Textbooks in Contemporary Dentistry

Mark Roettger *Editor*

# Modern Sports Dentistry

Springer

Textbooks in Contemporary Dentistry

This textbook series presents the most recent advances in all fields of dentistry, with the aim of bridging the gap between basic science and clinical practice. It will equip readers with an excellent knowledge of how to provide optimal care reflecting current understanding and utilizing the latest materials and techniques. Each volume is written by internationally respected experts in the field who ensure that information is conveyed in a concise, consistent, and readily intelligible manner with the aid of a wealth of informative illustrations.

*Textbooks in Contemporary Dentistry*

will be especially valuable for advanced students, practitioners in the early stages of their career, and university instructors.

More information about this series at http://www.springer.com/series/14362

Mark Roettger
*Editor*

# Modern Sports Dentistry

Springer

*Editor*
**Mark Roettger**
Division of Oral Surgery
University of Minnesota
Minneapolis, MN
USA

ISSN 2524-4612 ISSN 2524-4620 (electronic)
Textbooks in Contemporary Dentistry
ISBN 978-3-319-44414-7 ISBN 978-3-319-44416-1 (eBook)
https://doi.org/10.1007/978-3-319-44416-1

Library of Congress Control Number: 2018951073



Printed on acid-free paper

This Springer imprint is published by the registered company Springer International Publishing AG part of Springer Nature
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

This book is dedicated first to my family, my wife Michelle and my sons Broderick and Matthew who sacrificed family time as I built a practice and while I took care of sports teams.
Second, I dedicate this book to my personal mentor, Dr. Norman O. Holte, oral surgeon and team dentist at the University of Minnesota who introduced me to sports dentistry while I was in my third year of dental school.
Finally this work is dedicated to the dentists who laid the groundwork for structure and organization in the discipline of sports dentistry, the founding members of the Academy for Sports Dentistry, all of whom were also mentors to me:
Cosmo Castaldi
Spiro Chaconas
H. Hugh Gardy
William Godwin
William Heintz
Robert Morrow
William Olin
Donald Peterson
John Stenger
Edwin Whitman
Arthur Wood

# Preface

Sports Dentistry has been around for many years. There was knowledge that sports participation created opportunity for dental injuries, and that we should try to prevent these injuries. There was no organization to the practice of sports dentistry until the Academy for Sports Dentistry (ASD) was formed in 1983. ASD was created by many of the dentists that were instrumental in making mouthguard use mandatory for American high school football in 1962. Still there was room for more organization and structure to sports dentistry. I have been involved with ASD since its inception, and I was often asked by colleagues and patients what sports dentistry was as they tried to get a definition. In the late 1990s I had the opportunity to serve as President of ASD and one task that I charged the board to tackle was to define sports dentistry in a position statement. The definition has been modified a bit over the years and it provides a framework of organization and structure to the discipline of sports dentistry:

» *"Sports Dentistry is the branch of sports medicine that deals with the prevention and treatment of dental injuries and related oral diseases associated with sport and exercise." November 18, 2012 (Academy for Sports Dentistry)*

This book is original and designed to give the reader a detailed view of the science and art of sports dentistry that will fit the ASD definition of sports dentistry. This should give the reader a solid knowledge of sports dentistry and can also be used as a clinical reference.

It is intended to be a resource for dentists who want to become a team dentist and become a member of the integrated modern sports medicine team. It also serves as an introduction to dentofacial trauma recognition, evaluation, and management that will be valuable to certified athletic trainers, sports medicine physicians, and school nurses. The book was written by a capable group of contributors that bring much experience to the work.

After an introductory chapter, we will review the incidence and prevalence of sports dental injuries and examine what we can infer from the data available to us. This will be followed by chapters that detail dentoalveolar injuries, endodontic issues in dental trauma, and facial trauma. We then will review definitive restoration of traumatized teeth. Chapter seven deals with the prevention of athletic dental injuries and explains dentistry's major role in athletics: the athletic mouthguard. The next chapter deals with mouthguards, public health, and public policy. We then look at the hot topic of concussion with pathophysiology, evaluation, and management as well as a look at the role of dentistry in this area. We then turn attention to the sports and exercise oral related diseases, dental erosion, tobacco use and their sequelae. Drug use in athletes will then be examined (both prescribed and illicit) with a focus on doping and performance enhancing drugs. This is followed by a thorough review of what we know about the new topic of oral appliances and performance. The final chapter tells the interested reader how to become a team dentist and be a valued member of the sports medicine team.

**Mark Roettger**
Minneapolis, MN, USA

# Contents

# Contributors

**Mansur Ahmad**
Department of Diagnostic and Biological Sciences,
Division of Oral Medicine, Diagnosis and Radiology
University of Minnesota
Minneapolis, MN, USA

**Leon A. Assael**
University of Minnesota School of Dentistry
Minneapolis, MN, USA

Community Based Education and Practice
University of California, San Francisco
San Francisco, CA, USA

**Emilio Canal**
General and Sports Dentistry
Reston, VA, USA

**James R. Gambucci**
Department of Developmental and Surgical Sciences
Division of Oral and Maxillofacial Surgery (GPR)
University of Minnesota
Minneapolis, MN, USA
gambu001@umn.edu

**Dena P. Garner**
Department of Health and Human Performance
The Citadel
Charleston, SC, USA
garnerd1@citadel.edu

**Jeff Hoy**
General and Sports Dentistry
Torrance, CA, USA

**Kristine Knoll**
Endodontics Program
University of Minnesota
Minneapolis, MN, USA

**Douglas L. Lambert**
Cosmetic, Family and Sports Dentistry
Edina, MN, USA
DDSSmile@aol.com

**Michael Longlet**
General Dentistry
Bend, OR, USA

**Luke A. McMahon**
Oral and Maxillofacial Surgical Consultants
Edina, MN, USA
mcmah117@umn.edu

**Danette McNew**
Texas A&M College of Dentistry
Dallas, TX, USA

**Steve Mills**
Pediatric Dentistry
Scarborough, ME, USA
lmills5977@aol.com

**Paul Nativi**
Center for Advanced Dental Education
St. Louis University
St. Louis, MO, USA

**Jennifer Oberstar**
Department of Family Medicine and Community Health
University of Minnesota
Minneapolis, MN, USA
ober0042@umn.edu

**Ryan Pacheco**
General Dentistry
San Francisco, CA, USA
pache052@umn.edu

**Chris Rivers**
Oral and Maxillofacial Surgery Program
University of Minnesota
Minneapolis, MN, USA
river105@umn.edu

**Mark Roettger**
Department of Developmental and Surgical Sciences
Division of Oral and Maxillofacial Surgery (GPR)
University of Minnesota
Minneapolis, MN, USA
roett003@umn.edu

**Leslie Rye**
General and Sports Dentistry
Reston, VA, USA
leslierye2th@aol.com

**Zainah Shaker**
Gillette Children's Specialty Healthcare Dental Clinic
St Paul, MN, USA

**Scott Sutter**
Endodontics
Reno, NV, USA
sgs426@gmail.com

**Todd Thierer**
Clinical Affairs School of Dentistry
University of Minnesota
Minneapolis, MN, USA
tthierer@umn.edu

**Matthew Greaves**
Comprehensive Care Group, ATSU Missouri School of Dentistry and Oral Health
Ste Louis, MO, USA

**Vladamir Leon-Salazaar**
Private Practice in Orthodontics
Minneapolis, MN, USA

Department of Orthodontics, University of Detroit Mercy School of Dentistry
Detroit, MI, USA

Division of Orthodontics, University of Alberta School of Dentistry
Edmonton, AB, Canada

# Introduction to Sports Dentistry

*Mark Roettger and Steve Mills*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_1

## 1.1 History of Sports Dentistry

Sports dentistry was born from attempts to prevent oral and facial injuries suffered during athletic competition. There have been many attempts over the years to devise schemes to prevent all types of injury from sport, and oral injuries are no different. Oral and dental injuries in sport have been accepted in some sports in the past such as hockey, rugby, and American football (◘ Fig. 1.1), but dental injuries can carry significant emotional and esthetic ramifications. Boxing was one of the first sports to see the benefit of sports dentistry (see ▶ Chap. 7). More recent, there have been attempts to prevent and manage dental injuries in sport, and in the late 1950s and early 1960s, modern sports dentistry was founded. Injuries in American football in the 1950s were seen frequently in the head and facial region. Dentistry stepped up and challenged high school and college football organizations to prevent dental injuries by mandating the use of protective mouthguards, and a mouthguard mandate was adopted for these levels in 1962 [1]. Along with the use of face masks on the football helmet, mouthguards prevented most of the injuries experienced by these football players as facial injuries went from 50% of all injuries in football to .5% [2–4]. This was the beginning of modern sports dentistry. Sports dentistry in 2016 is considered one of the newest and up-and-coming fields in dentistry [5]. This is in part due to the ever-increasing popularity in sports participation. As we will see in ▶ Chap. 2, the participation numbers are steadily increasing with no signs of slowing. Athletes start competing at an earlier age, and Title IX in the USA assures equal opportunity for female sports participation (◘ Fig. 1.2). Sports today are becoming more extreme than ever before. Skiing, snowboarding, and cycling are all becoming more extreme in nature which in turn increases the chance of sustaining an injury (◘ Fig. 1.3). All these factors considered together assure us that the risk of injury in sports continues to increase each year. What then is the focus of modern sports dentistry?

◘ **Fig. 1.1** Common site in American football in the 1950s when dental injuries were expected. (Photo courtesy of ASD member slide series)

◘ **Fig. 1.2** Girls are participating in sports in numbers never before seen driving participation numbers ever higher. (Photo credit Mark Roettger DDS)

■ Fig. 1.3 Sports are rapidly becoming more extreme which increases the chances for injury. (Photo Credit Matthew Roettger)


■ Fig. 1.4 Academy for sports dentistry: a premier sports dentistry organization

## 1.2 Academy for Sports Dentistry

The Academy for Sports Dentistry (ASD) was formed in San Antonio, Texas, in 1983 by a dedicated group of dentists interested in protecting the oral health of athletes (■ Fig. 1.4). The Academy boasts an international membership of over 600 members, many of whom are former athletes involved in a wide variety of research activities or with a strong interest in this field of study. Each year the ASD hosts their Annual Symposium usually in a city in North America. The program is full of dental trauma and trauma prevention information as well as additional information pertinent to sports dentists, such as concussion, nutrition, and musculoskeletal injuries. Members receive the journal *Dental Traumatology* as a benefit of membership, which helps to keep members current in treatment of dental trauma. The ASD also holds a team dentist course that is aimed at preparing dentists to work as a part of a medical staffs of an athletic team from high school through professionals.

## 1.3 Definition of Sports Dentistry

For nearly 40 years, there was no real definition to the field of sports dentistry, and in 1998 the Academy for Sports Dentistry (ASD) created their first position statement that defined sports dentistry, and it has been revised to its current form today. "Sports Dentistry is the branch of sports medicine that deals with the prevention and treatment of dental injuries and related oral diseases associated with sport and exercise" [6]. The current definition of sports dentistry involves not only the prevention and treatment of dental injuries in sport but also related oral diseases associated with sport. Related oral diseases in sports may include dental erosion and dental caries from increased use of sports drinks or from emesis for weight control in body conscious sports such as gymnastics or weight class sports like wrestling (■ Fig. 1.5). The use of tobacco in the culture of some sports like baseball and hockey is aimed at getting the stimulant effects of nicotine, a potent mood stimulant, and many athletes wrongly believe that nicotine can improve performance. Nicotine is highly addictive, and other products in tobacco whether smoked or chewed are dangerous carcinogens (■ Fig. 1.6) [7]. Oral cancer has been a sequelae to athlete habits over the years, and dentistry must be involved to stop this phenomenon. Spit tobacco usage also causes caries and periodontal destruction and is harmful to oral health in addition to being carcinogenic. Periodontal disease and dental caries are the two diseases fought exclusively by dentistry, and we need to take a very active role in this fight in the athlete community. Sports dentistry has begun additional research into the possibility that specially designed oral appliances may improve athletic performance (▶ Chap. 13). This will negate the need to use nicotine stimulants to improve performance and could improve athlete oral health by eliminating carcinogen usage and protecting the teeth from traumatic injury. Preventing and managing dentoalveolar trauma from sports injuries is a large task, but sports dentistry has become much more. Modern sports dentistry's aim is to protect and improve the oral health of all athletes, and by improvement of oral health, we improve the total health of our athlete patients. We also cannot forget retired and aging athletes and their special oral health needs including dental solutions for managing the detrimental effects of obstructive sleep apnea. Obstructive

**Fig. 1.5** Female athlete with a history of drinking 4–5 energy drinks per day as a performance aid. Severe erosion and caries resulted carrying a consequence of very early tooth loss in an otherwise healthy young woman. (Photo credit Mark Roettger DDS)

**Fig. 1.6** Oral cancer from tobacco use. (Photo credit Mark Roettger DDS)

sleep apnea is seen as a growing problem in the athlete population especially in sports where size and body mass are important to success. Obstructive sleep apnea and sleep-disordered breathing carry significant health problems for those affected. Sleep-disordered breathing increases the risk of hypertension, cardiovascular disease, congestive heart failure, cardiac arrhythmias, cerebral vascular accident, as well as the dangers of excessive sleepiness such as motor vehicle accidents [8].

**Mission of Modern Sports Dentistry: Improve the Oral Health of Athletes**

1. Prevent injuries to the face, mouth, dentition, and jaws
2. Effectively manage these injuries when they do occur
3. Insure that athletes have the same preventative care that the general public has
4. Counsel athletes on the use of harmful product or practices
    1. Tobacco
    2. Sports and energy drinks
    3. Emesis to achieve a body image or weight
5. Look out for athletes of all ages
    - Sleep apnea
6. Use scientific facts and evidence whenever possible to make decisions

## 1.4 Evidence-Based Sports Dentistry

Sports dentistry can encompass quite a number of different topics, but at its core is the treatment and prevention of athletic injuries. These two areas should be approached scientifically, and dental intervention should be guided by the evidence-based literature. But what does that mean in the area of sports dentistry, and is it possible for us, as sports dentists, to function in an evidence-based way?

Evidence-based dental treatment is founded on the triad of dentist's experience and skill, the patient's needs and desires, and the scientific literature and knowledge base (Fig. 1.7, Venn Diagram). The scientific literature can be impossibly voluminous and the information contained can come in a variety of strengths and quality. This has led to the development of the "evidence-based pyramid" that stratifies the various sources of dental information that an individual has available to them to make clinical decisions (Fig. 1.8, pyramid).

At the top of the pyramid are randomized controlled clinical trials and systematic reviews of the literature. Both critical summaries of systematic reviews and meta-analyses can be included here as well. These forms of information should guide most clinical guidelines and indeed most clinical decisions. Unfortunately some areas of dentistry, and sports dentistry is one of these, do not lend themselves to randomized clinical trials and have few systematic reviews available.

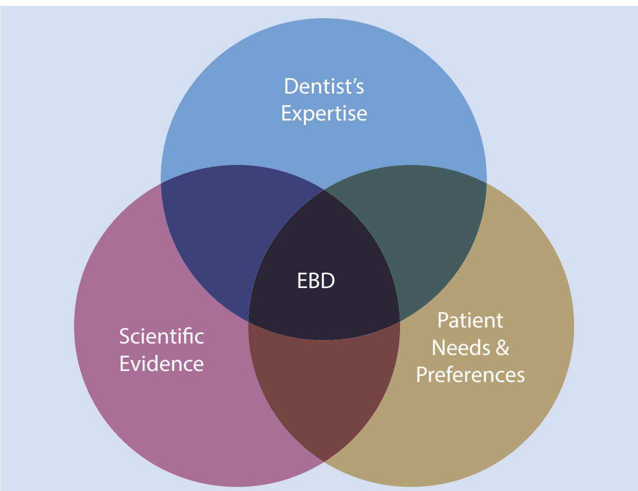


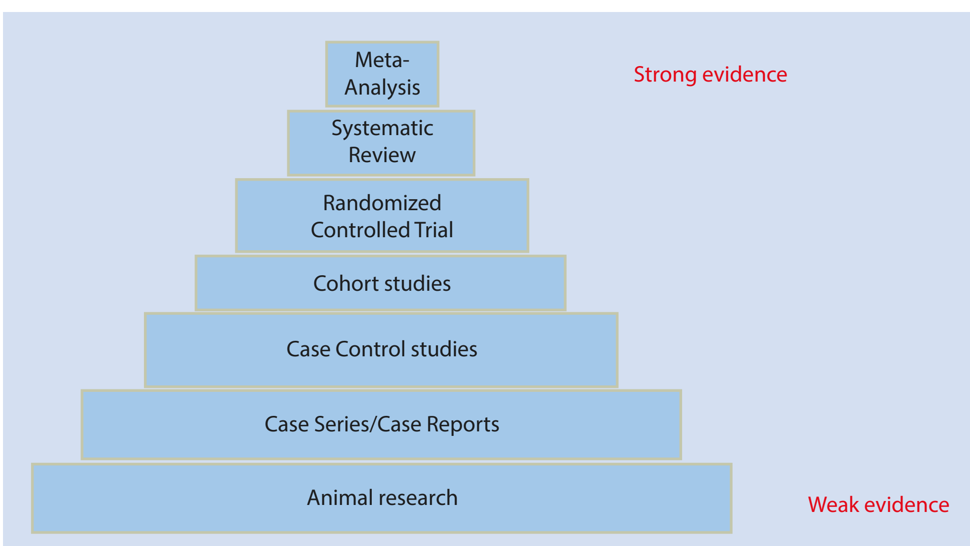

**Fig. 1.7** Foundation of evidence-based dentistry. (Copyright 2016 American Dental Association. All rights reserved. Reprinted by permission)

The lower half of the pyramid that contains examples such as expert opinions, in vitro trials, and case studies is where most of the literature of sports dentistry falls. This is not to say that this is unimportant or that it lacks value. In the absence of other forms of information, an individual must make use of what knowledge is accessible. In fact, in vitro testing of mouthguards and materials is critical to our everyday use of different techniques and in helping to define the best characteristics of mouthguards. Retrospective surveys of populations to assess the needs, knowledge, experiences, and desires of sports participants have also been of great value to our understanding of our subject. These are important but they do not quite reach the level of strong information.

Sigurdsson, in an article on evidence prevention of dental injuries, cites only two prospective studies and one systematic review that he deems useful when discussing the protective capabilities of mouthguards [9]. One of these, a systematic review and meta-analysis by Knapik et al in 2007, has also been critically reviewed by Mascarenes in 2012 [10, 11]. The systematic review was given a grade of B for inconsistent data and patient-reported information. The information contained was deemed limited-quality evidence, but it remains the most acceptable review of the sports dentistry literature.

Dentists in Japan investigated the lack of good clinical evidence in sports dentistry and responded in 2013 with an article proposing a long-term clinical prospective trial. This type of effort will add greatly to the strength of the science behind sports dentistry and particularly to the benefits of mouthguards [12]. Our directive to become evidence based in sports dentistry is clear, but the road ahead is difficult as these types of studies are large cumbersome and difficult to control as we will see in ▶ Chap. 2 of this book.

**Fig. 1.8** Evidence-based pyramid



## 1.5 Interprofessional Care Opportunities

We have entered a new phase in medical and dental practice. This is the time of collaborative practice and interprofessional care. We see this push on every medical or dental campus, in most if not all academic health centers across the globe and beginning to be a part of private practices of medicine and dentistry. Interprofessional care of athletes has been going on for some time. Sports medicine teams include athletic trainers, physicians of many specialties, dentists, physical therapists, and psychologists all working together as a team to keep athletes healthy and performing at the peak of their abilities. This book provides all members of the sports medicine team a more detailed look into the world of dentistry and how dentistry fits into the sports medicine team. Sports medicine teams should serve as a template for collaborative integrated healthcare practice everywhere.

Dentistry is sometimes overlooked in some sports medicine teams because many dental injuries do not limit the athlete from participation in competition. This does not mean that these injuries are not important to the athlete. Some injuries that don't require limited participation may be reinjured more easily if the original injury is not managed or protected properly. All dental injuries should be reviewed by a team dentist for plans to manage the injury going forward. This will happen as the sports medicine team evolves and becomes more collaborative and integrated. When this occurs, athletes receive better care and exhibit better overall health.

## References


1. American Dental Association. Report of the Joint Commission on Mouth Protectors of the American Association of Health, Physical Education and Recreation and the American Dental Association. Chicago, IL: ADA; 1960.
2. Cathcart J. Mouth protectors for contact sports. Dent Dig. 1951;57(8):346.
3. Flanders RBM. The incidence of orofacial trauma in sports: a pilot study in Illinois. JADA. 1995;126(4):491–6.
4. McCarthy M. Sports and mouth protectors. Gen Dent. 1990;38(5):343–6.
5. Saini R. Sports dentistry. Natl J Maxillofac Surg. 2011;2(2):129–31.
6. Academy for Sports Dentistry. Definition of sports dentistry. Springfield, IL: ASD; 2012.
7. Silverman S. Oral Cancer. 5th ed. Hamilton: B. C. Decker; 2003.
8. Fairbanks D, Mickelson S, Woodson B. Snoring and obstructive sleep apnea. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2003.
9. Sigurdsson A. Evidence based review of prevention of dental injuries. Pediatr Dent. 2013;35(2):184–90.
10. Knapik J, Stephan W, Marshall S, Lee RB. Mouthguards in sports activities: history, physical properties and injury prevention effectiveness. Sports Med. 2007;37(2):117–44.
11. Mascarenhas A. Mouthguards reduce orofacial injury during sport activities, but may not reduce concussion. J Evid Based Dent Pract. 2012;12(2):90–1.
12. Maeda Y, et al. Is mouthguard effective for preventing traumatic injuries during sports event?: A strategic protocol formulated by The Japanese Academy of Sports Dentistry (JASD) to accumulate scientific evidence. Int J Sports Dent. 2013;6(1):7–12.

# Epidemiology of Athletic Dental Injuries

*James R. Gambucci*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_2

2

## 2.1 Epidemiology

The World Health Organization defines *epidemiology* as the study of the *distribution* and *determinants* of health-related states or events (including diseases) and the *application* of this study *to the control of diseases and other health problems* [1].

**Epidemiology**

- The study of the distribution and determinants of health-related events and the application of this study to the control of these events.

Studies of the distribution of health-related events fall under the category of descriptive epidemiology.

**Descriptive Epidemiology**

- Purpose: To quantify the frequency and pattern of health-related events.
- Answer the following questions: *What* is the problem? *Who* is affected? *Where* does the problem exist? *When* does the problem occur?

Descriptive studies quantify the frequency and pattern of events in a population by answering the following questions: What is the disease or problem? Who is affected? Where does the problem exist? When does the disease or problem occur? A clear description of the problem is essential before any of the other questions can be answered, and that description should be universally accepted and used in all related studies. Personal characteristics such as age, sex, race, socioeconomic status, and level of education are common variables considered in describing the study population. A problem may be more prevalent in one location than another, such as urban vs. rural, developed vs. developing country, or one neighborhood, school, county, or states vs. another. Time variables may be seasonal, early or late in one particular season, or early or late during a particular activity [2].

Studies of the determinants of a disease or event consider the causes and contributing factors of occurrence.

**Central Tenet of Epidemiology**

- **Adverse health effects do not occur randomly but as a result of exposure of the individual to risk factors and other conditions that result in susceptibility to the disease or condition.**

Central to the study of determinants of disease is the presumption that adverse health effects do not occur randomly but as a result of exposure of the individual to risk factors and other conditions that result in susceptibility to the disease or condition [3]. Analytical epidemiology is the branch of the discipline that answers these questions: Why does the disease or condition occur in one population and not another? How does the disease or condition occur?

**Analytical Epidemiology**

- Purpose: Compare populations in which health-related problem occurs at different rates to determine if characteristics of the populations or risk factors to which they are exposed can explain the difference.
- Answer the following question: Why does the health-related condition occur in one population and not another?

Analytical studies compare populations in which disease or conditions occur at different rates to determine if characteristics of the populations or risk factors to which they are exposed can explain the differences [4].

Understanding the distribution and determinants of health-related states or events is useful only if the information can be applied to human populations for the prevention or control of those events, the ultimate goal of any public health undertaking. A broad understanding of cultural, behavioral, social, economic, and other characteristics of the population and condition is necessary because these and other factors unique to the population often determine whether individuals will adopt recommended interventions.

**Requirements for Successful Intervention**

- An understanding of the *distribution* of disease
- An understanding of the *determinants* of disease
- An understanding of *cultural*, *behavioral*, *social*, and *economic* characteristics of the population

## 2.2 Epidemiologic Research and Study Design

Since epidemiology is concerned with the distribution and determinants of disease in human populations, epidemiologic research is the application of scientific discipline to the study of those concerns.

**Epidemiologic Research**

- The application of scientific discipline to the study of the distribution and determinants of disease in a human population

Research can take place in a laboratory or in the field, can be descriptive or analytical, and can employ experimental or observational methods [5]. Laboratory studies involve manipulation of study populations in a tightly controlled setting. They are inherently experimental in design. In experimental studies, investigators separate a homogeneous population into two groups and apply a treatment or preventive measure to one, the study group, and not to the other, the control group. In randomized controlled experimental studies, the subjects are randomly assigned to one group or the other, and the investigator has control over confounding variables and all other aspects of the experiment. The results of studies that feature randomization of populations and control of variables are assumed to be valid for an entire population, not just for the particular group being studied.

**Laboratory Studies**

- Involve manipulation of study populations in a very controlled setting.
- Inherently experimental in design.
- Study populations are randomized.
- Results can be generalized over an entire population.

Field studies take place in a community setting during the course of normal activities in a population. Field studies are inherently observational in design. In observational studies, such as the injury surveillance studies that characterize most of the literature on dental trauma, investigators observe subjects during specified activities and measure predetermined variables. Because randomization of study populations and strict control of variables are not characteristics of observational studies, results cannot be generalized across an entire population. The results of any single study apply only to the specific population in that study.

**Field Studies**

- Take place in a community setting during the course of normal activities.
- Study populations are not manipulated.
- Inherently observational in design.
- Results cannot be generalized over an entire population.

Therefore, one of the tenets of observational epidemiologic studies is that pooling data from a large number of studies *of similar design and execution* is necessary to draw valid conclusions than can be applied with confidence across populations.

**Tenet of Observational Studies**

- Pooling of data from a large number of studies *of similar design and execution* necessary to draw valid conclusions that can be generalized across a population

Observational studies can be analytical if there is a search for association between a disease occurrence and possible causative, contributing or preventive factors that have been suggested by surveys or descriptive studies [4].

## 2.3 Epidemiology of Dental Trauma

In the area of sports-related dental traumatology the disease occurrence is traumatic injury to the teeth and related supporting structures and soft tissue. Since the etiology and immediate cause of sports-related traumatic dental injuries are well-established—the transfer of mechanical energy to the teeth and mouth due to contact with the ground or some other fixed structure, contact or collisions with another participant, or being struck by an object, either a projectile such as a ball or puck or the device used to launch the projectile, like a stick or racket [6]—attention in analytical observational studies focuses on contributing and preventive factors.

Analytical epidemiologic studies of preventive factors associated with sports-related traumatic dental injuries have traditionally focused on the use of intraoral mouthguards and/or face protection to prevent injuries. Since most high-risk sports, football, hockey, lacrosse, field hockey, and rugby, or high-risk positions in sports with a moderate risk of dental trauma—catchers in baseball and softball—require one or both types of protection for participants, attention has turned to sports with a comparatively moderate risk for traumatic dental injuries, such as soccer, baseball, and basketball, and to factors other than mouthguards that are associated with the prevention of traumatic dental injuries. While the dental injury rates for these sports do not approach the rate for the high-risk sports prior to mandatory mouth and face protection, they exceed the current rate of dental injury in protected athletes for high-risk sports [7].

Factors contributing to risk of sports-related dental trauma can be classified as intrinsic and extrinsic.

**Risk Factors**

- Intrinsic risk factors: Physical characteristics and psychological profile of individual athletes
- Extrinsic risk factors: Type of sport, level of competition, environment, coaching, and protective equipment

Intrinsic factors include the physical characteristics and psychological profile of individual athletes. Age, gender, anatomic anomalies, motor abilities, and sports-specific skills are examples of physical characteristics. Psychological profile includes motivation and risk-taking behavior. Of the intrinsic risk factors, sports-related traumatic dental injuries are associated most strongly with age (higher risk through adolescence, then decreasing with age) and anatomic features (higher risk with increasing occlusal overjet and lip incontinence). The association between gender and sports-related traumatic dental injuries is weakening steadily as sports participation by girls and women increases [8]. Extrinsic factors include type of sport, position played, exposure time, level of competition, coaching, referees, and protective equipment. The association between extrinsic risk factors and sports-related traumatic dental injuries is strongest for protective equipment (lower risk when using mouthguard and face mask protection) and type of sport (higher risk when competing in contact sports and sports involving sticks and balls). The list of factors contributing to the risk of traumatic dental injuries in sports underlines the complexity of the issue of risk mitigation [9]. Each athlete has a unique set of intrinsic variables that may confer greater or lesser risk for injury, and the interplay among and between intrinsic and extrinsic factors results in an almost infinite range of risk for participants in athletic events [10].

Formal analyses of observational studies of traumatic dental injuries are problematic for several reasons. There is tremendous diversity in study design and populations studied, which complicates combining studies for analysis and brings into question any conclusions drawn from such analysis [6]. Sports-related injuries are considered multi-risk phenomena, so it is difficult to isolate one cause from the many possible causes and confounding variables that coexist during athletic competition [10]. Finally, sports-related dental trauma is a relatively rare occurrence since face and teeth protection became common in the most high-risk sports [11] Studies of low-incidence phenomena often fail to reach a level of statistical significance, limiting the ability of interested parties to draw from these studies valid and defensible conclusions that could affect policy.

Inconsistency in the design and execution of epidemiologic studies of sports-related dental traumatic injuries is significant. The Task Force on Community Preventive Services of the *American Journal of Preventive Medicine* identified sports-related craniofacial injuries as preventable causes of health-care expenditures. Their search of the literature resulted in only four studies on the topic that met the criteria for inclusion in their report. They concluded that together these studies "provide insufficient evidence of the effectiveness of such programs in changing the behavior of players or in reducing the frequency of sports-related injuries to the head, face, and mouth" because "effectiveness could not be established, mainly because of inadequate number, design, or execution of studies" [12].

Studies that yield fruitful information on the topic of sports-related traumatic dental injuries vary in all of the ways mentioned in the previous section and also in scope. Some look at traumatic dental injuries during a single activity or specific sport [13]. Some document all sports-related traumatic dental injuries [14]. Some report on traumatic dental injuries from all causes, usually with sports-related injuries as one of the causes [15]. Some are interested in all sports-related traumatic injuries, not just dental injuries [16].

As in many areas of inquiry, most of these studies suffer from imperfections in design and methods, rendering the data imperfect or incomplete. Others are limited in scope. Together, however, a broad picture of the epidemiology of sports-related traumatic dental injuries can be drawn.

**Oral Trauma Statistics**

1. The oral region comprises 1% of total body area.
2. The oral region accounts for 5% of all body injuries.
   - In preschool children, oral injuries can account for as high as 17% of all body injuries.
3. Incidence of oral trauma is 1–3%.
4. Prevalence of oral trauma is 20–30%.
5. The annual cost to treat oral trauma in the United States alone is \$2–\$5 million per 1 million people.

J Endod. 2013 Mar;39(3 Suppl):S2-5. doi: 10.1016/j.joen.2012.11.021.

The number of people participating in sports, the prevalence and incidence of traumatic dental injuries in that group, and the risk factors associated with traumatic dental injuries all fall under the umbrella of epidemiology of traumatic dental injuries.

**Epidemiology of Traumatic Dental Injuries**

- Number of people participating in sports
- Prevalence and incidence of traumatic dental injuries
- Risk factors associated with traumatic dental injuries

### 2.3.1 Sports Participation

Estimates suggest that up to 45,000,000 youth and adolescents age 5–18 participate in organized sports activities in the United States [17].

**Participation in Organized Sports in the United States**

- Age 5–18: 45,000,000
- High school students: 7,800,000
- College students (NCAA): 482,000

The National Federation of High School Associations reports participation among high school students surpassed 7,800,000 in 2014–2015, which marks the 26th consecutive year participation has increased [18]. According to the National Collegiate Athletic Association (NCAA) participation in both men's and women's sports reached an all-time high for the 2014–2015 season. Approximately 482,000 student-athletes participated in sports in which the NCAA conducts championships that year [19].

### 2.3.2 Prevalence of Traumatic Dental Injuries

Prevalence of traumatic dental injuries is the proportion of a given population with evidence or history of a traumatic dental injury at a specific point in time. Since traumatic dental injuries are not necessarily sports-related, researchers must rely on the recollection of the subjects to record those injuries that are sports-related. As a result, prevalence of dental injuries in a population is of limited value in documenting sports-related dental injuries.

It is instructive to consider a prevalence study to demonstrate the scope and limitations of prevalence data. NHANES III, the Third National Health and Nutrition Examination Survey, 1988–1994, included dental examinations on and interviews with 13,057 individuals between 6 and 50 years old. This representative sample of the US population, adjusted for sociodemographic variables including gender and race-ethnicity, and occlusal characteristics, specifically overjet and overbite, yielded an overall prevalence for incisal trauma of 23.45%, although prevalence in subjects between 21 and 50 was 27.09%. The odds of traumatic injury increased with increasing overjet. The authors caution that these figures likely underestimate the actual prevalence due to reliance on recall of patients as to the history of trauma. The effect of overbite on trauma is also underestimated in this type of study because many subjects undergo orthodontic therapy after suffering a traumatic injury to the incisors. Since the excessive overbite that existed at the time of the traumatic injury no longer exists at the time of the survey, the relationship between overbite and injury is not recorded. It is also important to note that since prevalence data is cumulative, prevalence of traumatic dental injuries will be greater in older cohorts even though the incidence of dental trauma is greater in younger age groups [20].

### 2.3.3 Incidence of Traumatic Dental Injuries

Incidence of sports-related traumatic dental injuries is the number of injuries that occur in a specific at-risk population over a specified period of time. To record an incidence rate, an accurate count of the number of people at risk is essential, since the rate is reported as injuries per population at risk. Clinical incidence differs from incidence rate. Clinical incidence is simply the ratio between new injuries and the population at risk. It does not take into account time of exposure, so it does not give good information on actual risk. Incidence rate measures injuries against total time at risk, which gives a more accurate measure of true risk.

**Incidence of Traumatic Dental Injuries**

- Clinical incidence: Number of injuries per total population
- Incidence rate: Number of injuries per participant or per amount of time at risk

## 2.4 Injury Surveillance

Injury surveillance is the process of documenting information about the occurrence of injuries and the factors associated with those injuries [10]. The intent of injury surveillance is to identify high-risk activities and the factors that contribute to injuries, with the ultimate goal of instituting policies and protocols to mitigate risk.

**Injury Surveillance**

- The process of documenting information about the occurrence of injuries and the factors associated with those injuries

Surveillance studies of sports-related traumatic dental injuries, like all surveillance studies, employ observational methods.

**Injury Surveillance Studies**

- Are field studies.
- Are observational in design.
- Subjects observed during specified activities.
- Predetermined variables are measured.
- Study populations are not randomized.
- Variables cannot be strictly controlled.
- Results cannot be generalized over the entire population.

Because observational study design precludes the use of randomization and control of the study groups, data from numerous studies that employ similar methodologies need to be pooled in order to arrive at useful conclusions [21].

In prospective, or cohort, studies, where groups are followed over time, injuries can be defined clearly and documented with respect to exposure time, severity, etiology, and intrinsic and extrinsic risk factors. "Active" collection of information, which characterizes prospective studies, provides ongoing data on all injuries but also introduces risk of inaccuracies because of the number of judgments involved in classifying the injuries. "Passive" collection characterizes retrospective or case series study design and involves reviewing medical or insurance records of injured participants. Such studies provide accurate, thorough, and consistent information [10]. However, since these records tend to exist only in cases of severe injuries, they understate the actual incidence of traumatic injuries [8]. In the case of both active and passive collection of data, the fact that athletic trainers or emergency room physicians rather than dentists usually make these judgments increases the risk of inaccuracies and compromises the usefulness of such data.

### 2.4.1 Design and Execution of Surveillance Studies

Surveillance studies of traumatic dental injuries vary in the scope and depth of information gathered and the precision and methods used to collect and report the information. Studies have been reported from countries on every continent of the globe, on subjects of all ages, and on every conceivable sport at various levels of competition [22].

The literature on sports-related traumatic injuries is enriched by all of these of studies. They are observational in design, and one of the tenets of such studies is that many studies of similar design and execution need to be combined and data pooled to draw an accurate picture of the issues in question. However, because there is little consistency in study design and execution of these observational injury surveillance studies, confidence in the conclusions drawn from combining the data from them is low. Therefore, they are unlikely to drive or support action to prevent traumatic dental injuries [21]. Three issues in the design and execution of studies limit the impact such studies could have on policies regarding injury prevention: the definition of an injury, the denominator used in compiling the risk, and the method of data collection [8].

#### 2.4.1.1 Definition

Dental trauma is an injury to the enamel, dentin and/or pulp of the tooth, the periodontium, the alveolar bone, and surrounding soft tissues.

**Dental Trauma**

- Definition: Traumatic injury to the enamel, dentin and/or pulp of the tooth, the periodontium, the alveolar bone, and/or surrounding soft tissue
- Etiology: The transfer of mechanical energy to the teeth and mouth

Sports-related traumatic dental injuries have traditionally been defined as traumatic injuries to the teeth and oral cavity as described above but which occur during a formal practice, game, or performance which require medical attention by a team physician, certified athletic trainer, personal physician, or emergency department/urgent care facility and which prevent the athlete from returning to participation for one or more days.

**Sports-Related Dental Trauma**

- Dental trauma that occurs during a formal practice, game, or performance which require medical attention by a team physician, certified athletic trainer, personal physician, or emergency department/urgent care facility that may or may not result in time lost from participation
- The unintended consequences of individual actions in a risky environment

Recently the criteria were modified; any dental injury regardless of time lost from participation is reportable [23]. There are a number of widely accepted systems for classification of dental traumatic injuries in use and an almost infinite number of variations of these systems. All systems include various degrees of crown and/or root fracture and various degrees and types of luxation and avulsion. Some include fracture of the maxilla or mandible. Some include laceration of intraoral soft tissue. Some include lip laceration. Some include concussion without luxation but with marked sensitivity to percussion. Since sports-related traumatic dental injuries are relatively rare, aggregation of data from numerous studies is necessary for analysis to be carried out. Inconsistencies in how studies define and report dental injuries render such aggregation and analysis unreliable [24].

**Ellis Classification of Dental Injuries 1970**

It is a simplified classification, which groups many injuries and allows for subjective interpretation by including broad terms such as simple or extensive or extensive fractures.

- *Class I*—Simple crown fracture with little or no dentin affected
- *Class II*—Extensive crown fracture with considerable loss of dentin but with the pulp not affected
- *Class III*—Extensive crown fracture with considerable loss of dentin and pulp exposure
- *Class IV*—A tooth devitalized by trauma with or without loss of tooth structure
- *Class V*—Teeth lost as a result of trauma
- *Class VI*—Root fracture with or without the loss of crown structure
- *Class VII*—Displacement of the tooth with neither root nor crown fracture
- *Class VIII*—Complete crown fracture and its replacement
- *Class IX*—Traumatic injuries of primary teeth

**Classification by World Health Organization in Its Application of International Diseases of Dentistry and Stomatology (1994)**

1. Injuries to the hard dental tissues and the pulp
    1. Enamel infraction (N 502.50) An incomplete fracture (crack) of the enamel without loss of tooth substance.
    2. Enamel fracture (uncomplicated crown fracture) (N 502.50) A fracture with loss of tooth substance confined to the enamel.
    3. Enamel-dentin fracture (uncomplicated crown fracture) (N 502.51) A fracture with loss of tooth substance confined to enamel and dentin, but not involving the pulp.
    4. Complicated crown fracture (N 502.52) A fracture involving enamel and dentin and exposing the pulp.
    5. Uncomplicated crown-root fracture (N 502.54) A fracture involving enamel, dentin, and cementum, but not exposing the pulp.
    6. Complicated crown-root fracture (N 502.54) A fracture involving enamel, dentin, and cementum and exposing the pulp.
    7. Root fracture (N 502.53) A fracture involving dentin, cementum, and the pulp. Root fracture can be further classified according to displacement of the coronal fragment, as horizontal, oblique, and vertical.
2. Injuries to the periodontal tissues
    1. Concussion (N 503.20) An injury to the tooth-supporting structures with abnormal loosening or displacement of the tooth but with marked reaction to percussion.
    2. Subluxation (loosening) (N 503.20) An injury to the tooth-supporting structures with abnormal loosening but without displacement of the tooth.
    3. Extrusive luxation (peripheral dislocation, peripheral avulsion) (N 503.20) Partial displacement of the tooth out of its socket.
    4. Lateral luxation (N 503.20) Displacement of the tooth in a direction other than axially. This is accompanied by comminution or fracture of the alveolar socket.
    5. Intrusive luxation (central dislocation) (N 503.21) Displacement of the tooth into the alveolar bone. This injury is accompanied by comminution or fracture of the alveolar socket.
    6. Avulsion (exarticulation) (N 503.22) Complete displacement of the tooth out of its socket.
3. Injuries to the supporting bone
    1. Comminution of the mandibular (N 502.60) or maxillary (N 502.40) alveolar socket Crushing and compression of the alveolar socket. This condition is found concomitantly with intrusive and lateral luxations.
    2. Fracture of the mandibular (N 502.60) or maxillary (N 502.40) alveolar socket wall A fracture confined to the facial or oral socket wall.
    3. Fracture of the mandibular (N 502.60) or maxillary (N 502.40) alveolar process A fracture of the alveolar process which may or may not involve the alveolar socket.
    4. A fracture involving the base of the mandible or maxilla and often the alveolar process (jaw fracture). The fracture may or may not involve the alveolar socket.
4. Injuries to gingiva or oral mucosa
    1. Laceration of gingival or oral mucosa (S 01.50) A shallow or deep wound in the mucosa resulting from a tear and usually produced by a sharp object
    2. Contusion of gingiva or oral mucosa (S 00.50) A bruise usually produced by impact with a blunt object and not accompanied by a break in the mucosa, usually causing submucosal hemorrhage
    3. Abrasion of gingival or oral mucosa (S 00.50) A superficial wound produced by rubbing or scraping of the mucosa leaving a raw, bleeding mucosa

#### 2.4.1.2 Denominator

Measures of the rate of any occurrence require a numerator and denominator. The numerator alone, in studies of traumatic dental injuries always the number of injuries, tells you the frequency of an event but gives no information about the rate. A denominator, a function against which the number of injuries is expressed, is needed to indicate clinical incidence, the number of injuries per total population, and incidence rate, the number of injuries per participant or per amount of time at risk for being injured. Injuries per participant are usually expressed as injuries per athlete-year, and injuries per time of exposure are expressed per athlete exposure (AE), with one AE being a practice, game or performance/event, or per athlete-hour of exposure. Sports that involve many exposures or hours of competition or with many participants may have relatively low rates of injuries because of a large denominator but in fact may have a high incidence of injury. As with differences in how traumatic dental injuries are defined, differences in the denominator used to measure rate of injury make aggregation and analysis of data unreliable [8].

#### 2.4.1.3 Data Collection

Data on traumatic dental injuries can be obtained in several ways. In retrospective studies, for example, cross-sectional surveys using questionnaires and/or clinical examinations to document evidence of trauma or other oral conditions of interest can provide valuable information [25]. Records from emergency departments, medical or urgent care clinics, insurance companies, schools, or athletic organizations can be reviewed. Well-designed retrospective studies can provide information on prevalence of a condition, risk factors, and relative risk [26]. A national survey of a probability sample of US residents combined information from a questionnaire, an interview, and a clinical exam to document the overall prevalence of dental trauma and differences in prevalence based on age, gender, and other demographic factors [20]. Baseline information derived from such descriptive studies serve the important purpose of identifying the scope of a problem and susceptible individuals, providing a roadmap for further, more focused studies.

Prospective studies actively document injuries over time and afford the opportunity to record comprehensive information about the "who, what, where, when, and how" of the incidents. They also provide a denominator, which makes it possible to record and compare injury rates. Because prospective studies are time- and resource-intensive, they usually focus on small subgroups, or cohorts, of the population, usually groups identified through descriptive studies as being at higher risk for the condition. Prospective cohort observational studies are similar to experimental studies in that they track the transition from health to disease in a population subjected to risk factors. In observational cohort studies, however, exposure is observed but not determined by the investigator as they are in experimental studies [4].

Because of the lack of control over exposure and the many confounding variables that could contribute to the occurrence of traumatic dental injuries, the results of these studies lack validity and reliability in comparison to studies of classical "one cause, one disease" phenomena.

## 2.5 Injury Surveillance Systems

The impact of traumatic dental injuries incidence studies depends on their design and execution. A formal protocol using a well-designed instrument to study a representative and well-defined population is most likely to yield reliable data that can support policies for the reduction of traumatic dental injuries. There are three systems in use which demonstrate this ideal: the National Electronic Injury Surveillance System (NEISS), the National Collegiate Athletic Association Injury Surveillance System (NCAA ISS), and the High School Reporting Information Online (High School RIO).

**Instruments Used to Measure Traumatic Dental Injuries**

1. *NEISS*—National Electronic Injury Surveillance System
2. *NCAA ISS*—National Collegiate Athletic Association Injury Surveillance System
3. *High School RIO*—High School Reporting Information Online

The Consumer Product Safety Commission operates NEISS to collect data on consumer product-related traumatic injuries that occur in the United States. NEISS is used in the emergency departments in a national probability sample of hospitals in the United States and its territories, so the results obtained in this system can be extrapolated to provide an estimate of injuries nationwide. The system is designed to collect data on injuries associated with consumer products, including athletic and recreational products. A physician performs a thorough exam and provides an accurate diagnosis for each injury, and a very thorough and formal process to gather demographic and injury-specific information is employed, so data can be compared and easily pooled [27].

Data collected by NEISS are available to the public or other organizations and can be used to analyze data pertaining to consumer products and their association with injuries. Two retrospective studies that used NEISS data to document consumer product-related traumatic dental injuries demonstrate how NEISS data provide helpful information on the epidemiology of traumatic dental injuries in children.

In 1990, information on consumer product-related dental trauma was secured by surveying NEISS from 1979 to 1987 [28]. There were over 900 consumer products or activities associated with dental injuries, which the authors classified into 9 general categories. "Sports and Play" was one category, and "Bicycles and Wheeled Vehicles" was another. A total of 146,750 product-related traumatic injuries to teeth were recorded during this time. Children under 5 years old accounted for 40% of this total, those from ages 5–14 accounted for 35%, and 15% of the cases occurred in those from 15 to 24 years of age. Among children under 5 years of age, 70% of the injuries were associated with falls or collisions that occurred in the home. Among those 5–14 years old, 32% were associated with sports and play and an equal percentage with bicycles and wheeled vehicles. For those from 15 to 24 years of age, almost 50% of product-related dental trauma treated in emergency departments was associated with sports and play. Together, sports and play (25.1%) and bicycles and wheeled vehicles (18.51%) accounted for almost 40% of all dental injuries treated in emergency rooms during this time.

In 2009, another retrospective analysis of data from the NEISS, for the years 1990–2003, was conducted to describe the association of consumer products and activities and dental injuries among children under age 18 [15]. In this study products were divided into four categories: home structures/furniture, sports, outdoor recreation, and miscellaneous. Three age categories were used: under age 7, age 7–12, and age 13–17. Because NEISS data represent a statistically representative sample of hospital emergency departments in the United States, the epidemiology of injuries associated with consumer products and activities for the nation can be estimated from this sample data. The mean annual dental injury rate of children age 17 and under was 31.6 per 100,000 over the years of this study. Children under age 7 accounted for 59.6% of injuries, with 7–12-year-olds and 13–17-year-olds accounted for 29.5% and 10.9%, respectively. Sports accounted for 13.8% of the total number of injuries, with baseball (40.2%) and basketball (20.2%) accounting for the most injuries in this category.

