, 50
  diagnosis, 51
  examination, 51
  orthodontic extrusion, 55–59
  in primary teeth, 209, 210
  prognosis, 68
  with root fracture, 16
  surgical extrusion, 59–68
  treatment, 52–68
  uncomplicated, 50
Dental follicle, 3
Dental trauma
  classification, 4–7
  endodontic considerations in, 160, 162
  Dentin, 3
  Dentin-adhesive resin, 41, 42, 43
  Dentin bridge formation, 39
  Discoloration
    of primary teeth, 201–209
    in subluxation, 106–107
    in transient apical breakdown, 144, 145, 147, 148
Documentation, 11
Electric pulp testing (EPT)
  in avulsion, 169, 170, 175
  in crown fracture, 27, 31, 32, 37, 39
  in crown-root fracture, 52, 53, 54
  in examination, 10
  in intrusive luxation, 110, 112, 113, 115
  in lateral luxation, 136, 138
  in primary teeth injury, 198, 204
  in root fracture, 10, 72, 77, 80, 81, 82, 83–87
  in subluxation, 90, 92, 93, 94–100, 106
  in transient apical breakdown, 144, 145–148, 150, 152, 154
Enamel fracture, 4–5, 26
Enamel infarction, 5
Endodontic considerations, 160, 162
Epithelial debris of Malassez, 3
Examination
  clinical, 10–13
  cone beam computed tomography, 14–19
  radiographic, 13–14
Fibroblasts, 3
Fracture classification, 4–5
Gingival injury, 226–227
Hertwig epithelial root sheath, 3
Internal bleaching, 103–106
Laceration, 226–227
Laser Doppler flowmetry, for pulp diagnosis, 159
Luxation, 6, 7, 19
with crown fracture, 29
extrusive classification, 110
crown restoration, 111, 112–114
definition of, 110
examination, 111
fixation, 111, 112–114
follow-up, 111, 115–117
repositioning, 111, 112–114
root canal for, 111, 112–114
transient apical breakdown in, 149–152
treatment, 111–117
intrusive
age and, 132, 133
biologic width, 131
cementoenamel junction in, 131
classification, 130
on cone beam computed tomography, 132–133
diagnosis, 130–133
examination, 130–133
mobility in, 130
orthodontic extrusion for, 139–140
percussion sensitivity in, 131
percussion sound in, 131
periodontal ligament space in, 132
prognosis, 133
pulp necrosis and, 133
root development and, 133
spontaneous re-eruption, 134–139
tooth dislocation, 130, 131
treatment, 133–142
lateral
classification, 120
crown restoration, 121, 122, 126
definition of, 120
diagnosis, 19, 120, 122
examination, 120
fixation, 121, 122, 125
follow-up, 121, 123, 126, 127
healing, 19
repositioning, 121, 122, 125
root canal treatment, 121, 122, 126
treatment, 121–127
in primary teeth, 212–215
Mandibular fracture, 222–225
Maxillary fracture, 222–225
Mineral trioxide aggregate, 42, 141
Odontoblasts, 3
Oral mucosa injury, 226–227
Orthodontic extrusion
for intrusive luxation, 139–140
Orthodontic extrusion, for crown-root fracture, 55–59
Osteoblasts, 3
Osteoclasts, 172
Periodontal ligament space, in extrusive luxation, 132
Periodontal ligament, in avulsion, 164, 170–171, 175
Periodontal ligament, inner, 175, 176
Permanent teeth, malformation of, 200
Photographs, 10–12
Preservation, after avulsion, 164, 165, 166, 175–177
Primary dentition trauma
avulsion, 216–219
concussion, 209
crown fracture, 209
crown–root fracture, 209, 210
diagnosis, 198
discoloration, 201–209
effects of, on permanent tooth germ, 199
examination, 198
luxation, 212–215
root fracture, 209, 211–232
subluxation, 209
treatment, 196–219
Pulp canal obliteration, in subluxation, 106–107