Several factors underline disadvantages of using data from NEISS to document dental injuries. The data is skewed toward more severe injuries because most traumatic dental injuries that involve only the teeth are treated in private dental offices, not in hospital emergency rooms. As a result, data from NEISS underestimates the actual number of traumatic dental injuries. Also, there is no way to estimate rate or risk of injury from emergency department data because there is no data to document the number of exposures to the etiologic agent or number of people involved in a specific activity. Finally, emergency room physicians are less familiar with dental-oral assessment than are dentists, and emergency rooms lack some of the diagnostic tools available in dental offices such as intraoral radiology equipment. Together these factors result in less reliable data.

The National Collegiate Athletic Association Injury Surveillance System and the High School Reporting Information Online collect data on sports-related traumatic injuries from representative samples of their institutions. Certified athletic trainers (ATCs) who are on hand during all activities document the injuries and related demographic and incident-specific information. The NCAA ISS, which has been in effect since 1982, defines a reportable injury as "one that (1) occurred as a result of participation in an organized intercollegiate practice or competition, (2) required medical attention by a team certified athletic trainer or physician, and (3) resulted in restriction of the student-athlete's participation or performance for 1 or more calendar days beyond the day of injury." Since 1994, the definition of a dental injury was expanded to include any injury to the teeth regardless of time loss [29]. Injury rate in the NCAA ISS is based on injuries per athlete exposure (AE), defined as one athlete participating in one practice or competition that involved exposure to an athletic injury. Injury rates are reported as injuries per 1000 AEs. Separate injury report forms with data on the "who, what, where, when, and how" are generated for each injury in this system, and an exposure form with further information pertinent to the injury occurrence is provided to a central reporting institution each week (see ◘ Fig. 2.1).

The longevity of the NCAA ISS and its consistency in definition, denominator, and methods of data collection is unique in sports medicine literature as a source of information on the epidemiology of sports injuries in intercollegiate athletics. The High School RIO is patterned after the NCAA ISS and offers similar consistency in definition, denominator, and methods of data collection for high school athletics [30]. However, because athletic trainers rather than dentists gather the information in a setting without optimal diagnostic tools such as intraoral radiology, data from NCAA ISS and High School RIO lacks optimal validity and reliability.

Studies using NCAA ISS and High School RIO provide information on incidence of traumatic dental injuries, injury rates, and comparison of traumatic dental injuries to other types of injuries sustained by college and high school athletes and can identify trends in sports-related traumatic injuries because of consistent methods employed over time. This consistency is invaluable for those who want to use the data to support decisions to institute practices to prevent sports-related traumatic injuries.

Data from High School RIO has been used to describe the epidemiology of rare injuries sustained by high school athletes. A 2-year prospective study [11] identified five injuries or conditions considered to be rare or unusual but which can result in high morbidity and a heavy burden on health care. More common injuries, like sprains, strains, and fractures, have a much higher statistical profile, and therefore the identification of risk factors and efforts at prevention has been focused there. In this study rare injuries and conditions included dental, eye, and neck and cervical spine injuries and heat and dehydration illnesses. Although these injuries were rare when all sports-related injuries were pooled, they actually made up a high proportion of reported injuries in some sports. Rare injuries and conditions accounted for 321 injuries, approximately 3.5% of all athletic injuries. A total of 23 dental injuries occurred in this population during the 2-year study, translating to an injury rate (IR) of 0.65 per 100,000 athletic exposures (AEs), with baseball (IR of 3.22 per 100,000 AEs) and girls' basketball (IR of 1.12 per 100,000 AEs) having the highest rate of dental injuries. In this study only injuries to the teeth were counted as dental injuries. Other studies include soft tissue injuries to the lips and oral cavity as traumatic dental injuries, which render pooling of data or comparison of studies problematic.

High School RIO provided the data for another study, which looked only at dental injuries [23]. This study documented the rate of dental injuries, as injuries per athletic exposure, in various sports and under various conditions, the immediate cause of the injuries, and whether a protective mouthguard was worn at the time of injury. There were 222 dental injuries during 24,787,258 AEs, accounting for 0.5% of all sports-related injuries. The overall injury rate was 0.90 per 100,000 athletic exposures. Girls' field hockey had the highest rate of dental injuries (3.9 per 100,000 AEs) followed by boys' basketball (2.6), boys' baseball (1.5), and boys' wrestling (1.4). All dental injuries were recorded regardless of whether they resulted in restriction of participation. In this surveillance study, lip lacerations were the single most common dental injury, comprising 36.5% of all such events. Note that this was in contrast to the study reported above in which only injuries to the teeth were counted, not injuries to the soft tissue.

Data from the National Collegiate Athletic Association Injury Surveillance System (NCAA ISS) was used to report changes in incidence rate of traumatic dental injuries over a 10-year period at a single NCAA institution [31]. Incidence rates (IR) were calculated for 19 separate teams, involving 15 different sports, and were highest for men's and women's basketball. Of particular interest in this study was the fact that the women's basketball team instituted a mandatory

## 2003–04 Individual INJURY Form–Women's Volleyball
## NCAA Injury Surveillance System

INJURY DEFINITION: A reportable injury in the ISS is defined as one that:
1. Occurs as a result of participation in an organized intercollegiate practice or contest;
2. Requires medical attention by a team athletics trainer or physician; and
3. Results in any restriction of the athlete's participation or performance* for one or more days beyond the day of injury.
4. **Any dental injury regardless of time loss.**

* See POINTS OF EMPHASIS.

**School Code:** ____________________

**Select one:** Fall season Spring season

**1. Year:**
(1) FR (2) SO (3) JR (4) SR (5) Fifth

**2. Age:** ________ years **4. Weight:** ________ pounds

**3. Height:** ______ inches **5. Date of Injury:** ________ (month/day)

**6. Injury occurred during:**
(1) Preseason (before first regular-season match)
(2) Regular season
(3) Postseason (after final regular-season match)
(99) Other: ____________________

**7. Injury occurred in:**
(1) Competition–varsity
(3) Practice

**8. COMPETITION ONLY–Where did this injury occur?**
(1) Home (3) Neutral site
(2) Away (99) Other:_____

**9. Injury occurred during:**
**Competition:** (1) Warm-up (2) Game 1 (3) Game 2 (4) Game 3 (5) Game 4 (6) Game 5
**Practice:** (7) First half (8) Second half
(99) Other:_____

**10. This injury is a:**
(1) New injury
(2) Recurrence of injury from this season
(3) Recurrence of injury from previous season (this sport)
(4) Complication of previous injury (this sport)
(5) Recurrence of other-sport injury
(6) Recurrence of nonsport injury
(7) Complication of other-sport injury

**11. Has student-athlete had unrelated injury recorded this season?**
(1) Yes (2) No

**12. *Not applicable to this sport; proceed to next question.***

**13. How long did this injury keep student-athlete from participating in the sport?** (If end of season, give best estimate.)
(1) 1-2 days (4) 10 days or more
(2) 3-6 days (5) Catastrophic, nonfatal
(3) 7-9 days (6) Fatal

**14. This injury involved:**
(1) Contact with another competitor
(2) Contact with playing surface
(3) Contact with apparatus/ball
(4) Contact with other in environment (e.g., wall, fence, spectators)
(5) No apparent contact (rotation about planted foot)
(6) No apparent contact (other)
(99) Other: ____________________

**15. Principal body part injured** (for 1–10, complete Head-Injury Information; for 31 or 32, complete Knee-Injury Information):
(1) Head
(2) Eye(s)
(3) Ear(s)
(4) Nose
(5) Face
(6) Chin
(7) Jaw (TMJ)
(8) Mouth
(9) Teeth
(10) Tongue
(11) Neck
(12) Shoulder
(13) Clavicle
(14) Scapula
(15) Upper arm
(16) Elbow
(17) Forearm
(18) Wrist
(19) Hand
(20) Thumb
(21) Finger(s)
(22) Upper back
(23) Spine
(24) Lower back
(25) Ribs
(26) Sternum
(27) Stomach
(28) Pelvis, hips, groin
(29) Buttocks
(30) Upper leg
(31) Knee
(32) Patella
(33) Lower leg
(34) Ankle
(35) Heel/Achilles' tendon
(36) Foot
(37) Toe(s)
(38) Spleen
(39) Kidney
(40) External genitalia
(41) Coccyx
(42) Breast
(99) Other: ________

**HEAD INJURY** (answer only if response in question 15 was 1–10)

**16. This student-athlete was diagnosed as having:**
(1) 1° cerebral concussion. [No loss of consciousness, short post-traumatic amnesia (seconds up to two minutes).]
(2) 2° cerebral concussion. [Loss of consciousness (less than five minutes) and amnesia for up to 30 seconds].
(3) 3° cerebral concussion. [Loss of consciousness (more than five minutes) and extended amnesia.]
(4) No cerebral concussion
(5) Unknown

**17. Was a mouthpiece (MP) worn?**
(1) MP worn–dentist-fitted (3) MP not worn
(2) MP worn–self-fitted

**18. Type eye injury:**
(1) Orbital fracture (4) Soft tissue
(2) Cornea (99) Other:________
(3) Ruptured globe

**KNEE INJURY** (answer only if response in question 15 was 31 or 32)

**19. Circle ALL knee structures injured:**
(1) Collateral (5) Patella and/or patella tendon
(2) Anterior cruciate (6) None
(3) Posterior cruciate (99) Other:________
(4) Torn cartilage (meniscus)

**– Please Answer All Questions –**

**Fig. 2.1** NCAA ISS form

**– Please Answer All Questions –**

**20. Primary type of injury** (circle one):

(1) Abrasion
(2) Contusion
(3) Laceration
(4) Puncture wound
(5) Bursitis
(6) Tendinitis
(7) Ligament sprain (incomplete tear)
(8) Ligament sprain (complete tear)
(9) Muscle-tendon strain (incomplete tear)
(10) Muscle-tendon strain (complete tear)
(11) Torn cartilage
(12) Hyperextension
(13) AC separation
(14) Dislocation (partial)
(15) Dislocation (complete)
(16) Fracture
(17) Stress fracture
(18) Concussion
(19) Heat exhaustion
(20) Heatstroke
(21) Burn
(22) Inflammation
(23) Infection
(24) Hemorrhage
(25) Internal injury (nonhemorrhage)
(26) Nerve injury
(27) Blisters
(28) Boil(s)
(29) Hernia
(30) Foreign object in body orifice
(31) Avulsion (tooth)
(99) Other: __________

**21. Did a laceration or wound that resulted in oozing or bleeding occur as a part of this injury?**

(1) Yes (2) No

**22. Did this injury require surgery?**

(1) Yes, in-season (2) Yes, postseason (3) No

**23. Describe the joint surgery:**

(1) Arthrotomy
(2) Diagnostic arthroscopy
(3) Operative arthroscopy
(4) No joint surgery
(99) Other: __________

**24. Injury assessment (best assessment procedure):**

(1) Clinical exam by athletic trainer
(2) Clinical exam by M.D./D.D.S.
(3) X-ray
(4) MRI
(5) Other imagery technique
(6) Surgery
(7) Blood work/lab test
(99) Other: __________

**25. Injury occurred during:**

(1) Offensive play
(2) Defensive play
(3) Neither

**26. Type of surface:**

(1) Wood
(2) Composition
(99) Other: __________

**Additional comments (optional):** __________

**27. Injury was caused by:**

(1) Injured player coming down on another player
(2) Another player coming down on injured player
(3) Other contact with another player
(4) Contact with net
(5) Contact with standard
(6) Contact with floor
(7) Contact with ball
(8) Contact with out-of-bounds observers (team, fans, media, cheerleaders)
(9) Contact with out-of-bounds apparatus (tables, bleachers, cameras)
(10) No apparent contact
(99) Other: __________

**28. Injured player's activity:**

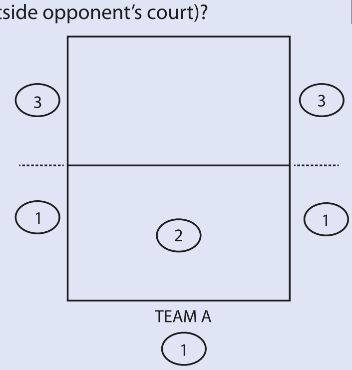
(1) Serving
(2) Spiking
(3) Setting
(4) Passing
(5) Digging
(6) Blocking
(7) Conditioning
(99) Other: __________

**29. Position played at time of injury (circle one):**

(1) Left front
(2) Center front (middle blocker)
(3) Right front
(4) Left back
(5) Center back (defensive specialist)
(6) Right back
(7) Nonpositional/conditioning drill
(99) Other: __________

**30. Assuming the athlete plays for Team A, which number best represents where the injury occured while she was playing the ball?**

(1) Area 1 (outside Team A's court)? ☐
(2) Area 2 (on court)? ☐
(3) Area 3 (across center line outside opponent's court)? ☐




**PRACTICE ONLY**

**31. Injury occurred during:**

(1) A triple session day
(2) A double session day
(3) A single session day
(99) Other: __________

**Fig. 2.1** (continued)

2

## 2003–04 Weekly EXPOSURE Form–Women's Volleyball
## NCAA Injury Surveillance System

EXPOSURE DEFINITION: An athlete exposure, the unit of risk in the ISS, is defined as one student-athlete participating in one practice or competition where he or she is exposed to the possibility of an athletics injury. Please report all weeks that include FORMAL team practices involving the entire team. Do not report optional or "captains" practices.

Note: Please be as accurate as possible in reporting number of participants. PRACTICE participants must be included in a majority of the drills, GAME participants must have **actual playing time**. In most cases, the number of game participants is *less than* the number of practice participants.

**School Code:** ______________________ **Week of:** ______________________
(Sunday to Saturday)

*Please answer all questions*

**PRACTICE**

1. **Number of practices this week (Sunday to Saturday):** ______________________
2. **This week was part of:**
   (1) Preseason (before first regular-season contest)
   (2) Regular season
   (3) Postseason (after final regular-season contest; includes conference, regional and national tournaments)
3. **Average number of participants per practice** (see instructions): ______________
4. **Number of practices on wood surface:** ______________
5. **Number of practices on composition (non-wood) surface:** ______________

**VARSITY CONTEST**

6. **Was a varsity contest played?**
   (1) No (stop)
   (2) Yes (go to next question)
7. **Number of varsity contests:** ______________
8. **For each varsity contest, provide the following information:**

| Contest | Total Number of Participants **With Actual Playing Time** (your Team) | Location (check one): Home | Away | Type of Surface (check one): Composition (non-wood) | Wood |
|---|---|---|---|---|---|
| No. 1 | ______ | ____ | ____ | ____ | ____ |
| No. 2 | ______ | ____ | ____ | ____ | ____ |
| No. 3 | ______ | ____ | ____ | ____ | ____ |
| No. 4 | ______ | ____ | ____ | ____ | ____ |
| No. 5 | ______ | ____ | ____ | ____ | ____ |

**Fig. 2.1** (continued)

mouthguard policy 4 years into the study. The IR before the policy was instituted was 8.3 injuries per 100 athlete-seasons. After all players were required to wear mouthguards, the IR dropped to 2.8. Due to the small sample size and relative infrequency of injuries, this difference was not statistically significant.

These examples of NEISS, NCAA ISS, and High School RIO in action point out that even when using a well-designed instrument on a representative population, variations can still occur in the design of studies and details of how the instruments are used. These variations rob the studies of universal applicability and make study-to-study comparison problematic. As a result, even studies that employ these ideal instruments often fall short of generating data reliable enough to drive policy.

## 2.6 Injury Prevention and Risk Compensation

The principle of Occam's razor suggests that the simplest solution to complex problems is the best and that if a cause is both true and sufficient to explain a phenomenon, there is no need to look further than addressing that single cause. In the case of traumatic dental injuries, this "true and sufficient cause" is the transfer of mechanical energy to the teeth and mouth. In the case of sports-related traumatic injuries, the mechanical energy is transferred from the ground or other immovable object, a projectile, or a device used to propel the projectile. Following the logic of Occam's razor, efforts to curtail or prevent athletic dental injuries should be focused simply on interrupting the transfer of energy rather than addressing the infinitely complex web of intrinsic and extrinsic factors associated with traumatic dental injuries.

Given that traumatic dental injuries, like injuries in general, are "the unintended consequences of individual actions in a risky environment" [32], there are three recognized strategies for injur prevention: persuade those at risk to change their behavior, require behavior change by law or rule, or provide automatic prevention through product and environmental design.

**Strategies for Injury Prevention**

- Persuasion to promote behavior change
- Require behavior change through enforced laws or rules
- Provide automatic prevention through product and environmental design

In general, rules work better than persuasion, and automatic protection works better than rules. Applying this model to the epidemiology of sports-related traumatic dental injuries and adhering to the principles of Occam's Razor, it is easy to conclude that since the automatic protection of the teeth and mouth provided by mouthguards and face masks is proven effective in the most high-risk sports, some variation of one or both should be written into the rulebooks of other less-risky activities. However, there is an intrinsic factor of human behavior that brings such a policy into question: risk compensation.

### 2.6.1 Risk Compensation

Risk compensation theory is based on the idea that people change their behavior in response to a real or perceived risk.

**Risk Compensation**

- Theory that people change behavior in response to real or perceived risk
  - Increased perceived risk, more cautious
  - Decreased perceived risk, less cautious

If risk increases, people become more cautious, as when drivers slow down when roads are icy to mitigate that risk. If risk decreases, however, do people consciously or subconsciously increase risky behavior? If so, does protecting one part of the body during sports participation lead to behavior that places other parts of the body at greater risk for injury? In the case of facemasks and mouthguards, does wearing them to eliminate the risk of injury to the face, eyes, and teeth increase the risk of other injuries due to carelessness or more aggressive play? The theory of risk compensation suggests that it does. Of particular interest in the field of sports medicine is the incidence of concussion and spinal cord injury.

The epidemiology of risk compensation and other behavioral factors in sports-related traumatic injuries is not conclusive. There are few studies of individual risk-taking behaviors before and after adoption of safety measures, so it is impossible to determine whether risk-taking behavior increases, decreases, or remains the same. The literature on this topic is characterized by conflicting results from a number of sources [16]. Bicycle helmet use corresponded with a greater level of caution in one study [33]. In another, helmeted bicyclists exhibited a much smaller risk of major trauma than non-helmeted bicyclists [34]. The findings of both of these studies are the opposite of what would be predicted by risk compensation theory. However, it was shown that when hard shell helmets and face protection were introduced into football, tackling fatalities increased dramatically, leading to the hypothesis that players felt so well-protected that they could use their head and face for tackling [35]. Rule changes making it illegal to "spear," or lead with the head while tackling, along with improvements in helmet construction resulted in a decrease in such fatalities. This suggests that risk behavior can be mitigated by rule changes and better safety equipment. In the sport of ice hockey, one study suggested that players with the greatest amount of protection, a helmet with full face shield, were less likely to suffer severe injuries or engage in illegal aggressive play than those wearing half-shields, a finding that is inconsistent with risk compensation theory [36]. On the other hand, a survey of 140 adolescent rugby players found that 2/3 of the respondents felt increased confidence in playing harder while wearing protective head gear [37]. A survey of a convenience sample of 190 recreational league hockey players with a mean age of 34 found that 70% of the players felt they played more aggressively when wearing full face protection and reported a higher incidence of injuries over time [38]. A study of the injury profile in ice hockey from 1976 to 1979, the 1988–1989 season, and the 1992–1993 seasons in Finland found that the incidence of severe injuries increased over time, though not to protected body parts like the face and head. The authors concluded that more aggressive and reckless play contributed to these findings [39].

While risk compensation theory suggests that greater protection from injury of one type increases the risk of injury of another due to increased risk taking, some argue that extrinsic factors, including better equipment and playing fields, new techniques, rule changes, and greater dedication to the intrinsic factors of physical fitness and strength training have, in the process of making modern games faster and more exciting for the players and spectators, made them more dangerous. In this model the proposal to introduce enhanced personal protective equipment is the result of an increased risk of injuries due to intrinsic and extrinsic factors. A descriptive epidemiological study to determine the incidence of head, face, and eye injuries in the sport of Women's Field Hockey in its modern form, with increased intrinsic and extrinsic risk factors as noted above, was conducted using NCAA ISS data from the 2004–2005 to 2008–2009 seasons [40]. Concussions were the most commonly reported injury, most of which resulted from direct contact with another player or contact with a stick. This is somewhat surprising because women's field hockey is a non-contact sport in which elevation of the stick to the level of the head is illegal. The author concluded that stricter enforcement of the rules to control overly aggressive play may be the best solution rather than introducing better protective equipment, which could result in more aggressive play. At this time the governing body of the sport in the United States is arguing against enhanced head protection for fear that it may lead to more reckless and aggressive play due to the false sense of security such equipment may impart.

These studies fall well short of offering enough information to guide policy. They are examples of how risk compensation may manifest in the area of sports-related traumatic injuries, however, and suggest that studies of the effectiveness of protective equipment and rules designed to protect players from specific injuries should take a broad enough view to consider whether negative side effects might accrue and increase the risk of other, more serious injuries.

A view of sport injury mitigation that considers all injuries and factors contributing to them holds more promise for making sports safer for participants than focusing on a series of isolated injuries. McIntosh [41] advocates taking a biopsychosocial approach to sport injury and considering the potential of extrinsic and intrinsic risk factors either increase or decrease the overall injury risk. This biopsychosocial model recognizes the primary contributors to disease—in the case of sports-related traumatic dental injuries, the mechanical forces transferred to the teeth and surrounding structures—but also acknowledges that environmental, social, and behavioral factors must be considered to devise effective methods for the prevention of sports-related traumatic injuries, including dental injuries.

This classic "chicken or the egg" dilemma—does enhanced protection lead to more aggressive play or does more aggressive play demand enhanced protection—is yet to be resolved. In the meantime, injury surveillance data from well-designed and executed studies offers the best hope to illuminate the issue of sports-related traumatic dental injuries and offer solutions that are consistent with the culture of the individual sports and which do not increase the risk of other sports-related traumatic injuries.

## 2.7 Conclusion

Epidemiology is the formal, systematic study of the distribution and determinants of health-related states or events and the application of this study to their prevention and control. In the context of this book, "health-related states or events" are sports-related traumatic dental injuries. Central to the study of the determinants of this condition is the presumption that traumatic dental injuries do not occur randomly but as a result of exposure of the individual to risk factors associated with such injuries. Analytical injury surveillance studies are employed to identify the factors that contribute to traumatic dental injuries, with the ultimate goal of instituting policies and protocols to mitigate risk. Injury surveillance studies are observational rather than experimental, so many studies of similar design and execution need to be combined and data from them pooled to draw an accurate picture of the issues. Since the etiology and immediate cause of sports-related traumatic dental injuries are well-established—the transfer of mechanical energy to the teeth and mouth due to contact with the ground or some other fixed structure, contact or collisions with another participant, or being struck by an object, either a projectile such as a ball or puck or the device used to launch the projectile, like a stick or racket—attention in analytical observational studies focuses on intrinsic and extrinsic risk factors for traumatic dental injuries. Intrinsic risk factors include the physical characteristics and psychological profile of individual athletes, including risk behavior and risk compensation. Extrinsic risk factors include type of sport, position played, exposure time, level of competition, coaching, referees, and protective equipment. The list of factors contributing to the risk of traumatic dental injuries in sports underlines the complexity of the issue of risk mitigation. Because each athlete has a unique set of intrinsic variables that may confer greater or lesser risk for injury, the interplay among and between intrinsic and extrinsic factors present an infinite range of risk for participants in athletic events. The simplest solution for mitigating sports-related traumatic dental injuries, protecting the mouth through the use of a mouthguard or facemask, is also the most effective irrespective of other factors. However, risk compensation theory suggests that face and mouth protection may increase risk for more serious injuries. The nature of sports makes it impossible to eliminate all risk of injury. Injury surveillance data from well-designed and executed studies that take a broad view of sports-related injuries and factors affecting them offers the best hope to illuminate the causes of sports-related traumatic dental injuries and offer solutions for their mitigation that are consistent with the culture of the individual sports and that do not increase the risk of other injuries.

## References


1. World Health Organization. Health topics: Epidemiology. 2016. http://www.who.int/topics/epidemiology/en/.
2. Centers for Disease Control and Prevention. Principles of epidemiology in public health practice, third edition: an introduction to applied epidemiology and biostatistics. Section 1: definition of epidemiology. 2012. http://www.cdc.gov/ophss/csels/dsepd/ss1978/lesson1/section1.html.
3. University of Houston College of Education (ND). Descriptive epidemiology: person, place, and time. http://viking.coe.uh.edu/~bsekula/pep7313/down/Ch.%204%20Friis.htm.
4. Centers for Disease Control and Prevention. Principles of epidemiology in public health practice, third edition: an introduction to applied epidemiology and biostatistics. Section 7: analytical epidemiology. 2012. http://www.cdc.gov/ophss/csels/dsepd/ss1978/lesson1/section7.html.
5. Centers for Disease Control and Prevention. Principles of epidemiology in public health practice, third edition: an introduction to applied epidemiology and biostatistics. Section 1: definition of epidemiology. 2012. http://www.cdc.gov/ophss/csels/dsepd/ss1978/lesson1/section4.html.
6. Glendor U. Aetiology and risk factors related to traumatic dental injuries—a review of the literature. Dent Traumatol. 2009;25:19–31.
7. Heintz WD. Mouth protectors: a progress report. Bureau of Dental Health Education. J Am Dent Assoc. 1968;77(3):632–6.
8. Goldberg A, Moroz L, Smith A, Ganley T. Injury surveillance in young athletes: a clinician's guide to sports injury literature. Sports Med. 2007;37(3):265–78.
9. Verhagen E, van Stralen M, van Mechelen W. Behaviour, the key factor for sports injury prevention. Sports Med. 2010;40(11):899–906.
10. Parkkari J, Kujala U, Kannus P. Is it possible to prevent sports injuries? Review of controlled clinical trials and recommendations for future work. Sports Med. 2001;31(14):985–95.
11. Huffman EA, Yard EE, Fields SK, Collins CL, Comstock RD. Epidemiology of rare injuries and conditions among United States high school athletes during the 2005-2006 and 2006-2007 school years. J Athl Train. 2008;43(6):624–30.
12. Task Force on Community Preventive Services. Recommendations on selected interventions to prevent dental caries, oral and pharyngeal cancers, and sports-related craniofacial injuries. Am J Prev Med. 2003;23(1 Suppl):16–20.
13. Chapman P. The prevalence of orofacial injuries and use of mouthguards in Rugby Union. Aust Dent J. 1985;30(5):364–7.
14. Beachy G. Dental injuries in intermediate and high school athletes: a 15-year study at Punahou School. J Athl Train. 2004;39(4):310–5.
15. Stewart G, Shields B, Fields S, Comstock R, Smith G. Consumer products and activities associated with dental injuries to children treated in United States emergency departments, 1990-2003. Dent Traumatol. 2009;25:399–405.
16. Hagel B, Meeuwisse W. Risk compensation: a "side effect" of sport injury prevention? Clin J Sport Med. 2004;14(4):193–6.
17. Merkel D. Youth sport: positive and negative impact on young athletes. Open Access J Sports Med. 2013;4:151–60. https://doi.org/10.2147/OAJSM.S33556.
18. 2014-15 High School Athletics Participation Survey conducted by the National Federation of State High School Associations based on Competition at the High School Level in the 2014-15 School Year. http://www.nfhs.org/ParticipationStatics/PDF/2014-15_Participation_Survey_Results.pdf.
19. The NCAA® Sports Sponsorship and Participation Rates Report 1981-82 – 2014-15 Report prepared by: Erin Irick, Assistant Director of Research. http://www.ncaa.org/sites/default/files/Participation%20Rates%20Final.pdf.
20. Kaste LM, Gift HC, Bhat M, Swango PA. Prevalence of incisor trauma in persons 6-50 years of age: United States, 1988-1991. J Dent Res. 1996;75:696–705.
21. Andersson L, Andreasen J. Important considerations for designing and reporting epidemiologic and clinical studies in dental traumatology. Dent Traumatol. 2011;27:269–74.
22. Kumamoto DP, Maeda Y. A literature review of sports-related orofacial trauma. Gen Dent. 2004;52(3):270–80.
23. Collins C, McKenzie L, Ferketich R, Huiyun X, Comstock R. Dental injuries sustained by high school athletes in the United States, from 2008/2009 through 2013/2014 academic years. Dent Traumatol. 2015. https://doi.org/10.1111/edt.12228.
24. Levin K. Study design VI: ecological studies. Evid Based Dent. 2003;7:60–1. https://doi.org/10.1038/sj.ebd.6400454.
25. Soriano E, Caldas A Jr, Carvalho M, Filho H. Prevalence and risk factors related to traumatic dental injuries in Brazilian schoolchildren. Dent Traumatol. 2007;23:232–40.
26. Gassner R, Bosch R, Tuli T, Emshoff R. Prevalence of dental trauma in 6000 patients with facial injuries: implications for prevention. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;87:27–33.
27. United States Consumer Products Safety Commission (n.d.). National Electronic Injury Surveillance System (NEISS). http://www.cpsc.gov/en/Safety-Education/Safety-Guides/General-Information/National-Electronic-Injury-Surveillance-System-NEISS/.
28. Bhat M, Li S. Consumer product-related tooth injuries treated in hospital emergency rooms: United States, 1979-87. Community Dent Oral Epidemiol. 1990;18:133–8.
29. Randall D, Agel J, Marshall S. National Collegiate Athletic Association injury surveillance system commentaries: introduction and methods. J Athl Train. 2007;42(2):173–82. Accessed from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1941300/.
30. Comstock R, Yard E, Knox C, Manring J. Summary report: High School Rio (Reporting Information Online): internet-based surveillance of injuries sustained by U.S. High School Athletes, 2005-2006. 2006. https://www.nata.org/sites/default/files/high-school-injury-surveillance.pdf.
31. Cohenca N, Roges RA, Roges R. The incidence and severity of dental trauma in intercollegiate athletes. JADA. 2007;138(8):1121–6.
32. Hedlund J. Risky business: safety regulations, risk compensation, and individual behavior. Inj Prev. 2006;6:82–90.
33. Farris C, Spaite D, Criss A, Valenzuela T. Observational evaluation of compliance with traffic regulations among helmeted and non-helmeted bicyclists. Ann Emerg Med. 1997;29:625–9.
34. Spate D, Murphy M, Criss E, Valenzuela T, Meislin H. A prospective analysis of injury severity among helmeted and non-helmeted bicyclists involved in collisions with motor vehicles. J Trauma. 1991;31:1510–6.
35. Mueller F. Fatalities from head and cervical spine injuries occurring in tackle football: 50 years experience. Clin Sports Med. 1998;17:169–82.
36. Benson B, Mohtad N, Rose M, Meeuwisse W. Head and neck injuries among ice hockey players wearing full face shields vs. half face shields. JAMA. 1999;282(24):2328–32.
37. Finch CF, McIntosh AS, McCrory P. What do under 15 year old schoolboy rugby union players think about protective headgear. Br J Sports Med. 2001;35(2):89–94.
38. Woods S, Aabat E, Daggy M, Diehl J, Engel A, Okragly R. Face protection in recreational hockey players. Fam Med. 2007;39(7):473–6.
39. Molsa J, Kujala U, Nasman O, Lehtipuu T, Airaksinen O. Injury profile in ice hockey from the 1970s through the 1990s in Finland. Am J Sports Med. 2000;28(3):322.
40. Gardner E. Head, face, and eye injuries in collegiate women's field hockey. Am J Sports Med. 2015;43(8):2027–34.
41. McIntosh A. Risk compensation, motivation, injuries, and biomechanics in competitive sport. Br J Sports Med. 2005;39:2–3.

Check for updates

# Sports-Related Oral and Dentoalveolar Trauma: Pathophysiology, Diagnosis, and Emergent Care

*Mark Roettger, Matthew Greaves, Mansur Ahmad, and Vladamir Leon-Salazaar*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_3

**Objectives**
- Describe the nature and effect of trauma.
- Describe the healing events after injuries to the maxillofacial region.
- Recognize injuries involving different parts of the tooth and supporting structures.
- Perform adequate clinical procedures to gather adequate information about the type and extent of injury.
- Compose an accurate diagnosis of the type of trauma.
- Formulate an emergency treatment plan.
- Perform adequate emergency treatment to maximize healing potential in all types of dentoalveolar trauma.
- Recognize the clinical and radiographic signs of healing: regeneration, repair, and failure.
- Know rationale and guidelines for post-trauma splinting.
- Be able to adequately manage trauma to the oral soft tissues.
- Be aware of the three big misconceptions of managing dental trauma.

## 3.1 Introduction to Dentoalveolar Trauma

Trauma to the dentoalveolar complex is a major focus of modern sports dentistry. These types of injuries are not uncommon in sports and athletic competition. Many times these are complex injuries of multiple types of tissues that require careful examination, thoughtful diagnosis, and formulation of a treatment plan, at times, all within a matter of minutes [1]. For this reason, it is imperative that all dental professionals as well as sports medicine personnel, coaches, school nurses, and parents be familiar with basics in dental anatomy and dental trauma first aid. Community dentists should stay abreast of the latest methods of managing dentoalveolar trauma and provide in-service lectures to school nurses, coaches, trainers, and parents. Team dentists must update their dental trauma management skills on a yearly basis as they have a duty to the athletes that they serve to provide optimal care when dental trauma occurs (Figs. 3.1 and 3.2). This chapter serves as a basis for such review for dentists as well as sports medicine personnel. It is estimated that half of all children will suffer a dental injury by the time that they graduate from high school and that sporting activities cause the greatest percentage of these injuries in children [2] (Fig. 3.3). This is a significant public health issue. See ► Chap. 2 for a more complete review of the epidemiology of dentoalveolar trauma.

**Nature of Dental Trauma**

*Complex Injuries*:
- Involving multiple tissues
- Damage to intercellular components; tearing
- Damage to cellular systems; crush, desiccation, contamination
- Tx aimed at resolving all damage

**Fig. 3.1** Dental trauma from sport: complex injury to the dental hard tissue, periodontal ligament, supporting bone, and the oral soft tissues. (Photo credit: Dr. Paul Nativi)

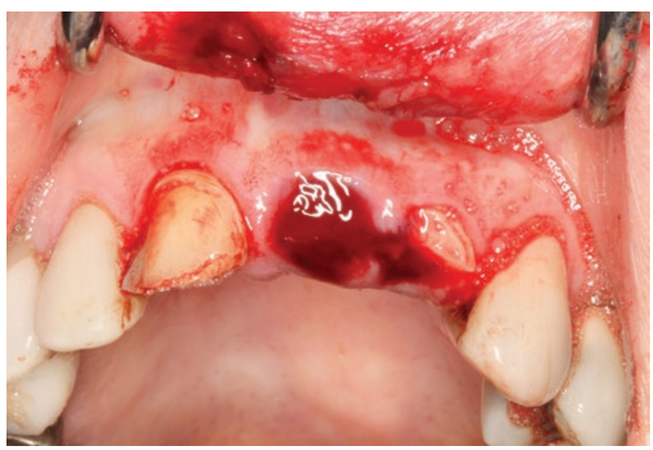


**Fig. 3.2** Complex dental trauma resulting from sport: crown fracture, avulsion, crown-root fracture, and soft tissue trauma. (Photo credit: Dr. Mark Roettger)


**Fig. 3.3** Participation in youth sports continues to grow exposing more young athletes both male and female to dental trauma from sport. (Photo credit: Dr. Mark Roettger)

### 3.1.1 Dentoalveolar Complex

This chapter focuses on trauma to the dentoalveolar complex. This complex of tissues consists of five tissue compartments, and together they make up the dentoalveolar complex. First the *gingival-periosteal complex* is composed of the gingiva, free gingiva, attached gingiva, and junctional epithelium as well as the periosteum covering the alveolar process. Second, the compartment is the *periodontal ligament cementum complex*. Third is the *alveolar bone and marrow complex*. The fourth is the *dentin-pulp complex*, and finally there is the *oral mucosa-skin complex*. The dentoalveolar complex is a very specialized area that allows the emergence of the dentition from the alveolar bone and consists of unique and specialized cells and tissues ([3], pp. 72–96).

## 3.2 Pathophysiology: Dentoalveolar Trauma

Energy is transferred from an object to the dentoalveolar structures, and as these structures absorb the energy transferred, the damage to normal anatomy occurs. Sporting objects such as balls, pucks, sticks, and opponents' body parts are the major causes of this energy transfer through the dentoalveolar complex, and these same sporting objects and body parts can trap the soft tissues of the oral cavity against the teeth causing soft tissue damage. These injuries to the dentoalveolar complex can occur in two basic ways, first by *direct trauma* where a ball, stick, elbow, knee, or other sports implement directly strikes the teeth. Direct trauma injuries usually affect the maxillary anterior teeth, and the injuries most likely encountered in direct trauma are luxation injuries, tooth avulsion, and all types of dental fractures. The energy of impact may also determine the type and the severity of damage as a result of trauma to the oral cavity. High-velocity low-mass-type injuries such as a ball striking the teeth tend to cause damage to the dental hard tissues and less damage to the supporting structure such as the periodontal ligament (PDL) or the alveolar process. The energy of the impact is dissipated in creating the tooth fracture and is not transferred to the supporting tissues. Conversely, low-velocity, high-mass-type injuries such as the teeth striking the ground or other playing surfaces tend to cause more damage to the supporting structures causing fewer dental fractures and more damage to the supporting structures leading to more luxation and avulsion injuries. A second mechanism of dental injury is *indirect trauma*. This is seen when the mandibular teeth are forcefully crashed into the maxillary teeth. Indirect trauma causes more damage to the posterior teeth than direct trauma, as well as trauma to other craniofacial structures. Injuries resulting from indirect trauma include crown/root fractures of posterior teeth, mandibular fractures [3], temporomandibular joint injuries, and brain concussion (see more in ▶ Chap. 5).

Dentoalveolar trauma creates complex injuries affecting multiple tissues. There is potential damage to dental hard tissues, osseous hard tissues, dental pulp, periodontal ligament, mucosa, nerves, vessels, intercellular components, and cellular systems. Successful treatment of dentoalveolar trauma must be aimed at resolving damage to all the cellular and intercellular systems. To do this effectively, the sports dentist needs to have a working knowledge of basic wound healing principles.

## 3.3 Principles of Wound Healing

All wounds ultimately heal; it is how they heal that determines the long-term function of damaged tissues and the outcome of treatment. The goal of all trauma management is to restore damaged tissues to original form and function; this is *regeneration* of damaged tissues. *Repair* of damaged tissues is where the damaged tissues are replaced by scar tissues which restores continuity of tissue but does not restore original form and function. In dentoalveolar trauma, we also identify failure healing especially when describing healing of the dental pulp and periodontal ligament. *Failure healing* in the pulp would be pulp necrosis or infection, and in the periodontal ligament, failure healing would be inflammatory resorption. There is another term used in wound healing and that is tissue metaplasia which is used when one type of tissue is replaced by another type of tissue after trauma occurs [1].

Healing of most wounds in the human body whether made by surgery or trauma is through the process of repair and the formation of scar tissue which can and does alter the structure and function of the organ affected. Dental tissues are unique when compared to the rest of the body due to their marked capacity for regeneration. Injuries to the dental pulp and periodontal ligament may heal by regeneration restoring normal form and function or by repair with scar tissue or bone. Repair of the injured dental pulp results in pulp canal obliteration (PCO), and repair of the periodontal ligament results in ankylosis and replacement resorption. Regeneration of dentoalveolar tissues is the key to complete recovery from trauma, and while the dental tissues exceed many other tissues in their capacity for regeneration, many oral wounds heal by repair as we will see later in this chapter

([3], pp. 62–132, [4]). There are research efforts underway presently that look into cell transplantation or use of bioactive molecules such as enamel matrix proteins to tip the scales in favor of regeneration over repair. Regenerative biology is a very active field within medicine today, and progress is being made on multiple fronts including dentoalveolar trauma healing [5]. Where we currently have some influence over healing in dental trauma cases, is the timing of treatment as in replantation of avulsed teeth and in the control of infection during healing in all types of dental trauma.

**Dental Trauma: Healing Outcomes**

| | |
|---|---|
| *Regeneration*: | Pulp → revascularization |
| | PDL → normal PDL |
| *Repair*: | Pulp → pulp canal obliteration |
| | PDL → replacement resorption (ankylosis) |
| *Failure*: | Pulp → pulp necrosis |
| | PDL → inflammatory resorption |

*All wounds heal*; it is how they heal that determines outcome.

## 3.4 Preparation: Dentoalveolar Trauma

Management of dental trauma requires some preparation in order to provide the best care to the patient when it happens. Dental trauma occurs with no regularity, and it cannot be anticipated, so having appropriate materials available in the dental office or in a team dentist kit is strongly suggested. Treatment planning resources are useful, and two recommendations would be "Traumatic Dental Injuries: A Manual; Third Edition" [6] and an online reference "IADT Trauma Guide" (▶ www.dentaltraumaguide.org) [7]. Both of these references in addition to this book are helpful because they are easy and quick to use for use at the time of trauma. The definitive resource for dental trauma remains "Textbook and Color Atlas of Traumatic Dental Injuries to the Teeth" [3]. The latter is a comprehensive volume on dental trauma and is recommended for study and preparation when no patient is in need of care. Additionally preparation for dental trauma requires appropriate materials, most of which can be found in a dental clinic: restorative materials, endodontic supplies and equipment, dental splint materials, dental storage media, and surgical supplies. There are recommendations for on-field dental emergency kits in the final chapter of this book.

## 3.5 Examination of the Dentoalveolar Trauma Patient

Dentoalveolar trauma is considered an emergency and requires thoughtful care to relieve pain, control bleeding, and replace teeth to their proper position in the dental arches. Treatment requires diagnosis, and diagnosis requires thorough examination of the injured patient. Serious dental trauma is an emotional injury and often patients, parents, or concerned onlookers bring a high level of anxiety to the operatory where the examination is taking place. This requires the practitioner to be comfortable and confident in his or her ability to examine and ultimately manage the trauma patient. Inaccurate examination of a trauma patient can lead to improper diagnosis and treatment of a patient. To avoid inaccuracy in examination, we need to take a systematic approach to the evaluation of the trauma patient.

One area where we may need to deviate from our systematic approach to the trauma patient will be in the case of tooth avulsion. These injuries as we will see later in this chapter require immediate treatment to maximize healing potential. After appropriate tooth reimplantation is completed, and systematic examination of the patient can continue.

Dentists should never treat a stranger. This concept was made famous by William Osler who was a Canadian physician who was instrumental in the formation of Johns Hopkins Hospital. We need to know a significant amount of history medical, dental and personal, about any patient that we treat and this holds true for the trauma patient. We accomplish this in the trauma patient, who may be a new patient to the treating dentist, through systematic examination using a trauma checklist.

**Trauma Checklist**

| | |
|---|---|
| – Consent for treatment (emergency) | – Clinical exam extraoral |
| – Emergent care CAB | – Facial bones |
| – Airway and vitals | – Nose |
| – Medical history | – Eyes |
| – Dental history | – Clinical exam intraoral teeth and soft tissue |
| – Previous injury? | – TMJ exam |
| – HPI | – Imaging |
| – When | – Conventional |
| – Where | – CBCT |
| – How | |

### 3.5.1 Medical History

This important part of the examination is where the clinician gets to know the patient. This also is where the important process of emergency consent for treatment is accomplished. We also inquire about patient allergies to medications, past bleeding disorders, the need for antibiotic prophylaxis, history of seizures and medications currently being taken by the patient, cardiovascular conditions, and potential airway issues ([8], pp. 105–131).

### 3.5.2 Dental History

In the dental history, we inquire about the patient's past dental care, treatments in progress, and about any past traumatic dental injuries (Fig. 3.4). Past dental trauma may affect the way that we approach the treatment of the current injury and give clues when considering diagnosis. Second, we question the patient or the parent in the case of a young child the history of the present injury. During this part of the history, we ask about the three Ws: When? What? Where?

1. When did the injury occur? This gives us important information of time passed which will give us insight as to the prognosis of our treatment.
2. What happened or how did the injury occur gives information and acuity on the mechanism of the trauma and what type of tissue damage we should be looking to find. Also, these questions can shed light on the possible severity of the injuries.
3. Where did the injury occur can tell us about the severity of the injury as well as the issues of contamination of

**Fig. 3.4** Record of dental trauma. (Credit: Academy for Sports Dentistry)

**Record of Traumatized Teeth**

Patient's name: ______________________ Date of birth: ______________
☐ Male ☐ Female Age: ______________

Initial examination time and date: ______________________

Referring dentist (or physician): ______________________

**Past trauma (if any)**
Time and date: ______________ Teeth involved: ______________

**Present trauma**
Time and date: ______________ Place: ______________
Cause: ______________________
______________________

**General findings**
Headache: ☐ Yes ☐ No Loss of consciousness: ☐ Yes ☐ No Nauses: ☐ Yes ☐ No

**Intraoral findings**
Teeth involved: ☐ Primary ______________ ☐ Permanent ______________
Spontaneous pain: ☐ Yes ☐ No Cold sensitivity: ☐ Yes ☐ No
Percussion sensitivity: ☐ Yes ☐ No Pulp exposure: ☐ Yes ☐ No
Electric pulp test (EPT): + / –
Discoloration of crown: ☐ Yes ☐ No Tooth mobility: ☐ 0 ☐ 1 ☐ 2 ☐ 3
Damage: ☐ To oral mucosa ☐ Laceration of the lips ☐ Other ______________

**Radiographic findings**
Completion of root formation: ☐ Complete ☐ Incomplete (apical foramen ________mm)
Root fracture: ☐ Yes ☐ No Apical lesion: ☐ Yes ☐ No
Obliteration of pulp cavity: ☐ Yes ☐ No Root resorption: ☐ Yes ☐ No
Type of root resorption: ☐ Surface ☐ Inflammatory ☐ Replacement
Widening of periodontal membrane (luxation): ☐ Yes ☐ No
Alveolar bone fracture: ☐ Yes ☐ No

**Condition of avulsed tooth**
Duration of time out of oral cavity: (________minutes)
Stored: ☐ Dry ☐ In water ☐ In saliva ☐ In milk

**Diagnosis**
☐ Crown fracture ☐ Crown-root fracture ☐ Root fracture
☐ Concussion ☐ Subluxation ☐ Extrusive luxation
☐ Lateral luxation ☐ Intrusive luxation ☐ Avulsion

**Treatment plan**

**Prognosis**

the traumatized tissues and tell us about the need for antibiotic coverage and need for tetanus booster.

Asking these open-ended questions of the patient is obviously important information as described above, but in addition, the accuracy of the answers can be the beginning of the next phase of our systematic examination of the trauma patient by providing clues to the patient's cerebral function.

### 3.5.3 Brief Neurologic Examination

Trauma to the dentoalveolar structures which are close to the brain means that there can be concomitant neurological damage in these patients. If patients come directly to the dental office and are not cleared by medical personnel, we need to conduct a brief neurological exam as a part of a complete trauma examination. Neurological deficits will require referral to appropriate medical providers for evaluation. This brief neurological exam should begin with a cursory cranial nerve assessment where we can test selected cranial nerves and determine if they are intact or weak and if this is asymmetrical ([8], p. 684, 702). More information on cranial nerve evaluation can be found in ▶ Chap. 5.

A quick neurological history can also be taken from the patient. A series of questions can help determine the need for medical referral. Question the patient as to presence of headache, lethargy, nausea and vomiting, loss of consciousness, and amnesia. A positive response to presence of any of the above should generate a medical consult. It is also useful to determine the patient's orientation to their surroundings. Orientation × 3 represents that the patient knows who they are, where they are, and the approximate time. Any deficit to orientation should also generate a medical referral.

Any suspicion of intracranial bleeding needs to be followed by appropriate medical personnel *immediately*.

### 3.5.4 Clinical Exam of the Head and Neck

This topic is covered more completely in ▶ Chap. 5 on maxillofacial injuries but is mentioned here as a part of the systemic evaluation of the dentoalveolar trauma patient. We begin this part of the assessment with a look at the skin, looking for disruptions causing bleeding. We will look for abrasion, contusion, laceration, edema, and ecchymosis. The patterns of soft tissue injuries to the face may give clues to how the trauma occurred as well as potential underlying bone trauma ([9], pp. 491–518).

As we survey the skin of the head and neck, we also begin our evaluation of the underlying bony structures, maxilla, mandible, bony orbit, zygoma, and frontal bones and sinuses. At this level of the evaluation, we look for signs of fracture such as mobility, crepitus, tenderness, and facial asymmetry. Signs of facial fractures will trigger referral to oral surgery personnel, remembering that facial fractures can compromise the patient's airway ([9], pp. 491–518, [10]).

Nasal fractures are some of the most common of the facial fractures, and the nose should be evaluated looking for epistaxis or displacement of the nasal complex.

Trauma to the face can and does result in eye injuries. These can be caused by direct trauma or by fractures of the bony orbit. The two most common sports injuries to the eye are corneal abrasion and hyphemia. Corneal abrasion is usually a minor, although painful, injury to the eye. Most minor corneal abrasions will heal in a few days, but more severe abrasions may cause permanent damage to the eye, so they should be evaluated by an ophthalmologist. Hyphema is bleeding between the cornea and the iris and is often caused by blunt force trauma to the eye. Again most hyphemas resolve with no treatment; traumatic hyphema has an increased chance to raise intraocular pressure, so they should be followed by ophthalmology [8].

Examination of the ears can help rule out more serious injuries. Check to see if the ears are clear of fluid. If there is fluid in the ears, it may be cerebrospinal fluid, and the patient should be immediately referred to the appropriate medical providers.

### 3.5.5 Temporomandibular Joint (TMJ) Evaluation

Again this topic will be addressed more completely in ▶ Chap. 5 but is mentioned here as a part of our dentoalveolar trauma checklist. We will do a cursory TMJ evaluation to see if further evaluation is indicated. Have the patient open and close a few times and inquire about joint pain on opening or closing. Measure the inter-incisal opening in mm to see if there is normal range of motion. As the patient opens and closes, look for deviations to the right or to the left during both opening and closing. Listen and feel for clicks, pops, or disruptions in smooth rotation and translation of the temporomandibular joint. Check the occlusion of the teeth for open bites anterior or posterior and deviations in occlusion right or left. We are, in this cursory examination, ruling out condylar fractures or hemarthrosis of the temporomandibular joint ([9], p. 627).

### 3.5.6 Intraoral Examination

Looking inside the mouth for damage from trauma should continue systematically. The intraoral examination begins by inspecting the soft tissues. Look for abrasions, contusions, and lacerations. Through and through, lacerations of the lips require meticulous care to avoid disfigurement. It is imperative that both the cutaneous and the mucosal sides of these wounds are closed to insure proper healing. This will be discussed in more detail later in this chapter. Any time there are fractured teeth with tooth fragments that are not located, it must be assumed that they may be embedded in the soft tissue wounds. Careful exploration and imaging will help to locate or rule out embedded tooth fragments in soft tissue. Lacerations of the gingiva are often associated with tooth luxation. Contusions may signal more injuries below the surface tissue. Contusion of the floor of the mouth may indicate a mandibular fracture.

A check of the patient's occlusion will help to locate possible displaced teeth or disruptions that could indicate a bony fracture. Open bites may indicate fractures or possibly hemarthrosis of the temporomandibular joint. Assessing the overjet may indicate the possibility of previous injury, considering the propensity of traumatic injury to maxillary incisors in patients with severe overjet.

Many dental injuries will be evident upon initial inspection of the teeth. Luxations and most dental fractures are obvious and usually easy to find. Cleansing the teeth and careful examination of the teeth using direct vision and transillumination can reveal more subtle damage to the dental tissues. While evaluating dental fractures, it is important to record whether the fractures involve enamel only, enamel and dentin (simple fractures), or enamel, dentin, and pulp (complex fractures). Examination of complex dental fractures that involve the dental pulp includes an assessment of the pulp seen. Determination of the condition of the pulp from bleeding to ischemic to necrotic can indicate the possibility of concomitant periodontal ligament issues and may alter treatment plans. Fractures that also include cementum are crown-root fractures and should be evaluated to determine the restorability of the tooth.

Examination of the dental trauma patient also includes mobility tests for all of the teeth, also noting loose and missing teeth. If any teeth are missing and unaccounted for, the patient should be referred for chest and abdominal imaging to rule out aspiration or swallowed teeth. Loose teeth are assessed for displacement that requires reduction and splinting. Displaced teeth that interfere with normal occlusion require more immediate treatment than those that do not interfere with biting or chewing.

Percussion tests are helpful to assist in the examination of the dental trauma patient. Finding pain on percussion helps to determine the imaging plan for the patient. High ankylotic tone can indicate lateral luxation or intrusive luxation injuries [6]. Dull ankylotic tone may indicate luxation-type injuries.

Pulp sensitivity is usually a help in determining the extent of a dental injury. Thermal and electronic testers assess the nerve supply to the dental pulp. Pulp testing following traumatic dental injuries is controversial. There are many false-negative and false-positive reading early in the post-trauma time period. Some may be due to the nature of the trauma, while other failures may result from the fact that a calm relaxed patient is needed to get accurate pulp sensibility readings, and this is usually not the case in the immediate post-trauma time period. Patients and family members tend not to be calm and relaxed. Pulpal sensibility tests immediate post-trauma may be helpful to determine a baseline when accurate readings can be obtained [11].

### 3.5.7 Imaging of Dentoalveolar Trauma

Initial radiographic examination for suspected dental trauma should include intraoral periapical radiographs. In cases of suspected concussion, a periapical radiograph may reveal no change or slight widening of the PDL spaces. Follow-up radiographic examinations conducted months or years after the trauma may demonstrate reduction in the size of the pulp chamber and root canals. In addition, such radiographic examination may also reveal refining osteitis or internal root resorption. Although a periapical radiograph may provide important diagnostic information about luxation, a CBCT scan helps in ruling out labial or palatal displacement of a root. In addition to the status of the tooth, a CBCT scan is more reliable in ruling out fracture of the alveolar bone. However, scans of patients with multiple coronal or endodontic restorations may be degraded due to image artifacts. Small fractures can remain undetected in scans with image artifacts.

For patients with suspected horizontal root fracture, multiple periapical radiographs at different vertical angulation should be acquired. Such multiple exposures can reveal a fracture which may remain undetected with a single exposure if the central beam of the X-ray did not travel through the line of fracture.

For vertical root fracture, a CBCT scan at a high resolution is a better examination than periapical radiography when the tooth is not endodontically treated. However, the superiority of a CBCT scan is not proven when a tooth is endodontically treated.

Since a large number of sports-related dental traumas occur in young children, CBCT scan time should be short to minimize motion. In addition, the field of view should be limited to area of question. A smaller field of view can also be obtained at a higher resolution. A large field of view scan often can be obtained only at a lower resolution.

## 3.6 Classification of Dental Trauma

Diseases and conditions are classified to allow study and definition of pathology, treatment, and prognosis. Dental trauma has been classified in a number of ways over the years regarding factors such as anatomic location, urgency of treatment, etiology, treatment, and pathology. We will review two methods of classification that can be useful to interprofessional teams of providers responsible for the oral care of athletes.

### 3.6.1 World Health Organization Classification

This classification uses anatomic factors in classifying dental trauma. It is from the *Application of* the *International Classification of Diseases to Dentistry and Stomatology* adopted by the World Health Organization (WHO) [12]. The WHO classification includes injuries to the dental hard tissues and dental pulp, injuries to periodontal tissues, injuries to the supporting bone, and injuries to the gingiva or oral mucosa (see "WHO Classification of Dental Trauma" in ► Chap. 2).

### 3.6.2 Classification of Dental Trauma by Treatment Urgency

A classification of dental trauma by treatment urgency can be extremely helpful to the non-dental members of the sports medicine team especially athletic trainers, team physicians, and also school nurses. This classification uses the need (or not) of acute care in management of the injury. The categories are injuries requiring acute care (minutes to hours), injuries that require subacute care (within 24 h), and injuries manageable with delayed care (over 24 h to weeks). Another urgency classification uses simply urgent care needed and delayed care (Fig. 3.5). Ability to classify dental injuries in this manner can assist non-dental sports medicine providers an idea of the urgency of dental consultation and treatment. If there is ever any doubt as to the urgency of treatment needed, the consulting dentist should always err on the side of urgency and see the patient immediately.

**Classification of Dental Injuries: Anatomical**

1. Injuries to dental hard tissues and pulp
   - Enamel fracture
   - Enamel-dentin fracture (simple)
   - Complex crown fracture
   - Crown-root fracture
   - Root fracture
2. Injuries to the periodontal tissues
   - Concussion
   - Subluxation
   - Extrusive luxation
   - Lateral luxation
   - Intrusive luxation
   - Avulsion
3. Injuries to gingival or oral mucosa
   - Laceration
   - Contusion
   - Abrasion

Always look for combination of injuries!!!

## 3.7 Injuries to the Hard Dental Tissues and the Pulp

This section will describe fractures to the tooth in all areas and covers fractures to the crown of the tooth, fractures that involve the crown and root, and fractures that involve the root of the tooth.

### 3.7.1 Crown Fractures

Crown fractures are among the most common dental injuries encountered in sports dentistry. Crown fractures may result in no loss of tooth structure and are termed dental *infractions*. Most crown fractures involve loss of tooth structure. They can involve enamel only, enamel and dentin, and enamel, dentin, and pulp. When the pulp is not involved, they are termed *simple crown fractures*, and when the pulp is involved, they are called *complex crown fractures* (Fig. 3.7).

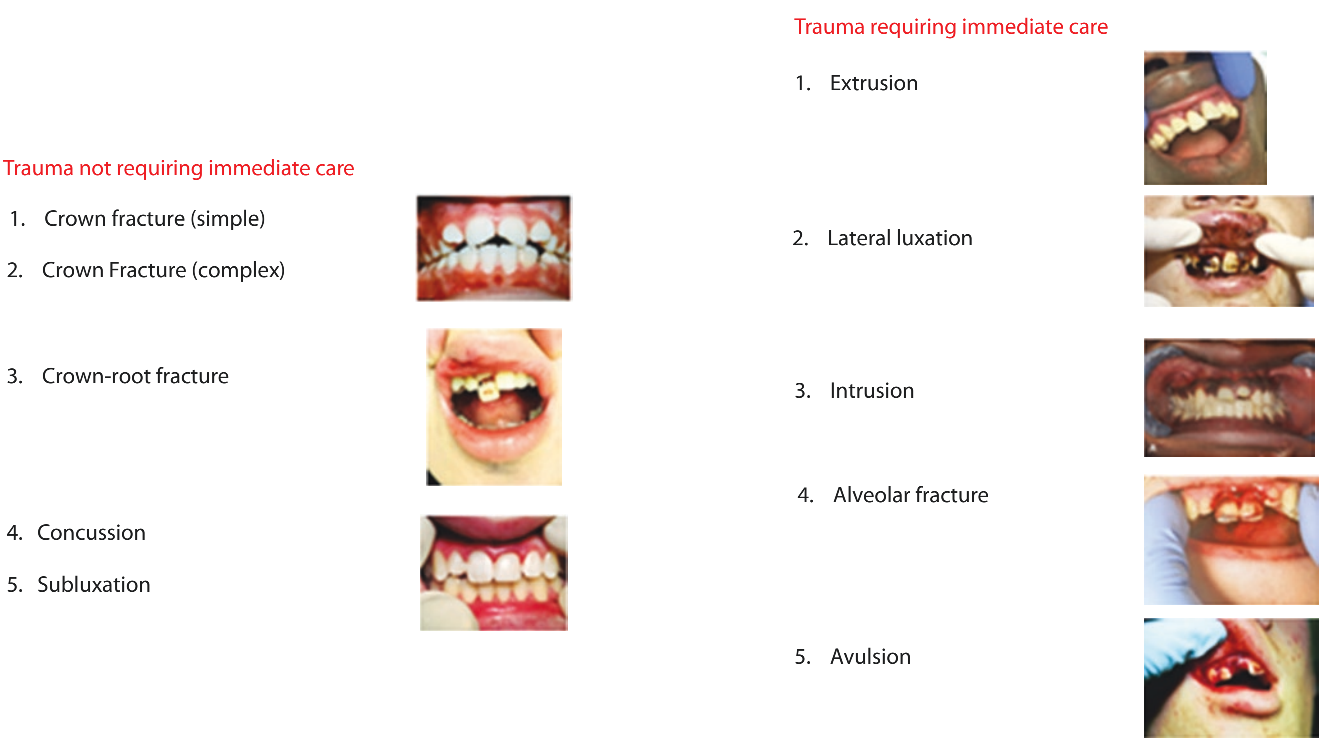



**Fig. 3.5** Duty instruction: dentoalveolar trauma. (University of Minnesota Medical Center Dental Service)

Crown fractures even when they are complex, involving the dental pulp, are not usually emergent. Exposure of the dental pulp by a complex fracture may cause temperature sensitivity, but the athlete can usually finish a competition before being referred to the dentist for treatment. The most common complaints after crown fracture are sensitivity and roughness to the oral soft tissues. So crown fractures are considered to be injuries requiring only delayed treatment [13]. The cases where there is intense pain may signal a concomitant injury and should be referred to dentistry immediately. The clinical appearance is the expected loss of tooth structure as seen in Figs. 3.6 and 3.7. In the case of a complex fracture when the dental pulp is visible to the eye, examination can reveal injuries to the periodontal tissues as well. A bleeding pulp noted red blood means that periodontal injuries are less likely, while ischemic or cyanotic look to the dental pulp may indicate concomitant periodontal damage or perhaps a prior injury or prior infection of the dental pulp, which will alter treatment plans. Also test the mobility of the tooth while examining the fracture which also could indicate periodontal damage [6].

**Fig. 3.6** Simple crown fracture of maxillary central incisors; note no exposure of the pulp is seen. (Photo credit: Mark Roettger DDS)

**Fig. 3.7** Complex fracture of left central incisor; note exposure of the dental pulp. (Photo credit: Mark Roettger DDS)

**Radiographic Findings of Crown Fracture** The lost part of the tooth can be seen on normal dental radiographs, but infractions are often times not seen on dental radiographs.

**Pathophysiology of Crown Fractures** Crown fractures alone have few biologic issues. Exposed dentin after a fracture can allow bacteria or their toxins to transport the dentinal tubules to the pulp to cause pulpal inflammation. The severity of the inflammatory reaction is dependent on the health of the pulp tissue. Covering the dentin with the tooth fragment or dental restorative materials will usually allow complete pulp healing after a simple crown fracture. There is significant confusion in the dental community on the proper management of complex crown fractures which leads us to: *Common Misconception #1*: Every traumatic pulp exposure requires endodontic treatment.

### Misconception #1: Every Exposed Pulp Needs to Be Extirpated

In fact, most traumatic exposure of the pulp can be treated by direct pulp capping or by partial pulpotomy, and a 90–99% success rate can be expected even if the pulp has been exposed to the oral environment for multiple days [14].

**Emergent Treatment of Traumatic Crown Fractures** Complex crown fracture requires minor pulpal therapy prior to the dental restorative work. *Direct pulp cap* involves placement of calcium hydroxide over the pulp exposure prior to restoration. *Partial pulpotomy* sometimes called the Cvek-type pulpotomy (after Dr. M. Cvek) involves removing some of the pulp tissue at the site of exposure. A new round diamond bur in a high-speed handpiece is used to remove 2–3 mm of pulp tissue. When pulpal hemostasis is achieved, calcium hydroxide or mineral trioxide aggregate (MTA) is placed over the pulpal wound. Resin-modified glass ionomer cement is placed over the calcium hydroxide or the MTA. Calcium hydroxide has high success rates in traumatic pulp exposures, and MTA has slightly improved success rates in the same traumatic exposure category [15–17]. MTA has a much higher cost per treatment than calcium hydroxide and has the issue of tooth discoloration after use. When the pulp treatment is accomplished, the tooth fracture can be restored like any other by bonding the fragment, composite restoration, or fabrication of an esthetic crown. This will be covered in detail in ▶ Chap. 6.

**Treatment Schemes Traumatic Pulp Exposure**

A hard tissue barrier can be expected to form over a traumatic exposure if:

1. Pulp was normal prior to trauma.
2. Intact vascular supply to pulp after trauma.
3. Use of appropriate capping or amputation technique.
4. Exclusion of bacteria during treatment and healing.
5. RCT is *not* automatically indicated for tx of traumatic exposures!!!
6. If absence of pulp is desired (post), the pulp should be extirpated.

**Pulp Capping vs. Partial Pulpotomy**

| Pulp capping | Partial pulpotomy |
|---|---|
| 1. Isolate with rubber dam | 1. Isolate with rubber dam |
| 2. Fx surface cleaned with CHX and hypochlorite | 2. Fx surface cleaned with chx |
| 3. Ca(OH)2 or MTA is applied to pulpal wound | 3. Use round diamond bur to prep to a depth of 2–3 mm |
| 4. Restore with bonding; fragment bonding | 4. Hemostasis |
| | 5. Thin layer of Ca(OH)2 or MTA |
| | 6. Layer of resin-modified glass ionomer |
| | 7. Restore with bonding or bonding fragments |

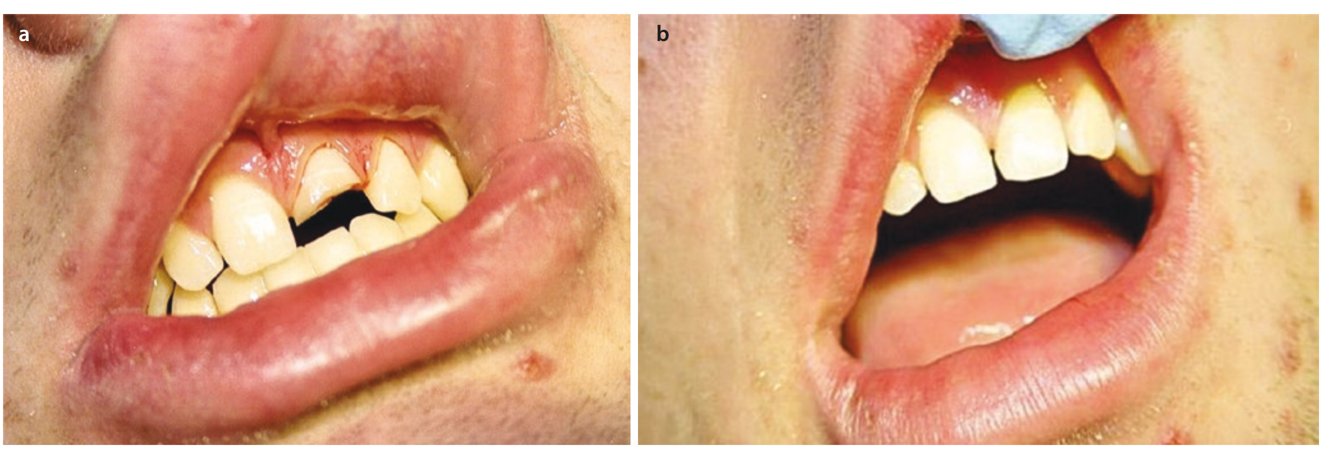
**Outcomes of Crown Fracture Treatment** Pulpal and periodontal healing is expected in most cases of crown fracture if proper management guidelines are followed. The size of the exposure and the time between injury and treatment are not as important as many assume [14]. Partial pulpotomy shows slightly better pulpal healing rates than direct pulp capping, but both have success rates in the 90–99% range [15] (◘ Figs. 3.8 and 3.9).

**Pulp Capping vs Partial Pulpotomy**

| Pulp capping | Partial pulpotomy |
|---|---|
| 1. Long-term studies show very high success rates with respect to pulp survival 90–95% | 1. Long-term studies show very high success rates with respect to pulp survival 95–99% |
| 2. That is without concomitant luxation injuries | 2. That is without luxation injury |

### 3.7.2 Crown-Root Fractures

Crown-root fractures also occur with some regularity in sports. They are usually caused by direct trauma in the anterior teeth, where they are most common, and by indirect trauma when seen in posterior teeth. These injuries involve the enamel, dentin, and cementum of the tooth and may or may not involve the dental pulp. Crown-root fractures in the anterior teeth seen in sports trauma usually begin a few millimeters incisal to the marginal gingiva, and a visible fracture line is seen on the facial aspect of the tooth, and the incisal part of the crown is usually displaced but remains attached to the gingiva or alveolar bone on the palatal side of the tooth (◘ Fig. 3.10). Bleeding is encountered from the periodontal tissues disrupted by the trauma and may make it difficult to assess the involvement of the dental pulp in the fracture. It is important to determine the depth of the fracture below the gingival margin or alveolar bone as this will help to establish the prognosis in treating this type of injury. These injuries



◘ **Fig. 3.8** **a** Simple crown fracture from a basketball injury. **b** Treatment of crown fracture by bonding fragment. (Photo credit: Mark Roettger DDS)


**Fig. 3.9** **a** Softball injury: hit by the ball creating a luxation injury to the right central incisor and a complex fracture to the left central incisor. **b** Luxation injury has been splinted to stabilize, and in this view, the pulp exposure on the left central incisor is evident. **c** The luxated right central incisor splinted and the fractured left central incisor has been treated with calcium hydroxide Cvek pulpotomy and composite bonding to restore tooth structure. (Photo credit: Mark Roettger DDS)

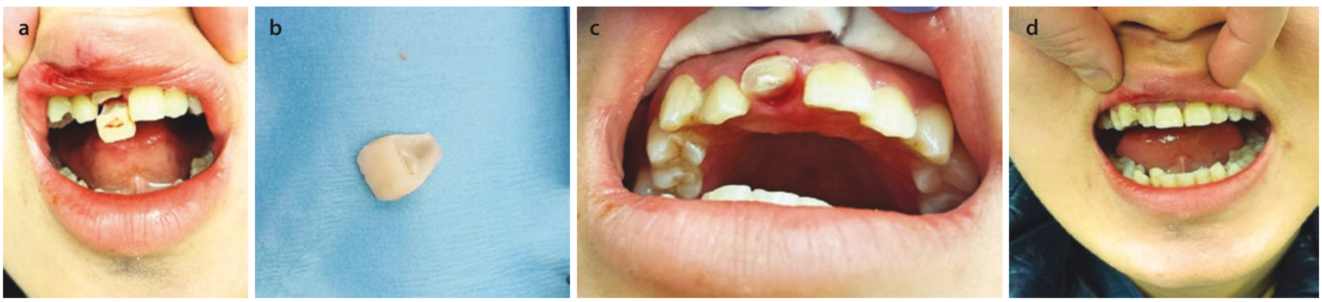



**Fig. 3.10** **a** Crown-root fracture from a curling injury; note the hinge axis on the palatal side of the tooth. **b** Coronal fragment removed and cleaned. **c** Fragment removed, hemostasis achieved in palatal tissues, and Cvek pulpotomy completed. **d** Fragment bonded back over the root. The tooth is awaiting definitive restoration. (Photo credit: Mark Roettger DDS)

are usually in the delayed treatment category not requiring immediate care. Even when the pulp is exposed, there are few symptoms in crown-root fractures, and athletes can usually finish their competition if the mobility of the coronal segment is not too great.

**Radiographic Findings Crown-Root Fracture** Radiographic examination of crown-root fractures is rarely helpful in making a diagnosis on in establishing a prognosis. The radiograph will likely show the facial fracture line but seldom show the apical extent of the hinge portion of the fracture. Radiographic examination can be helpful in seeing the extent of a proximal crown-root fracture in an anterior tooth. Also radiographs can be used to evaluate the root of the tooth.

**Pathophysiology of Crown-Root Fractures** The force of trauma generates an oblique fracture through the tooth from midfacial on the buccal to below the gingiva or bone on the palatal side of the tooth. Many of the same biologic issues are seen in crown-root fractures as are encountered in crown fractures mentioned above. Additionally, because these fractures involve the cementum on the root of the tooth, there is need to evaluate the biologic width in planning treatment [18]. *Biologic width* describes the combined heights of the connective tissue and epithelial attachments to a tooth. The dimensions of the attachment were described in 1961 by Gargiulo, Wentz, and Orban in a classic article on cadavers [19]. Their work showed the connective tissue attachment having an average height of 1 mm and the epithelial attachment also having an average height of 1 mm, leading to the 2 mm dimension often quoted in the literature for *biologic width*. In addition, they found the average facial sulcus depth to be 1 mm, leading to a total average gingival height above bone of 3 mm on the face (see figure from ► Speareducation.com biologic width ► http://www.speareducation.com/spear-review/2014/12/biologic-width-part). Crown-root fractures are evaluated with this 2–3 mm biologic width requirement for restoration in mind. If there is insufficient tooth structure available after the fracture, the crown-root fractured tooth should not be restored without surgical or orthodontic intervention prior to restoration (Fig. 3.11).

**Emergent Treatment of the Crown-Root Fractured Tooth** In almost every case, the loose tooth fragment will need to be removed to enable full examination of the extent of the fracture. After the fractured fragment is removed, the tooth will be examined for pulp involvement and the apical extent of the fracture. If the pulp is involved, the treatment will start with direct pulp cap or partial pulpotomy (see "Pulp Capping vs Partial Pulpotomy"). If the apical extent of the fracture violates biologic width and there is less than 2–3 mm of sound tooth apical to the crest of the alveolar bone, this must be remedied before definitive restoration of the tooth can proceed. Biologic width can be restored in one of five ways:

1. Fragment removal only
2. Fragment removal and ostectomy

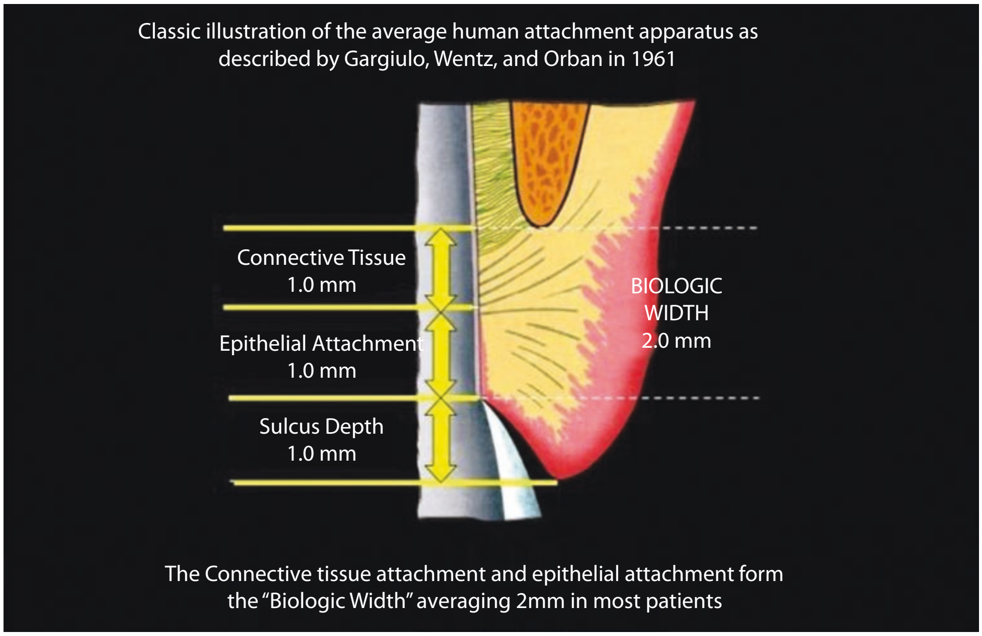



**Fig. 3.11** Biologic width graphic showing the relationship of the sulcus to the crest of the alveolar bone. (Credit: ► www.speareducation.com)

3. Fragment removal and surgical extrusion of the tooth
4. Fragment removal and orthodontic extrusion of the tooth
5. Extraction of the tooth and placement of dental implant

Fragment removal only is reserved for cases where the apical extent of the fracture is near the cementoenamel junction (CEJ) and where there is no pulp exposure. In these cases, the loose fragment is removed as soon after the injury as possible, and the tooth is restored with composite only supragingivally (Fig. 3.10). Meticulous oral hygiene is required to allow appropriate gingival healing after restoration.

Surgical exposure of the fractured surface is also known as crown lengthening surgery. This surgical procedure may be a total soft tissue surgery or soft tissue and bone removal surgery. The objective of this surgery is to convert a subgingival fracture to a supragingival fracture. This procedure should be used only when it does not compromise the final esthetic result of the treatment. The procedure is completed by removing gingival tissue to expose the apical extent of the fracture. If removal of soft tissue is insufficient to expose the fracture and create biologic width, some of the alveolar bone is removed with a surgical handpiece or hand instruments to create the proper access. One advantage to this treatment is minimal effect on crown-root ratio. After emergent care, the tooth is restored with either composite bonding or full-coverage restorations (Fig. 3.12).

Surgical extrusion of the injured tooth uses partial extraction of the tooth and repositioning of the tooth within the alveolus with the fracture sufficiently above the alveolar bone. The timing of this treatment is not critical so it can be done days or weeks after the injury. Surgical repositioning of the injured tooth will cause the loss of pulp vitality, and endodontic treatment will be required after the procedure. This procedure works best with more conical shaped roots. After local anesthesia administration, the tooth is luxated carefully to preserve the thin buccal plate of the alveolar bone. After sufficient luxation, the tooth is repositioned to expose the fracture and create biologic width. The tooth is then splinted to adjacent teeth to stabilize as the periodontal ligament regenerates. Endodontic treatment is initiated in 3–4 weeks after the injury ([3], p. 328). There is a slight chance of root resorption and ankylosis with this option. The tooth can be temporarily restored with composite. The definitive restoration of traumatized teeth will be discussed in ► Chap. 6 (Fig. 3.13).

Orthodontic extrusion moves the tooth slowly out of the bone to expose the fracture to enable restoration of the tooth after crown-root fracture. This is the preferred method of moving the fracture when pulp vitality needs to be preserved. This is important in young athletes especially when the roots are immature. Orthodontic extrusion is slower than surgical extrusion but has the advantage of guiding osseous or gingival tissues to create a better esthetic result. Refer to the orthodontics in dental trauma section later in the chapter for additional information and cases.

A final consideration in crown-root fractured teeth is to extract the tooth and restore the defect with a dental implant and implant crown. This option is reserved for adult nongrowing patients. Dental implants have become mainstream in dentistry and will continue to affect out treatment decisions now and in the future. See more on dental implants in ► Chap. 6.

In deciding which treatment options are available for each individual patient, we need to consider the age of the patient, and with the age, we need to know if the patient is still growing. Younger and growing patients are best served by orthodontic extrusion. We need to know the status of the patient's athletic career whether or not the patient is still active in athletics and at what level. We need to assess the need to expeditious care in which case surgical extrusion may be the best option. There are a number of questions to be answered before making treatment decisions for crown-root fracture cases, and the dentist should have frank conversation with patients or parents when finalizing treatment plans.

3

**Fig. 3.12** **a** Bicycle accident simple crown fracture #7 and crown-root fractures on #8 and 9 with subluxation. **b** Trauma from incisal view. **c** Crown fragments removed and pulp is exposed. **d** Teeth #8 and 9 have been treated with Cvek pulpotomy with white MTA and covered. **e** Crown fragment #8 bonded back and a denture tooth added to the splint for #9 to allow possible eruption. **f** Temporary restoration; plans considered for orthodontic extrusion at a later date. (Photo credit: Mark Roettger DDS)

**Fig. 3.13** **a** Male athlete with a baseball injury a few weeks before senior pictures scheduled in his school, and he was most anxious to get his smile in order. Tooth #8 had a previous crown fracture and tooth #9 had a recent crown-root fracture with the palatal margin not restorable. **b** Pretreatment radiograph shows the root length was favorable for surgical extrusion of the tooth to create an accessible margin on the palatal side of the tooth. **c** Surgical repositioning of the tooth begins with a careful luxation of the tooth, trying never to remove the tooth completely from the socket. **d** Shows the tooth has now been repositioned and the restorative margin access is verified; once verified, the splint can be placed. **e** Shows the splint in place to stabilize the intentionally luxated and extruded tooth. **f** Shows the splint is removed 2 weeks after placement. **g** The patient has been restored and is ready to smile for senior pictures on time. (Photo credit: Mark Roettger DDS)

**Outcomes of Treatment for Crown-Root Fractures** Expectations are high for success if all the biologic principles are carefully considered and followed when treatment is rendered. Pulp healing in complex crown-root fracture is similar to the outcomes of complex crown fractures if surgical extrusion is not done. Surgical extrusion requires endodontic treatment since pulp blood supply will be disrupted. The overall prognosis after surgical or orthodontic extrusion is better for teeth that maintain adequate crown-root ratios.

### 3.7.3 Root Fractures

Root fractures are relatively uncommon dentoalveolar injuries. They are complex injuries of the dentin, cementum, and pulp. They are usually classified by the location on the root where the fracture occurred, coronal, middle, or apical third. Knowing the location of the root fracture is of utmost importance in determining the prognosis and treatment of these injuries. These fractures can be horizontal or oblique across the root of the tooth. The coronal segment of the fractured tooth may or may not be mobile and the degree of mobility may affect treatment decisions. Root fractures create clinical management problems for many dentists confronted with these injuries, and many root fractured teeth are improperly treated by premature extraction. This is *common misconception #2* all teeth with middle and apical third root fractures that heal with a dark line require extraction.

**Trauma Misconception #2**

If you see this after 2–4 weeks of healing, you need to extract this tooth.

The fact is that most middle or apical root fractured teeth can survive a long time if properly managed [20].

**Radiographic Findings in Root Fractures** Root fractured teeth cannot be properly and completely diagnosed without adequate imaging. Most root fractures are oblique fractures across the root from facial to palatal and are easily detected by normal periapical dental imaging. Cone beam CT is rapidly becoming the standard of care for these types of injuries as the 3D view of the root allows more information to aid in diagnosis (Fig. 3.14).

**Pathophysiology of Root Fracture** The root fracture of a tooth is a complex injury of dentin, cementum, and pulp with the coronal segment of the fractured tooth suffering a luxation injury to the periodontal ligament and likely severing of the neurovascular supply to the dental pulp. The apical segment of the root is virtually uninjured and intact. The coronal pulp may or may not survive this injury, and long-term monitoring must be done to follow the healing of the dental pulp. Simplified, root fractures are a luxation injury of the coronal fragment of a tooth with periodontal ligament and pulp sequelae and no injury to the apical fragment. Crown-root ratio of the coronal fragment becomes important in the treatment and prognosis of the root fractured tooth.

**Emergent Treatment of the Root Fractured Tooth** Fractures in the coronal part of the root have the poorest prognosis of all root fractures and many times will need to be extracted, especially in the adult patient. We may try more heroic measures to save coronal one third root fractured teeth in growing patients to facilitate growth and development if possible. For teeth fractured in the middle or apical one third of the root, reduction of the fracture and splinting of the coronal fragment are indicated. Splinting can be accomplished with a rigid or semirigid splint, and the teeth can be splinted for 4 weeks up to 4 months. Monitor healing by mobility tests, sensibility tests, and radiographic imaging (Fig. 3.15).

**Healing of Root Fractured Teeth** Andreasen et al. published a paper on the survival of root fractured teeth in 2008 [20] where they found the survival of root fractured teeth was as high as

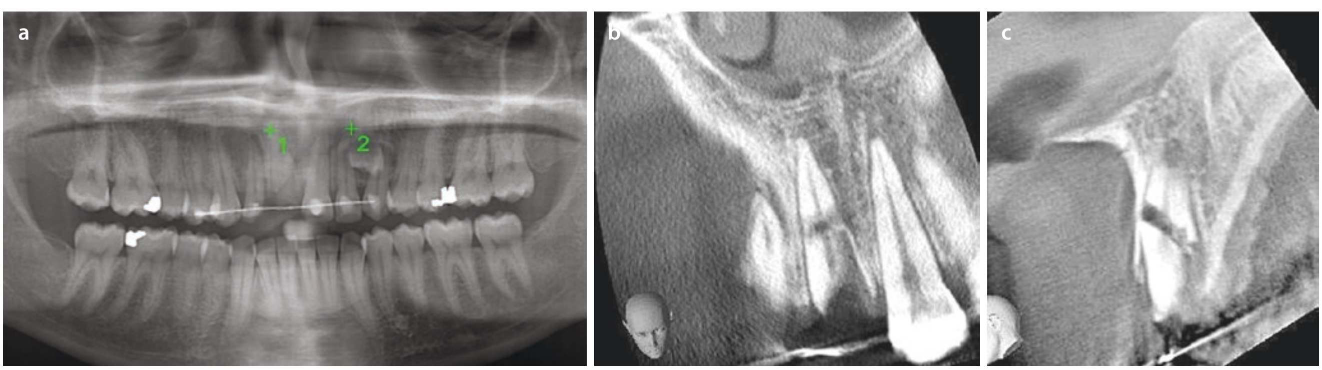



**Fig. 3.14** **a** Conventional dental radiography provides images in two dimensions which can make diagnosis difficult. This soccer player was kicked in the mouth during a game. It is difficult to determine the precise injury. **b** CBCT allows us a three-dimensional look at the same soccer injury. **c** CBCT of the same injury from a different plane reveals more information about the injury and aids in the treatment planning. (Photo credit: Eric Grutzner DDS)

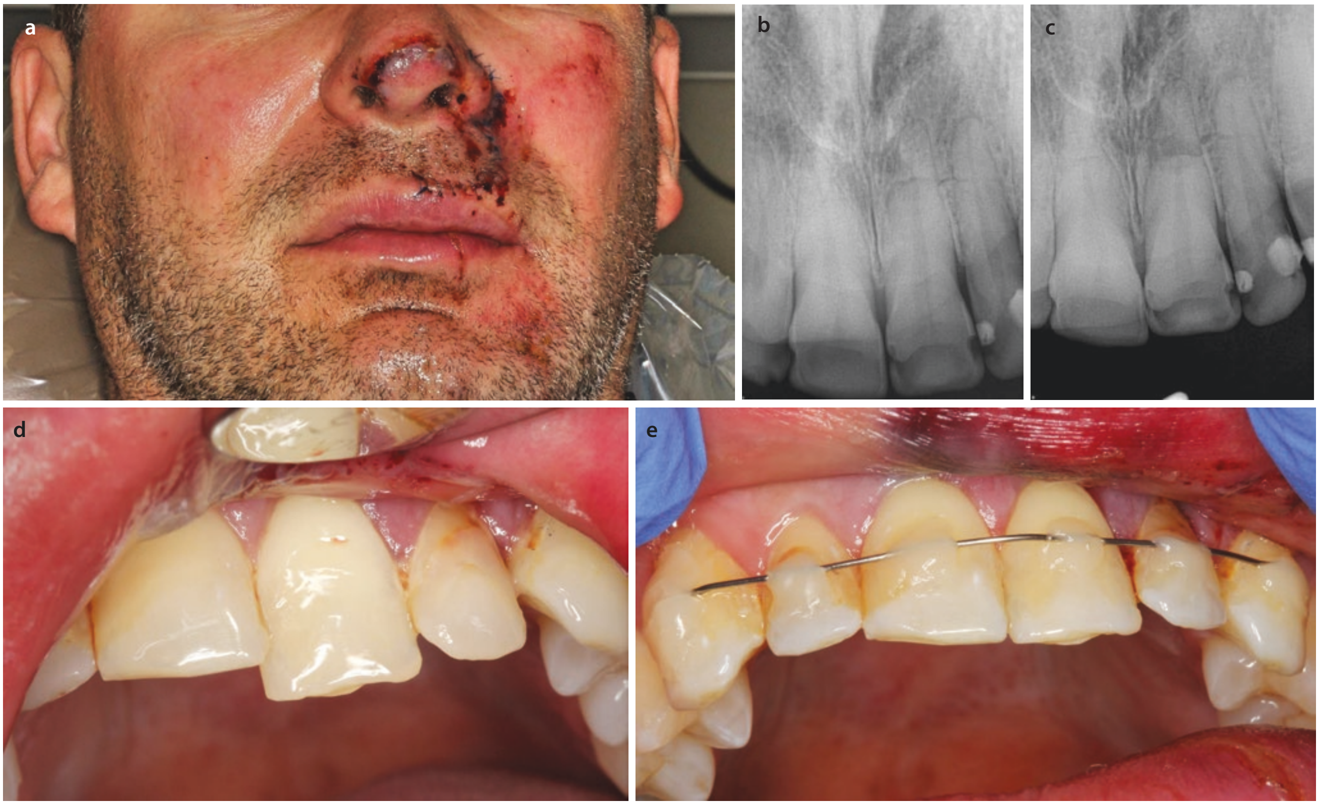



**Fig. 3.15** **a** Competitive bicycle accident resulted in the displacement of tooth #9 with tenderness present on teeth #8 and 10. **b** Radiograph #1 shows possible root fracture lines possible on #8, 9, and 10. **c** Second radiograph at a different vertical angle shows the fracture more obvious on tooth #9 and still possible on teeth #8 and 10. **d** Clinical intraoral finding is that tooth #9 is displaced and #8 and 10 are not. Slight mobility was noted on teeth #8 and 10. **e** Shows reduction of root fracture and splint in place. The splint will be worn for 1–2 months. Teeth will be monitored for appropriate healing at the fracture site(s). (Photo credit: Mark Roettger DDS)

88% for up to 10 years. There are three healing possibilities in root fractures.

- *Hard tissue healing*: where dentin and cementum bridge the fracture space. Here normal tooth mobility is observed, a thin radiographic fracture line is seen on imaging, and the coronal pulp canal is intact.
- *Connective tissue healing*: where periodontal ligament cells fill the space between coronal and apical segments. Here tooth mobility is increased, a larger darker fracture line is seen radiographically, and pulp canal obliteration in the coronal fragment is seen.
- *Granulation tissues interposition*: where granulation tissue fills the space between apical and coronal fragments due to infection of the coronal fragment pulp tissue. Here increased to even extensive tooth mobility is seen, absent pulp sensibility is noted, the distance between the fragments may grow as seen on imaging, and bone resorption is seen at the level of the fracture line.

Both hard tissue healing and connective tissue healing are considered successful resolution of a root fractured tooth. Granulation tissue interposition with infection is considered failed healing. Failed healing does not require immediate extraction of the tooth especially if the fracture is in the middle or apical third of the tooth root. Proper endodontic treatment of the coronal fragment many times will revert granulation tissue interposition to connective tissue or hard tissue healing vastly improving the survival of these teeth avoiding extraction. Every effort should be made to keep teeth with root fractures in the mid to apical thirds of the root, especially in growing patients. Remember the apical fragment is almost never injured and requires no treatment. If endodontic treatment is required to treat fracture healing by granulation tissue or infection, endodontic treatment is limited to the coronal fragment only!

## 3.8 Injuries to the Periodontal Tissues

This section of dentoalveolar trauma describes injuries to the supporting structures of the tooth especially the periodontal tissues. Trauma to the alveolar bone will be covered in the next section. Luxation injuries cover a wide variety of dentoalveolar trauma, and we categorize them as to the damage caused and the treatment needed to manage the injuries properly. Luxation injuries as a result of sports trauma are almost exclusively seen in the maxillary anterior region, more specifically the maxillary right and left central incisors. Luxation injuries many times occur to multiple teeth in the arch. These injuries will be discussed one by one to shed light on the proper management of each luxation injury. The more severe luxation injuries cause significant damage to the periodontal ligament and to the dental pulp as well.

### 3.8.1 Concussion Injury to the Periodontal Ligament

This is a minor trauma to the periodontal ligament (PDL) where the tooth is not loosened or displaced from its normal anatomic position. The tooth has been struck but not moved and is usually tender to touch, percussion, or biting. This is caused by edema and possible bleeding in the PDL. There is no treatment indicated for this minor injury of the PDL. Radiographic imaging is done to rule out more serious injury to the tooth. The pulp will almost always recover normally without treatment.

### 3.8.2 Subluxation of the Periodontal Ligament

This is also a minor luxation injury of the PDL that exhibits increased mobility of the tooth without displacement. Bleeding around the marginal gingiva and the gingival sulcus is noted on clinical examination. The tooth is mobile and tender to palpation, percussion, and bite if in occlusion. From the trauma, there is edema, bleeding, and some tearing of the PDL. There is also a slight chance of disruption of the neurovascular supply to the pulp of the tooth in this injury as well. Radiographs are taken to rule out more serious damage to the tooth. The treatment for subluxation injuries is often no treatment. In the case of very tender or painful teeth, an occlusal adjustment of the opposing teeth can be done, or the teeth can be splinted with a nonrigid splint for a week or 2 to allow healing of the PDL. Monitor the dental pulp until a definitive diagnosis can be made. There are very few instances of pulp necrosis resulting from subluxation injuries. Pulp canal obliteration may also be seen in response to the trauma.

### 3.8.3 Extrusive Luxation

The tooth in extrusive luxation is partially displaced from its socket usually in an axial direction. These teeth look longer than the adjacent teeth and are many times displaced toward the palate. There is usually significant mobility on clinical examination. There is always bleeding from the periodontal tissues, and the percussion sound is dull (Fig. 3.16).

**Radiographic Features** The radiographic appearance of an extrusive luxated tooth is one of displacement with the socket empty at the apex.

**Pathophysiology of Extrusive Luxation** The traumatic force transmitted through the tooth tears many of the fibers that make up the periodontal ligament, leaving a few mostly near the gingival margin to hold the luxated tooth partially in its socket. The coronal movement of the tooth out of the socket severs the neurovascular supply to the dental pulp, creating the potential for pulp necrosis. Extrusive luxation of an open apex immature tooth has the potential to revascularize keeping the pulp vital but ending up ultimately with pulp canal obliteration (PCO). Most teeth that suffer extrusive luxation will show pulp necrosis from the lack of blood supply to the pulp.

**Emergent Treatment of Extrusive Luxation Injuries** The emergent care of this injury is to replace the tooth to its normal anatomic position, checking occlusion and function, and splint

**Fig. 3.16** **a** College basketball injury: extrusive luxation from an elbow to the mouth under the basket; tooth is loose and extruded. **b** Tooth repositioned and ready to splint. **c** Tooth splinted; the season was a few weeks from the end, so a mouthguard was made for the patient, and the splint was left in place until the season ended

the tooth in place with a nonrigid splint for a period of 2 weeks. A radiograph is taken to insure the tooth is completely replaced in the original bony socket. The tooth is then monitored to follow the pulp condition with sensibility tests and the root and periodontal condition by taking periodic radiographs. In teeth with closed apices at the time of injury, there is very little chance for revascularization so endodontic treatment can be initiated when the splint is removed.

**Healing of Extrusive Luxation Injuries** Proper and adequate splinting assures complete healing of the periodontal ligament. Pulpal healing is dependent on the root development at the apex at the time of injury. Open apex teeth have a good chance to revascularize the pulp tissue during and after splinting. The likely ultimate healing course for an open apex tooth with an extrusive luxation injury is pulp canal obliteration. The tooth with a closed apex at the time of trauma has virtually no chance to revascularize after extrusive luxation and endodontic treatment is recommended.

**Extrusive Luxation Expected Outcomes**

*Pulp*

- Immature roots: PCO is frequent and pulp necrosis is rare.
- Mature roots: Pulp necrosis is frequent and PCO is rare.

*PDL*

- Root resorption is rare; however, inflammatory resorption can be seen in association with pulp necrosis.

### 3.8.4 Lateral Luxation Injuries

Displacement of a tooth, usually maxillary anterior tooth, in a direction that does not extrude it from the socket. These injuries typically include a fracture of the alveolar socket wall in addition to the luxation of the tooth. The tooth usually appears to be slightly intruded and has a palatal inclination to the crown of the tooth. The tooth is not loose unlike teeth with extrusive luxation injuries. Additionally the tooth exhibits a high ankylotic tone on percussion.