Pulp capping materials, 41–44
Pulp cells, 3
Pulp necrosis
cocagulation necrosis vs, 144
in intrusive luxation, 133
ischemic, 144
root canal in, 160
in subluxation, 93–96
in transient apical breakdown, 144, 152, 153
transient apical breakdown vs, 154–159
Radiographic examination, 13–14
Record of traumatized teeth, 11
Replantation
delayed, 165, 177–192
alveolar socket in, 178
auxiliary procedures, 185–192
cleaning of avulsed tooth, 177–178
fixation, 178, 179
follow-up, 178, 179, 185–192
in postpubertal patients, 177–179
in prepubertal patients, 180, 181
in pubertal patients, 180, 181–185
root canal, 178
immediate, 165, 166–177
bleaching, 168
cleaning of alveolus, 167
cleaning of tooth, 166
crown restoration, 168
diagnosis of, 166
examination, 166
fixation, 167
Index

fixation removal, 167, 168
of immature teeth, 169–170
periodontal ligament healing, 170–171, 175
preservation, 166, 175–177
pulpal healing in, 174–175
root canal, 167
root resorption in, 172–174
wound healing, 170–177
Root canal
in delayed replantation of avulsed tooth, 178
disinfection of, 160
for extrusive luxation, 111, 112–114
in immediate replantation of avulsed tooth, 167
for lateral luxation, 121, 122, 126
in pulp necrosis, 160
recontamination prevention in, 160
for subluxation
immature teeth, 93–96
mature teeth, 100
for transient apical breakdown, 144, 147
Root fracture, 4–5, 17, 18
autotransplantation for, 75
classification, 72
definition of, 72
diagnosis, 17, 72–73, 86–87
examination, 72–73, 86–87
extraction for, 75
fixation, 88
healing patterns, 18, 76–85
with calcified tissue, 76–79
with interposition of bone and connective tissue, 82–84
with interposition of connective tissue, 80–82
with interposition of granulation tissue, 85–86
postoperative follow-up, 88
in primary teeth, 209, 211–232
repositioning, 88
shallow, 74–75
temporary apical breakdown and, 160, 161
treatment, 73–75, 86–88
Root resorption
after subluxation, 154, 157–158
ankylosis-related, 172, 173
in avulsion, 172–174
infection-related, 173, 174
repair-related, 172, 173
S
Sharpey fibers, 3
Spontaneous re-eruption, in intrusive luxation, 134–139
Subluxation, 6, 7, 21–23
apexification, 100–102
classification, 90
definition of, 90
diagnosis, 21–22, 90
discoloration in, 106–107
examination, 21–22, 90
follow-up, 23
immature teeth, 91, 92, 93–96
fixation, 93
root canal for, 93–96
internal bleaching, 103–106
mature teeth, 91, 97–100
fixation, 97–98
follow-up, 98–99
root canal for, 100
in primary teeth, 209
pulp canal obliteration in, 106–107
pulp necrosis, 93–96
root resorption after, 154, 157–158
temporary apical breakdown and, 145
treatment, 93–100
Supernumerary teeth, 176
Surgical extrusion
for crown-root fracture, 59–68
for intrusive luxation, 139–140
T
Tooth anatomy, 2–3
Transient apical breakdown (TAB)
age and, 154
case reports, 144–148
classification, 144
don cone beam computed tomography, 149–152
definition, 144
differential diagnosis, 154–159
discoloration in, 144, 145, 147, 148
in extrusive luxation, 149–152
follow-up, 149–152
injury criteria for, 154
laser Doppler flowmetry and, 159
mechanism of, 152–153
pulp necrosis, 144
pulp necrosis in, 152, 153
pulp necrosis vs., 154–159
pulpal healing in, 152, 153
root canal for, 144, 147
root fracture and, 160, 161
in subluxation, 145