**Radiographic Features of Lateral Luxation Injuries** In normal periapical radiographs, the tooth may not look displaced in the socket, but a steep vertical angle on a periapical radiograph could disclose the displacement. Cone beam CT scans can also be very useful in capturing the damage in lateral luxation injuries.

**Pathophysiology of Lateral Luxation Injuries** Traumatic force against the facial of the crown of an upper incisor causes palatal movement of the crown with labial movement of the tooth root apex, pushing the apex of the root out through the buccal plate of bone in the apical part of the alveolar socket wall. This locks the tooth into the eccentric position with the root apex outside of the bone. There are injuries to the PDL, bony socket wall, cementum at the root apex, as well as the cementum at the cervical area of the palatal side of the tooth. The neurovascular supply to the dental pulp is also severed creating ischemia of the dental pulp. This is a very complex injury involving many tissues.

**Treatment of Lateral Luxation Injuries** The displaced tooth is usually locked into its ectopic position by the root apex protruding through the buccal cortical plate of the alveolus. The treatment like for extrusive luxation is reduction and splinting. The locked position complicates simple reduction of these luxation injuries. Reductions are painful, so administration of local anesthetic is required prior to the procedure. The reduction can be completed with digital pressure at the root apex in a coronal direction with simultaneous facially directed digital pressure on the palatal side of the crown of the tooth. With the appropriate force on the tooth, it should pop back into the socket and the reduction is complete (Fig. 3.17). Verify occlusal interferences. After reduction, the tooth should be splinted with a nonrigid splint for 4 weeks. Monitor at regular intervals to follow healing. Closed apex teeth at the time of injury should have endodontic treatment started within 2–3 weeks of trauma. Open apex teeth that are followed to

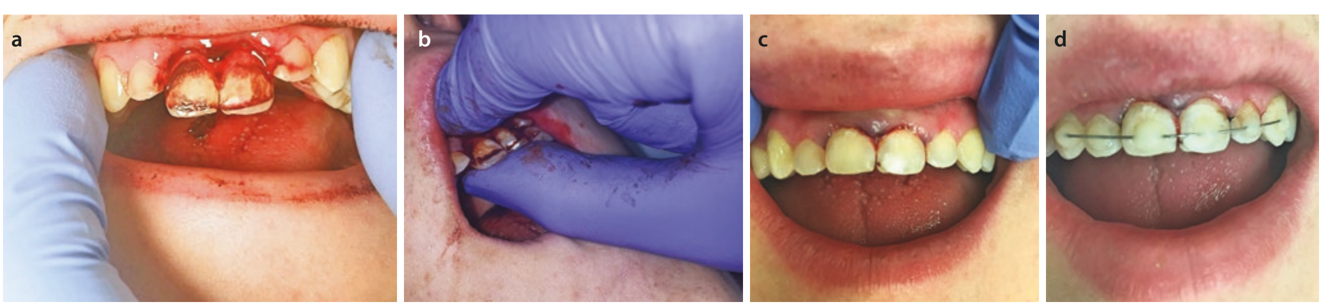



**Fig. 3.17** **a** College basketball injury: elbow to the mouth caused a lateral luxation injury. Teeth were locked in place with no mobility. **b** Reduction of the lateral luxation: fingers push the roots coronally while the thumb pushes the crowns anteriorly. **c** Luxation reduced and ready to apply the splint. **d** Splint in place; the splint was left on for 4 weeks, endodontic treatment was started 2 weeks after injury. (Case courtesy of Mark Roettger DDS)

observe pulp regeneration need close monitoring with radiographs and sensibility testing to diagnose regeneration or necrosis.

**Healing of Lateral Luxation Injuries** These are damaging injuries and have multiple healing consequences. The pulp may survive in an open apex tooth with a lateral luxation. The ultimate disposition of the pulp in these cases is usually pulp canal obliteration. Teeth with closed apices at the time of injury will not experience pulp regeneration and will experience pulp necrosis if endodontic treatment is not completed. Teeth with lateral luxation injuries have areas on the root that receive trauma from crushing the root against the alveolar bone in the apex area and the cervical area of the root. These areas could be susceptible to replacement resorption and ankylosis.

**Lateral Luxation Expected Outcomes**

*Pulp*
- Immature teeth: PCO is frequent and pulp necrosis is rare.
- Mature teeth: Pulp necrosis is frequent and PCO is rare.

*PDL*
- Due to compression of the PDL, inflammatory and replacement resorption may occur (rarely).
- Surface resorption is frequent and is seen apically.

### 3.8.5 Intrusive Luxation Injuries

These relatively rare injuries result in the tooth being displaced apically in an axial direction and cause devastating injuries to the periodontal ligament and its cells, the alveolar bony socket, and the neurovascular supply to the pulp and to the dental pulp. The tooth appears shorter than adjacent teeth and will exhibit a high ankylotic tone when tested for percussion. The teeth usually show no mobility on examination as they are locked in place due to the fracture of the alveolar socket.

**Radiographic Features of Intrusive Luxation** The tooth will appear to be apically dislocated with possible disappearance of the periodontal ligament.

**Pathophysiology of Intrusive Luxation Injuries** This injury occurs when the energy of trauma is mostly in an apical direction pushing the tooth in an apical direction. This traumatic displacement causes multiple damaging injuries. The periodontal ligament suffers a serious crush injury as well as the cementum of the tooth. The alveolar bony socket suffers a comminuted fracture, and the neurovascular bundle is crushed and severed. Spontaneous re-eruption can be anticipated only in teeth with open apices; all mature teeth must be repositioned as a part of active treatment either by orthodontic or surgical methods.

**Treatment of Intrusive Luxation Injuries** Intrusive luxation injuries are very serious injuries, and a number of factors need to be considered in planning treatment in order to maximize healing potential. Healing of the periodontal ligament is dependent of the size of the surface area of the cementum damage on the root surface. The healing of the pulp will depend on the root development stage and size of the apical foramen. Since spontaneous re-eruption will happen only in teeth with open apices and then only some of the time. Tooth repositioning in most cases will need to be done by the managing dental team.

- *Teeth with open apices*: These teeth have a modest chance of spontaneous eruption, so immature teeth that suffer intrusive luxation can be monitored for eruption over the next 3 weeks; if no eruption is noted, repositioning can be initiated. Repositioning can be accomplished by orthodontic or surgical procedures. If no repositioning is seen in 3 weeks, orthodontic extrusion can be initiated. If there are multiple teeth with intrusive luxation injuries, or if the intrusion is over 6 mm, surgical repositioning can be done [21]. In this case, the area is anesthetized with local anesthetic and the tooth grasped by a surgical forceps and gently loosened to unlock the tooth from the bone and allow the tooth to erupt while closely monitoring the patient. If pulpal necrosis occurs, endodontic treatment needs to be done. See ► Chap. 5 for endodontic treatment detail.
- *Teeth with mature closed apices*: In these teeth, there is little or no chance of spontaneous eruption after intrusion, and the teeth need to be orthodontically or surgically repositioned. Studies indicate that orthodontic repositioning may be the best choice for many of these injuries [22]. This repositioning should begin as soon as possible, and the teeth may need to be surgically luxated prior to orthodontic repositioning. When multiple teeth are intruded or when the intrusion is 6 mm or greater, surgical repositioning should be considered. Again the area is anesthetized and the tooth is luxated with a forceps and repositioned and splinted in the proper position. If there is gingival laceration present, the lacerations need to be sutured to repair and seal the periodontal ligament from the oral flora. After repositioning, endodontic treatment needs to be initiated within 2 weeks to avoid inflammatory internal resorption. Careful monitoring of teeth with intrusive luxation injuries needs meticulous follow-up to watch for complications such as ankylosis, pulp necrosis, and inflammatory resorption.

**Healing of Intrusive Luxation Injuries** There are multiple healing complications due to the tremendous damage done by these injuries. Pulp necrosis, root resorption, and marginal bone loss are frequent complications of intrusive luxation injuries. *Pulp necrosis* can happen in any intrusive luxation injury, and frequency is related to the stage of root development at the time of injury. Open apex teeth at the time of injury have a better chance of pulp regeneration than teeth with closed apices. *Internal root resorption* is seen in these injuries when endodontic treatment is delayed in cases of open apex spontaneous eruption after late occurrence of pulp necrosis. *Replacement resorption* is seen in the intrusive luxation injured teeth and is related to the surface area of cementum damage on the root surface. The larger the surface area of cementum damage, the higher the incidence of replacement resorption. Replacement resorption is common in these types of injuries. Survival of teeth after intrusive luxation despite root development at time of injury and type of treatment rendered is less than 70% over the 15 years after injury. These are very damaging injuries and require careful emergent care and long-term follow-up. The prognosis of these injuries is significantly related to the stage of root development and the degree of intrusion of the tooth at injury [22]. Ankylosis and pulp sequelae are less common in teeth with one quarter to three quarter root development and a mild degree of intrusion.

**Intrusive Luxation Expected Outcomes**

*Pulp*

- Immature teeth: PCO is a relatively frequent sequelae.
- Mature teeth: Pulp necrosis is almost universal.

*PDL*

- Due to excessive compression of the PDL, both inflammatory and replacement resorption may occur; surface resorption is very frequent and is seen apically.

### 3.8.6 Avulsion Injuries

Avulsion injuries are the complete luxation or exarticulation of the tooth from the alveolar bone. This is a relatively uncommon injury although some sources claim that as many as 5 million teeth are avulsed in the USA each year. These statistics are difficult to verify, and the actual number of avulsed teeth in the USA is likely much smaller than 5 million. These injuries occur like most traumatic injuries to the maxillary central incisors. The most common etiologies of avulsions are sports injuries and fights. These are complex injuries that require acute care, and most of these injuries occur within a short distance of home, school, or sports venue, so if workers at such locations were educated on the emergent care of these injuries, we might expect better outcomes [23]. Avulsion injuries are complicated by the fact that the tooth is traumatically separated from its normal environment and is subjected to exposure to strange bacteria from the environment, desiccation if not replaced in the socket or stored in an appropriate storage medium, and chemical irritants from the environment such as fertilizer from a sporting field. Many groups and individuals have offered management strategies for the avulsed tooth, and these strategies are not always in sync with each other, leaving the clinician to decide between conflicting ideas for management and naturally leading to confusion in the treatment of these injuries. This leads to *common misconception #3* that 60 min of dry time for an avulsed tooth is a magic treatment deadline and that less than 60 min of dry time before replantation of an avulsed tooth will result in regeneration of periodontal ligament, while greater than 60 min of dry time before replantation will lead to ankylosis of the avulsed tooth.

**Misconception #3. 60 Min of Dry Time is a Magic Number in Avulsion Care**

The literature will be reviewed here and guidelines for treatment and management will be offered. The American Association of Endodontists (AAE), the International Association of Dental Traumatology (IADT), and the American Academy of Pediatric Dentistry (AAPD) all use the 60 min dry time in their recommendations [24, 25]. They state that there can be viable PDL cells (fibroblasts) on the root of the avulsed teeth that can survive the 60 min of dry time to repopulate the root and form a complete periodontal ligament and avoid ankylosis of the tooth after replantation. Other authors have made claims that even if the PDL fibroblast cells are non-vital after dry time, soaking the tooth in Hank's balanced salt solution, a cell preservation medium, will reconstitute the non-vital cells and result in regeneration of a complete periodontal ligament [26]. Periodontal ligament cells and progenitor cells on the root of an avulsed tooth must remain *vital* (alive) to repopulate the root surface [27, 28]. Progenitor cells are able to *differentiate* to form fibroblasts [29] with the appropriate *phenotype* to create periodontal ligament fibers [30]. If all of this happens, the regeneration of the periodontal ligament is likely. Extraoral dry time is the most critical factor in ultimate survival of an avulsed tooth. If the tooth is placed in a proper storage medium (reviewed later in the chapter), one that has the proper osmolality, the tooth has a better prognosis if the replantation is delayed [31]. Extraoral dry time of greater than 5 min negatively affects the vitality of periodontal ligament cells and has an osteogenic effect on the remaining vital cells on the avulsed tooth root which will favor replacement resorption [27, 28, 30, 32].

### PDL Cells After Avulsion: Cell Biology

- *Vitality*: Are the cells alive?
- *Differentiation*: What kind of cells are they?
- *Function*: What are the cells doing?
- If we know the answers to these questions, we can predict what will happen to the PDL.

If excessive drying occurs to the periodontal ligament cells on the root of the avulsed tooth, these cells may not repopulate the root or may exhibit an osteogenic phenotype [30]. They may also create an inflammatory response over a large area of the root surface as faster-moving osteoblasts will cover the root instead of cementoblasts, and large areas of the root are not covered by the periodontal ligament [23]. When the root is not covered by cementum and periodontal ligament, the body cannot differentiate between the root and bone, and the root will be remodeled as if it were bone succumbing to replacement resorption [23]. In order for the tooth to successfully and predictably be replanted in the socket and have the periodontal ligament regenerate over the entire root surface, we need to reimplant the teeth within 5 min of the injury [27, 28, 30, 32, 33].

**Radiographic Appearance of Tooth Avulsion** Radiographs of the traumatized area will show an empty socket in the bone. The images can be useful in evaluation of the socket as replantation of the tooth is planned.

**Pathophysiology of Avulsion Injuries** The energy of trauma in these cases causes the tooth to be exarticulated from its bony socket in the alveolar bone. The periodontal ligament is torn over the entire root surface, and the cementum on the root may be damaged from the trauma. The root of the tooth is further damaged by desiccation and possible environmental, chemical, or bacterial contamination. The neurovascular supply to the pulp is completely severed, and the bony socket in the alveolar bone may be fractured. There is damage to nearly all of the dentoalveolar tissues including nerves and blood vessels. There is irreversible damage to the dental pulp in mature teeth and guarded to poor prognosis for the pulp in immature teeth. There is complete destruction of most of the dentoalveolar structures, and careful timely management is the only way to get successful regeneration of the PDL and management of the dental pulp.

**Treatment of Avulsion Injuries** Prompt treatment is of the utmost importance in treating avulsion injuries. The tooth must be replanted or placed in an appropriate storage medium within 5 min of the injury in order to insure regeneration of the periodontal ligament and thus inhibiting the process of replacement resorption of the tooth root [27, 28, 30, 33]. Immediate replantation of the tooth is the ideal treatment for avulsion and transport to a dentist for splinting and definitive care. These injuries most often happen when no dental personnel are present, and many times coaches and parents are hesitant to replant teeth at the scene of the trauma. Also severe injuries where multiple teeth are avulsed and the sockets are fractured and severely traumatized, immediate replantation may be impossible. Studies show that most avulsed teeth are replanted between 1 and 4 h after injury [34]. This is clearly well beyond the recommended 5 min that is required for the best outcomes. Avulsed teeth should never be allowed to be stored dry for over 5 min [33].

### Research Summary: Effects of Dry Time on PDL Regeneration

- *Less than 5 min*. Extraoral dry time, PDL cells maintain vitality and fibrogenic phenotype.
- *Result*: regeneration of PDL likely
- *Greater than 5 min but less than 15 min* of extraoral dry time, cells maintain vitality but begin to exhibit osteogenic phenotype.
- *Result*: Ankylosis likely
- *Greater than 15 min* extraoral dry time, cells lose vitality and die, losing the ability to make clones to repopulate the root surface.
- *Result*: Ankylosis

**Storage Media for Avulsed Teeth** The ideal storage medium would be compatible with the cells from the periodontal ligament that remain on the tooth root. This medium would provide compatible pH and osmolarity and nutrition for the cell's metabolism needs, be free of bacteria and contaminants, and be immunologically neutral. The ideal storage medium must also be available at the site of the trauma and therefore must be relatively inexpensive. Many solutions have been looked at as storage media for avulsed teeth from tap water to expensive tissue culture media used in transport of organs for transplant. Currently, the most widely used storage media for avulsed teeth are tap water, saliva, isotonic saline, milk, and Hank's balanced salt solution (HBSS). *Tap water* has been ruled out as a storage medium due to its hypotonicity which leads to rapid lysis of periodontal ligament cells on the tooth root [35]. *Saliva* has also been used as a storage medium in avulsion injuries. Saliva also has a nonphysiologic osmolarity for the periodontal ligament cells on the root as well as many bacteria. For these reasons, saliva has been discounted as an appropriate storage medium, although it is better than dry storage. *Physiologic isotonic saline* has also been tested as a storage medium in tooth avulsion cases and had been found to be useful for shorter storage periods because of its physiologic osmolarity [35]. Milk has been studied extensively as a storage medium for avulsed teeth. Cold skimmed milk has many of the properties of an ideal storage medium. It has a physiologic osmolarity, it has relatively few bacteria and near neutral pH, and it is readily available around most sporting venues. Additionally it has been suggested that milk contains growth factors such as platelet-derived growth factor (PDGF), transforming growth factor beta (TGF-β), and fibroblast growth factor (FGF) as well as other growth factors that are useful in promoting development of a new periodontal ligament [36, 37]. *Hank's balanced salt solution* was introduced as a storage medium by Save-A-Tooth™ system. Balanced salt solutions are solutions made to a physiologic pH and isotonic salt concentration. They also routinely contain sodium, potassium, calcium, magnesium, and chloride. Hank's solution also contains glucose to nourish cells metabolic needs [38]. Hank's salt solution was developed as a cell culture medium and should be a good storage medium for avulsed teeth. The drawback of Hank's salt solution is that it is usually not readily available at sporting venues. Comparison of milk and Hank's salt solution found that milk performed as well if not better than Hank's solution for period up to 4 h [37]. With this information, we can assume that in considering all factors, cold skimmed milk is the best storage medium for avulsed teeth [36, 37] (◘ Fig. 3.18). This is an active area of current research and recommendations may change in the future.

Ideal treatment of the avulsed tooth involves immediate replantation of the tooth into the empty socket. The tooth needs to be retrieved for the sporting surface being careful to touch only the crown part of the tooth and avoid touching the root surface. Rinse debris from the root surface with saline if possible or water if that is all that is available. If this is at the sporting event, replant the tooth with digital pressure until the tooth is completely back into the socket. Use the adjacent teeth as a guide for the depth of replantation required. After the tooth has been replanted, have the patient bite on a clean gauze or washcloth and transport the patient immediately to the dental office for definitive care. Once the patient gets to the dental office, if the tooth has been replanted, the dentist needs to check the position of the tooth and the occlusion. If this is correct, the tooth can be splinted with a semirigid splint. If the patient is transported to the dental office with the tooth in an appropriate storage medium, the tooth can be retrieved from the storage container holding by the crown part of the tooth. The tooth then is rinsed with physiologic saline and the socket gently curetted or flushed with saline. Holding the tooth by the crown, the tooth is replanted with digital pressure using adjacent teeth as a guide. After position is correct in the socket, the tooth can be splinted using semi-rigid splint (see the splint section later in this chapter) (◘ Fig. 3.19). After stabilizing the tooth with a splint, we need to inquire about the patient's tetanus status. After a tooth has been out of the mouth for a time, especially on the ground, there is danger of tetanus infection after replantation so tetanus vaccine status is discussed with the patient. Often the patient is placed on an antibiotic for 7–10 days to assist healing. Bacterial presence is always a detriment to healing. The tooth will remain splinted for 1–2 weeks, and in any closed apex tooth, endodontic treatment is initiated at the time of splint removal [23]. Teeth with open apices may experience pulp revascularization and should be carefully followed for signs of pulp vitality [25]. If ankylosis occurs, the tooth will ultimately be lost since the root will be completely replaced by bone over time. This is of most consequence in a growing patient when an ankylosed tooth will adversely affect the growth and development of the jaw [23].


◘ **Fig. 3.18** **a** Cold milk. **b** Sterile saline (isotonic). **c** Save-A-Tooth (HBSS)


**Fig. 3.19** **a** Baseball injury: 12-year-old male hit in the mouth by a batted ball; tooth avulsed and immediately retrieved and placed in milk at the scene. **b** At the dental office less than 30 min after injury, the tooth was dry for less than 5 min and placed in milk, the tooth replanted after gentle rinsing with isotonic saline. **c** The tooth splinted with a Pro-Temp semirigid splint that was left in place for 1 week, endodontic treatment was started 1 week later. (Case courtesy of Mark Roettger DDS)

**Avulsion Ideal Treatment**

- *Immediate reimplantation!!! 5 min*.
- Rinse tooth with physiologic saline holding tooth by the crown.
- Flush socket coagulum with saline.
- Slowly replant the tooth with gentle digital pressure.
- Splint 1 week semirigid or nonrigid splint.
- Antibiotic coverage, i.e., amoxicillin 1000 mg and then 500 mg tid × 7 d.
- Inquire as to pt tetanus status.
- Initiate RCT at 1 week, when splint is removed.

**Delayed Replantation** In a nongrowing patient, a tooth that has been dry for greater than 30 min can be replanted knowing that the tooth will heal by repair and will become ankylosed. Steps in the replantation process can be modified to slow the inevitable resorption of the root to delay tooth loss for as long as possible. The goal in delayed replantation is to restore esthetics and function for a temporary time and give the patient time to prepare for definitive treatment, perhaps after the sports career has ended. In this case, the root is scaled and all remnants of the periodontal ligament are removed. Then endodontic treatment is carried out extraorally. After administration of local anesthetic, the coagulum is removed from the socket with a curette, and examine and revise the socket as needed. If you desire to slow the root replacement for as long as possible, the tooth can be soaked in 2% sodium fluoride for 20 min prior to replantation. Replant the tooth with digital pressure aligning it with adjacent teeth, and stabilize the tooth with a flexible splint for 4 weeks. Administer antibiotics as above, and inquire as to the patient's tetanus status. Follow the tooth at subsequent recalls clinically and radiographically to monitor for infection or resorption [25].

Remember that at the scene of the trauma, the tooth should be replanted or placed in milk or Hank's balanced salt within 5 min of the incident if at all possible. Record the time of injury and the time when the tooth was placed in the storage medium.

**Healing Outcomes of Tooth Avulsion Injuries** We are concerned with pulpal healing and periodontal ligament healing in these injuries. The age of the patient, stage of root development, and extraoral dry time will be important in each of these healing outcomes. We need to consider pulpal healing and periodontal ligament healing as separate issues.

- *Pulpal healing after avulsion injuries*: The dental pulp will not heal after avulsion in a tooth with a closed, mature apex. The blood supply will be lost, and the pulp will become necrotic if the tooth is not treated endodontically [25]. Infection of the root canal space can result in inflammatory resorption of the tooth root and cause failure in healing. Immature teeth have a chance to revascularize the pulp after avulsion injuries. Revascularization is not predictable, so teeth replanted with open apices need close monitoring after initial treatment to look for signs of tooth vitality. Immature teeth that do not revascularize require additional regenerative endodontic care that is covered in ▶ Chap. 4.
- *Periodontal ligament healing after avulsion injuries*: Periodontal healing is almost completely dependent on extraoral dry time. Extraoral dry time of over 5 min leads to increased chances of healing the periodontal ligament by repair which means cementum and periodontal ligament do not cover the root and the tooth becomes ankylosed [23]. Ankylosis of a tooth always means the tooth will ultimately be lost. Ankylosis has additional consequences for a growing patient. When a tooth becomes ankylosed, it can no longer continue to erupt. When a growing patient has an ankylosed tooth, it retards the eruption of the tooth as the patient's jaws continue to grow and infraposition of the tooth is seen (Figure). Ankylosis of a tooth in a growing patient usually causes disturbances in the jaw growth and needs to be managed to insure normal craniofacial growth and development ([18], pp. 179–184, [25]). Ankylosed teeth in growing patients need treatment, and many procedures have been tried over the years. Currently decoronation is the recommended treatment for teeth exhibiting advancing infraposition after replantation [39].

3

**Current and Past Management Options for the Ankylosed Incisor**

*Adult*:

- Wait it out; minimal Tx with minor cosmetic adjustment

*Growing patients*:

- Early Ext with a series of transitional prostheses
- Intentional luxation and Sx repositioning*
- Decoronation with prosthesis is the current treatment of choice
- Intentional replantation with Emdogain*
- Alveolar distraction osteogenesis and ridge augmentation

**Recent research suggests these Tx are not justified; all are based on case reports with little evidence of support.*

### 3.8.7 Injuries to the Alveolar Bone

Injuries to the supporting bone of the teeth consist of comminution of the socket wall seen in intrusive of lateral luxation injuries, fracture of the socket wall, fracture of the alveolar process, and fracture of the mandible or maxilla. These injuries are similar to lateral luxation injuries but many times affect multiple teeth along with the fracture of the alveolar bone on the palatal side of the teeth (refer to ◘ Fig. 3.17).

**Radiographic Findings** Fracture lines may or may not be seen on images.

**Pathophysiology of Fractures of the Supporting Bone** Usually all fractures to the alveolar process are caused by damage to the teeth in the area. Jaw fractures will be discussed in more detail in ▶ Chap. 5. Fractures to the alveolar process will almost always be seen with concomitant tooth injuries. Caused by damage and movement of the teeth. Oftentimes, we see multiple teeth displaced as a unit and may or may not be locked into the improper position. We therefore have similar healing issues for the pulp and periodontal ligament tissues that we have in other tooth luxation injuries, and these need to be treated as well as the injury to the alveolar bone.

**Treatment of Fractures to Alveolar Process** The treatment of most alveolar fractures is reduction by repositioning the teeth involved and splinting for a period of 4 weeks. Endodontic treatment is required for all teeth involved with closed apices. Multi-rooted teeth in the fracture area enhance the chances of complications in these types of injuries ([3], pp. 499–501).

**Healing Outcomes of Alveolar Process Fractures** If the bone maintains adequate blood supply, it will heal after fractures are reduced. Additionally, we need to insure the healing of the pulp and periodontal ligament as well in these injuries. The injuries to the pulp and periodontal tissue are similar to those that the teeth experience in lateral luxation injuries.

## 3.9 Splinting in Dentoalveolar Trauma

Dental trauma that involves damage to the periodontal ligament may require splinting to assure complete healing after trauma. It has been found that no splint is better for healing than having a splint placed in experimental conditions ([3], pp. 842–851). Too rigid a splint will have damaging effects on the gingiva, pulp, and periodontal ligament. In clinical settings, it is practical to splint teeth in order to stabilize them during healing, provide patient comfort during healing, and improve function. The smaller the splint the better. For years, it was thought that rigid splints were required to heal luxation dentoalveolar injuries and arch bars were used to stabilize injured teeth. As new materials and techniques were discovered, especially adhesive dentistry, arch bars fell out of favor for most if not all of dentoalveolar trauma, due to adverse effects on the pulp and periodontium [40]. Most splints currently accepted for use in dentoalveolar trauma are bonded wire splints using adhesive dentistry to hold them in place [41]. Our goal in splinting of traumatized teeth is to use flexible splints for the least amount of time. The ideal splint should be easily applied to the teeth, be easy to cleanse, have no impingement on gingival tissue, be passive with no occlusal interference, allow for endodontic access, allow physiologic tooth movement, and be easily removed. This rules out the mostly outdated use of arch bars to stabilize dentoalveolar trauma. Arch bars are much too rigid to stabilize most dentoalveolar trauma cases, and the design brings them over the gingival margin making it impossible to keep the area under arch bard clean which in turn interferes with normal healing (◘ Fig. 3.20).

### 3.9.1 Splint Timing for Different Injuries

We need splints for stabilization and comfort during healing. We need to use the splint only for the time necessary to assist in the healing process. Splinting too short a time can cause reinjury to the area, and splinting too long can impede healing by not allowing physiologic movement of teeth and by inability to maintain proper oral hygiene [21, 25].

**Splinting Guidelines**

| *Injury type* | *Splint time* |
|---|---|
| 1. Concussion | No splint |
| 2. Subluxation | 2 weeks |
| 3. Extrusive luxation | 2 weeks |
| 4. Avulsion | 2 weeks |
| 5. Root fracture mid to apical 1/3 | 4 weeks |
| 6. Alveolar fracture | 4 weeks |
| 7. Root fracture coronal 1/3 | 4 months |

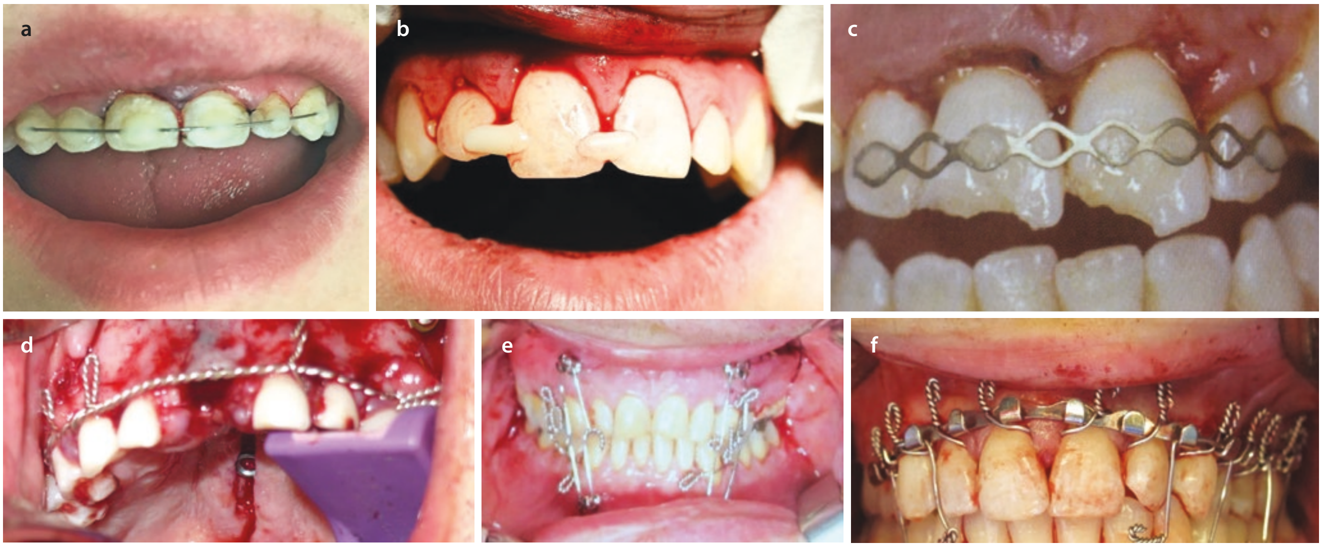



**Fig. 3.20** **a** Semirigid wire splint; preferred for most dentoalveolar trauma splinting; ease of placement and removal with access to the patient for cleansing. **b** Pro-Temp bond splint; semirigid; when composite is used for this type of splint, there are some thoughts that it is too rigid a splint. **c** Titanium mesh splint: semirigid and easy to place and remove, allows cleansing, and is most costly of these first three splint types. **d** Risdon wires used mainly to stabilize jaw fractures has no real use in dentoalveolar trauma with difficulty in placement and removal along with poor patient access for cleansing. **e** Intermaxillary fixation: no use in dentoalveolar trauma. **f** Arch bars; sometimes used in alveolar bone fractures, this splint also has very limited use, if any, in dentoalveolar trauma. Difficult to place and remove, and poor access for the patient to cleanse makes this a poor choice for most dentoalveolar trauma. (Photos courtesy of Mark Roettger DDS and Luke McMahon DDS)

## 3.10 Soft Tissue Injuries

See ▶ Chap. 5 for more discussion of soft tissue injuries. It is common to have associated soft tissue injuries when dentoalveolar trauma is encountered. The teeth are surrounded by the soft tissues of the lips, cheeks, gingiva, and the tongue. The soft tissues will inevitably absorb some of the energy of trauma, causing damage to these tissues. The soft tissues are also subjected to penetration injuries from the dental hard tissues. These penetration injuries are often through and through lacerations of the lips or tongue, and proper repair is required for appropriate healing to occur. Soft tissue injuries are classified into the following descriptive categories and can be seen both extraorally and intraorally.

**Abrasion** Abrasion is a superficial wound caused by scraping of the outer layers of the epithelium away exposing deeper layers of the skin or mucosa and underlying connective tissue. This leaves a raw bleeding surface. This type of injury is usually seen extraorally around the nose, mouth, and chin.

**Contusion** A contusion is a bruise caused in these cases by blunt force trauma that causes subcutaneous bleeding. Contusions are also caused by trauma from fractured bones below the surface.

**Laceration** Laceration is a shallow or deep wound resulting in a tear in the tissue usually caused by a sharp object, and in the oral region, this is most likely to be teeth. Lacerations are most commonly seen in the lips, oral mucosa, and gingiva. Deeper lacerations can involve additional structures such as blood vessels, nerves, and salivary glands. Through and through lacerations of the oral region, most commonly the lip, require careful repair to insure and esthetic result. These wounds are closed in layers with all through and through lacerations closed on both the cutaneous as well as the mucosal side of the laceration.

**Avulsion** Avulsion means tissue loss. Tissue avulsion is differentiated from tooth avulsion. Tissue avulsion is rare in the oral region but can be seen in bite injuries. Tissue avulsion can create difficult revision and management.

**Emergent Care for Soft Tissue Injuries in the Oral Cavity** ([42], pp. 627–635).

**Wound Assessment** Always be aware and look clinically and radiographically for additional injuries to underlying structures such as the bone, blood vessels, nerves, and salivary glands.

**Documentation** Documentation with diagrams and high-quality photographs will document the damage and deficits prior to treatment.

**Local Anesthesia** Adequate local anesthesia is required to repair soft tissue wounds in a clinical setting when the operating room is not required.

**Hemostasis** Adequate hemostasis is necessary to successfully treat soft tissue injuries. In most sports injuries, a vasoconstrictor in the local anesthetic is adequate to control hemorrhage.

**Decontamination** Cleansing of the wound is aimed at decreasing the bacterial counts and removing debris from the damaged tissue. This is done with normal saline.

**Debridement** Debridement is the surgical exploration and removal of foreign bodies and debris from the wound. Scalpel blades, forceps, and even scrub brushes can be used in the debridement process. Remember to look for tooth fragments in the debridement process in the soft tissue wound care protocol.

**Wound Closure** Lacerations require closure for esthetic healing. Dermal support and tension-free closure are required for proper healing. Oral cavity through and through lacerations must be repaired on the mucosal and cutaneous sides of the wound. Repair of only the cutaneous wound allows oral bacteria to percolate through the lesion and can cause extraoral scarring.

**Soft Tissue Wounds: Essentials in Summary**

| *Penetrating lip wounds* | *Tongue wounds* |
|---|---|
| – Antibiotics as indicated | – Antibiotics as indicated |
| – Soft tissue radiograph 25% | – Soft tissue radiograph 25% of normal exposure |
| – Regional anesthesia | – Regional anesthesia |
| – Rinse wound and surrounding area with detergent | – Rinse with saline |
| – Remove foreign bodies | – Remove foreign bodies |
| – Suture labial mucosa | – Rinse again with saline |
| – Rinse again | – Suture mucosal wound |
| – Suture cutaneous wound 6.0 nylon or Prolene | – Remove sutures in 4–5 days |
| – Remove sutures in 4–5 days | |

## 3.11 Orthodontic Considerations in Dental Trauma

In this section, we discuss the predisposition of some malocclusions for the occurrence of incisor trauma, the consequences of dentoalveolar trauma in the developing dentition, and the orthodontic management of the trauma-related tooth displacement.

### 3.11.1 Introduction

Dentoalveolar trauma is more prevalent in children and adolescents, particularly in boys, than in adults. This higher prevalence may be related to the active nature of children and their involvement with collective or individual high-impact sports. It is during these periods of life that the majority of them will be receiving orthodontic treatment with removable or fixed appliances such as brackets, maxillary expanders, or functional appliances, among others.

The presence of orthodontic appliances during the occurrence of a traumatic injury can reduce or increase the extension of lesions observed. For instance, the presence of brackets or any other appliance with metal components during a traumatic event may increase the severity of the soft tissue lacerations. On the other hand, the same appliances may decrease the extension of the dentoalveolar injuries because they may act as a splint. In the case of full fixed appliances, the brackets are ligated together by the archwires and this ligation may prevent the occurrence of severe tooth displacements decreasing the severity of the dental injury. This splint effect is more evident during the final stages of the orthodontic treatment when the wires are more rigid. Nevertheless, severe traumatic forces will eventually overpower the most rigid orthodontic wire and produce major injuries; therefore, the use of a mouthguard is even more important during the orthodontic treatment.

### 3.11.2 Class II Malocclusion and Incisor Trauma

Excessive overjet with flared maxillary incisors have been associated with an increased risk for dentoalveolar trauma. These two dental features are characteristics of the Class II malocclusion which is present in about 15% of the US population. Recent systematic reviews that include large randomized clinical trials have demonstrated that reducing the overjet of children with Class II malocclusion in the mixed dentition may reduce the incidence of incisor trauma in more than 40% [43]. Although the Class II malocclusion is more efficiently corrected in the young permanent dentition, the early treatment of Class II should be indicated in children with large overjet and proclined incisors especially for those that have other risk factors for dentoalveolar trauma such as participation in high-impact sports.

### 3.11.3 Dentoalveolar Trauma During Orthodontic Treatment

During the initial phase of the orthodontic treatment with brackets, flexible archwires are used for the alignment, frequently made of nickel titanium alloys (NiTi). Due to its flexibility, the NiTi will have minimal “splinting” effect. Therefore, severe impacts during this phase will affect individual teeth close to the affected area, which are usually the incisors. When the orthodontic treatment is more advanced, after 6 months of initiating the treatment, more rigid archwires are used, usually made of stainless steel alloys (SS). The rigidity of the SS archwires may potentially decrease the displacement of individual teeth during trauma, when the forces are mild to moderate. During severe impact; however, this rigid archwire can transmit the forces of the impact to teeth that are away from the affected area injuring them as well.

Injuries in the orolabial region during orthodontic treatment can produce undesired tooth movements as a consequence of the deformation of the appliance during the trauma.

The forces of the impact also can produce permanent deformation of the archwire, changing its form and adding undesirable forces to the affected teeth. The deformation of the SS archwire will continue to produce active forces on the affected teeth increasing the periodontal and pulp damage already produced by the trauma. Essentially, this deformation is similar to the intentional activation that is done by the orthodontist during active treatment; however, when it is caused by the trauma, the movement is toward an undesired and uncontrolled direction and with a force greater than the therapeutic orthodontic force.

Therefore, as part of the post-trauma clinical evaluation, it is fundamental to remove the archwire to confirm that it has not deformed and then replaced for a passive one.

### 3.11.4 Clinical Assessment of the Acute Trauma when Orthodontic Appliances Are in Use

When trauma occurs in the middle of the orthodontic treatment, the following considerations should be incorporated to the normal trauma protocol at the emergency care:

1. Careful examination to assess for missing components of the appliances that could have been aspirated or ingested. Brackets, bands, and coil springs are small enough that might not be identified during the clinical exam. A radiographic evaluation of the affected area including the radiographs of the soft tissue will be necessary to identify them because most of the current orthodontic materials are radiopaque. The clinical and radiographic findings need to be corroborated by the anamnesis.
2. Evaluate the integrity of the appliances. Remove the archwires, and replace it in a passive position if possible, or substitute it by a splint. Remove or trim sharp components that may cause further lacerations on the soft tissue. Instruct the patient in the use of orthodontic wax to reduce the sharp edges of the appliance to help with the healing of soft tissues.
3. Brackets can be used to retain the splints after the manual reposition of displaced teeth, but the archwire needs to be passive in the brackets. SS archwires can be bended and customized into the desired passive position. A 0.018″ round stainless steel wire will fit with any type of bracket prescription, therefore, should be part of the dental trauma emergency kit.

### 3.11.5 Consequences of Trauma in the Developing of the Dentition

Two of the most common dentoalveolar injuries, lateral luxation and intrusive luxation, of the primary teeth may cause a disruption on the path of eruption of the forming permanent teeth resulting on its impaction or dilacerations of their roots. Surgical exposure followed by orthodontic forced eruption is many times needed to resolve the trauma-related impaction of the permanent teeth. When the root dilacerations are severe, further endodontic treatment including apicoectomy may be also necessary to reduce the risk of buccal cortical bone perforation while trying to align the tooth in the proper position within the alveolar process. *Ortho Case 1: Surgical exposure and traction of a "scorpion" root dilaceration* (Fig. 3.21).

### 3.11.6 Orthodontic Movement Immediately After Dentoalveolar Trauma

In ideal circumstances, the traumatic tooth displacement, including lateral luxation, intrusion, and extrusion, should be treated during the immediate care by the digital reposition or surgical reposition. However, orthodontic movement may be indicated as part of the immediate care when the digital repositioning or surgical repositioning of the affected teeth is not possible [44].

It is not uncommon for trauma patients to receive the first care in the ER where dental repositioning or splinting is not accomplished due to the need for treatment of other major injuries, lack of patient cooperation, and lack of trained providers, among other limitations. Delaying the dental repositioning increases the difficulty of the digital or surgical repositioning in the ambulatory setting because of the tissue growth resulting from the reparative process in the periapical area of the affected teeth. Therefore, orthodontic repositioning may be an alternative in cases where the lateral, intrusive, or extrusive luxation cannot be addressed during the acute care of the dentoalveolar trauma or when the position achieved was not ideal.

*General principles of orthodontic repositioning of traumatized teeth*:

- Apply light orthodontic forces in the displaced teeth.
- Provide anchorage control to avoid undesirable movements of the unaffected teeth.
- Monitor pulp vitality closely when endodontic treatment has not been performed before starting the orthodontic tooth repositioning.
- Use a semirigid splint after the tooth is repositioned into their original position.

#### Key Concepts

The orthodontic extrusion as a mean to correct the intrusive luxation, crown fracture, or crown-root fracture can be performed in a rapid or a slow rate of activation.

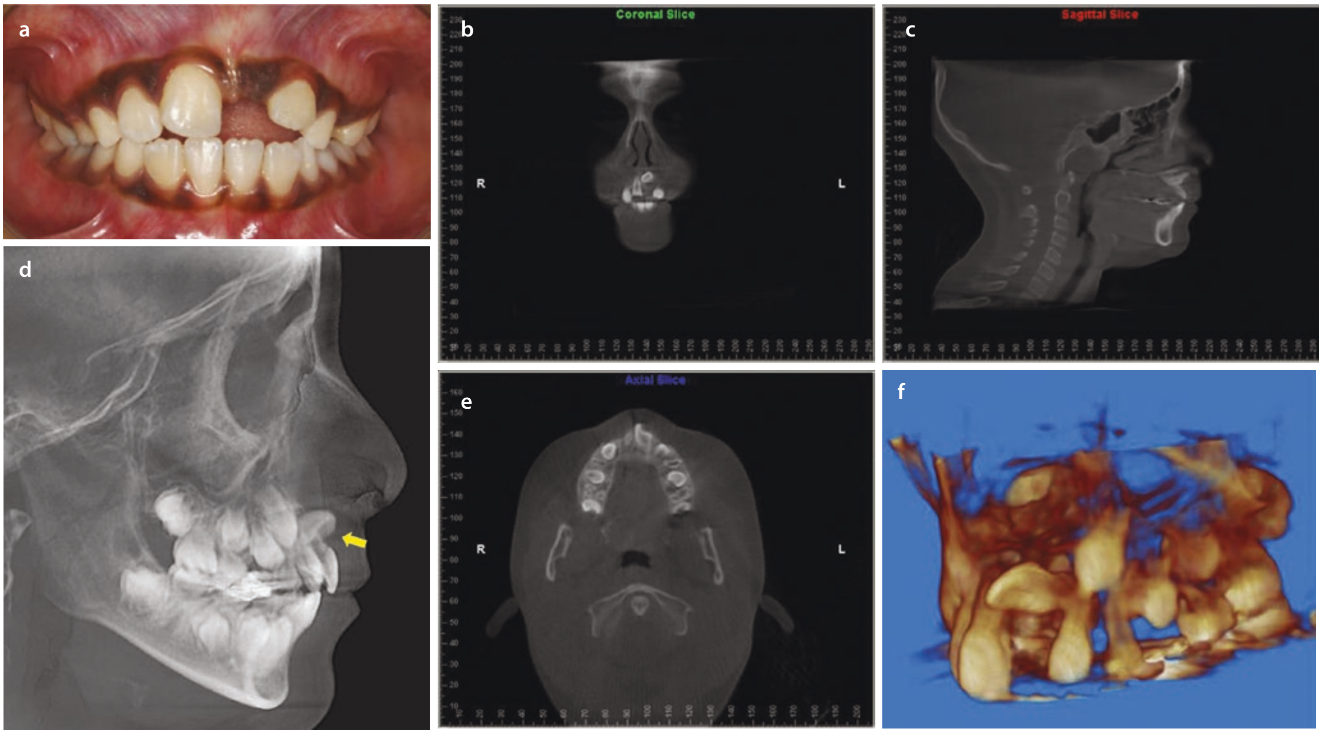



**Fig. 3.21** 9-year-old patient with previous history of dental trauma during the primary dentition. **a** Clinically, there is an absence of the right maxillary central incisor. **b**–**e** The coronal, sagittal, and axial views of the CBCT show the change of the eruption path of the maxillary central incisor. The labial and superior orientation of tooth #9 was caused by a previous traumatic event at age 4. The 3D rendering of the CBCT shows a horizontally positioned and upturned tooth #9 with a "scorpion"-shaped root due to an apical dilaceration. A surgical exposure and orthodontic slow extrusion will be necessary to align the tooth in the correct position. Due to the apical dilaceration, an apicoectomy may also be needed after the tooth is aligned in the arch

*Rapid orthodontic forced extrusion* is when the orthodontic force is applied in the occlusal direction at a rate of 1 mm per week to promote dental extrusion without promoting a vertical growth of the alveolar bone. Rapid orthodontic eruption is indicated for the treatment of intrusive luxation and for the orthodontic crown lengthening in cases of crown or crown-root fractures.

*Slow orthodontic forced eruption* is when the orthodontic force is applied in the occlusal direction at a rate of 1 mm per month to promote dental extrusion and vertical growth of the alveolar bone and gingival tissue. Slow orthodontic forced eruption is indicated for the treatment of non-restorable crown-root fractures and the delayed treatment of intrusive luxation.

### Orthodontic Treatment of the Intrusive Luxation

The traumatic intrusion of permanent teeth is classified according to the severity and the stage of development of the root of the affected tooth. In cases when the apices are open, the spontaneous eruption of the teeth can be expected even when the intrusion has been up to 7 mm. When the apices are closed, spontaneous eruption is recommended only if the intrusion is 3 mm or less [3]. Greater amounts of intrusion can be treated with surgical or digital repositioning in the acute phase and orthodontic repositioning in the acute or delayed treatment (delayed considered more than 3 days after the traumatic event). Rapid orthodontic forced extrusion is used to treat the intrusive luxation because it is not necessary for the alveolar bone to accompany the tooth movement in the occlusal direction. The objective of the orthodontic treatment is to recover the normal biological width that was invaded when the tooth was intruded in the socket placing the alveolar crest occlusal to the original position. The amount of extrusion required is about 1 mm per week to finalize the tooth repositioning with the alveolar crest in the ideal position, 2–3 mm apical to the cementoenamel junction, and to maintain the gingival contour symmetric with the non-affected teeth.

> » Orthodontic extrusion is appropriate when the intrusive luxation is greater than 3 mm, when the spontaneous eruption does not occur, or when the surgical repositioning is not feasible.

During the orthodontic forced extrusion, it is important to avoid applying active forces in traumatized teeth contiguous to the intruded tooth. During the clinical exam, teeth that were not injured need to be identified to serve as the anchorage teeth for the orthodontic forced extrusion (Fig. 3.22a–e). Usually posterior teeth are not affected by trauma and will serve as anchorage. However, to simplify the mechanics, it is preferable to use the closest non-affected teeth. For instance, in a case of a central maxillary incisor intrusive luxation, the appliance can extend only from canine

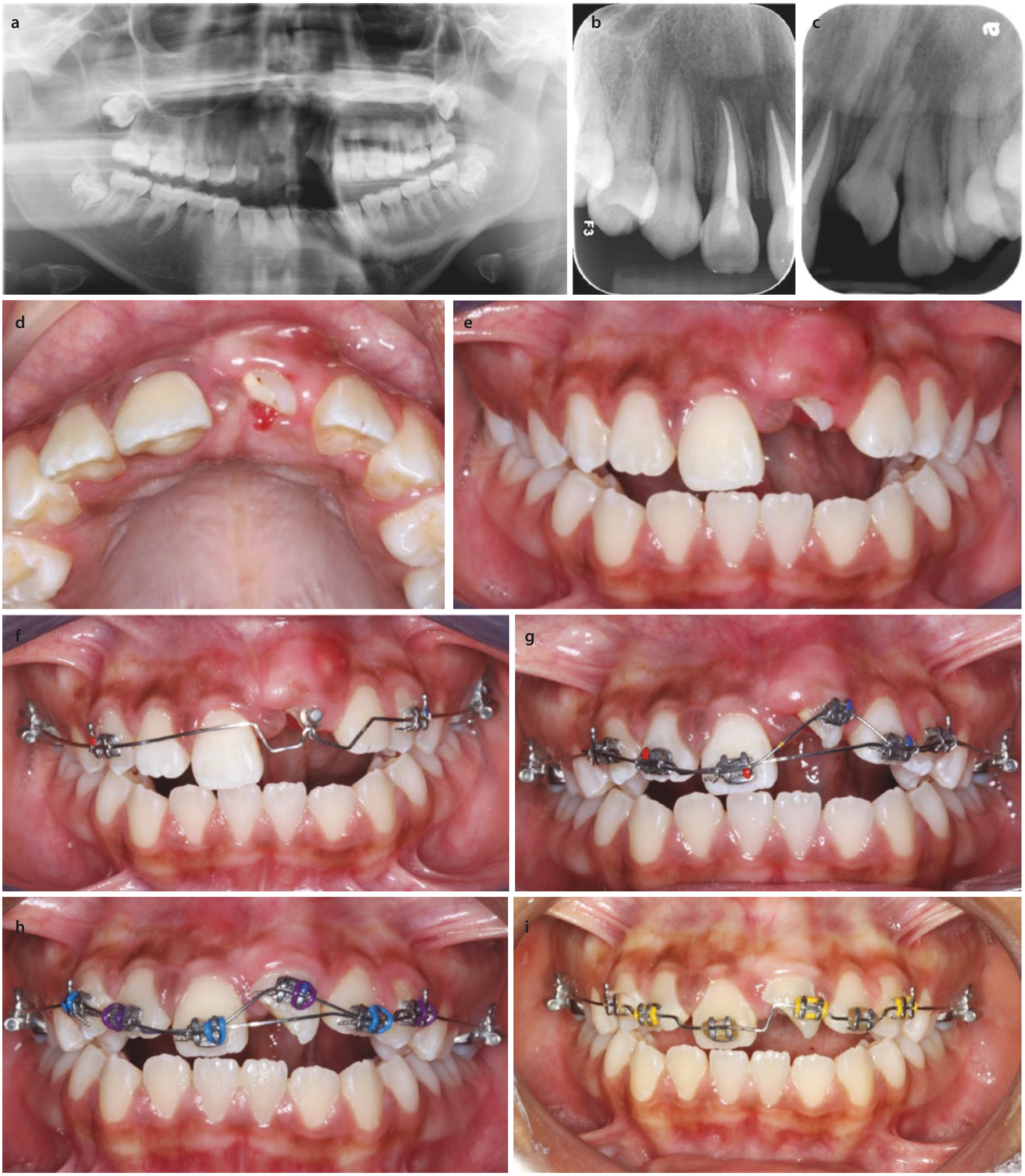



**Fig. 3.22** **a** Panoramic radiograph of an adolescent with avulsion (tooth #8), intrusive luxation, and complex crown fracture (tooth #9). **b** and **c** Periapical radiographs showing the reimplantation of tooth #8 and endodontic treatment of teeth #7 and #8. Endodontic treatment on tooth #9 was not possible due to the difficult access. **d** and **e** Frontal and occlusal views of the case before the orthodontic extrusion of tooth #9. **f** The orthodontic extrusion is initiated using a single stainless steel archwire with an extrusion "W" bend. Brackets are not placed initially on the incisors to avoid increasing the tissue damage produced by the trauma. **g** After initial extrusion, brackets are bonded in all anterior teeth. Two archwires are used: A passive stainless steel to stabilize the traumatized teeth and one auxiliary 0.16″ NiTi archwire that will continue to produce extrusive forces on the intruded tooth #9. **h** Since the crown fracture has eliminated the incisal reference, the gingival contour is used to align tooth #9 with the contralateral incisor. **i** After the desired amount of extrusion is achieved, a passive stainless steel archwire is used as a retainer which was maintained during the endodontic treatment. **j** and **k** Frontal and occlusal views of the results of the orthodontic extrusion after removal of the fixed retainer and before the esthetic restoration. **l** The final vertical position of the crown and the root parallelism can be assessed using a panoramic radiograph. **m** Soundness of the root structure was preserved during the treatment. The round apex in tooth #9 shows a stable and arrested root resorption

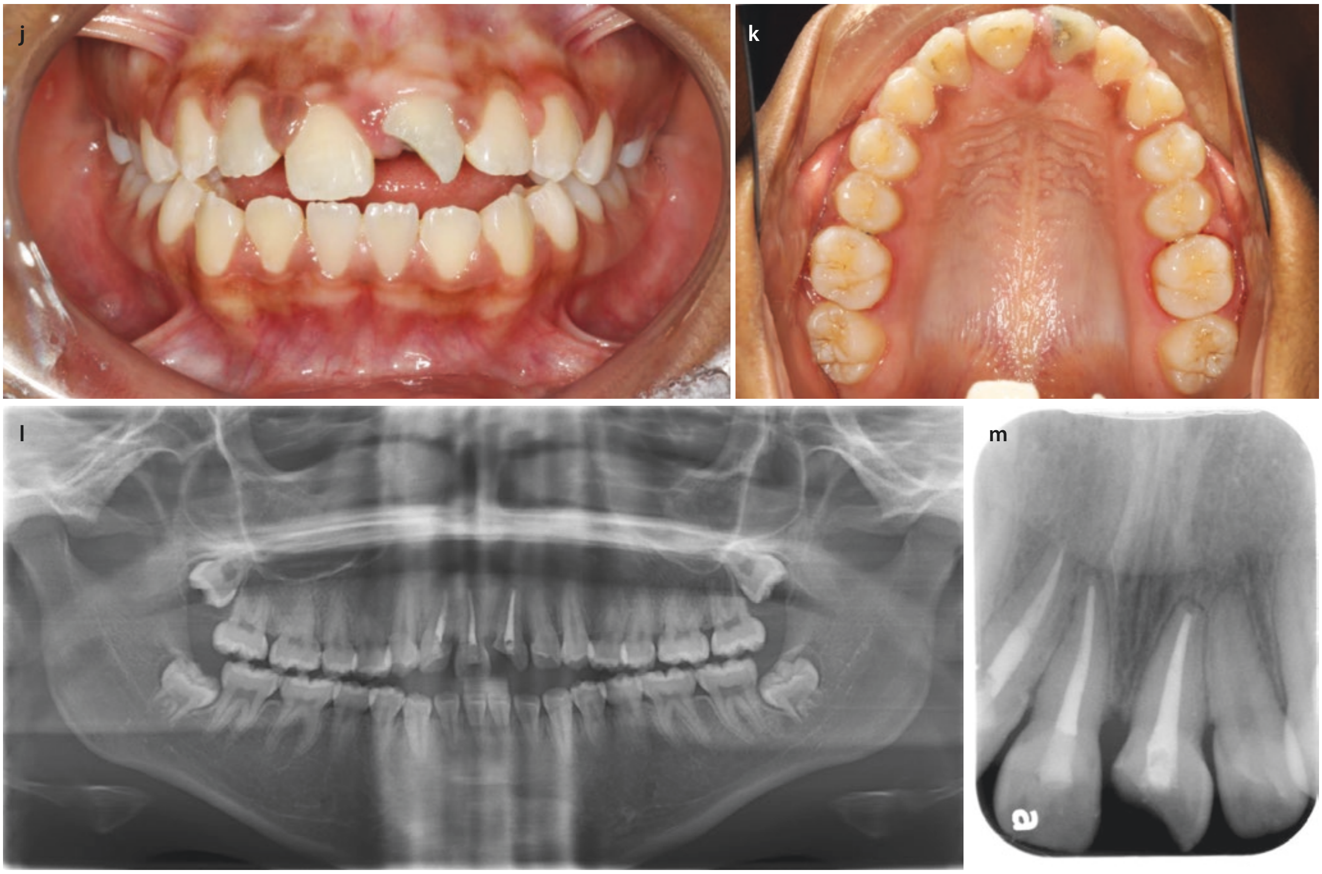



Fig. 3.22 (continued)

to canine if the lateral incisors where not affected, and it can be used as part of the anchorage.

When all incisors are affected by the trauma. The design of the appliance needs to extent to the premolar or molars to prevent the application of orthodontic forces in the incisors which has the potential to increase the inflammatory response and risk for root resorption and further pulp damage (Fig. 3.22f).

*The sequence for bonding the appliances includes*:

- Using a prophy cup eliminates the biofilm and any blood from the visible portion of the surfaces of the tooth to be bonded.
- Etch for 15–30 s the visible enamel of the intruded tooth and the adjacent teeth that will work as the anchorage. Then dry until the chalky appearance is observed in the enamel. Self-etching bonding agents can also be used to avoid the bleeding produced during the rinsing of the etching gel.
- Using filled composite, bond the brackets in the affected tooth and the teeth that will serve for anchorage. It is important to eliminate any excess of composite between the teeth and the base of the bracket to avoid undesirable movements. The brackets need to be placed in a position that will promote extrusive forces in the intruded tooth while allowing passive engagement of the archwire in the anchor teeth. Having a preset stainless steel wire with the brackets can facilitate the passive bracket positioning in the anchor teeth (*Figure*).

Since all the forces applied to an intruded tooth will generate reciprocal forces in the opposite direction in the anchor teeth, it is important to eliminate the undesired effects by using a main archwire that is rigid and passive (Fig. 3.22g). The main archwire will work as a semirigid splint to maintain the position of the unaffected teeth, while the extrusion force will be produced by the flexible wire overlaying on the main wire and only activated on the intruded tooth. A 0.014” NiTi archwire is usually used for the rapid orthodontic extrusion (Fig. 3.22h). After the NiTi wire is fully engaged in the bracket (avoid 90° bends), no further activation is needed because this wire will produce a continuous extrusive force. Adjustment in the main stainless steel wire can be made to allow for the ideal positioning of the wire and to maintain it passive. The extrusion will be normally completed at a 1 mm/week rate. After the intrusion is resolved, the tooth should be splinted and keep in retention for at least 3 months (Fig. 3.22i). The gingiva around the intruded tooth sometimes will move incisally following the extrusion, but it will self-correct since the biological width was not changed (Fig. 3.22j–m).

## Orthodontic Treatment of Crown and Crown-Root Fracture

The treatment of crown and crown-root fractures requires a multidisciplinary evaluation to determine the viability and long-term plan for the fractured tooth. The decision regarding preserving or extracting the remnant tooth or root will determine whether the orthodontic extrusion will be performed at a rapid or slow rate (◘ Fig. 3.23a–e).

When the treatment plan includes maintaining and restoring the tooth, a rapid orthodontic extrusion is indicated to bring any subgingival line of fracture occlusally which will reestablish the normal biological width and will allow to finish the restoration with supragingival borders (◘ Fig. 3.23f–i).

Clinical and radiographic assessment with periapical radiographs will help to determine the amount of extrusion

◘ **Fig. 3.23** Emergent treatment of a crown-root fracture. **a**–**e** Initial clinical and radiographic assessment shows the extension of a central incisor fracture beyond the CEJ and with pulp exposure. **f** and **g** After initial pulpotomy, the rapid orthodontic extrusion is initiated with an auxiliary NiTi archwire. **h** The extrusive force produced by the NiTi archwire is continuous; therefore, a modification is made in the passive stainless steel archwire to avoid any interference. **i**. Occlusal view of the initial endodontic access. **j** Stainless steel archwire with bends is used as a temporary retainer. **k** and **l** Final positioning of the extruded tooth. An asymmetric gingival contour due to an hyperplastic gingiva around tooth #9 can be observed. The multidisciplinary treatment plan will consider a gingivoplasty before the definitive esthetic restoration. **m** A radiographic assessment shows that the CEJ of tooth #9 is 6 mm more occlusal than the CEJ of tooth #8 confirming the rapid orthodontic extrusion


Fig. 3.23 (continued)

achieved, the location of the fracture line in reference of the crestal bone, and the crown-root ratio (Figs. 3.23j and 3.23m). For better long-term prognosis, the final crown-root ratio should be of at least 1:1.

Occlusal adjustment and coronoplasty are part of the orthodontic extrusion of fractured teeth. Every time the extrusion is achieved, the remnant crown moves toward the occlusal plane producing premature occlusal contacts. Using articulating paper, it is possible to identify and eliminate any premature contact and create enough space to continue with the extrusion of the tooth.

Although the alveolar bone does not follow the coronal movement of the tooth during the rapid orthodontic forced extrusion, it is not uncommon for the gingiva to migrate coronally during the extrusion (Figs. 3.23k and 3.23m). After obtaining the desired extrusion, a gingivoplasty procedure may be necessary to maintain a symmetrical gingival contour.

## Treatment of Extrusive Luxation

Orthodontic intrusion is mostly indicated for the delayed treatment of extrusive luxation. However, it can be an option when the tooth cannot be repositioned manually during the acute phase due to the accumulation of reparative tissue in the alveolus. The magnitude of the force and the amount of the activation needs to be carefully considered to avoid increasing the periodontal and the pulp damage that has already occurred or to disrupt the ongoing reparative process in the traumatized area. Light orthodontic intrusive forces can be applied with fixed appliances and the use of 0.014” NiTi wires with minimum activation of 1–2 mm per month.

## Orthodontic Movement of Previously Traumatized Teeth

Dental trauma unless severe, involving bleeding or fracture, for instance, goes mostly underreported. Enamel fracture or large composite restorations are sometimes the only clinical indicators of the previous history of trauma. Tooth mobility and root shortening can also be incidental findings during the clinical and radiographic assessments. Previously traumatized teeth will have an increased risk for severe root resorption during orthodontic movement. Although it is still possible to orthodontically move traumatized teeth, it will require a close radiographic monitoring during the orthodontic treatment to detect severe root resorption. When severe resorption occurs during the orthodontic treatment, it is advisable to stop any active tooth movement and to place the teeth in passive retention for 3 months. Before restarting the tooth movement, the treatment objectives should be reevaluated to balance the risk and benefits. Teeth that finish with severe root resorption should be splinted permanently to increase their longevity [45].

## References


1. Andreasen JO. Pulp and periodontal tissue repair-regeneration or tissue metaplasia after dental trauma. A review. Dent Traumatol. 2012;28:19–24.
2. Gutmann JL. Cause, incidence and prevention of trauma to the teeth. Dent Clinic N Am. 1995;39(1):1–13.
3. Andreasen JO, Andreasen FM, Andersson L. Textbook and color atlas of traumatic injuries to the teeth. 4th ed. Copenhagen: Blackwell; 2007.
4. Olson JL, Atala A, Yoo JJ. Tissue engineering: current strategies and future directions. Chronnam Med J. 2011;47:1–13.
5. Huang GT-J, Gronthos S, Shi S. Mesenchymal stem cells derived from dental tissues vs. those from other sources: their biology and role in regenerative medicine. J Dent Res. 2009;88:792–806.
6. Andreasen JO, Bakland LK, Flores MT, Andreasen FM, Andesson L. Traumatic dental injuries: a manual. 3rd ed. Oxford: Wiley-Blackwell; 2011.
7. IADT Dental Trauma Guide. www.dentaltraumaguide.org. n.d.
8. Bickley LS. Bates guide to physical examination and history taking. 11th ed. Philadelphia: Lippencott Williams & Wilkins; 2013.
9. Hupp JR, Ellis E, Tucker MR. Contemporary oral and maxillofacial surgery. 6th ed. St Louis: Elsevier; 2014.
10. Allareddy V, Nalliah RP. Epidemiology of facial fracture injuries. J Oral Maxillofac Surg. 2011;69:2613–8.
11. Gibson-Levin L. Pulp and periradicular testing. J Endod. 2013;39:s13–9.
12. WHO, World Health Organization. Application of the international classification of diseases to dentistry and stomatology ICD-DA. 3rd ed. Geneva: WHO; 1992.
13. Olsburgh S, Jacoby T, Krejci I. Crown fractures in the permanent dentition: pulpal and restorative considerations. Dent Traumatol. 2002;18:103–15.
14. Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated fracture. J Endod. 1978;4:232–7.
15. Cvek M. Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys. J Endod. 1982;8(9):391–7.
16. Marques MS, Wesselink PR, Shemesh H. Outcome of direct pulp capping with mineral trioxide aggregate: a prospective study. J Endod. 2015;41(7):1026–31.
17. Mente J. Mineral trioxide aggregate or calcium hydroxide direct pulp capping: an analysis of the clinical treatment outcomes. J Endod. 2010;36(5):806–13.
18. Tsukiboshi M. Treatment planning for traumatized teeth. 2nd ed. Hanover Park: Quintessence; 2012.
19. Gargiulo AW, Wentz FM, Orban BJ. Dimensions and relations of the dentogingival junction in humans. J Periodontol. 1961;32:261–7.
20. Cvek M, Tsilingardis G, Andreasen JO. Survival of 534 incisors after intra-alveolar root fracture in patients aged 7-17 years. Dent Traumatol. 2008;24:379–08.
21. DiAngelis AJ, Andreasen JO, Ebelseeder KA, Kenny DJ, Trope M. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of permanent teeth. Dent Traumatol. 2012;28:2–12.
22. Tsilingaridis G, Malmgren B, Andreasen J, Malmgren O. Intrusive luxation of 60 permanent incisors: a retrospective study of treatment and outcome. Dent Traumatol. 2012;28:416–22.
23. Trope M. Avulsion of permanent teeth: theory to practice. Dent Traumatol. 2011;27:281–94.
24. AAE, American Association of Endodontists. The recommended guidelines of the American Association of Endodontists for the treatment of traumatic dental injuries. AAE; 2013.
25. IADT. Guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth. Dent Traumatol. 2012;28:88–96.
26. Krasner P, Rankow H. New philosophy fir the treatment of avulsed teeth. Oral Surg Oral Med Oral Pathol. 1995;79:616.
27. Leckic P, Kenny D, Moe HK, Barrett E, McCullough CAG. Relationship of clonogenic capacity to plating efficiency and vital dye staining of human periodontal ligament cells: implications for tooth replantation. J Periodontal Res. 1996;31:294–300.
28. Lekic PC, Kenny DJ, Barrett EJ. Influence of storage conditions on the clonogenic capacity of periodontal ligament cells: implications for tooth replantation. Int Endod J. 1998;31:137–40.
29. McCullough CAG. Origins and functions of cells essential for periodontal repair: the role of fibroblasts in tissue homeostasis. Oral Dis. 1995;1:271–8.
30. Lin DG, Kenny DJ, Barrett EJ, Leckic P, McCullough CAG. Storage conditions of avulsed teeth affect the phenotype of cultured human periodontal ligament cells. J Periodontal Res. 2000;35:42–50.
31. Blomlof L. Milk and saliva as possible storage media for traumatically exarticulated teeth prior to replantation. Swed Dent J Suppl. 1981;8:1–26.
32. Andreasen JO, Barrett EJ, Kenny DJ. Is anti-resorptive regenerative therapy working in case of replantation of avulsed tooth. Dent Traumatol. 2005;6:344–6.
33. Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed permanent incisors: factors related to periodontal ligament healing. Endod Dent Traumatol. 1995;11(2):76–89.
34. Panzarini SR, Gulinelli JL, Poi WR, Sonoda CK, Pedrini D, Brandini DA. Treatment of root surface in delayed tooth replantation: a review of the literature. Dent Traumatol. 2008;24:277–82.
35. Blomlof L, Otteskog P, Hammarsrom L. Effect of storage in media with different ion strengths and osmolalities on human periodontal ligament cells. Scand J Dent Res. 1981;89:180–7.
36. Souza BDM, Luckemeyer DD, Felippe WT, Simoes CMO, Felippe MCS. Effect of temperature and storage media on human periodontal ligament fibroblast viability. Dent Traumatol. 2010;26:271–5.
37. Wang JW, Zhao YM, Feng XY, Jia WQ, Ge LH. Effect of skimmed pasteurized milk and Hank's balanced salt solution on viability and osteogenic differentiation on human periodontal ligament stem cells. Dent Traumatol. 2013;29:365–71.
38. Jacoby W, Pastan I. Cell culture. In: Colwick S, Kaplan N, editors. Methods in enzymology. San Diego: Academic Press; 1979.
39. Andersson L, Malmgren B. The problem of dentoalveolar ankylosis and subsequent replacement resorption in the growing patient. Aust Endod J. 1999;25:57–61.
40. Ngassapa DN, Freihofer HP, Maltha JC. The reaction of the periodontium to different types of splints. (I). Int J Oral Maxillofac Surg. 1986;15:240–9.
41. Andreasen JO. Adhesive dentistry applied to the treatment of traumatic dental injuries. Oper Dent. 2001;26:328–35.
42. Kademani D, Tiwana PS. Atlas of oral and maxillofacial surgery. 1st ed. St Louis: Elsevier; 2016.
43. Thiruvenkatachari B, Harrison J, Worthington H, O'Brien K. Early orthodontic treatment for class II malocclusion reduces the chance of incisal trauma: results of a Cochrane systematic review. Am J Orthod Dentofac Orthop. 2015;148(1):47–59.
44. Fields HW, Christensen JR. Orthodontic procedures after trauma. Pediatr Dent. 2013;35(2):175–83.
45. Owtad P, Papademetriou M, Park JH. Management guidelines for traumatically injured teeth during orthodontic treatment. J Clin Pediatr Dent. 2015;39(3):292–6.

# Role of Endodontics in Dental Trauma

*Scott Sutter and Kristine Knoll*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_4

4

## 4.1 Introduction

Endodontics plays a crucial role in the diagnosis, treatment, timing, and restoration of traumatized teeth. The primary goal of the clinician is to maintain pulp vitality and provide the patient with a functional tooth. This chapter will provide the clinician with the tools to make a proper pulpal diagnosis and provide treatment in a timely manner to obtain the best possible outcome for the patient. In addition, special care will be given to emerging topics in vital and non-vital pulpal therapy, as well as diagnosis and treatment of the resorptive sequelae that can occur following trauma.

## 4.2 Diagnosis

Multiple changes occur to the pulp and periodontal ligament (PDL) following trauma. One of the most difficult aspects of managing traumatic injuries is establishing a correct diagnosis prior to providing definitive treatment [1]. A systematic approach is presented which encourages the practitioner to perform a thorough history and radiographic and clinical examination and to use multiple methods of pulpal vitality testing to make an accurate diagnosis.

**Steps to Proper Pulpal Diagnosis in Traumatic Injuries**

- History
  - Present trauma
  - Previous trauma
- Radiographic
  - Multiple angles of periapical radiographs
- Clinical examination
  - Pulp tests may be unreliable close to the time of trauma.

### 4.2.1 History

Several questions should be asked prior to proceeding with the radiographic and clinical examination to narrow down the affected area or tooth. Following a thorough medical history, the next questions should focus on the time of the injury, where the injury took place, and what was done at that time. For example, the treatment is very different if the patient fell and concussed their tooth outside of your office than if they fractured the tooth 3 months prior. The energy, shape, and resiliency of the impacting force also determine the type of injury. If the surface is resilient and there is a low-velocity impact, such as when the patient has collided with another player and hit his or her mouth, there will probably be multiple teeth affected. A hard surface with a high velocity, such as a baseball hitting the oral cavity, will usually result in a single tooth injury. In addition to the number of teeth affected, harder surfaces will usually result in a focused force on a smaller area, and the clinician should suspect a higher likelihood of crown or root fractures. However, if the lip cushioned the impact, or if the surface was resilient, one would look for luxation or avulsions of multiple teeth [2].

Age also plays a role in the etiology and outcome of traumatic dental injuries. Younger patients have less dense bone, and the clinician is more likely to see avulsions or PDL injuries. Older patients have higher density in their bone, and as such, their teeth or roots tend to fracture [2]. Most dental trauma occurs in the 7- to 12-year-old age group and is primarily caused by falls or accidents at home or school [3]; however serious accidents can affect all ages.

### 4.2.2 Radiographic Examination

Radiographs are invaluable in the assessment of dental trauma, as they can reveal root fractures, bone fractures, tooth displacement, foreign body impactions, resorption, and other pathologies. One radiograph is not sufficient to adequately diagnose the condition of an area following dental trauma. The International Association of Dental Traumatology (IADT) recommends at least 3–4 radiographs be taken following trauma, including multiple angled periapical images (PA), a straight on image, and an occlusal film [4].

In addition to traditional radiography, some authors recommend the use of cone beam computed tomography (CBCT) following traumatic injuries to further increase the accuracy of the diagnosis [5] (Fig. 4.1). CBCT imaging allows for three-dimensional viewing of an area and is very useful in diagnosing the location of resorption or presence of bone fractures.

If lacerations are present and sections of the traumatized tooth are missing, it is advisable to take a soft tissue radiograph of the lacerated area prior to suturing to ensure there is no foreign body present in the soft tissue. To perform this, place a normal digital sensor over the lacerated area and use a reduced kilovoltage to expose the area. Cone beam computed tomography has also been shown to be useful in identifying fragments located in the soft tissue [6]. These techniques should allow the clinician to visualize the presence or absence of multiple foreign bodies, including missing tooth fragments (Fig. 4.2).

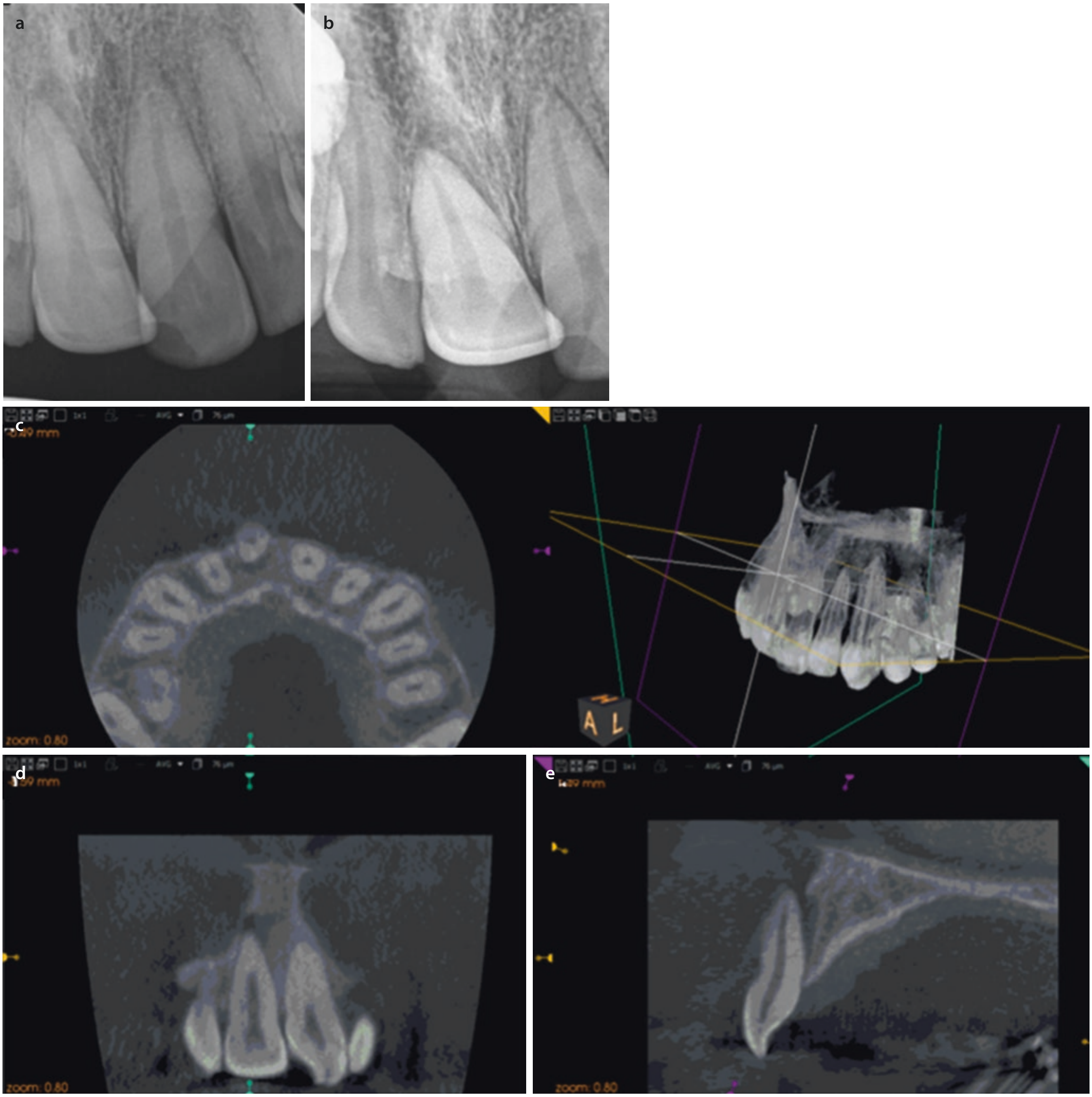



**Fig. 4.1** **a**, **b** Pre-operative images of lateral luxation on tooth #8 and uncomplicated crown fracture on tooth #9. Soft-ware imaging from Kodak depicting the CBCT axial **c**, sagital **d**, and coronal **e** orthogonal planes of tooth #8, which aided in gathering more diagnostic information for ideal treatment planning. (Courtesy of Kristine Knoll, DDS, MS)

### 4.2.3 Clinical Examination

The reader is directed to ► Chap. 3 for a full description of the clinical examination following trauma, but some discussion on the changes in the pulpal and periapical responses to testing is warranted to further the understanding of the results.

It should be remembered that vitality testing is not a test of the vitality of the tooth; rather it is a response to a stimulus and does not indicate the presence or absence of a blood supply. As such, teeth that initially respond positively may not retain their vitality with time, and conversely, teeth that respond with a negative test may not be necrotic. It may take

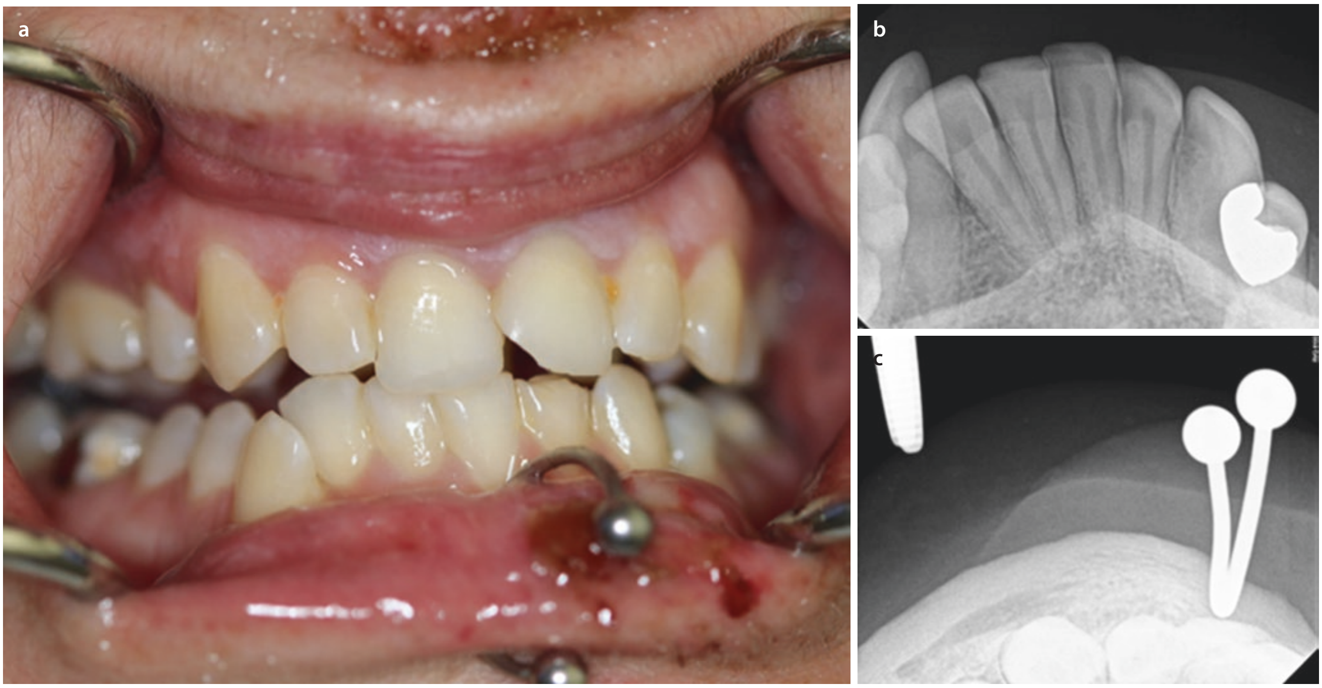



**Fig. 4.2** **a** Intra-oral photograph of lateral luxation with crown infraction on tooth #8, subluxation with uncomplicated crown fracture on tooth #9, cutaneous lacerations in philtrum region, and vermillion lacerations on mandibular lip. Occlusal films of mandibular anterior **b**, **c** teeth taken to rule out presence of foreign body. (Courtesy of Kristine Knoll, DDS, MS)

up to 9 months for the blood supply to return to normal in a traumatized tooth, and the importance of follow-up cannot be stressed enough [7]. Laser Doppler flowmetry and pulse oximetry have both been shown to aid in the diagnosis of the pulpal vitality rather than sensitivity [8]; however the disadvantages include the cost of these instruments and reproducibility, as the absorbed light may be affected by restorations or height of the pulp horns (Fig. 4.3).

## 4.3 Clinical Management of Traumatic Injuries

Following a definitive diagnosis, it is important to provide the correct treatment to allow the patient to return to function on the tooth for an extended period of time.

### 4.3.1 Crown Fractures Classification and Pulpal Treatment

The majority of crown fractures occur in caries-free healthy teeth [9, 10]. As such, maintaining the pulp vitality is essential.

#### 4.3.1.1 Crown Infraction

Crown infractions are incomplete fractures involving the enamel, without loss of any tooth structure [1]. The risk for pulpal necrosis is low following these types of injuries.

#### 4.3.1.2 Uncomplicated Crown Fractures

Uncomplicated crown fractures are when the fracture involves either the enamel or dentin but does not involve the pulp. The patient will usually present with sensitivity to cold but otherwise normal pulp vitality testing. Soft tissue radiographs should be taken in order to locate any missing fragments if lacerations are present [4]. If the fragment is available, bonding it to the tooth has a good long-term success rate with an acceptable esthetic result [11]. In cases where the tooth fragment is not available, conventional restorative dentistry using bonded composite or ceramic crowns should act as the definitive treatment. If the dentinal fracture is close to the pulp (within 0.5 mm), a base with hard-set calcium hydroxide is recommended to decrease the reactive inflammation associated with bonding agents [12–14]. More recent studies have also recommended the use of mineral trioxide aggregate (MTA) [15, 16] or other bioceramic materials, Biodentine, along with a resin modified glass ionomer (RMGI) [17], as indirect pulp capping materials.

#### 4.3.1.3 Complicated Crown Fractures

A complicated crown fracture is when the fracture involves the enamel, dentin, and pulp. The most important factor in the treatment of this injury is the time since the trauma. If left untreated, these teeth will always progress to pulpal necrosis due to the presence of bacteria in the oral cavity [18]. Bacteria are not expected to travel more than 2 mm apically within the first 48 h after a pulpal exposure [19], which gives the clinician many different treatment options to maintain pulp vitality, depending on the level of development of the root and the size


**Fig. 4.3** **a** Nellcor OxiMax N-65 pulse oximeter; after removal of the sedation external finger cover, the individual sensors can be placed on the tooth in question. **b** White sensor transmits both infrared and red light wavelengths, which is absorbed by the tissue and the amount depends on the ratio of oxygenated and deoxygenated hemoglobin in the blood. The black sensor detects the absorbed light, which can also be affected by restorations or pulp horn heights (Nellcor Puritan Bennett, Boulder, CO; now part of Covidien). (Courtesy of Scott B. McClanahan, DDS, MS and the *Endodontic Department at the University of Minnesota*)

of the exposure. In cases where the root has not completed development, the goal is to maintain pulpal vitality to ensure continued root formation and increased thickening of the dentinal walls, which reduce the chance of fracture, in a process called apexogenesis [20]. These procedures include pulp capping, indirect or direct, and pulpotomies. The severity of removing the pulp in a mature root is much less than that of an immature tooth due to the high success rates of pulpectomy [21] and traditional root canal therapy [22]. However, vital pulp therapy has been shown to be a successful procedure in mature teeth [23], especially in younger patients [24].

**Vital Pulp Therapies Used in Dental Trauma**

1. Indirect pulp cap
2. Direct pulp cap
3. Cvek type pulpotomy
4. Apexogenesis

Once the pulp has been addressed, the tooth is restored in the same manner as the uncomplicated crown fracture (Fig. 4.4).

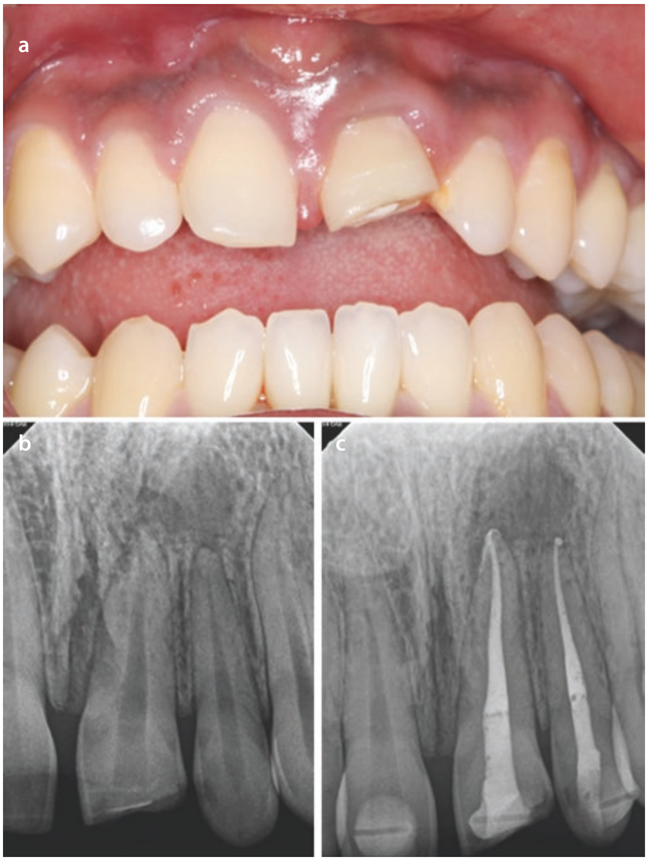



Fig. 4.4 Pre-operative image **b** of tooth #9, with history of complicated crown fracture **a** and previously initated therapy. Endodontic therapy was treated in multiple visits **c**. (Courtesy of Chris Saylor, DDS, MS)

#### 4.3.1.4 Pulp Capping (Vital Pulp Therapy)

When a vital and asymptomatic pulp becomes exposed to the external environment, the least invasive procedure is the placement of a pulp capping material directly onto the surface of the exposed pulp. The procedure is as follows:

After anesthesia and placement of a dental dam, the exposed pulp tissue should be gently rinsed with diluted (1.5%) sodium hypochlorite (NaOCl). A sterile cotton pellet soaked in NaOCl is then placed over the exposure for 5 min. Bleeding should be assessed at this point, if it is controlled by a moist cotton pellet, placement of a capping material can then occur. If the bleeding is still significant, a pulpotomy is recommended. Acceptable pulp capping materials are MTA or other bioceramic materials. Calcium hydroxide was historically used as a capping material but has been shown to have a higher failure rate. As such, its use should be curtailed in favor of more modern materials [23]. Both the original gray MTA and white MTA have been associated with staining, so in cases where there is an esthetic concern, newer bioceramic materials such as Biodentine should be utilized [25]. A final restoration is then placed over the bioceramic.

When performed under a sterile field using a rubber dam and modern bioceramic materials, direct pulp caps have a very high success rate [23]. Factors that decrease the long-term success include amount of time the tooth has been exposed to the oral environment, use of non-biocompatible materials as a pulp capping material (calcium hydroxide, glass ionomer, composites), nonmechanical pulpal exposures, and lack of an adequate coronal seal [26].

#### 4.3.1.5 Cvek Pulpotomy (Vital Pulp Therapy)

A pulpotomy is the surgical removal of the coronal portion of vital pulp tissue that is then covered by a biocompatible material and restoration. A variation of this procedure is called a Cvek pulpotomy, which is used specifically for traumatically exposed pulps. In this procedure, a smaller portion of the inflamed pulp tissue is removed compared to the full pulpotomy. The long-term success of this procedure is high and should be the first procedure attempted in fracture cases with higher levels of inflammation [27].

In this procedure, the patient is anesthetized, and a dental dam is placed. The exposed pulp tissue is then removed using a sterile bur or endodontic spoon to the level of the cementoenamel junction (CEJ). The tissue is then gently irrigated with diluted 1.5% NaOCl, and a cotton pellet soaked in NaOCl is placed over the exposed site for 5 min. A bioceramic material is placed over the remaining pulp. The aforementioned esthetic concerns should be taken into account. A final restoration is then placed over the pulp capping material.

#### 4.3.1.6 Apexogenesis (Vital Pulp Therapy)

Apexogenesis is a form of a regenerative procedure where the goal is the preservation of vital radicular pulp tissue to encourage continued root development with apical closure [28]. This procedure is performed on teeth with vital (or inflamed) pulps with open apices.

After anesthesia and rubber dam isolation, the tooth is accessed, and the majority of the coronal pulp tissue is removed to the level of the canal orifices using a high-speed diamond bur with water-cooling. [29]. The chamber should be rinsed with sterile saline or water to remove debris, and then the chamber is dried using cotton pellets. Once the bleeding is controlled, calcium hydroxide or MTA is placed over the canals, and a long-term coronal restoration is placed. Follow-up should occur every 3 months to ensure successful root formation and no signs of pulp necrosis, resorption, or periapical pathosis.

Once the roots have finished development, the operator can then continue monitoring the tooth or can elect to perform conventional root canal therapy. Although there is some controversy over which approach is superior, the inability to determine pulp vitality and the ability to initiate the endodontic therapy prior to the development of root resorption, canal obliteration, or the development of apical periodontitis should result in a higher long-term success rate [21, 30].

#### 4.3.1.7 Crown-Root Fractures

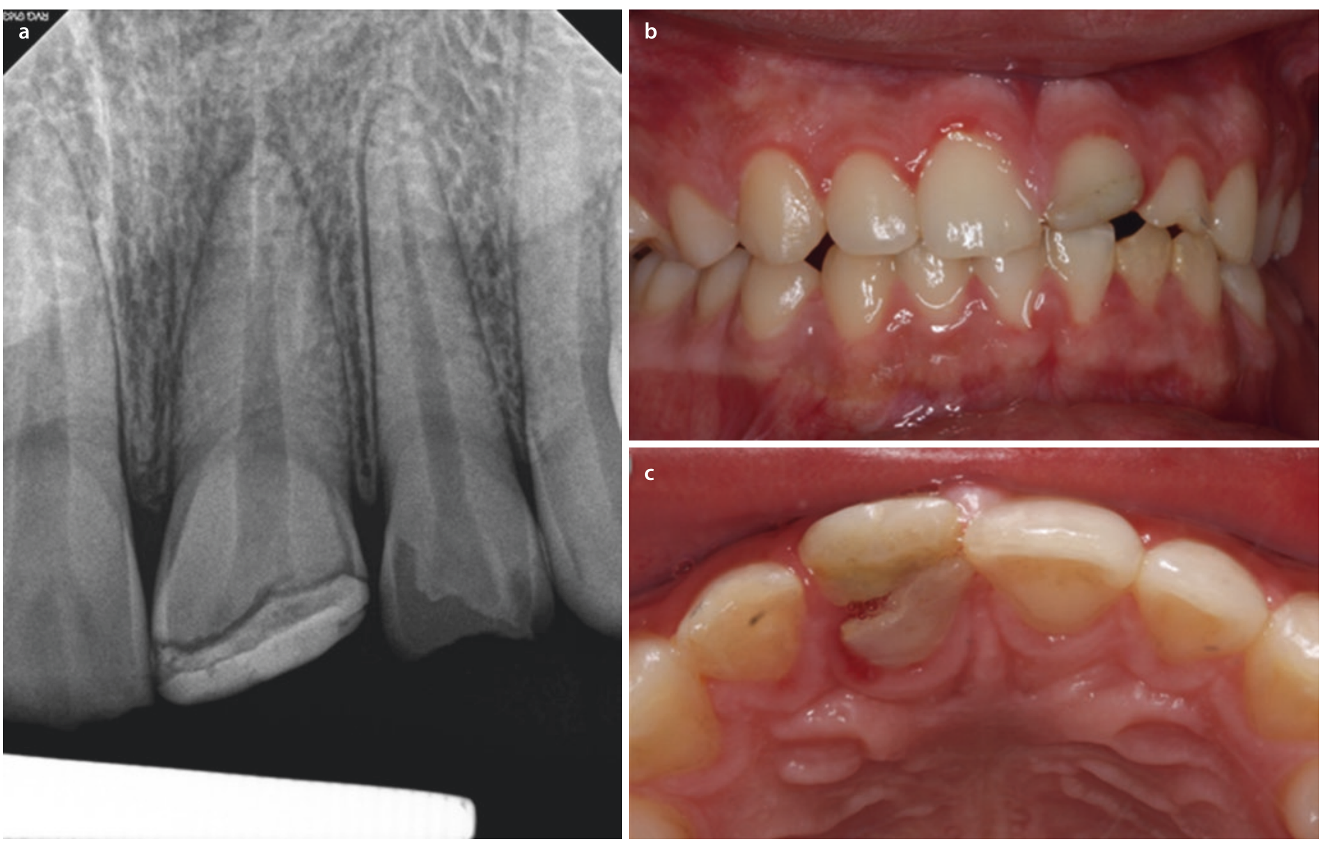
A crown-root fracture is when the fracture involves the enamel, cementum, dentin, and sometimes the pulp. The apical location of the fracture should be confirmed using multiple periapical radiographs or CBCT imaging and influences the prognosis of the tooth. Emergency treatment includes stabilization of the fractured segment and either placement of a pulp cap (◘ Fig. 4.5), pulpotomy, or root canal therapy depending on the stage of development and size of the exposure. Final treatment will depend on the location of the fracture and can include surgical or orthodontic extrusion, gingivectomy with crown lengthening, decoronation, or extraction [4] (◘ Fig. 4.6) (see ▶ Chap. 3 for details of decoronation treatment).

#### 4.3.1.8 Root Fractures

A root fracture involves the fracture of cementum, dentin, and pulp and is most commonly seen in mature teeth with closed apices [31]. In certain cases, the coronal fragment will be displaced coronally and will be mobile. This displacement disrupts the pulpal blood circulation and causes pulpal necrosis in the displaced fragment in approximately 25% of cases [32, 33]. The apical fragment is rarely affected. Multiple radiographs are necessary to visualize the fracture line on traditional periapical films. Occlusal films and CBCT imaging are also useful to localize the fracture site.

Treatment for root fractures begins by repositioning the fragments as close as possible to their original position using radiographic imaging. Once the fragments are repositioned, a flexible splint should be placed for 4 weeks [34]. In cases where the fracture is located in the coronal region of the root, the splint can be left for up to 4 months [4].

There are four different responses to root fractures that have been described in the literature: healing with hard tissue, healing with connective tissue, healing with the bone and connective tissue, and interposition of granulation tissue [35, 36]. The proximity of the two fragments influences what type of material is formed between the apical and coronal sections [37]. The first three are considered successful outcomes and should be monitored closely to ensure proper healing is achieved. Coronal pulp obliteration is possible and can result in yellowing of the tooth [38]. In cases where the coronal fragment becomes infected, the bacterial by-products cause an inflammatory response which forms a radiolucency at the fracture line [35]. If the fragment is stable, root canal therapy on the coronal fragment alone should be sufficient.




◘ **Fig. 4.5** Pre-operative image **a** of tooth #9, with history of complicated crown-root fracture and mobility of coronal segment **b**, **c**. Endodontic therapy was recommended, as well as possible crown lengthening after prosthodontics consultation. (Courtesy of Kristine Knoll, DDS, MS)

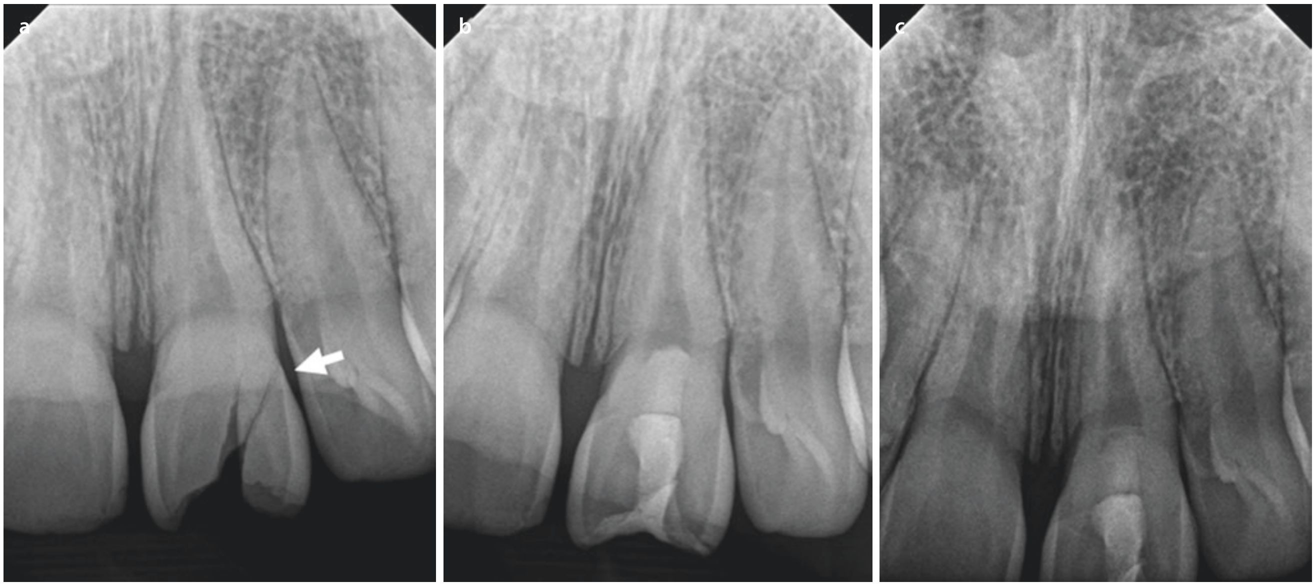



**Fig. 4.6** Pre-operative image **a** of tooth #9, with complicated crown-root fracture (*white arrow*). Endodontic therapy was completed in multiple visits **b**, and pt is asymptomatic at 6 month recall show **c**. (Courtesy of Chris Saylor, DDS, MS)

The size of the canal at the apical extent of the coronal segment is often quite large, and thus apexification with long-term calcium hydroxide treatment or placement of an MTA is indicated. In cases where both segments have become infected, surgical removal of the apical portion is suggested in addition to the aforementioned treatment of the coronal segment.

## 4.3.2 Luxation Injuries

Luxations occur when the tooth is traumatized and sometimes moved from its normal location in the socket. These include the following types of injuries: concussion, subluxation, extrusive luxation, lateral luxation, avulsion, and intrusive luxation. The reader is directed to ▶ Chap. 3 for specific definitions of the different types of luxation injuries.

### 4.3.2.1 Concussions and Subluxations

Teeth that have concussion or subluxation injuries generally do not require any treatment beyond vitality testing and monitoring. It should be noted that these teeth can have irregular responses to vitality testing, and the responses should be noted and compared to testing at later dates [39, 40].

### 4.3.2.2 Extrusive Luxations

Extrusive luxation is a severe type of injury and requires immediate treatment. These teeth should be repositioned into the original site as soon as possible. Extrusive luxations occur when the tooth is partially displaced out of the socket or in a coronal direction. The patient will note occlusal changes, and severe mobility is possible. Following local anesthesia, the exposed surfaces of the tooth should be cleaned using saline or chlorhexidine, the tooth repositioned using digital pressure, and a flexible splint applied for 2 weeks [4].

### 4.3.2.3 Lateral Luxations

Lateral luxation is also a severe type of injury, which requires immediate treatment. Lateral luxations occur when the tooth has been laterally repositioned, usually with a concomitant alveolar process fracture. The tooth is often nonmobile due to the displacement of the tooth into the bone. For these luxations, the treatment of choice is to apply local anesthesia and reposition the tooth out of the bony lock using digital pressure or by using forceps. Once the location is verified using clinical and radiographic evidence, the tooth should be splinted for 4 weeks using a flexible splinting material [4] (Fig. 4.7).

### 4.3.2.4 Avulsions

Avulsions occur when the tooth is completely displaced out of the socket and results in attachment damage due to the tearing of the periodontal ligament and possible crushing of the cementum. If the ligament does not dry out and is still attached to the root surface, the outcome is generally favorable [41, 42]. In cases where there is a large amount of damage, the area affected must be repaired by new tissue. In many cases, the cementoblasts are not able to mobilize as quickly as the osteoclasts present in the bony socket, and a common response in these cases is replacement of the root by the bone [43, 44]. This is described as replacement resorption and will be discussed in ▶ Sect. 4.4.4.

Treatment of this injury depends on the stage of development of the root and the speed at which the tooth is replanted [42].

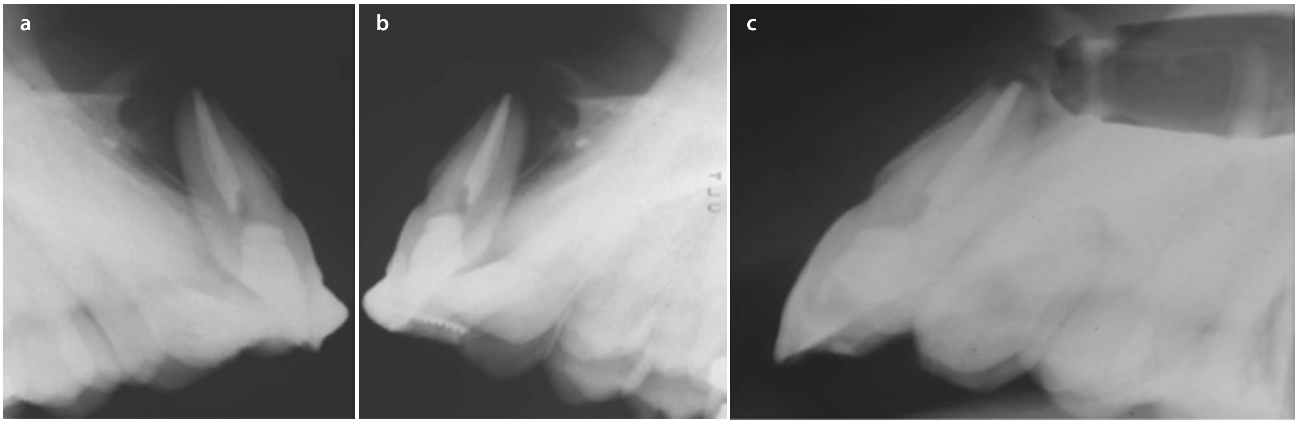



Fig. 4.7 Pre-operative images **a**, **b** of tooth #9, with lateral luxation and a concomitant alveolar process fracture. Post-operative image **c** after reduction. (Courtesy of Scott B. McClanahan, DDS, MS)

#### 4.3.2.5 Avulsion Immature Root

Avulsions that occur while the roots are still developing have a better chance for revascularization to occur than those whose roots have closed [45]. Root canal therapy should be delayed unless definitive signs of pathosis occur. When dealing with avulsions, the time the tooth has been exposed to the external environment is the most critical factor in the prognosis and treatment.

Ideally, the tooth will have been replanted immediately at the site of the accident. When the patient arrives at the office, the area should be inspected for proper position of the tooth, both visually and radiographically. A flexible splint should be applied and left in place for 1–2 weeks. Systemic antibiotic coverage with either tetracycline (if permanent tooth development is completed and there is no risk of staining) or amoxicillin (if permanent tooth buds are still growing) is recommended, along with verifying tetanus coverage and referring to the patient's physician as needed. The patient should avoid any contact sports, should be on a soft food diet for 2 weeks, and should rinse with chlorhexidine bid for 1 week. Follow-up includes radiographic and clinical examination at 2 weeks, 4 weeks, 3 months, 6 months, and yearly to verify continued development of the root and monitor for possible pulpal necrosis or resorption [46] (Figs. 4.8 and 4.9).

In cases where the extraoral time is over 60 min, it is presumed that the periodontal ligament is no longer viable [47] (Fig. 4.9), and different surface treatments should be utilized to reduce the chance of resorption and increase the success of the replantation. Minocycline and doxycycline soaks prior to replantation have been shown in animal studies to increase the chance of revascularization [45, 48]. The tooth should then be gently rinsed and replanted, with splinting and follow-up instructions the same as if the tooth was replanted at the site [46].

#### 4.3.2.6 Avulsion Mature Root

In a mature root, it is presumed that there is no chance for revascularization [48]. However, if the extraoral dry time is under 60 min, there is a chance for the PDL cells to remain viable. Endodontic treatment should be initiated within 7–10 days, utilizing an interappointment medicament for 2–4 weeks such as calcium hydroxide to reduce the incidence of resorption [49]. Although not available in the United States at this time, a combination of triamcinolone and demeclocycline has been utilized as an intracanal medicament for immediate treatment of avulsed teeth with high success rates [50]. Treatment for this type of injury is then identical to that of the avulsed tooth with an open apex.

If the avulsed tooth has been dry for greater than 60 min, it is assumed that the PDL has no viable cells and no regeneration is possible. The goal of treatment in these cases is to preserve the tooth in function while maintaining the alveolar contour with the knowledge that the ultimate fate of this tooth is ankylosis and resorption [46]. The final restoration in these cases will often be an implant or a fixed partial denture.

#### 4.3.2.7 Intrusive Luxation

Intrusion of a tooth occurs when the tooth is forced into the socket. Intruded teeth will appear to be shorter than the surrounding teeth, are nonmobile, and will give an ankylosed metallic sound upon percussive tests. Intrusions are the most severe of the luxations due to the crushing of the blood supply and periodontal ligament. Treatment of these injuries depends on the stage of development of the root and the level of intrusion. In cases of incomplete root development and the intrusion is less than 7 mm, the tooth can be monitored for spontaneous eruption [4, 51]. In teeth with immature roots but over 7 mm of intrusion, orthodontic or surgical extrusion is recommended due to the severity of the intrusion.

If intrusion occurs in mature roots, root canal therapy should be initiated within 3–4 weeks after the trauma regardless of the level of intrusion due to the crushing of the blood supply [4, 51]. If the intrusion is minor (<3 mm), the clinician is advised to monitor the tooth for eruption. More severe intrusions of 3–7 mm should be orthodontically or surgically repositioned, and intrusions of greater than 7 mm should be surgically repositioned [4, 51, 52].

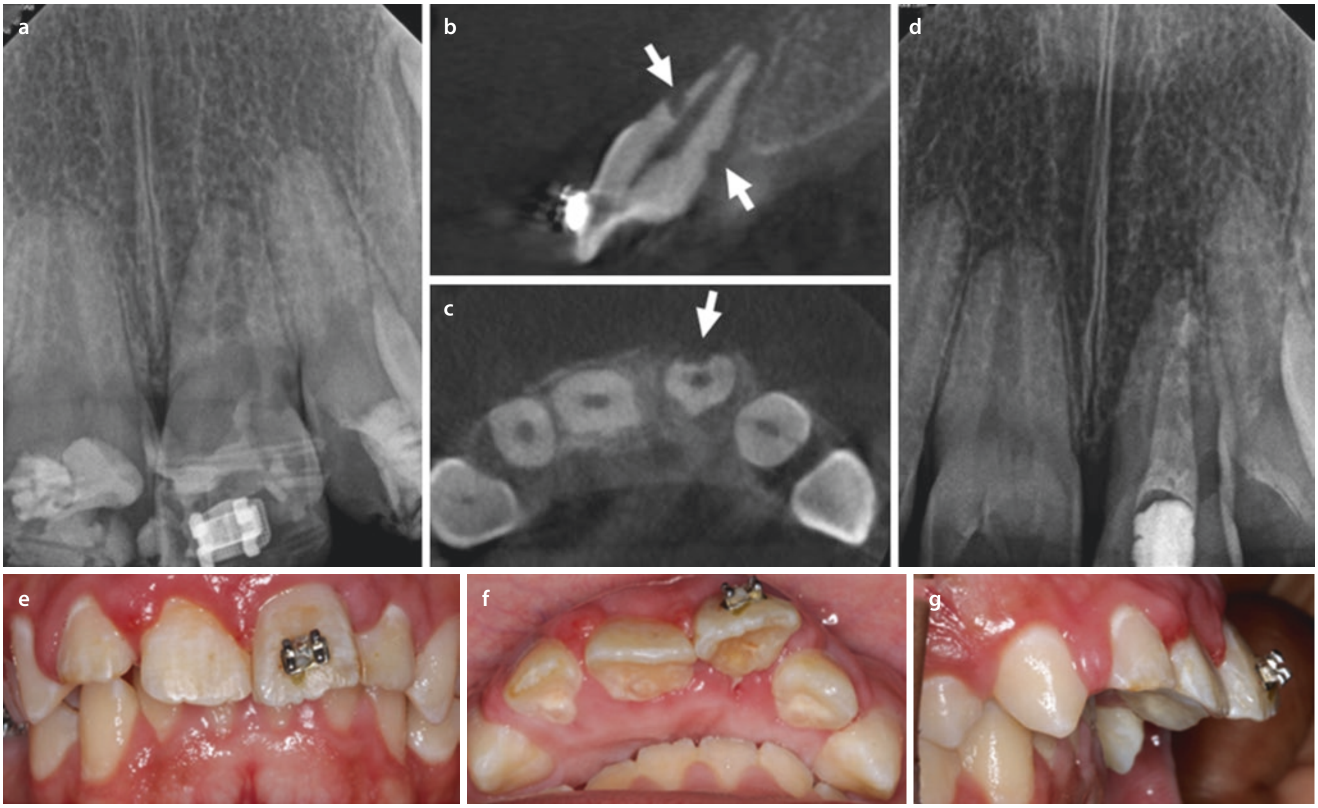



**Fig. 4.8** Pre-operative image **a** of tooth #9, with history of recent avulsion and rigid splint placement at a hospital **e**–**g**. EODT was <60 min. CBCT coronal **b** and axial **c** orthogonal planes showing presence of external resorption (*white arrows*), consistent with disclosure of previous traumas to area in past. Endodontic therapy was recommended and completed in multiple visits **d**. (Courtesy of Tyler Schuurmans, DDS, MS)

## 4.4 Resorption

Normal physiological root resorption occurs during the event of exfoliation in the primary dentition, whereas root resorption in the permanent dentition is the result of a pathological event, which may cause a progressive loss of tooth structure and could lead to premature loss. In order for pathological resorption to occur, two events must happen:

1. There is injury that removes or alters the protective covering of the dentin, the cementum on the external surface, or the odontoblast layer on the internal surface, which allows clastic cells from the circulatory system to access the dentin [53, 54].
2. Inflammation, from the pulp or periodontal tissue, must occur to the site of the unprotected root surface [55].

The primary etiology of injury to the non-mineralized, protective predentin or pre-cementum layers is from trauma [56], although heat from restorative procedures [57], excessive forces during orthodontics [58], and improper use of internal bleaching materials [59] have also been associated. The primary etiology of inflammation is from intrapulpal or periodontal infections, where adequate removal of bacteria can arrest the resorption process [60].

Although there is no universal classification system for resorption, generally it is classified by two broad categories, internal or external and the location, apical or cervical, for example. Resorption associated with a radiolucent pathosis is referred to as inflammatory. Often a sequela to avulsion is replacement resorption, where the cementum and dentin of the root is replaced by the bone. Schwartz recently classified pathologic resorption into four basic types [61].

**Schwartz Classification of Dental Resorption**

1. Internal resorption
2. Invasive cervical resorption
3. External inflammatory resorption
4. Replacement resorption

### 4.4.1 Internal Resorption (IR)

Internal resorption originates from the loss of the protective odontoblastic layer and predentin, either from trauma or an unknown factor, which allows for direct access to the dentin [62]. This process may be transient or may become progressive from continued stimulation of bacteria from the infected, necrotic pulp tissue coronal to the resorptive lesion [62]. This process requires vital tissue; although coronal to the resorption defect it may be necrotic and infected, the rest of the pulp tissue must remain vital in order for the resorption process to continue.

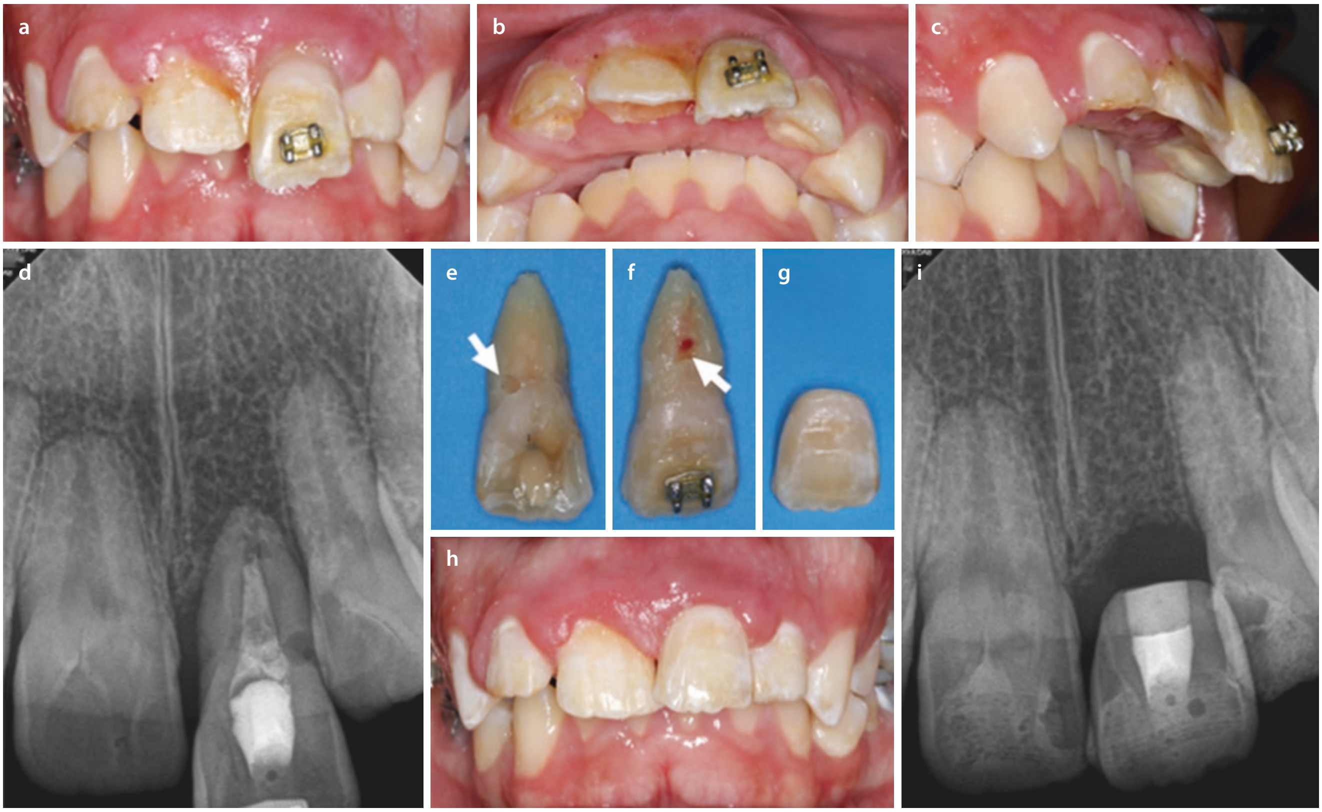


**Fig. 4.9** Three months post-op, pt reported to dental office for a second avulsion of tooth #9 with EODT >180 min **a**–**c**. Pre-operative image **d** revealed marked incisal edge discrepancy and socket seen clinically as well with severe Class III mobility. An esthetic ribbon bridge was recommended, utilizing tooth #9 as a pontic **g**–**i**, note the external resorption on the lingual **e** and buccal aspect **f** (*white arrows*). (Courtesy of Tyler Schuurmans, DDS, MS)

Recognition of internal resorption is either during routine radiographic examination or clinically, if the resorption becomes large that perforation occurs and the patient develops symptoms. Radiographically, this resorption pattern produces a sharp outline, and the canal cannot be traced through the lesion [63]. Teeth are usually asymptomatic, but if symptoms do arise, it is likely related from the existing pulp condition and not the resorptive process itself [64].

The treatment of choice for non-perforating internal resorption is conventional root canal therapy and for perforating internal resorption extraction, nonsurgical or surgical repair [65]. The goal is to deprive the resorptive cells from the pulpal blood supply, whereby performing conventional endodontic treatment cuts off the nutrients available to these resorbing cells. Calcium hydroxide should be utilized as an interappointment medicament to aid in the removal of tissue from the irregular defect or used long-term for remineralizing perforated defects [65]. The use of ultrasonics with irrigation [66] can chemically debride the tissue in these irregular defects, and obturation using a thermoplasticized gutta-percha injection technique, such as Obtura (Coltene/Whaledent, Cuyahoga Falls, OH), can be utilized to fill the irregularities [67] (Fig. 4.10).

### 4.4.2 Invasive Cervical Resorption (ICR)

Invasive cervical resorption occurs when clastic cells from the PDL invade the tooth, apical to the epithelial attachment. Predisposing factors to ICR include trauma, orthodontic treatment, bruxism, or bleaching [68, 69]. Anatomically, the CEJ may also predispose this area for ICR due to the presence of microscopic gaps of cementum, exposing unmineralized dentin to clastic cells [70]. The pulp tissue plays no role in the etiology of ICR, and clinical signs of pulpal involvement occur when the lesion progresses [71]. Usually, ICR is first detected radiographically. The radiolucent lesion can vary from barely discernible to dramatic, well-delineated to poorly defined with irregular borders or may even be mistaken for caries. When a resorptive lesion becomes large, it may be clinically detected by a pink hue, similar to internal resorption, or present with localized gingival overgrowth [72] (Fig. 4.11). ICR is often misdiagnosed as IR because the resorptive defect primarily occurs inside the tooth. Radiographically, ICR is distinguished by absence of resorptive defect symmetry and by the presence of an outlined pulp chamber (Fig. 4.12). ICR is usually localized, but it can affect multiple teeth. Therefore, the clinician is encouraged to rule out generalized occurrences by reviewing a full-mouth series of radiographs. When radiographically detected, small lesions can be treated conservatively with a

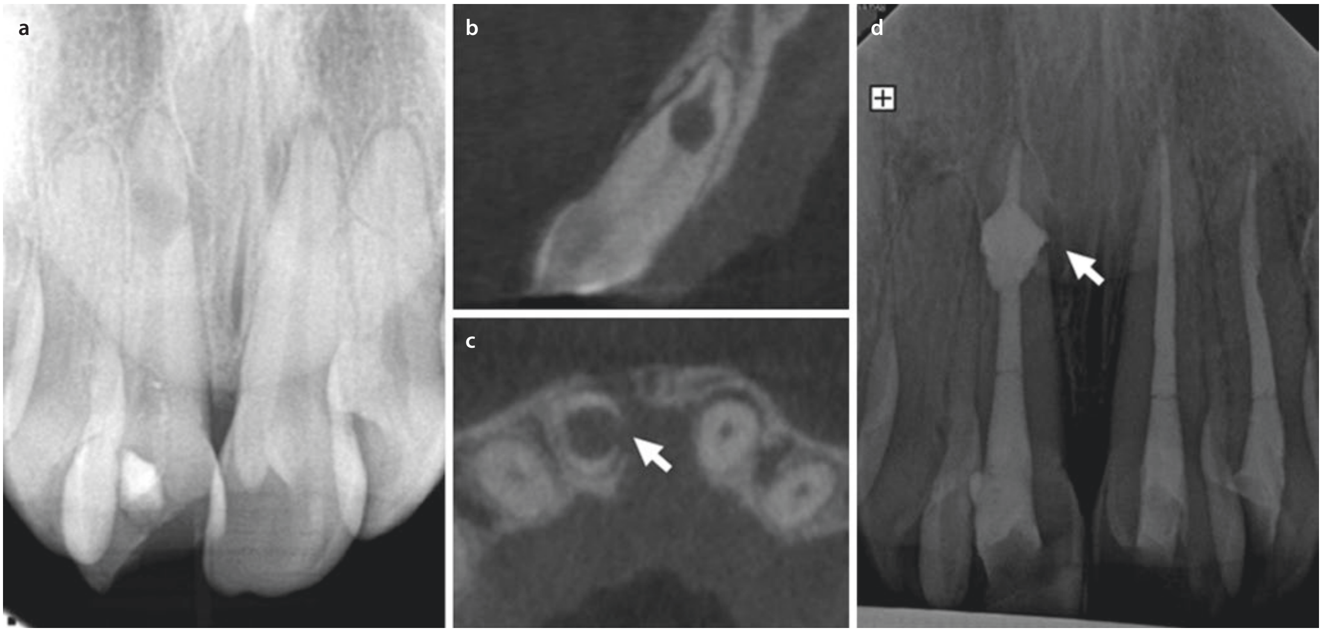



**Fig. 4.10** **a** Pre-operative radiograph of IR on tooth #9 with history of lateral luxation and complicated crown fracture; previously initiated by referring dentist. The CBCT coronal **b** and axial **c** orthogonal planes show the presence of mesial perforation in the apical third (*white arrows*), which was treated non-surgically with MTA **d**. (Courtesy of Chris Saylor, DDS, MS)

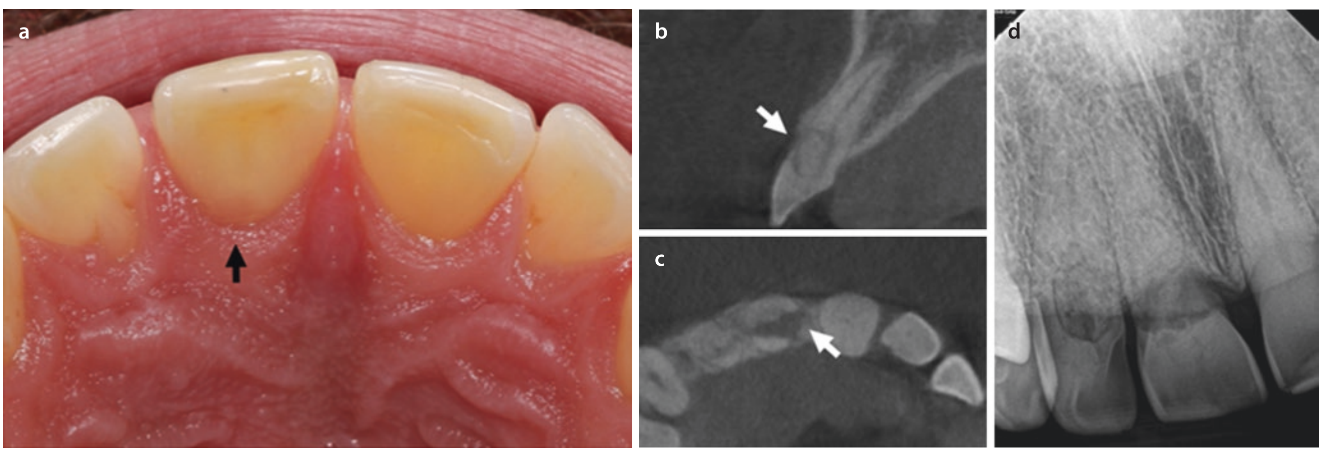



**Fig. 4.11** **a** Intra-orally, a pink hue (*black arrow*) is observed on tooth #8 with Class 3 ICR. The CBCT coronal **b** and axial **c** orthogonal planes show the portal of entry on teeth #7 and #8, respectfully (*white arrows*). No treatment, with eventual extraction when teeth become symptomatic was recommended, as shown in the PA **d**. (Courtesy of Kristine Knoll, DDS, MS)

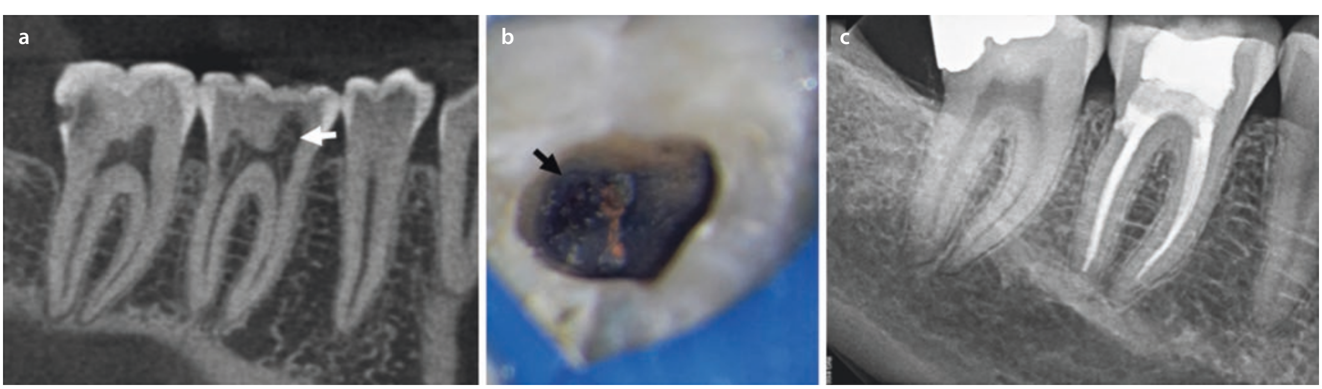



**Fig. 4.12** CBCT coronal **a** orthogonal plane shows Class 3 ICR on tooth #30 with presence of intact pre-dentin layer (*white arrow*). Endodontic therapy was completed, then perforated defect in distal canal (*black arrow*) was internally repaired with MTA **b**. Post-operative image with Paracore restoration **c**. (Courtesy of Michael Regan Anderson, DDS, MS)

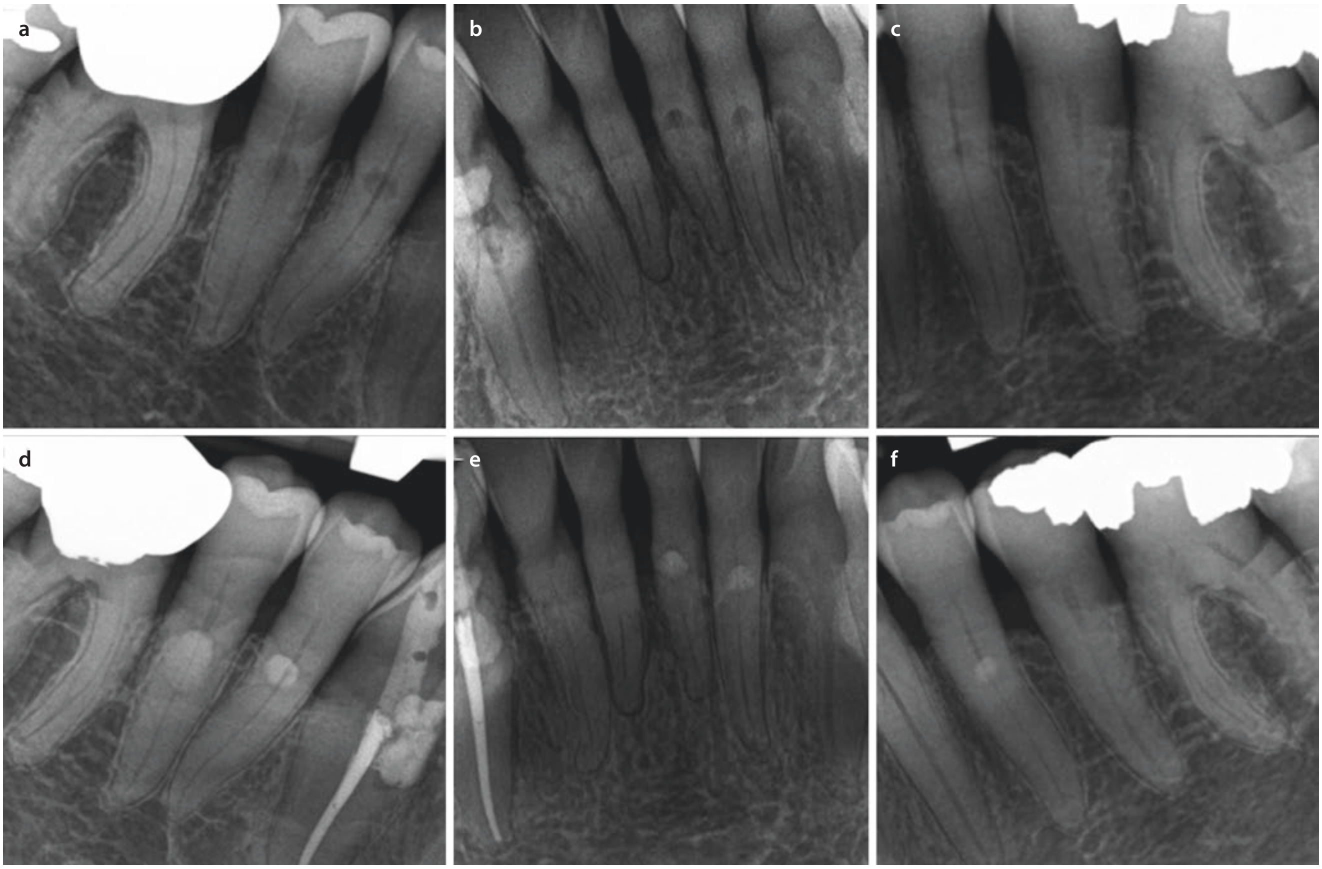



**Fig. 4.13** **a**–**c** Pre-operative radiographs showing Class 1–2 ICR on teeth #21, 23, 24, 27, 28, & 29. **d**–**f** Endodontic treatment was completed in multiple visits for tooth #27. A surgical approach was recommended for access to the remaining lesions cervical to the alveolar crest; tissue was removed with round bur, cauterized with TCA, and restored with glass ionomer. (Courtesy of Phil McKenzie, DDS, MS)

restoration and may or may not require endodontic therapy. Early detection and treatment of these small lesions are recommended, whereas the "wait-and-see" approach may lead to more tooth destruction and loss of structural integrity due to the aggressive nature of ICR [73]. Preoperative CBCT imaging is useful for identifying the progression and extent of lesions as well as aids in the decision-making process for a surgical or nonsurgical approach of therapy.

ICR treatment recommendations and prognosis were described by Heithersay using a classification system based on the extent of the resorption lesion [69]. *Class 1 defects* are those with small, shallow dentin penetration and an associated soft tissue defect. *Class 2 defects* penetrate closer to the pulp but do not extend into the radicular dentin (Fig. 4.13). *Class 3 defects* extend into the coronal third of the root in addition to the coronal dentin. *Class 4 defects* extend beyond the coronal third of the root and have the worst prognosis. Treatment of Class 1 or 2 defects have a very high success rate (~100%), treatment of Class 3 defects have a moderate success rate (78%), and Class 4 defects have a poor success rate (12.5%)[72]. Depending on the location of the portal of entry, treatment options for ICR can be surgically, from an external approach, or nonsurgically, from an internal approach [74] The resorptive tissue is removed using a round bur until the dentin is smooth and clean. Chemical debridement using 90% trichloroacetic acid (TCA) will help cauterize any remaining resorptive tissue as well as aid in identification of remaining viable tissue for further removal with a round bur. Multiple case reports on both internal and external treatment approaches are available and describe the process in greater detail [71, 75–78]. The author notes that TCA is extremely caustic and needs to be handled carefully.

### 4.4.3 External Inflammatory Resorption (EIR)

External inflammatory resorption (EIR) generally occurs following trauma. The loss of the pre-cementum, cementoblasts, and epithelial rests of Mallassez results in exposure of the root surface to resorptive cells [79]. This break in the cementum allows the clastic cells to initiate the resorption, which continues if it is stimulated by a necrotic pulp [62] (Fig. 4.14). Thus, there must be necrosis and infection present in order for the EIR to progress. Clinically these teeth may appear normal, but a radiographic moth eaten appearance is possible, and the canal can usually be traced through the lesion. This type of inflammation can progress rapidly following trauma, and treatment should be initiated as soon as possible. Treatment options depend on the extent of the resorption. In cases that have large amounts of root structure

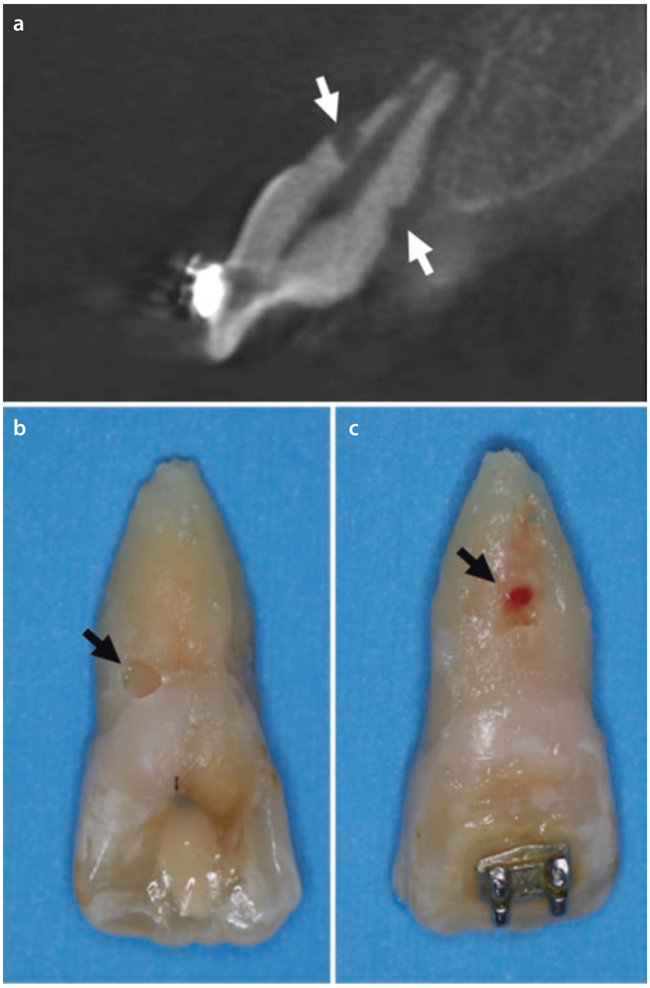


Fig. 4.14 CBCT coronal **a** orthogonal plane shows EIR on the lingual and buccal (*white arrows*) aspect of tooth #9 with history of second avulsion >90 min. Resorption defects could be visually seen (*black arrows*) on lingual **b** and buccal **c** aspects of avulsed tooth. (Courtesy of Tyler Schuurmans, DDS, MS)

missing, often extraction is the only option. In cases with smaller resorptive defects, endodontic therapy with multiple changes of calcium hydroxide (until the bone has healed) is the treatment of choice. In cases where a perforation from the external resorption is present, MTA should be the obturation material of choice.

### 4.4.4 Replacement Resorption

Replacement resorption occurs when there has been extensive damage to a large area of the periodontal ligament or if the PDL has died due to improper storage following an avulsion [47]. Adjacent PDL fibers will attempt to heal over the area with cementum and Sharpey's fibers, while bone marrow-derived cells from the socket will attempt to create bone to fill in the area. If a large area is involved, the bone marrow cells are able to repopulate the area faster, and root dentin is resorbed and replaced by bone [79].

Treatment options are limited for replacement resorption, as the tooth will progressively become weaker as the resorption advances, eventually resulting in a fracture. Treatment options include no treatment, extraction, orthodontic extrusion, or decoronation. Decoronation is a surgical method for treating ankylosed incisors in children and adolescents (growing patients) and involves the removal of the crown and root canal contents, leaving the root to be resorbed [80]. The goal is to maintain the height and width of the bone until the patient has completed the rapid growth phase to facilitate the eventual placement of an implant. Decoronation is indicated when there are clinical signs of replacement resorption and an incisal edge discrepancy of greater than 1 mm [46]. Decoronation usually requires a full-thickness flap to be reflected, but a unique technique has been recently described wherein a flapless approach is utilized to decrease postoperative pain and decrease the working time, beneficial in children and adolescents [81].

## 4.5 Non-vital Pulp Therapies on Immature Teeth

**Non-vital Pulp Therapies Used in Dental Trauma**

1. Apexification and Apical Barrier
2. Regeneration

The completion of root development and closure of the apex occurs up to 3 years following eruption of the tooth [82]. Because the incidence of trauma is relatively high in patients aged 7–15, there is a high probability of a clinician needing to treat teeth with immature roots or open apices [83]. The most important factor in determining the proper treatment of immature teeth is determining the pulp vitality, as the treatment options for vital teeth are different from those of non-vital teeth. As mentioned previously, vital pulp therapy on immature teeth are indicated when the blood supply is still intact or vital. Pulpal diagnosis, whether reversible or irreversible, and the absence of periapical pathosis are candidates for vital pulp therapy and, indirectly, apexogenesis.

When the status of the pulp has transitioned to pulpal necrosis with or without apical periodontitis, the treatment options become very limited for these immature teeth. The regenerative cells that were utilized in vital pulp therapies and apexogenesis are no longer available, and therefore the immature tooth is at a higher risk for fracture due to canal wall thinness and short root length. The treatment options for an immature tooth with non-vital pulp or apical periodontitis are apexification, apical barrier, and regeneration (also known as revascularization, revitalization, or pulpal repair by various authors).

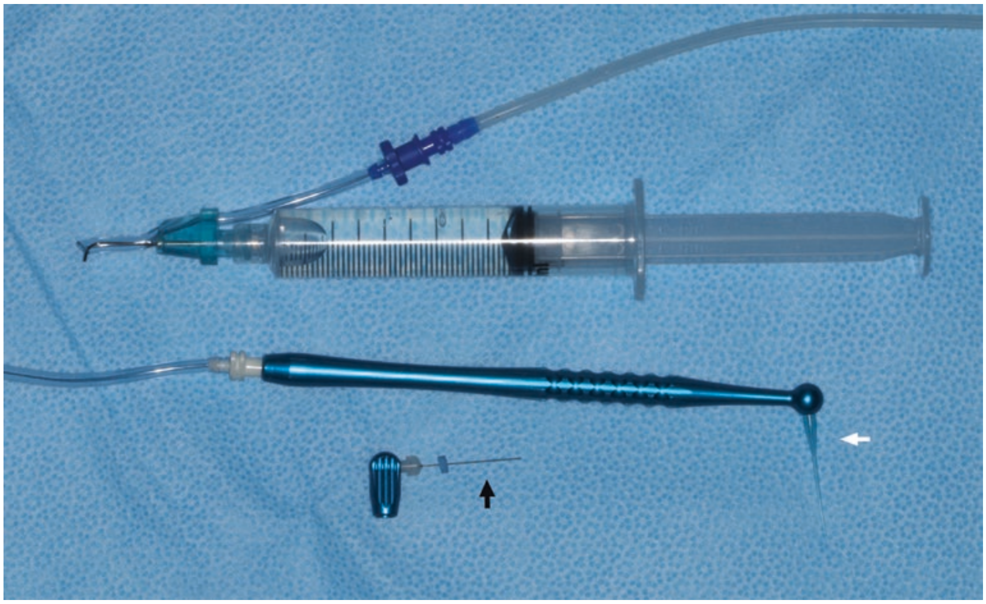


**Fig. 4.15** EndoVac system is a commercially available negative apical pressure system for improved delivery and safety; the macrocannula (*white arrow*), equivalent to ISO size #55.02 taper is introduced for bulk debris removal, then closed-end microcannula (*black arrow*), equivalent to ISO size #32.02 taper is introduced for debris removal closer to working length (Discus Dental Inc., Culver City, CA). (Courtesy of Kristine Knoll, DDS, MS)

### 4.5.1 Apexification and Apical Barrier

When pulp necrosis occurs in immature teeth, the roots are no longer able to continue their normal development in canal length, canal wall thickness, and apical closure. Treating these teeth conventionally is very difficult due to the large canal size, thin root walls, and a large open apex. The clinician is encouraged to utilize a safe delivery of their irrigants in order to chemically debride the infected canal space, without further thinning the canal wall thickness with the use of mechanical preparation. Therefore, the clinician's irrigation protocols are crucial and should include taking advantage of the antimicrobial and tissue-solvent properties of sodium hypochlorite, ultrasonic devices or other devices capable of activating the irrigant, and EndoVac (Discus Dental Inc., Culver City, CA), which is capable of delivering the irrigant safely in an open apex (Fig. 4.15).

Apexification is a method to induce a calcified barrier in a root with an open apex or the continued apical development of an incomplete root in teeth with necrotic pulp [84]. Traditionally, calcium hydroxide paste has been considered the gold standard to form a hard tissue barrier [85, 86]. This method seeks to create a stop to minimize overfill of gutta-percha by using multiple changes of calcium hydroxide over the span of 5–20 months. Disadvantages to this treatment include multiple appoints, potential for coronal leakage [87], and possible increase in the risk of future fracture from the long-term exposure of calcium hydroxide due to weakening of the root dentin [88, 89]. Alternatively, creation of a hard tissue barrier in the apical region can be achieved with materials such as MTA or Biodentine [90, 91] (Fig. 4.16). This procedure has the benefit of requiring only 1–2 appointments and allows for the immediate restoration of the tooth, without the chance for continued development of root canal length and thickness. Due to these issues, it has been suggested that a regenerative approach should be an alternative to apexification due to its desired outcome of increasing root thickness and length [92, 93].

### 4.5.2 Regeneration

Regeneration is the process of encouraging the development of root length and thickness in a necrotic, immature tooth. It was determined three factors are necessary for proper regeneration of the tooth: a blood supply, which requires an apical foramen size of at least 1 mm [94], a scaffold, and a sterile environment [95].

The size of the apex can be estimated using a traditional PA radiograph or measured using CBCT imaging, and larger apical sizes have been associated with higher success rates [95]. However, recent animal studies showed positive results with apical sizes much smaller than the historically recommended 1 mm [96].

A scaffold has been found to encourage growth of the blood supply to the tooth and traditionally was created by forming a blood clot in the tooth to the level of the CEJ [97]. More recent studies have shown success with platelet-rich plasma (PRP) or platelet-rich fibrin (PRF), with PRF having a very high potential to accelerate the growth characteristics needed for regeneration [98]. Other studies have shown success using artificial scaffolds of materials such as polylactic acid (PLA), polyglycolic acid (PGA), or synthetic hydrogels of polyethylene glycol (PEG) polymers [99]. Collagen materials (CollaPlug TM, CollaTape, or others) have also been shown to be useful as a matrix [99, 100].

To create the sterile environment, multiple combinations of antibiotics or chemicals have been tested, including triple antibiotic paste (TAP; ciprofloxacin,

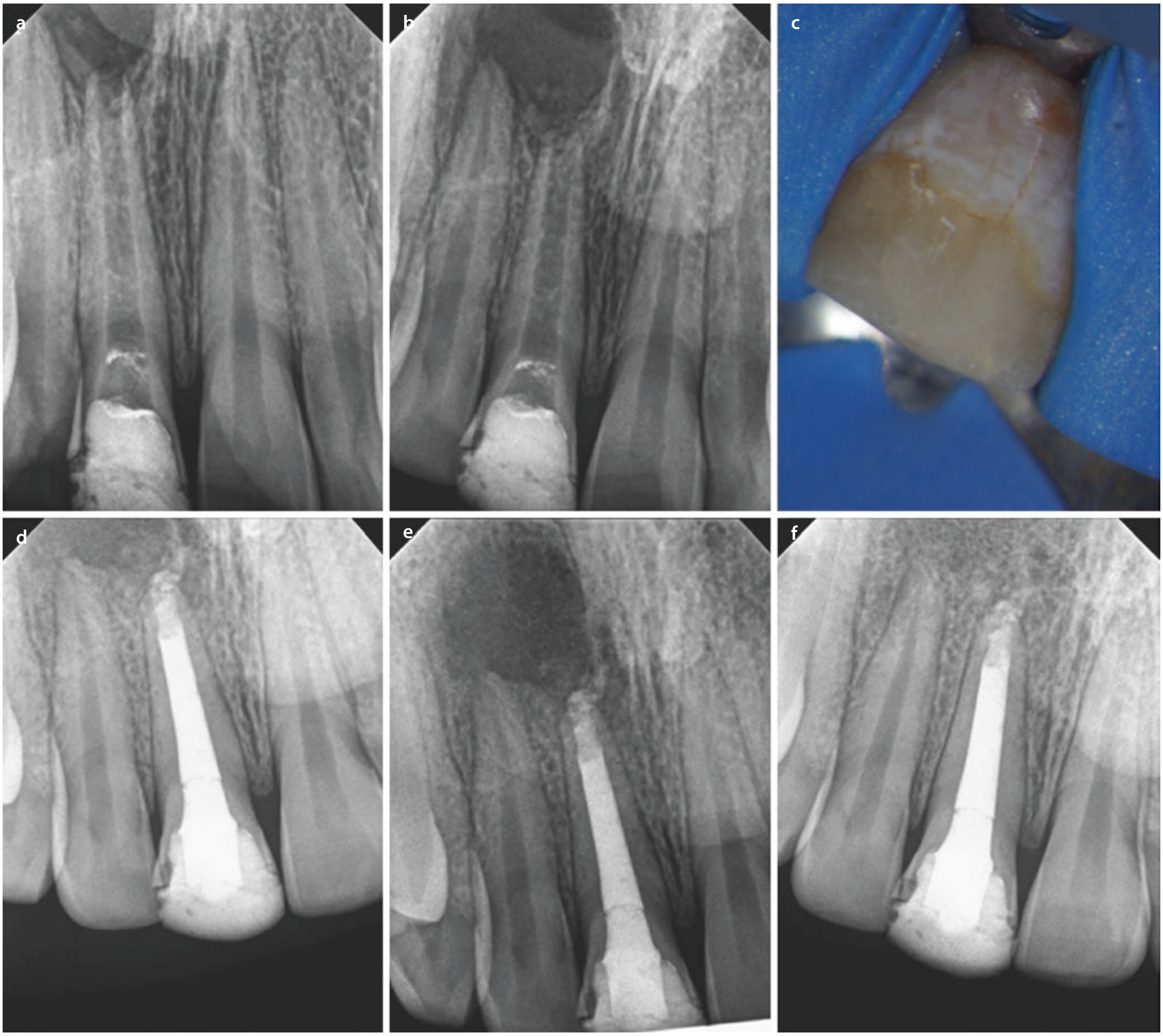



**Fig. 4.16** **a**, **b** Pre-operative radiographs showing immature tooth #8 with pulpal necrosis and asymptomatic apical periodontitis. History of complicated crown fracture that was previously treated and marked coronal staining **c**. Endodontic therapy was completed, using MTA as an apical barrier **d**, **e**. 6 month follow-up radiograph showing healing and reduction of radiolucent lesion **f**. (Courtesy of Chris Saylor, DDS, MS and Tyler Schuurmans, DDS, MS)

metronidazole, and minocycline), double antibiotic paste (DAP; clindamycin and amoxicillin), calcium hydroxide, and other agents [101, 102]. Large-scale clinical trials have not been able to adequately address the best agent, but a continuously updated protocol can be found on the American Association of Endodontist's (AAE) website, ► www.aae.org. Calcium hydroxide or low concentrations of TAP are the current recommendations due to their lower cytotoxicity.

As of this writing, the most recently updated protocol is as follows: if a compliant patient presents with a tooth that has a necrotic pulp and an immature apex and a post/core is not required for the final restoration, then the patient should be given the option of regeneration versus the traditional MTA apexification. Possible side effects include staining, lack of response to treatment, and pain or infection. The patient should also understand the need for two or more appointments.

**First Visit** Following anesthesia, a dental dam is placed on the tooth. Access to the pulp chamber is then achieved, and 20 ml of a lower concentration of 1.5% of NaOCl should be used as an irrigant. A final rinse with 20 ml of saline or ethylenediaminetetraacetic acid (EDTA) should then be used to minimize toxicity from the NaOCl. The canals are then dried and either calcium hydroxide or an antibiotic mix is placed into the canal system. A temporary restoration is then placed in the access, and the patient is dismissed for 4 weeks.

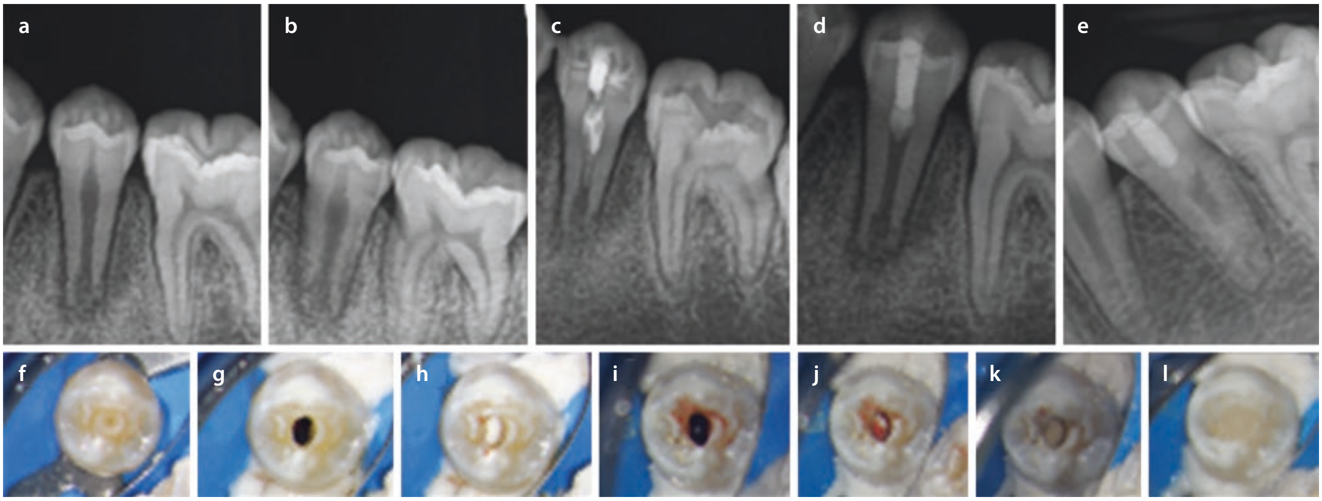



Fig. 4.17 **a**, **b** Pre-operative radiographs of immature tooth #20 **f** with necrotic pulp and chronic apical abscess. Pulpal repair was recommended and completed in two visits. First visit: accessed, chemically derided **g**, and placement of CaOH **c**, **h** for 4 weeks. Second visit: absence of sinus tract observed and blood clot induced **i**, Collaplug scaffold **j** placed over clot, 2 mm of Biodentine **k**, and final restoration **l** placed. Post-operative image of tooth #20 **d** and at 1-year recall **e**. (Courtesy of Michael Regan Anderson, DDS, MS)

**Second Visit** Look for signs of persistent infection. Additional treatment with the same or alternate antimicrobial agent(s) may be necessary. Once the signs and symptoms have abated, anesthesia is achieved with 3% mepivacaine without a vasoconstrictor in order to facilitate the creation of a blood clot. Irrigation is achieved with 20 ml of 17% EDTA, and the canal is then dried. Bleeding is created by over instrumenting with a K-file 2 mm beyond the apex and allowing the entire canal to fill with blood to the level of the CEJ. Fibrils from a collagen product are then placed over the blood clot, and white MTA or another bioceramic is placed as a capping material. As mentioned previously, both the original gray MTA and white MTA have been associated with staining [103] (Fig. 4.17), so in cases where there is an esthetic concern, newer bioceramic materials such as Biodentine should be utilized [104]. A final restoration with either composite or a resin modified glass ionomer is then placed.

Follow-up is key, and one can expect resolution of an apical lucency between 6 and 12 months after the treatment and increased root width 1–2 years after treatment. Short-term success rates for this procedure are quite high [93] when compared to MTA or calcium hydroxide apexifications, but long-term studies have not yet been completed.

## References


1. Andreasen JO, Andreasen FM, Andersson L. Textbook and color atlas of traumatic injuries to the teeth. Hoboken: Wiley; 2013.
2. Oikarinen K. Pathogenesis and mechanism of traumatic injuries to teeth. Dent Traumatol. 1987;3(5):220–3.
3. Andreasen JO, Ravn JJ. Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample. Int J Oral Surg. 1972;1(5):235–9.
4. Diangelis AJ, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of permanent teeth. Dent Traumatol. 2012;28(1):2–12.
5. Cotti E, et al. Comprehensive management of a complex traumatic dental injury. Dent Traumatol. 2014;30(5):400–5.
6. Cohenca N, et al. Clinical indications for digital imaging in dento-alveolar trauma. Part 1: traumatic injuries. Dent Traumatol. 2007;23(2):95–104.
7. Gazelius B, Olgart L, Edwall B. Restored vitality in luxated teeth assessed by laser Doppler flowmeter. Endod Dent Traumatol. 1988;4(6):265–8.
8. Yanpiset K, et al. Efficacy of laser Doppler flowmetry for the diagnosis of revascularization of reimplanted immature dog teeth. Dent Traumatol. 2001;17(2):63–70.
9. Jarvinen S. Fractured and avulsed permanent incisors in Finnish children. A retrospective study. Acta Odontol Scand. 1979;37(1):47–50.
10. Love RM, Ponnambalam Y. Dental and maxillofacial skeletal injuries seen at the University of Otago School of Dentistry, New Zealand 2000-2004. Dent Traumatol. 2008;24(2):170–6.
11. Andreasen FM, et al. Long-term survival of fragment bonding in the treatment of fractured crowns: a multicenter clinical study. Quintessence Int. 1995;26(10):669–81.
12. Murray PE, et al. Remaining dentine thickness and human pulp responses. Int Endod J. 2003;36(1):33–43.
13. Ersin NK, Eronat N. The comparison of a dentin adhesive with calcium hydroxide as a pulp-capping agent on the exposed pulps of human and sheep teeth. Quintessence Int. 2005;36(4):271–80.
14. Subay RK, Demirci M. Pulp tissue reactions to a dentin bonding agent as a direct capping agent. J Endod. 2005;31(3):201–4.
15. Leye Benoist F, et al. Evaluation of mineral trioxide aggregate (MTA) versus calcium hydroxide cement (Dycal((R)) ) in the formation of a dentine bridge: a randomised controlled trial. Int Dent J. 2012;62(1):33–9.
16. Monea M, Monea A, Stoica A. Histological evaluation of indirect pulp capping procedures with calcium hydroxide and mineral trioxide aggregate. Eur Sci J. 2014;10(24):1–10.
17. Kotsanos N, Arizos S. Evaluation of a resin modified glass ionomer serving both as indirect pulp therapy and as restorative material for primary molars. Eur Arch Paediatr Dent. 2011;12(3):170–5.
18. Kakehashi S, Stanley H, Fitzgerald R. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. Oral Surg Oral Med Oral Pathol. 1965;20(3):340–9.

19. Cvek M, et al. Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys. J Endod. 1982;8(9):391–7.
20. Katebzadeh N, Dalton BC, Trope M. Strengthening immature teeth during and after apexification. J Endod. 1998;24(4):256–9.
21. Sjögren U, et al. Factors affecting the long-term results of endodontic treatment. J Endod. 1990;16(10):498–504.
22. Salehrabi R, Rotstein I. Endodontic treatment outcomes in a large patient population in the USA: an epidemiological study. J Endod. 2004;30(12):846–50.
23. Mente J, et al. Mineral trioxide aggregate or calcium hydroxide direct pulp capping: an analysis of the clinical treatment outcome. J Endod. 2010;36(5):806–13.
24. Marques MS, Wesselink PR, Shemesh H. Outcome of direct pulp capping with mineral trioxide aggregate: a prospective study. J Endod. 2015;41(7):1026–31.
25. Berger T, Baratz AZ, Gutmann JL. In vitro investigations into the etiology of mineral trioxide tooth staining. J Conserv Dent. 2014;17(6):526.
26. Hilton TJ. Keys to clinical success with pulp capping: a review of the literature. Oper Dent. 2009;34(5):615–25.
27. Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture. J Endod. 1978;4(8):232–7.
28. Barrington C, Barnett F. Apexogenesis in an incompletely developed permanent tooth with pulpal exposure. Oral Health. 2003;93(2): 49–56.
29. Granath LE, Hagman G. Experimental pulpotomy in human bicuspids with reference to cutting technique. Acta Odontol Scand. 1971;29(2):155–63.
30. Mejare I, Cvek M. Partial pulpotomy in young permanent teeth with deep carious lesions. Endod Dent Traumatol. 1993;9(6):238–42.
31. Andreasen FM. Pulpal healing after luxation injuries and root fracture in the permanent dentition. Endod Dent Traumatol. 1989;5(3):111–31.
32. Andreasen FM, Andreasen JO. Resorption and mineralization processes following root fracture of permanent incisors. Endod Dent Traumatol. 1988;4(5):202–14.
33. Andreasen FM, Andreasen JO, Bayer T. Prognosis of root-fractured permanent incisors—prediction of healing modalities. Endod Dent Traumatol. 1989;5(1):11–22.
34. Andreasen JO, et al. Healing of 400 intra-alveolar root fractures. 2. Effect of treatment factors such as treatment delay, repositioning, splinting type and period and antibiotics. Dent Traumatol. 2004;20(4):203–11.
35. Andreasen JO, Hjorting-Hansen E. Intraalveolar root fractures: radiographic and histologic study of 50 cases. J Oral Surg. 1967;25(5):414–26.
36. Cvek M, Mejare I, Andreasen JO. Healing and prognosis of teeth with intra-alveolar fractures involving the cervical part of the root. Dent Traumatol. 2002;18(2):57–65.
37. Herweijer JA, Torabinejad M, Bakland LK. Healing of horizontal root fractures. J Endod. 1992;18(3):118–22.
38. Jacobsen I, Zachrisson BU. Repair characteristics of root fractures in permanent anterior teeth. Scand J Dent Res. 1975;83(6):355–64.
39. Skiellkr V. The prognosis for young teeth loosened after mechanical injuries. Acta Odontol Scand. 1960;18(2):171–81.
40. Andreasen FM, Pedersen BV. Prognosis of luxated permanent teeth—the development of pulp necrosis. Endod Dent Traumatol. 1985;1(6):207–20.
41. Soder PO, et al. Effect of drying on viability of periodontal membrane. Scand J Dent Res. 1977;85(3):164–8.
42. Andreasen JO, Kristerson L. The effect of limited drying or removal of the periodontal ligament. Periodontal healing after replantation of mature permanent incisors in monkeys. Acta Odontol Scand. 1981;39(1):1–13.
43. Andreasen JO, Hjorting-Hansen E. Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss. Acta Odontol Scand. 1966;24(3):263–86.
44. Trope M. Root resorption of dental and traumatic origin: classification based on etiology. Pract Periodontics Aesthet Dent. 1998;10(4):515–22.
45. Ritter AL, et al. Pulp revascularization of replanted immature dog teeth after treatment with minocycline and doxycycline assessed by laser Doppler flowmetry, radiography, and histology. Dent Traumatol. 2004;20(2):75–84.
46. Andersson L, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth. Dent Traumatol. 2012;28(2):88–96.
47. Andreasen JO. Effect of extra-alveolar period and storage media upon periodontal and pulpal healing after replantation of mature permanent incisors in monkeys. Int J Oral Surg. 1981;10(1):43–53.
48. Cvek M, et al. Effect of topical application of doxycycline on pulp revascularization and periodontal healing in reimplanted monkey incisors. Endod Dent Traumatol. 1990;6(4):170–6.
49. Trope M, et al. Short vs. long-term calcium hydroxide treatment of established inflammatory root resorption in replanted dog teeth. Endod Dent Traumatol. 1995;11(3):124–8.
50. Bryson E, et al. Effect of immediate intracanal placement of Ledermix Paste® on healing of replanted dog teeth after extended dry times. Dent Traumatol. 2002;18(6):316–21.
51. Wigen TI, Agnalt R, Jacobsen I. Intrusive luxation of permanent incisors in Norwegians aged 6-17 years: a retrospective study of treatment and outcome. Dent Traumatol. 2008;24(6):612–8.
52. Caliskan MK, Turkun M, Gomel M. Surgical extrusion of crown-root-fractured teeth: a clinical review. Int Endod J. 1999;32(2):146–51.
53. Patel S, Kanagasingam S, Pitt Ford T. External cervical resorption: a review. J Endod. 2009;35(5):616–25.
54. Patel S, et al. Internal root resorption: a review. J Endod. 2010;36(7):1107–21.
55. Trope M. Root resorption due to dental trauma. Endod Top. 2002;1:79–100.
56. Wedenberg C, Zetterqvist L. Internal resorption in human teeth—a histological, scanning electron microscopic, and enzyme histochemical study. J Endod. 1987;13(6):255–9.
57. Wedenberg C, Lindskog S. Experimental internal resorption in monkey teeth. Endod Dent Traumatol. 1985;1(6):221–7.
58. Talic NF. Adverse effects of orthodontic treatment: a clinical perspective. Saudi Dent J. 2011;23(2):55–9.
59. Bahuguna N. Cervical root resorption and non vital bleaching. Endodontology. 2013;25(2):106.
60. Fuss Z, Tsesis I, Lin S. Root resorption—diagnosis, classification, and treatment choices based on stimulation factors. Dent Traumatol. 2003;19:175–82.
61. Schwartz R. In: Schwartz R, editor. Best practices in endodontics. Hanover Park: Quintessence; 2015. p. 245–52.
62. Tronstad L. Root resorption—etiology, terminology and clinical manifestations. Endod Dent Traumatol. 1988;4(6):241–52.
63. Gartner AH, Mack T, Somerlott RG, Walsh LC. Differential diagnosis of internal and external root resorption. J Endod. 1976;2:329–34.
64. Bakland LK. Root resorption. Dent Clin N Am. 1992;36:491–508.
65. Caliskan MK, Turkun M. Prognosis of permanent teeth with internal resorption: a clinical review. Endod Dent Traumatol. 1997;13:75–81.
66. Stamos DE, Stamos DG. A new treatment modality for internal resorption. J Endod. 1986;12:315–9.
67. Goldberg F, Massone EJ, Esmoris M, Alfie D. Comparison of different techniques for obturating experimental internal resorptive cavities. Endod Dent Traumatol. 2000;16:116–21.
68. Kandalgaonkar SD, et al. Invasive cervical resorption: a review. J Int Oral Health. 2013;5(6):124–30.
69. Heithersay GS. Invasive cervical resorption: an analysis of potential predisposing factors. Quintessence Int. 1999;30(2):83–95.
70. Neuvald L, Consolaro A. Cementoenamel junction: microscopic analysis and external cervical resorption. J Endod. 2000;26:503–8.
71. Umer F, Adnan S, Raza Khan F. Conservative management of invasive cervical resorption: a case report. J Dent (Tehran). 2013;10(3):289–95.

72. Heithersay GS. Treatment of invasive cervical resorption: an analysis of results using topical application of trichloracetic acid, curettage, and restoration. Quintessence Int. 1999;30(2):96–110.
73. Patel S, Dawood A. The use of cone beam computed tomography in the management of external cervical resorption lesions. Int Endod J. 2007;40(9):730–7.
74. Frank AL, Torabinejad M. Diagnosis and treatment of extracanal invasive resorption. J Endod. 1998;24:500–4.
75. Schwartz RS, Robbins JW, Rindler E. Management of invasive cervical resorption: observations from three private practices and a report of three cases. J Endod. 2010;36(10):1721–30.
76. Harris BT, et al. Treatment of a maxillary central incisor with class III invasive cervical resorption and compromised ferrule: a clinical report. J Prosthet Dent. 2014;111(5):356–61.
77. Krishnan U, Moule AJ, Alawadhi A. Cone beam CT assisted retreatment of class 3 invasive cervical resorption. BMJ Case Rep. 2015;2015:bcr2014204615.
78. Salzano S, Tirone F. Conservative nonsurgical treatment of class 4 invasive cervical resorption: a case series. J Endod. 2015;41(11):1907–12.
79. Finucane D, Kinirons MJ. External inflammatory and replacement resorption of luxated, and avulsed replanted permanent incisors: a review and case presentation. Dent Traumatol. 2003;19(3):170–4.
80. Cohenca N, Stabholz A. Decoronation—a conservative method to treat ankylosed teeth for preservation of alveolar ridge prior to permanent prosthetic reconstruction: literature review and case presentation. Dent Traumatol. 2007;23(2):87–94.
81. Buck J. Flapless decoronation for teeth with replacement resorption. In: Schwartz R, editor. Best practices in endodontics. Hanover Park: Quintessence; 2015. p. 269–72.
82. Nolla CM. The development of permanent teeth. University of Michigan, 1952.
83. Bastone EB, Freer TJ, McNamara JR. Epidemiology of dental trauma: a review of the literature. Aust Dent J. 2000;45(1):2–9.
84. American Association of Endodontists. Glossary of Endodontic Terms. 7th ed. Chicago: American Association of Endodontists; 2003.
85. Dominguez Reyes A, Munoz Munoz L, Aznar Martin T. Study of calcium hydroxide apexification in 26 young permanent incisors. Dent Traumatol. 2005;21(3):141–5.
86. Rafter M. Apexification: a review. Dent Traumatol. 2005;21(1):1–8.
87. Heling I, et al. Endodontic failure caused by inadequate restorative procedures: review and treatment recommendations. J Prosthet Dent. 2002;87(6):674–8.
88. Andreasen JO, Farik B, Munksgaard EC. Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture. Dent Traumatol. 2002;18(3):134–7.
89. Yilmaz S, Dumani A, Yoldas O. The effect of antibiotic pastes on microhardness of dentin. Dent Traumatol. 2016;32:27.
90. Holden DT, et al. Clinical outcomes of artificial root-end barriers with mineral trioxide aggregate in teeth with immature apices. J Endod. 2008;34(7):812–7.
91. Witherspoon DE, et al. Retrospective analysis of open apex teeth obturated with mineral trioxide aggregate. J Endod. 2008;34(10):1171–6.
92. Lee BN, et al. A review of the regenerative endodontic treatment procedure. Restor Dent Endod. 2015;40(3):179–87.
93. Jeeruphan T, et al. Mahidol study 1: comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: a retrospective study. J Endod. 2012;38(10):1330–6.
94. Kling M, Cvek M, Mejare I. Rate and predictability of pulp revascularization in therapeutically reimplanted permanent incisors. Endod Dent Traumatol. 1986;2(3):83–9.
95. Garcia-Godoy F, Murray PE. Recommendations for using regenerative endodontic procedures in permanent immature traumatized teeth. Dent Traumatol. 2012;28(1):33–41.
96. Laureys WG, et al. The critical apical diameter to obtain regeneration of the pulp tissue after tooth transplantation, replantation, or regenerative endodontic treatment. J Endod. 2013;39(6):759–63.
97. Nosrat A, et al. Tissue engineering considerations in dental pulp regeneration. Iran Endod J. 2014;9(1):30–9.
98. Narang I, Mittal N, Mishra N. A comparative evaluation of the blood clot, platelet-rich plasma, and platelet-rich fibrin in regeneration of necrotic immature permanent teeth: a clinical study. Contemp Clin Dent. 2015;6(1):63–8.
99. Saber SE. Tissue engineering in endodontics. J Oral Sci. 2009;51(4):495–507.
100. Dabbagh B, et al. Clinical complications in the revascularization of immature necrotic permanent teeth. Pediatr Dent. 2012;34(5):414–7.
101. Sato I, et al. Sterilization of infected root-canal dentine by topical application of a mixture of ciprofloxacin, metronidazole and minocycline in situ. Int Endod J. 1996;29(2):118–24.
102. Bose R, Nummikoski P, Hargreaves K. A retrospective evaluation of radiographic outcomes in immature teeth with necrotic root canal systems treated with regenerative endodontic procedures. J Endod. 2009;35(10):1343–9.
103. Krastl G, et al. Tooth discoloration induced by endodontic materials: a literature review. Dent Traumatol. 2013;29(1):2–7.
104. Utneja S, et al. Current perspectives of bio-ceramic technology in endodontics: calcium enriched mixture cement-review of its composition, properties and applications. Restor Dent Endod. 2015;40(1):1–13.

# Facial Trauma: Oral and Maxillofacial Surgical Issues

*Luke A. McMahon, Mansur Ahmad, and Leon A. Assael*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_5

5

## 5.1 Introduction

Due to our upright posture, binocular vision, and facial structures, trauma to the oral and maxillofacial region is a frequent occurrence in athletic activity. By its nature, sport involves action, speed, and some danger. Collisions, projectiles, water, air, acceleration/deceleration forces in sport all promote an environment for risk for facial trauma. Parents, coaches, trainers, dentists, and physicians all play integral roles in the evaluation and management of facial and dental trauma sustained during sporting events. Conn et al. reported that 7 million Americans (25.9 per 1000 persons) were treated annually for sports-related injuries [1]. Of these injuries, 1.1 million (14.8%) involved the head/neck region. 3–29% of facial injuries are related to sports [2]. Given the possibility for injuries to occur in a wide variety of events, especially in contact sports, it is important to understand the evaluation of individuals who suffer from sports-related trauma [1]. The goal of this text is to outline these concepts in relation to the treatment of these injuries. The reader is referred to other chapters within this text for a detailed discussion of dentoalveolar trauma.

Various causative factors are implicated in facial trauma. These vary according to age and sex of the patient. Males are more likely to sustain facial trauma as well as sports-related facial trauma than females [3–9, 10–12, 13]. Conn et al. [1] reported that injuries were more likely to occur in 5–14-year-olds (59.3 per 1000 persons) and 15–24-year-olds (56.4 per 1000 persons). The cause of injuries was different in these age groups as 5–14-year-olds were most likely to be injured while cycling or playing basketball. 15–24-year-olds were more likely to be injured in basketball or football. MacIsaac et al. [14] indicated that 13–15-year-olds (40.6%) had the highest incidence of sports-related injury followed by 10–12-year-olds (23.4%). Nalliah et al. [10] again demonstrated the propensity for injuries to occur in young teens (13–15) in a review of emergency department (ED) visits due to sports injuries. This large-scale review of over 430,000 ED visits identified open wounds (10.7%), concussions (6.8%), and skull and facial fractures (3.1%) as common head and neck injuries sustained while participating in sports [10].

The most common causes of facial fractures vary by region. In the United States, the three most common reported causes include assault (37%), falls (24.6%), and motor vehicle accidents (12.1%) [12]. Various sources have reported other figures, but these remain the three most common causes. Other, less common, causes include bicycle accidents (1.6–2%) and gunshot wounds (0.5–2%) [11, 12]. MacIsaac et al. [14] evaluated sports-related craniofacial fractures and indicated that the most commonly related sports were baseball and softball (44.3%), skateboarding (8.4%), soccer (7.8%), and basketball (7.2%).

**Most Common Sports for Craniofacial Fractures [14]**

| | |
|---|---|
| – Baseball/softball | 44.3% |
| – Skateboarding | 8.4% |
| – Soccer | 7.8% |
| – Basketball | 7.2% |

*Note*: Baseball/softball and skateboarding have use of protective equipment

This study indicated that fractures from sporting events were reported to most likely occur in 13–15-year-olds (40.7%). Nasal, orbital, and skull fractures were the most commonly sustained fractures in this review. Maxillary (12.6%), mandibular (7.2%), zygomaticomaxillary (4.2%), and naso-orbital-ethmoid (1.2%) fractures were less likely to occur. However, varying distributions have been reported in the literature. Sports-related injuries and distribution of the related fractures vary by region and sport preferences. Studies from abroad have also had high proportions of injuries related to cricket, rugby, handball, equestrian, and martial arts [5–7].

Changes in sport are exposing the face to more injuries. With the emergence of freestyle skiing and snowboarding, with airborne high-speed maneuvers, facial, head, and neck injuries are on the rise with 245 facial trauma injuries in World Cup events [15]. As with other high-speed injuries, many of these (58) had severe neurotrauma. Competitive platform diving is another example in which changes in sport have resulted in new types and patterns of injury, such as orbital and sinus fracture from high-speed impact with the water [16]. Deepwater exploration in scuba diving can potentially cause temporomandibular joint dysfunction or barodontalgia [17].

There is continued emphasis on prevention of injuries in sports. The use of protective gear including helmets, face masks, and mouth guards is endorsed for decreasing injury rates. The effectiveness of these measures is mostly positive, but there is evidence that helmeted participants in sport are more likely to employ risky behaviors. Distracted cycling is an emerging concern along with the rates of helmet use [18]. Protective gear promotes the perception of safety and can cause the participant to minimize risk [19]. This may partially explain why the use of protective gear is variable in its impact on injury reduction.

## 5.2 Evaluation of Patients with Facial Trauma

**Evaluation of Patients with Facial Trauma**

- Immediate assessment
  - Advanced trauma life support (ATLS)
- History and physical examination and acute care
- Imaging

### 5.2.1 Immediate Assessment

The initial assessment of facial sports injuries must follow the recommendations of the American College of Surgeons (ACS) Advanced Trauma Life Support (ATLS) management to assess systemic, potentially life-threatening emergency. Injuries seemingly isolated to the face are commonly associated with neurotrauma, cervical spine injury, and pulmonary barotrauma. Treating providers must remember the systematic evaluation of airway, breathing, circulation, and disability (ABCDs) of ATLS trauma management [20]. Careful attention should be turned to assuring a patent airway (A). If an injury to the patient's cervical spine is suspected, the patient's cervical spine should not be manipulated. Per ATLS protocol, patients with maxillofacial injuries or head trauma should be presumed to have a cervical spine injury, and the neck should be immobilized until fully evaluated [20]. The provider must also assure that breathing (B) is unobstructed and that circulation (C) is maintained. Any life-threatening hemorrhage should be managed at this point in time. It should be remembered that highly conditioned athletes might not have the same physiologic response (i.e., tachycardia) to blood loss that the normal population may have [20]. The patient should then be evaluated for any neurologic deficit (D—Disability) with a focused neurologic exam including level of consciousness, orientation to person, place, and time, visual acuity, pupillary size and reactiveness, visual changes, or numbness or weakness in the extremities. Glasgow Coma Scale (GCS) assessment of patients with a head injury will assist in developing an evaluation and triage plan for the patient. Facial fractures, particularly high-energy zygoma and midface fractures, are associated with lower GCS scores and cervical spine injuries [21]. Concerning symptoms include loss of consciousness, altered consciousness, nausea, or vomiting. If a head injury is suspected, prompt transfer to a medical facility for neurosurgical consultation is recommended. Again, it is imperative that cervical spine precautions be maintained in the transport period.

Many concomitant injuries are noted along with maxillofacial trauma. The treating provider must have a high suspicion of index of other injuries whenever a patient presents with facial trauma. Allareddy et al. [12] reported common associations with intracranial injury (12.3%), skull fractures (7.2%), and spinal cord injury (0.2%). Cervical spine injuries (2.6–6.5%) have also been associated with facial fractures [22, 23].

### 5.2.2 History and Physical Examination and Acute Care of Facial Injuries

Once the patient has been evaluated with the primary survey in a systematic fashion and it has been noted that he or she is stable from a cardiorespiratory standpoint and intracranial injury or cervical spine injury has been ruled out, the secondary survey should then be completed. This involves completing a review of the patient's history and performing a thorough physical examination. Obtaining a patient's history can be performed in the AMPLE (A, allergies; M, medications; P, past history; L, last meal; E, events/environment related to the injury) format [20]. Pertinent findings in the medical history should be noted and discussed with any subsequent providers.

The physical examination should be performed in a systematic and thorough manner. The head and neck exam will evaluate for facial asymmetry. The skin of the head and face is then evaluated for abrasions, lacerations, or contusions. The forehead is examined for symmetry, depression, or neurosensory changes. As the exam moves inferiorly, the eyes are palpated for steps of the orbital rims, pupillary size, light reactivity, peri- orbital ecchymosis, subconjunctival hemorrhage, chemosis, extraocular eye movements, visual acuity, diplopia, entrapment, or enophthalmos. Enophthalmos, steps of the orbital rim, or entrapment are indicators of orbital fractures. If entrapment (limitation of the movement of the globe) is noted, the patient should be referred for immediate surgical evaluation. Additionally, if bilateral periorbital ecchymosis (i.e., "raccoon eyes") is noted, there may be an injury to the anterior cranial fossa, frontobasilar injury, and naso-orbital-ethmoid or Le Fort fractures. Any concern for fracture should prompt a referral for further evaluation.

The external ear evaluation is also important [24]. The region is evaluated for lacerations or hematoma formation. Left untreated, a hematoma can lead to permanent deformity, such as "cauliflower ear." The external auditory canal should be inspected and the tympanic membrane evaluated for perforation or hemotympanum (blood along the tympanic membrane). Any changes in hearing should be noted. When evaluating the mastoid region, the examiner should be mindful that if there is any bruising in this region (i.e., Battle's sign, [25]), it may indicate a basilar skull fracture.

Fig. 5.1 Clinical findings consistent with a mandibular fracture. Floor of mouth hematoma, gingival laceration, and occlusal step in the interproximal region of teeth 23 and 24

Nasal fractures have been reported to be the most common sports-related facial fracture [14]. Therefore, a thorough nasal evaluation should also be performed. Epistaxis in the sports injury patient is a nasal fracture unless proven otherwise. The dorsum should be evaluated for deviation or crepitus. The septum is evaluated for a septal hematoma or deviation. Septal hematomas will require prompt treatment in order to prevent the possibility for necrosis and subsequent septal perforation. This may ultimately lead to a saddle nose deformity [26]. Epistaxis may be initially managed with compression; if this is unable to manage the initial hemorrhage, the patient should be referred to a facility for further management with anterior or posterior nasal packing as indicated. Within this region, the midface is palpated for stability or depression along the malar (cheek) region, zygomatic arch, and maxilla.

An oral evaluation is then completed. The mucosa is examined for lacerations. Teeth are evaluated for fractures, avulsions, and/or mobility. The maxilla is assessed for stability via manual palpation for mobility and assessment of occlusion. Clenching the teeth may reveal vertical instability associated with maxillary fracture. The patient's occlusion (bite) is evaluated. Any changes in occlusion and fractured, missing, or loose teeth should be noted and referred for further clinical and radiographic evaluation. Any missing teeth should be accounted for in the immediate area; otherwise a chest radiograph should be obtained to rule out aspiration. The mandible is put through an active range of motion; any mobility of mandibular segments, occlusal steps, floor of mouth ecchymosis, bony steps along the inferior border, gingival lacerations, (Fig. 5.1), or trismus (limited opening) is concerning for mandibular fracture.

Temporomandibular joint (TMJ) evaluation is for the purpose of diagnosing both hard and soft tissue injuries of the temporomandibular joint. Inspection of the mandible may demonstrate preauricular facial edema in the patient with TMJ injury, such as contusion or fracture of the condyle. Bleeding from the external auditory canal may be a finding in tympanic plate (posterior glenoid fossa) fracture or in hemarthrosis. Open bite on the ipsilateral to the side of injury is a sign of hemarthrosis or lateral dislocation of the condyle. Ipsilateral occlusal prematurity in the molar occlusion and deviation to the side of injury on opening and protrusion are signs of condylar or subcondylar fracture. Articular disc displacement or tears can occur in concussive and deceleration injuries. Tenderness on lateral capsule TMJ palpation and with gentle digital pressure in the anterior portion of the external auditory canal is associated with capsular tears of the articular disc. Due to edema, clicking of the disc is rarely noted in the examination shortly following injury when the disc is torn but often emerges in the days after which acute edema has abated. Anterior disc displacement without reduction can cause immediate mandibular hypomobility. Contusion of the muscles of mastication as well can produce hypomobility. These injuries may be isolated or concomitant.

Open lock (when the patient is unable to close the jaw) may be related to mandibular dislocation or due to spasm of contused muscles. The patient may require mandibular reduction with inferior and posterior pressure on the external oblique ridge of the mandible. Because of the possibility of associated fractures, when practical, preoperative imaging of the injury is often utilized. The patient with open lock normally benefits from sedation with a short-acting benzodiazepine, such as Versed, to allow for completion of this maneuver.

Increased pain on biting (i.e., a tongue blade) may be indicative of a mandibular fracture. If an intraoral laceration approximates the region of the parotid ducts or submandibular ducts, one should attempt to express saliva from the respective gland to assess for ductal injury. Paresthesias in the upper lip and cheek (cranial nerve (CN) V2 distribution) may indicate an orbital floor or maxillary fracture. Paresthesia in the lip or chin (CN V3 distribution) may indicate a mandibular fracture.

Further neurologic evaluation of the patient with a maxillofacial injury includes a thorough cranial nerve exam testing visual acuity (CN II), pupillary responses (CNs II and III), and ocular motility (CNs III, IV, VI). The trigeminal nerve (CN V) controls facial sensation, the muscles of mastication, anterior digastric, mylohyoid, tensor tympani, and the tensor veli palatini. Facial sensation in the ophthalmic (V1), maxillary (V2), and mandibular (V3) branches should be examined with a light touch. Facial movement can be evaluated along the branches of the facial nerve (CN VII). Raising the eyebrows tests the temporal (frontal) branch. Tight eye closure tests the zygomatic branch. A smile is used to evaluate the buckle branch. A frown or lip pucker is used to evaluate the marginal mandibular branch [24]. Function of the patient's hearing should be evaluated (CN VIII). This can be accomplished with a finger rub bilaterally. Palatal elevation and symmetric position of the uvula are also evaluated (CNs IX and X). Shoulder shrug (trapezius innervation) or head turn (sternocleidomastoid innervation) are evaluated for any asymmetries (CN XI). Finally the tongue is protruded (CN XII), and any asymmetry should prompt further evaluation. Following completion of the focused history and physical, referral and imaging (if indicated) should then be obtained.

## 5.3 Imaging of Facial Trauma

Different types of facial fractures have been described below (▶ Sects. 5.4 and 5.5). Ideal imaging depends on the initial clinical diagnosis of the trauma.

**Mandibular Facture** A panoramic radiograph is the best initial examination to evaluate mandibular fracture. Radiographic appearance of mandibular fracture can be of three types: (1) a linear radiolucency limited to the bony outline of the mandible indicating separation of the fracture fragments, (2) a linear radiopacity limited to the bony outline of the mandible indicating overlapping of the fracture fragments, and (3) a step deformity in the outline of the mandible. For fracture diagnosis, the mandible is considered as a ring that often fractures in two different places, e.g., subcondylar fracture may be associated with a fracture of the contralateral parasymphysis. In addition to a panoramic radiograph, a CBCT scan is particularly helpful in determining displacement of a fractured condylar head. Plain film radiographic examinations, such as open-mouth Towne's view or transorbital view, have been replaced by CBCT or CT scans. An MRI is useful when an edema is suspected in the temporomandibular joint or when multiple fractures are present in the facial skeleton in addition to mandibular fracture.

If healing is uncomplicated, postreduction evaluation of the mandibular fracture is best performed by panoramic radiography, particularly when metallic hardware is used. In case of suspicion of infection or inadequate reduction, cross-sectional views from a high-resolution (0.1–0.2 mm) CBCT scan can help to rule out periosteal new bone formation. A CBCT scan is also helpful in identifying the relationship of metallic screws of bone plates with the inferior alveolar canal. Compared to a CBCT scan, a CT scan is likely to generate extensive image artifacts when metallic bone plates are present.

**Midface Fracture** Midface fracture provides challenging radiographic findings, due to the presence of multiple bones, sutures, and critical structures in the midface region. Panoramic radiography is moderately useful in determining fracture involving the zygomatic processes and floors of the orbits. Most plain film radiographic examinations only provide minimal information on the complex anatomy of the maxillofacial skeleton. Although a CBCT scan of the maxillofacial trauma can provide adequate information of the bony fractures, it does not provide reliable data on the soft tissue status. In contrast, CT images in soft tissue and bone algorithms are most useful to evaluate midface trauma. Optimal protocols for CT imaging should include 0.6–1.2-mm-thin slices in axial and coronal orientation with 3D reformatting. The field of view should extend from the frontal sinus to the inferior border of the mandible. An MRI should be acquired when there is suspicion of trauma to the brain tissues and if there are penetrating wounds. Imaging for brain tissues is beyond the scope of this text.

Each imaging study for maxillofacial trauma should accompany a thorough radiology report that identifies the location and extent of the fracture(s), whether favorable or unfavorable, critical structures that may be involved, dental tissues that may be in the path of the trauma, any dental fracture in addition to jaw fracture, and recommendation for further imaging. To assist the surgeon in proper planning, 3D reconstruction of the CT or CBCT data should be provided in addition to multiplanar reconstruction.

## 5.4 Mandibular Fractures

### 5.4.1 Classification

Of all facial fractures related to sports, 7–41% [6, 7, 14, 13] of these fractures occur in the mandible. The site of mandibular injury is related to the mechanism. The type of injury will vary depending on the circumstances at the time of the injury and the force sustained by the participant. Common mandibular injury locations have been described by several sources [3, 11, 12, 27, 13]. Fractures may occur at the condyle, ramus, coronoid process, angle, body, alveolus, or symphysis. Antoun and Lee [13] evaluated the distribution of mandibular fractures in sports-related maxillofacial fractures in New Zealand, and the angle was noted to be the most frequently affected region (◘ Fig. 5.2). When evaluating and treating mandibular fractures, it is important to consider other possible injuries. If one mandibular fracture is noted, there is a high probability of a second fracture [28]. Additionally, cervical spine injuries (5.1%) can be encountered even with isolated mandibular fractures [23].

When diagnosing fractures, they can be categorized by the type of fracture. Often, when children sustain mandibular fractures, they can occur in a greenstick fashion (◘ Fig. 5.3a), meaning they are incomplete. Simple fractures are a complete separation of two pieces of the bone (◘ Fig. 5.3b). When multiple fragments of bone are present at the fracture site, this is termed comminuted (◘ Fig. 5.3c). A compound, or open, fracture communicates with the external environment via skin or oral lacerations (◘ Fig. 5.1). Fractures that occur in the region of the teeth are also considered open fractures due to communication to the oral cavity through the periodontal ligament.

If the fracture is reduced by the muscle pull of the mandible in relation to the geometry of the fracture, it is considered to be a favorable fracture. If the bones are further displaced by the muscle pull, the fracture would be considered unfavorable (◘ Fig. 5.4).

Lastly, if one wishes to evaluate severity of mandibular injuries in a more objective fashion, there are tools to do this. The mandibular injury severity score was developed to address this need [29]. This scale has been used since its development in areas of research.

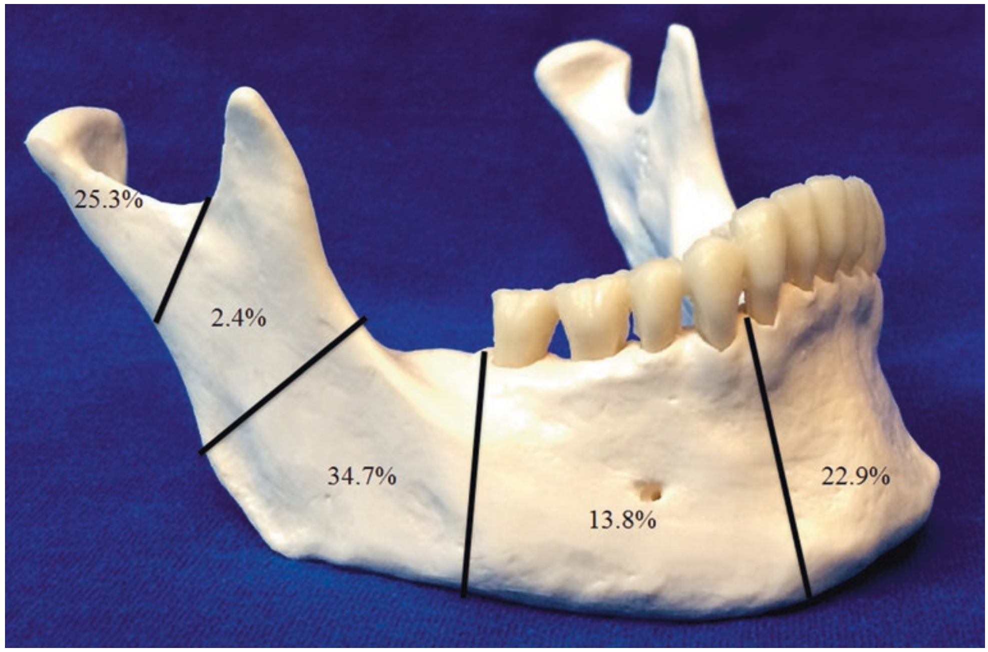



**Fig. 5.2** Distribution of sports-related mandibular fractures in the condyle (25.3%), ramus (2.4%), angle (34.7%), body (13.8%), and parasymphysis/symphysis (22.9%) [13]

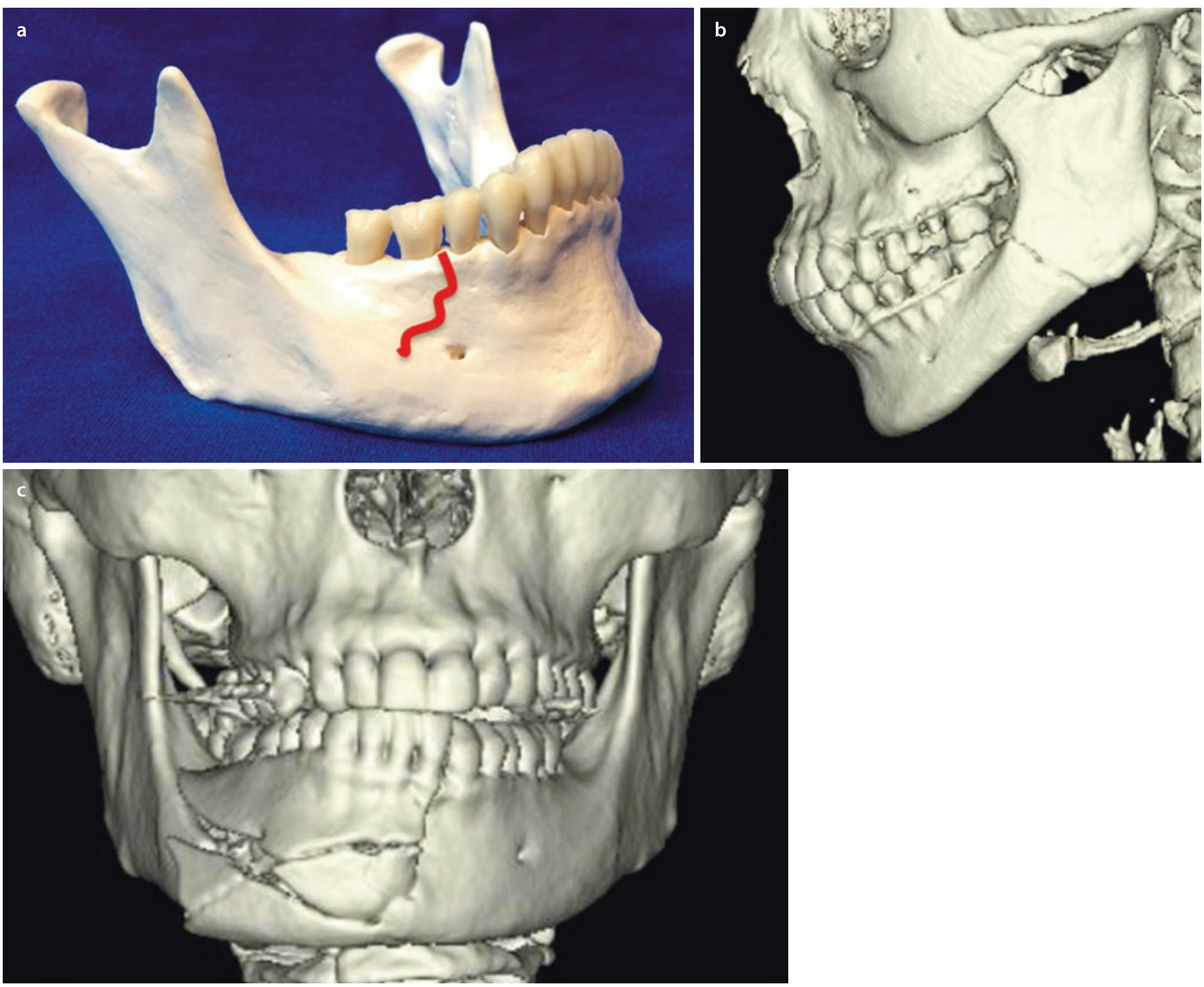



**Fig. 5.3** Diagram and 3D CT reconstructions demonstrating **a** a greenstick (incomplete) mandibular fracture, **b** a simple mandibular angle fracture, and **c** a comminuted mandibular fracture

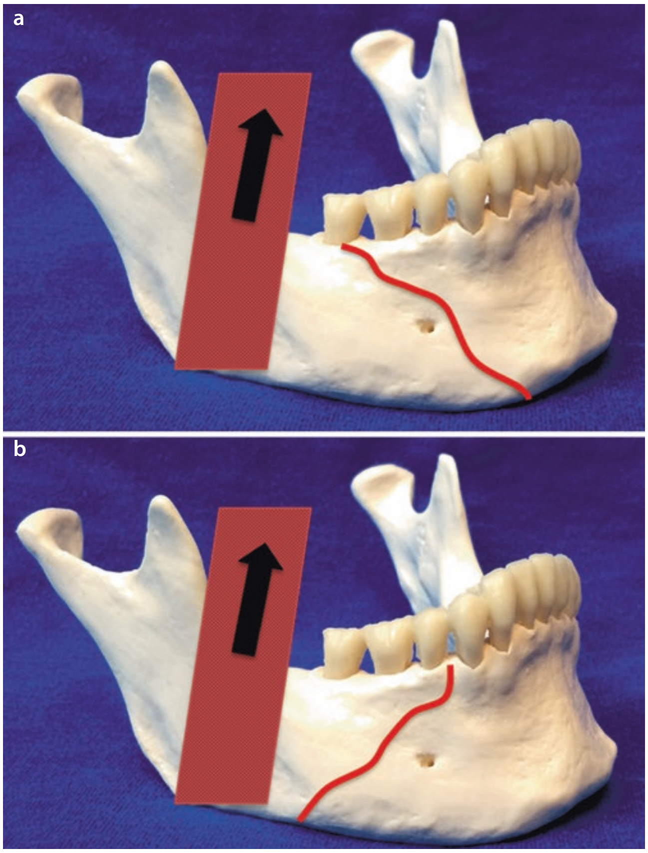



**Fig. 5.4** Demonstration of masseter pull (*red hash with bold arrow*) on **a** favorable and **b** unfavorable fractures

### 5.4.2 Treatment of Mandibular Fractures and TMJ Injuries

The goal of treatment of mandible fractures is directed toward restoration of form and function at the fracture site. In the mandible, this includes the restoration of the patient's occlusion along with the ability to eat and speak. For all patients, but especially the athlete, return to immediate full function of the mandible is advantageous. It is important to do all of these things while providing the patient with minimal inconvenience and maximum esthetics.

Timing of repair is variable and is highly dependent on any associated injuries or medical comorbidities. These concerns must be addressed prior to proceeding with treatment. Generally, it is preferred to treat fractures in a timely manner. Postoperative edema, typically peaking at 48–72 h following the injury, may complicate surgical access and reduction. The treating surgeon must consider this fact. Most mandibular fractures are compound fractures, communicating with the oral cavity or the skin. The most typical site of communicating to the external environment is via the gingival sulcus in the dentate portion of the mandible. These, as well as compound fractures through the skin, are at higher risk of infection if treatment is delayed. Antibiotics covering oral and skin flora, as appropriate, and an antimicrobial rinse (i.e., Peridex 0.12%) are recommended beginning at the time of patient evaluation.

Treatment principles of fractures include fracture reduction and immobilization. This can be completed in several ways in the mandible. Patients may be treated closed (i.e., without incisions) or open. Internal fixation is typically provided with plates and screws. Internal fixation provides fixation forces across the fracture site to permit undisturbed healing with an early return to function. Closed treatment provides immobilization of the entire body part with maxillomandibular fixation. The treatment chosen depends on the age and physical status of the patient, type of injury, patient desires, local anatomic factors, and risks of treatment.

Closed reduction was originally used with a bandage, known as the Barton bandage [30]. This gave way to wiring techniques to align the teeth into the proper occlusal relationship. This can be accomplished with arch bars (Fig. 5.5a) and Risdon wires (Fig. 5.5b). Patients are either wired together into maxillomandibular fixation or placed in elastics (heavy or guiding). Orthodontic brackets or intermaxillary fixation (IMF) screws (Fig. 5.5c) can also be used in the appropriate clinical situations.

Splints may also be used to treat mandibular fractures. Significantly displaced fractures, or those with concomitant midfacial fractures, may require lingual or occlusal splint fabrication to establish the proper mandibular width. Presurgical records from the patient's dentist or orthodontist may assist with this process.

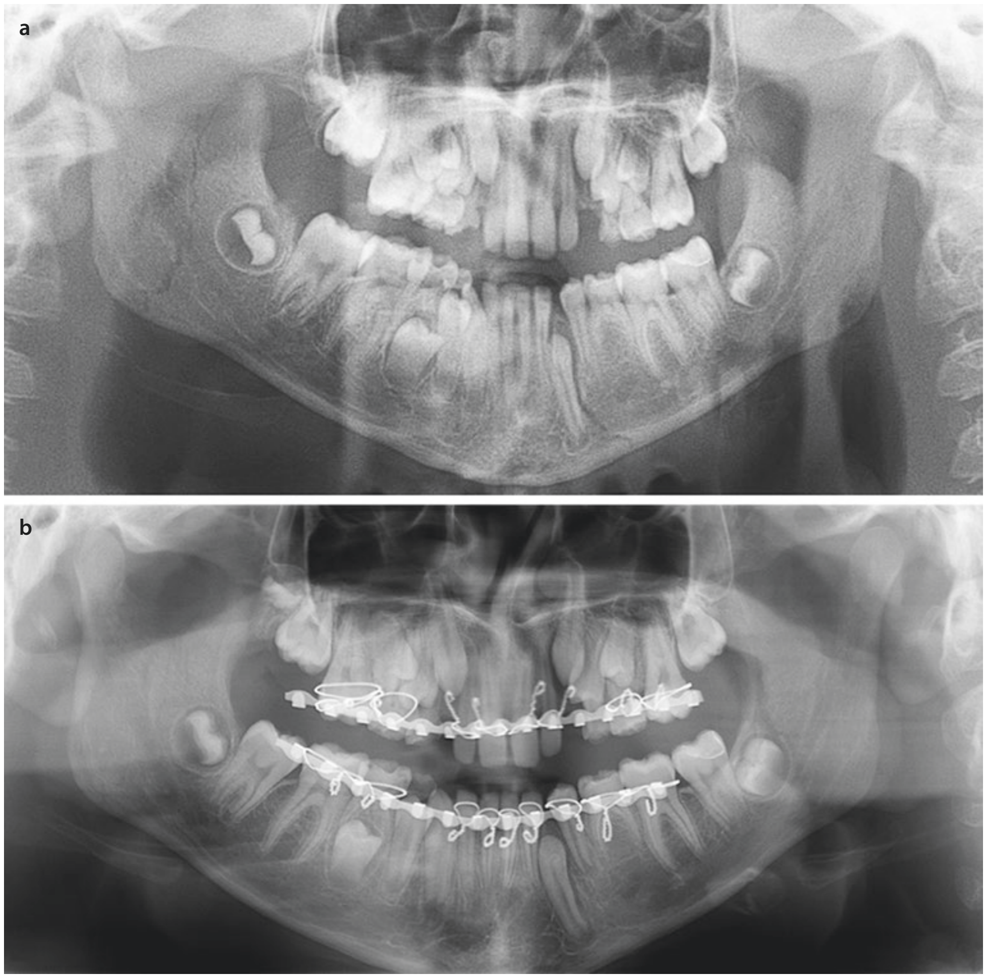
When undergoing closed reduction, the period of maxillomandibular fixation (MMF) is usually 6–8 weeks for adults. Pediatric fractures (Fig. 5.6) are preferably treated closed, as open treatment has risks, including damage to the developing tooth buds. Growing patients are treated with a shorter period of MMF than adults [31]. Patients maintain a strict liquid diet throughout the period of MMF.

Treatment protocols differ for mandibular condylar head and neck fractures depending on institutional experience and preferences. Protocols may range from 2 to 4 weeks of MMF in adults and 7 to 10 days in children. Aggressive physiotherapy is recommended in the posttraumatic period for these cases. Fractures of the condyle can be treated closed or via open reduction. Closed treatment is most commonly used when the patient can obtain good intercuspation on attempted closing, and there is no fracture dislocation of the condyle. If the ramus is functionally shortened by the fracture, the patient will be unable to gain maximum intercuspation. This normally occurs if the condyle is dislocated from the glenoid fossa and is thus a fracture dislocation. Open reduction with plate and screw fixation is normally employed for such condylar fractures. Ideal treatment of condylar fracture treatment is a controversial topic; however, recent studies suggest that open treatment provides better outcomes in these patients [32]. Patients that are averse to a period of MMF may also elect to undergo open reduction and internal fixation (ORIF), which will allow earlier function.

When patients require treatment, the range of options, along with the risks and benefits, should be presented. For patients desiring open treatment, direct visualization, reduction, and fixation of the fractures will occur. This can be

**Fig. 5.5** Multiple methods of intermaxillary wiring techniques including **a** arch bars, **b** Risdon wires, and **c** intermaxillary fixation (IMF) screws

accomplished through existing lacerations or surgical incisions via intraoral or extraoral approaches. Transcutaneous trocars can also be used to minimize extraoral incisions in the mandibular body and angle regions. Surgical access may put vital structures (nerves, blood vessels, and/or salivary ducts) at risk, and proper informed consent is necessary in the treatment process. When considering the risks of open treatment, one must weigh the benefits. These include an earlier return to function, decreased period of MMF, decreased discomfort, improved postoperative hygiene, and improved postoperative nutrition. Patients with medical comorbidities such as patients with a history of seizures, alcoholism, psychiatric illness, or compromised pulmonary function may be more safely treated with open procedure in order to avoid MMF [33, 34].




■ **Fig. 5.6** A left mandibular parasymphysis and right mandibular ramus fracture sustained by a 13-year-old during a baseball game **a** treated with closed reduction for 4 weeks **b**

■ **Fig. 5.7** Open reduction internal fixation of a left mandibular fracture sustained from blunt trauma

Historically, open treatment consisted of mandibular wiring. Transosseous wiring is very rarely employed today. Plates and screws are used to fixate bony segments (■ Figs. 5.7 and 5.8). It is important that the patient be placed into MMF during the fixation to establish the proper occlusion; this can be done manually [35], with arch bars, wires, or IMF screws.

With both open and closed treatments, the management of teeth in the line of the fracture, and their contribution to morbidity, has been debated. If the teeth meet the criteria for extraction (i.e., non-restorable due to caries, periodontal mobility, root fracture), they should be removed. However, maintaining teeth at the fracture site has not been shown statistically to influence the complication rate [36–38].

Contusions of the temporomandibular joint are treated with ice for the first 2 days and followed by heat prior to

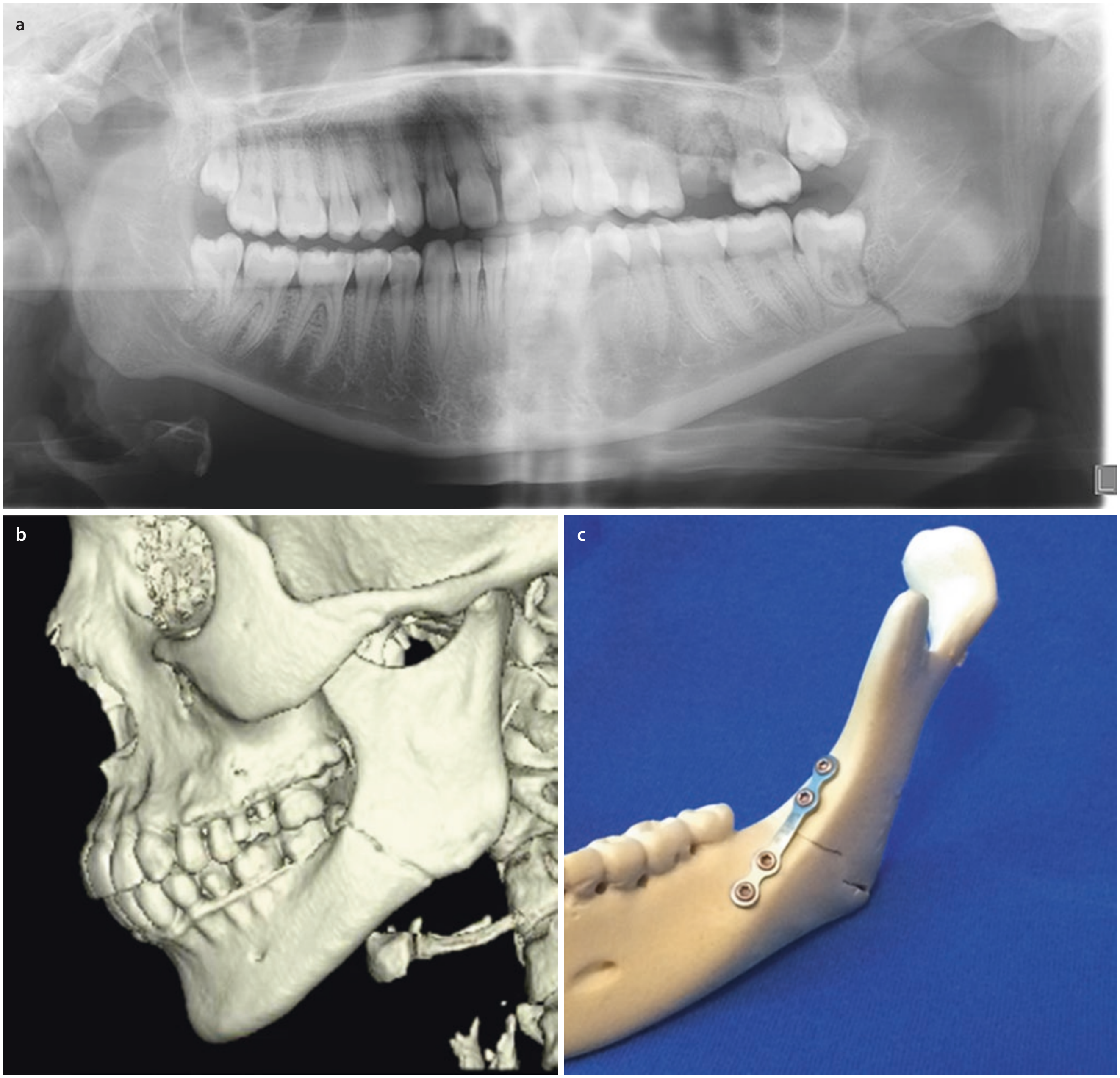


**Fig. 5.8** Demonstration of a left mandibular angle fracture on a panoramic radiograph **a**, a 3D reconstruction of this patient **b**, and a model demonstrating of fixation of the left mandibular angle using a Champy technique **c**

physical therapy thereafter until range of motion is restored. If imaging establishes a simple contusion, range of motion is usually restored with gentle range of motion exercises in the few weeks following injury. Acute dislocation and tears of the articular disc are also treated with physical therapy. Arthrocentesis to lavage hemarthrosis may be of assistance. Early arthroscopic or open repair of torn disc may be considered before scarring, shortening, and adhesion of the disc occur. This is usually performed 6 weeks or greater after the acute injury.

## 5.5 Midfacial Trauma and Management

**Midface Fractures**
- Le Fort fractures
- Zygomatico-orbital fractures
- Orbital fractures
- Nasal and naso-orbital-ethmoid (NOE) fractures
- Frontal sinus fractures

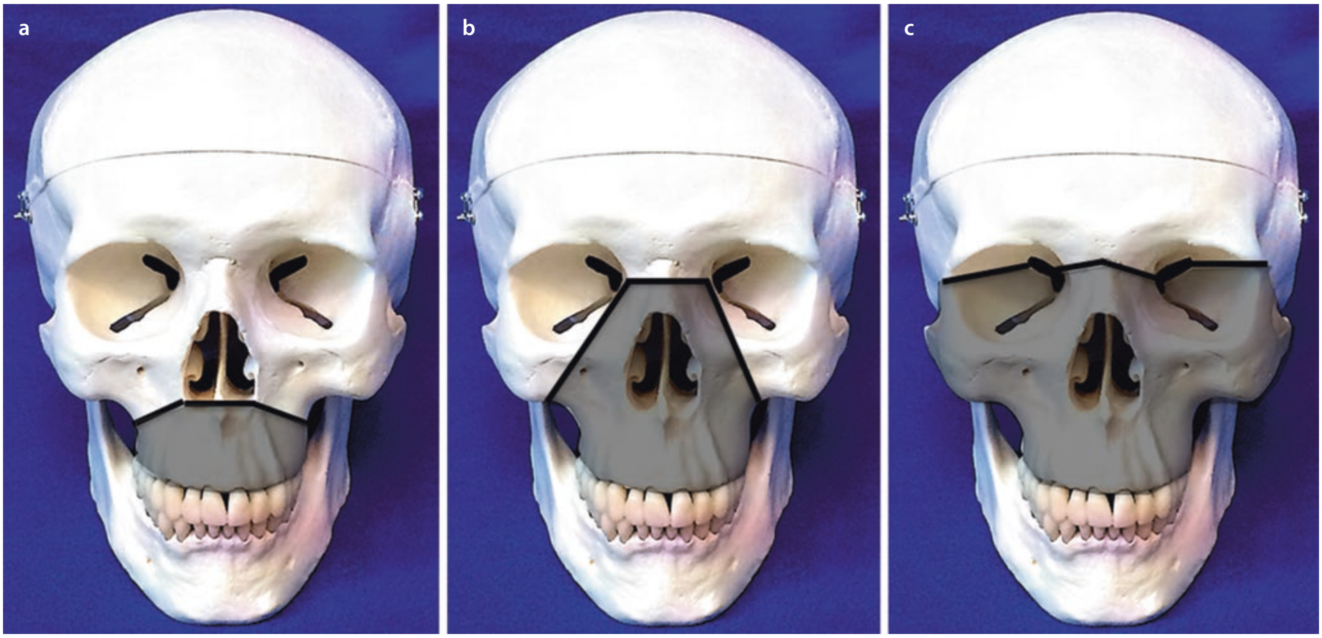



**Fig. 5.9** Differentiation of **a** Le Fort I, **b** Le Fort II, and **c** Le Fort III midfacial fractures

### 5.5.1 Le Fort Fractures

Midfacial fractures are classified on their location and frequently by degrees of comminution. Rene Le Fort described a common pattern of fractures of the midface [39]. The Le Fort I fracture separates the maxilla horizontally from the anterior maxillary sinus walls, along the lateral nasal wall, to the posterior maxillary sinus walls and the pterygoid plates (Fig. 5.9a). A Le Fort II fracture results in a fracture of the maxilla at the nasal-frontal junction extending inferiorly through the orbit to the zygomaticomaxillary region (Fig. 5.9b). The Le fort III fracture, or craniofacial separation, occurs when the nasal, zygomatic, and maxillary structures are separated from the cranial base (Fig. 5.9c).

**Fig. 5.10** Patient presented following blunt trauma to the right orbit with periorbital ecchymosis and a right orbital floor fracture. Upon superior gaze, the right globe is restricted as compared to the left. Clinical examination is consistent with entrapment of the right inferior rectus. This clinical finding requires immediate surgical evaluation and treatment

### 5.5.2 Zygomatico-orbital Fractures

Fractures of the zygoma can occur along the arch or at the articulating surfaces of the zygoma along the maxilla, frontal, sphenoid, and temporal bones. All fractures involving the frontal and maxillary articulation of the zygoma are zygomatico-orbital fractures, in that they injure the internal osseous and soft tissue structures of the orbit. These fractures are frequently related to blunt trauma to the cheek region and may be sustained during baseball, cycling, martial arts, or other activities.

### 5.5.3 Orbital Fractures

Orbital fractures can be seen in the sports setting from blunt trauma injuries to the globe. These are sustained from projectiles or person-to-person contact. Baseball and softball players are commonly affected [8]. Trauma to this region can cause visual disturbances, which can be temporary or permanent. All patients with orbital and zygomatico-orbital injuries require early ophthalmologic evaluation. Noted findings on exam may include entrapment of extraocular muscles with diplopia, vitreous hemorrhage, hyphema, retinal detachment, or proptosis with ischemic disc, among others.

When acute ophthalmologic injury is noted, the patient must undergo emergent surgical evaluation. For example, early management of entrapment may prevent external ocular muscle ischemia (Fig. 5.10).

### 5.5.4 Nasal and Naso-orbital-ethmoid (NOE) Fractures

Nasal fractures are very common fractures and can affect patient's esthetics and nasal function. Additionally, they can be associated with significant epistaxis. Pressure, and then nasal packing, is recommended for managing epistaxis. NOE fractures occur with significant force to the midfacial region and involve the nasal bones along with the orbital and ethmoidal complex. They are classified based on their level of comminution and the integrity of the medial canthal tendon.



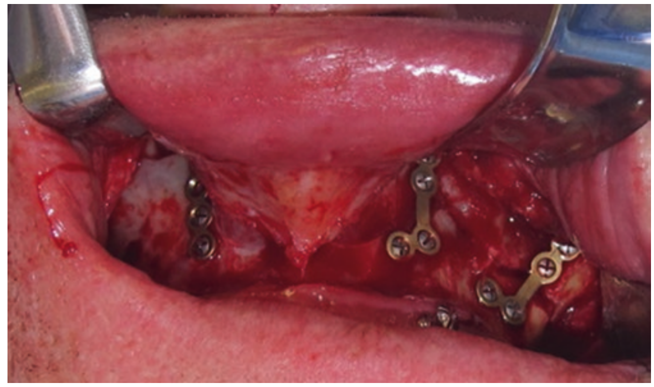


Fig. 5.11 Fixation of a Le Fort I midfacial fracture

### 5.5.5 Frontal Sinus Fractures

Fractures of the frontal sinus require a high-velocity impact to the region. The fractures can include the anterior or posterior table of the sinus. These fractures, along with NOE fractures, require monitoring for cerebrospinal fluid (CSF) leakage. Consultation with a neurosurgeon should be obtained if this is suspected. These patients frequently have other associated facial injuries (70%) and cervical spine injuries (7–14%) [40].

Fig. 5.12 Loss of left malar projection in a patient with a depressed left zygoma fracture

### 5.5.6 Management

Midfacial trauma management depends on the type of injury, the amount of displacement, as well as the associated injuries. Significant levels of comminution complicate the treatment of these fractures due to the inability to reduce and fixate the fractures. Additionally, involvement with additional facial injuries involves additional planning and sequencing to properly reestablish the patient's occlusion and facial width and height. Structure can be established in a *top-to-bottom*, *bottom-to-top*, *out-to-in*, or *in-to-out* fashion. The facial width and height can be reestablished from the stable structures that are present at the outset of the case. It is critical to reestablish the maxillomandibular complex and the patient's occlusion during the treatment.

Isolated maxillary sinus wall fractures will typically not require treatment beyond observation and sinus precautions. Maxillary fractures can be treated in a closed fashion or through open reduction internal fixation and reestablishing the occlusal relationship (Fig. 5.11). Palatal fractures can be treated with maxillomandibular fixation, transpalatal wiring, palatal splint fabrication, or plate fixation [41].

Fractures of the zygoma are treated to restore facial contours. Also, treatment can ensure proper mandibular range of motion if the displaced bone limits the motion of the coronoid process. Operative intervention is performed due to orbital floor defects, loss of malar projection (Fig. 5.12), or trismus. External (Gillies) or internal (Keen, Carmody-Batson) approaches can be used to reduce the zygomatic arch fractures. Intraoral and extraoral approaches are combined to treat zygomaticomaxillary complex fractures. A combination of screws and thin plates (<1 mm thickness) are used to fixate the fractured segments (Fig. 5.13). A coronal approach may be required when projection and facial width require complex assessment and fixation, such as when the zygomatic arch requires plate fixation. Intraoperative imaging is playing a larger role in treatment of these fractures [42].

Isolated orbital fractures are treated through incisions on the periocular skin or through the conjunctiva. The orbital floor can be reconstructed with plates (Fig. 5.14), autogenous bone, or porous polyethylene. Before and after fracture reduction, a forced duction test is performed.

Nasal fractures are largely treated through closed methods in a delayed fashion following resolution of edema. Postsurgical splints (Fig. 5.15) may be placed during a period of healing to provide support to the nasal septum (internal splints) and nasal dorsum (external splints).

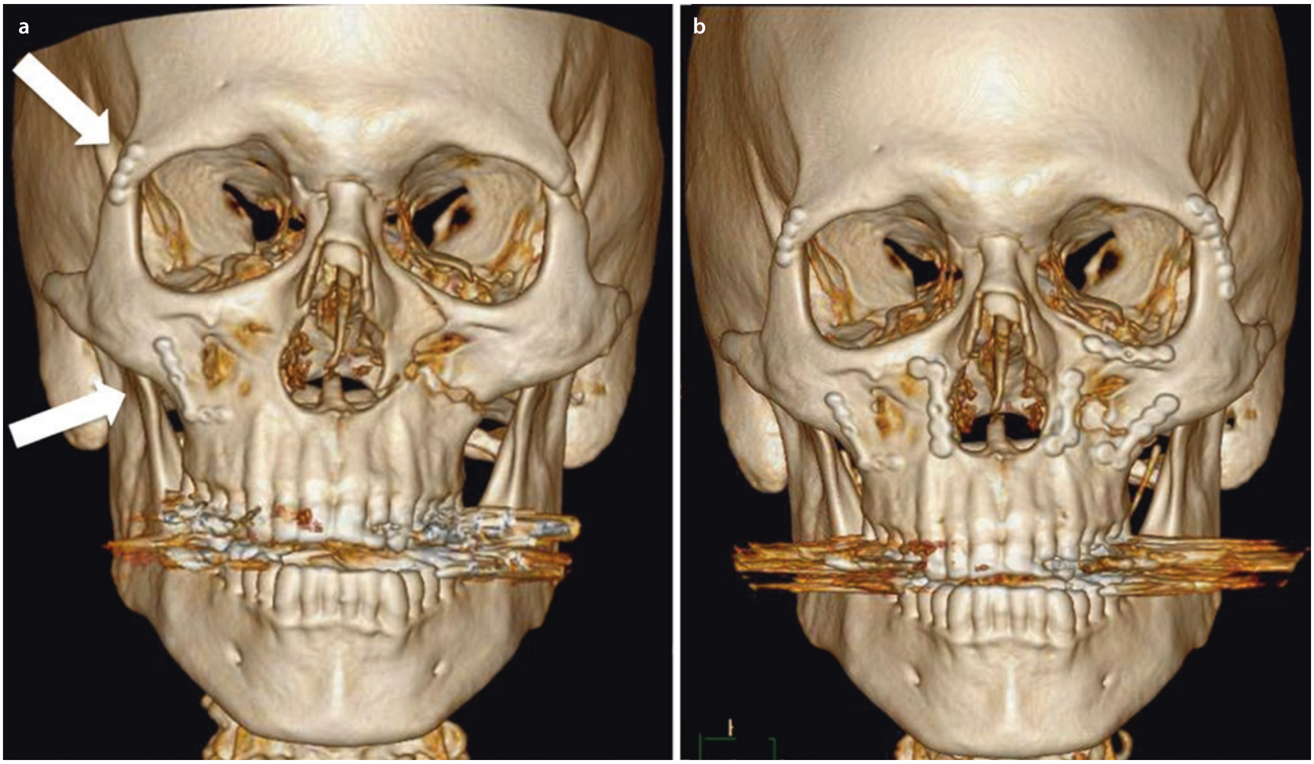



**Fig. 5.13** A 3D reconstruction of patient with previous fixation of a right zygomatico-orbital fracture (*white arrows*) who sustained multiple midfacial fractures at the time of presentation **a** and postoperatively **b**

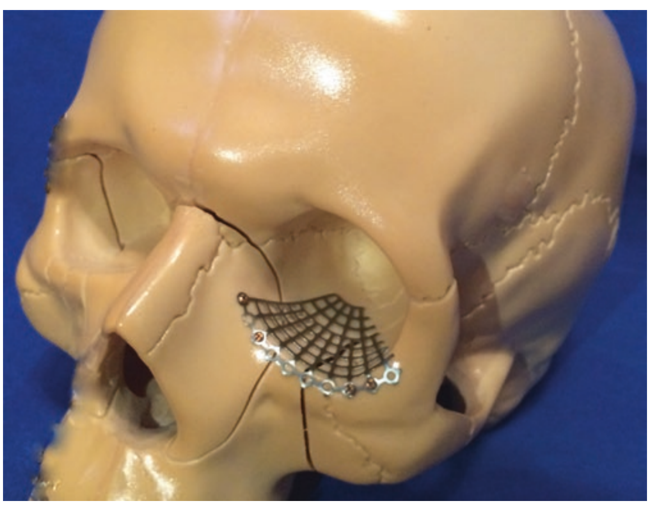


**Fig. 5.14** Plating of an orbital floor fracture

Naso-orbital-ethmoid fractures are managed through open approaches through existing lacerations or a coronal approach through the patient's hairline. Fixation plates are used to secure the bony fragments in the appropriate positions. The canthal tendon may need to be resuspended, and this can be done with wires or a canthal barb.

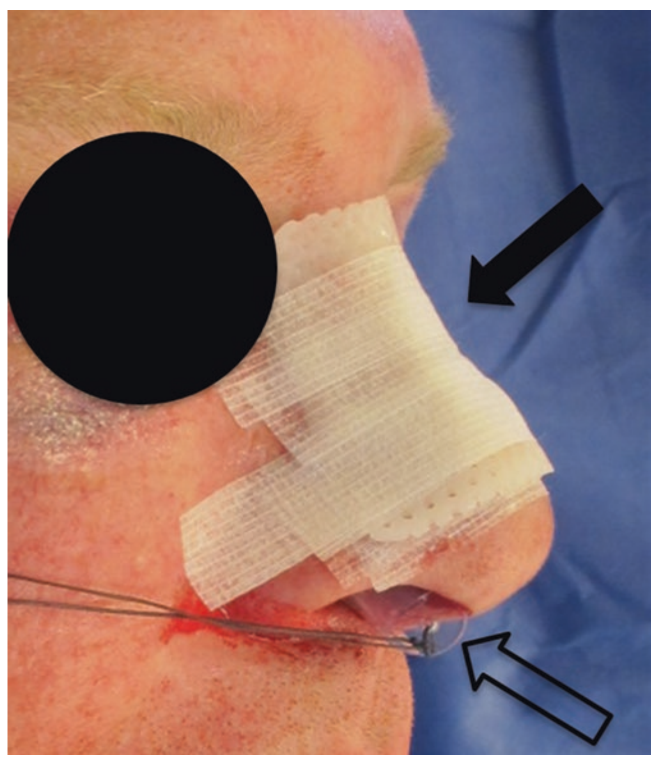


**Fig. 5.15** Placement of external (*bold arrow*) and internal (*outlined arrow*) following reduction of nasal bone fractures

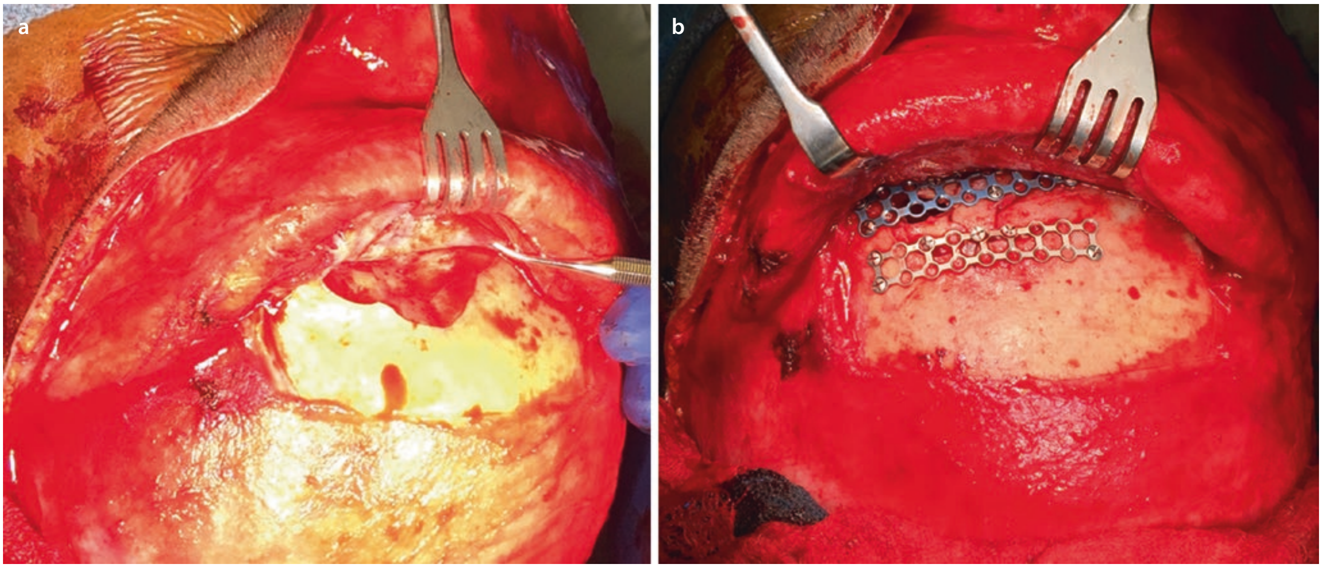


Fig. 5.16 Frontal sinus fracture exposure **a** with a coronal approach and **b** fixation of the fracture



Treatment of frontal sinus fractures is determined based on the level of displacement of the fractures as well as patency of the nasofrontal duct. Untreated or mistreated frontal sinus fractures may lead to a cosmetic deformity, meningitis, or mucopyocele formation. Fractures may require treatment along with a neurosurgical team. Plating of the frontal sinus (Fig. 5.16) is performed with thin plates (0.4–0.8 mm) to prevent visualization or palpation of the hardware through the overlying skin. This type of fracture, like NOE fractures, can be accessed through existing lacerations or a coronal approach.

## 5.6 Facial Soft Tissue Injuries

### 5.6.1 Abrasions

Abrasions involve friction along the skin or mucosa, which may completely or partially remove the epithelium depending on the depth of the injury. This type of injury is commonly encountered in the extremities of athletes when diving along artificial turf or gymnasium floors. When encountered in the facial region, they are painful and esthetically challenging for the patient. Bleeding is generally controlled with pressure. It is important for the provider to key in on areas of abrasions as potential injury clues. Chin abrasions may occur injunction with dentoalveolar trauma or mandibular fractures. Cheek abrasions in bicycle accidents may be accompanied with zygoma fractures.

Careful cleansing of abrasions is important to prevent foreign material from becoming embedded in the skin. Local or general may be required for large, deep, or contaminated abrasions in order to ensure complete debridement. Treatment consists of application of a topical antibiotic ointment (i.e., bacitracin). Dressings may be applied for patient comfort. Tetanus and antibiotic prophylaxis is recommended in contaminated wounds; however, antibiotics are not routinely administered.

### 5.6.2 Lacerations

Lacerations of the head and neck can involve many important structures in the head and neck. The face has a rich anastomosis of vessels, and hemostasis may be challenging to achieve. Direct pressure is recommended to limit any bleeding. Neurologic structures including sensory branches of the trigeminal nerve (V1, V2, V3) may be damaged with a laceration. Additionally, motor branches of the facial nerve may be affected with resulting facial paresis (Fig. 5.17). Stensen's duct or Wharton's duct may be injured in facial or floor of mouth lacerations, respectively. Periorbital lacerations must require careful attention to the lacrimal gland, nasolacrimal ducts, canthal attachments, and palpebral musculature. It is important to ensure that a thorough examination of the pretreatment state is completed and documented. Local anesthesia may complicate this exam and should not be administered prior to a comprehensive examination. Clinical photographs can also be a valuable portion of the patient's record; consent for these should be obtained according to institutional guidelines.

Lacerations can come in a variety of shapes and patterns from simple and linear to stellate and deep. As more structures are involved (vessels, nerves, muscle, vermilion border, eyelids, eyebrow), the repair becomes more involved, and specialist consultation should be sought. Patients with contaminated wounds should be treated with tetanus and antibiotic prophylaxis. Soft tissue injuries are treated following the management of the hard tissue injury, whether this includes dentoalveolar trauma or facial fractures. Treatment of the soft tissue wounds includes cleansing and debridement, hemostasis, and closure in a tension-free fashion. Primary closure may not always be desirable if avulsion injuries are also associated with the laceration.

Cleansing and debridement of the wound involves administration of local anesthesia for patient comfort. The wound is then inspected for foreign bodies (i.e., teeth, glass, grass). This may be performed with soap and brush if necessary. Pulsed irrigation with saline in a syringe is effective for this procedure. Devitalized wound margins may be conservatively removed to facilitate closure. Hemostasis can then be achieved at this point. First, direct pressure is recommended. If this is unsuccessful, the provider can proceed with vessel ligation or the use of electrocautery.

**Fig. 5.17** A laceration of the right temporal region requiring evaluation of the right frontal nerve (cranial nerve VII) function. Any laceration that lies along Pitanguy's line (white line, 1.5 cm superior to the lateral brow and 0.5 cm inferior to the tragus [43]) requires proper evaluation and treatment of any nerve injury (if present)

Following these proper steps of treatment, the provider can then close the wound. This is best accomplished in layers with the wound closed in a deep to superficial fashion, if necessary. Small wounds can be reapproximated with tissue adhesive [44]. Superficial lacerations of the intraoral tissues (buccal sulcus, buccal mucosa, gingiva) can be closed in one layer with resorbable (i.e., chromic gut) suture. Layered closure is performed if the perioral or tongue musculature is involved. Full-thickness lip lacerations (Fig. 5.18) are closed in a layered fashion proceeding from the mucosa, to the muscle, to the skin. If the vermilion border is involved, this is reapproximated first to prevent a posttraumatic deformity. The muscular layer is closed with resorbable suture (i.e., Vicryl), and the skin is closed with monofilament (5-0 or 6-0 nylon) or resorbable suture (5-0 or 6-0 fast gut). Nonresorbable sutures should be removed in 5–7 days. Antibiotic ointments can be applied to the lacerations following repair.

### 5.6.3 Bite Wounds

Animal bite injuries are beyond the scope of this text; however, athletes that participate in martial arts, wrestling, or contact sports with improper facial protection may sustain inadvertent bite injuries or penetrating tooth injuries. *Streptococcus*, *Staphylococcus*, *Eikenella*, *Prevotella*, *Peptostreptococcus*, *Fusobacterium*, and *Candida* spp. are frequently associated with human bite injuries. Baker & Moore [45] reported that sports-related human bite injuries have a higher rate of infection. There is also a concern for transmission of communicable diseases such as hepatitis B, hepatitis C, tuberculosis, syphilis, and HIV [46]. Empiric antibiotic therapy is recommended to include amoxicillin with clavulanic acid. For penicillin-allergic patients, one may consider fluoroquinolones or carbapenems [46]. Any evidence of infection should be referred for appropriate surgical management.

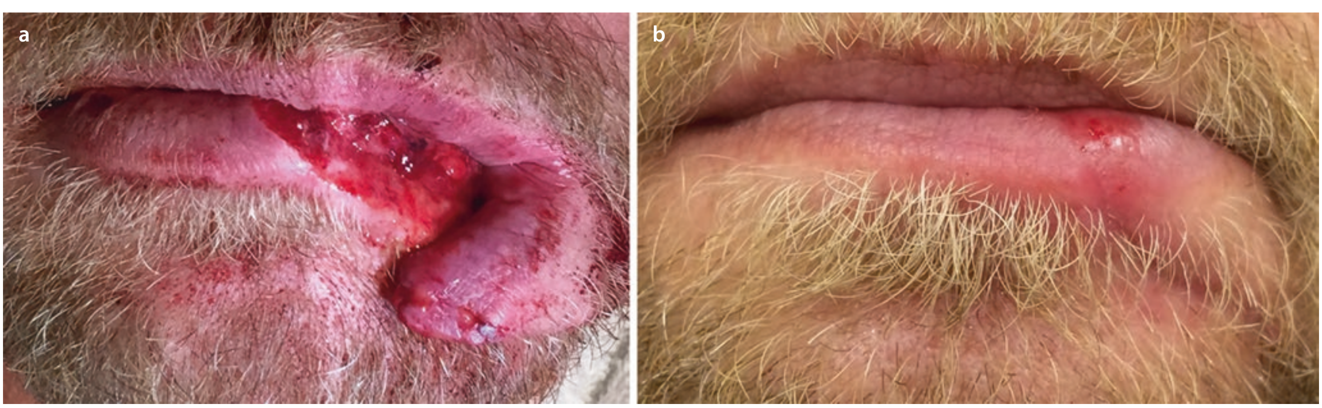


**Fig. 5.18** A laceration involving the mucosa, muscle, and skin layers of the lower lip on presentation **a** and 3 weeks following repair **b**. Residual edema exists at the site of injury

5

## 5.7 Conclusion

Athletes sustain a wide variety of facial trauma. Soft tissue injuries, dentoalveolar injuries, and facial fractures all require appropriate evaluation and treatment in order to ensure good patient outcomes. Health care providers play the important role of treating these injuries; however, the participants, parents, coaches, and providers must all continue to encourage prevention by the proper use of mouth guards, face masks, helmets, and safety restraints when participating in sporting activities.

## References


1. Conn JM, Annest JL, Gilchrist J. Sports and recreation related injury episodes in the US population. Inj Prev. 2003;9:117–23.
2. Reehal P. Facial injury in sport. Curr Sports Med Rep. 2010;9:27–34.
3. Allareddy V, Itty A, Maiorini E, et al. Emergency department visits with facial fractures among children and adolescents: an analysis of profile and predictors of causes of injuries. J Oral Maxillofac Surg. 2014;72:1756–65.
4. Hill CM, Eppley BL, Thomas DW, Bond SE, Monaghan AM, Dover MS. Etiology and prevention of craniomaxillofacial trauma. In: Booth PW, Eppley BL, Schmelzeisen R, editors. Maxillofacial trauma and esthetic facial reconstruction. 2nd ed. St. Louis: Saunders/Elsevier; 2012. p. 3–18.
5. Hwang K, You SH, Lee HS. Outcome analysis of sport-related multiple facial fractures. J Craniofac Surg. 2009;20:825–9.
6. Maladiere E, Bado F, Meninguad JP, Guilbert F, Bertrand JC. Aetiology and incidence of facial fractures sustained during sports: a prospective study of 140 patients. Int J Oral Maxillofac Surg. 2001;30:291–5.
7. Murphy C, O'Connell JE, Kearns G, Stassen L. Sports-related maxillofacial injuries. J Craniofac Surg. 2015;26:2120–3.
8. Perkins SW, Dayan SH, Sklarew EC, Hamilton M, Bussell GS. The incidence of sports-related facial trauma in children. Ear Nose Throat J. 2000;79:632–4, 636, 638.
9. Roccia F, Diaspro A, Nasi A, Berrone S. Management of sport-related maxillofacial injuries. J Craniofac Surg. 2008;19:377–83.
10. Nalliah RP, et al. Epidemiology of hospital based emergency department visits due to sports injuries. Pediatr Emerg Care. 2014;30:511–5.
11. Afrooz PN, Bukowski MR, James IB, Daniel LN, Blavijo-Alvarez JA. The epidemiology of mandibular fractures in the United States, part 1: a review of 13,142 cases from the US National Trauma Data Bank. J Oral Maxillofac Surg. 2015;73:2361–6.
12. Allareddy V, Allareddy V, Nalliah RP. Epidemiology of facial fracture injuries. J Oral Maxillofac Surg. 2011;69:2613–8.
13. Antoun JS, Lee KH. Sports-related maxillofacial fractures over an 11-year period. J Oral Maxillofac Surg. 2008;66:504–8.
14. MacIsaac ZM, Berhane H, Cray J Jr, et al. Nonfatal sport-related craniofacial fractures: characteristics, mechanisms, and demographic data in the pediatric population. Plast Reconstr Surg. 2013;131:1339.
15. Steenstrup SE, Bere T, Bahr R. Head Injuries among FIS World Cup alpine and freestyle skiers and snowboarders. Br J Sports Med. 2014;48(1):41–5.
16. Chua DY, Lo S. Orbital fracture in a professional diver. J Maxillofac Oral Surg. 2015;14(Suppl 1):81–3.
17. Brandt MT. Oral and maxillofacial aspects of diving medicine. Mil Med. 2004;169:137–41.
18. Ethan D, Basch CH, Johnson GD, Hammond R. An analysis of technology related distracted biking behaviors and helmet use among cyclists in New York City. J Community Health. 2016;41:138.
19. Gamble T, Walker I. Wearing a bicycle helmet can increase risk taking and sensation seeking in adults. Psychol Sci. 2016;27:289.
20. American College of Surgeons. ATLS: advanced trauma life support. Chicago: American College of Surgeons; 2012.
21. Halsey JN, Hoppe IC, Marano AA, Kordahl AM, Lee ES, Granich MS. Characteristics of cervical spine injury in pediatric patients with facial fractures. J Craniofac Surg. 2016;27:109–11.
22. Elahi MM, Brar MS, Ahmed N, et al. Cervical spine injury in association with craniomaxillofacial fractures. Plast Reconstr Surg. 2008; 121:201.
23. Mulligan RP, Mahabir RC. The prevalence of cervical spine injury, head injury or both with isolated and multiple craniomaxillofacial fractures. Plast Reconstr Surg. 2010;126:1647.
24. Erbele ID, Sorensen MP, Rivera A. Otologic and temporal bone injuries, triage, and management. Atlas Oral Maxillfac Surg Clin North Am. 2013;21:117–25.
25. Watanabe K, Kida W. Battle's sign. N Engl J Med. 2012;367:1135.
26. Kass JI, Ferguson BJ. Treatment of hematoma of the nasal septum. N Engl J Med. 2015;372(22):e28.
27. Olson RA, Fonseca RJ, Zeitler DL, et al. Fractures of the mandible: a review of 580 cases. J Oral Maxillofac Surg. 1982;40:23.
28. Kelly DE, Harrigan WF. A survey of facial fractures: Bellevue hospital 1948-1974. J Oral Surg. 1975;33:146–9.
29. Shetty V, Atchison K, Der-Matirosian C, Wang J, Belin TR. The mandible injury severity score: development and validity. J Oral Maxillofac Surg. 2007;65:663–70.
30. Barton JR. A systematic bandage for fractures of the lower jaw. Am Med Recorder Phila. 1819;2:153.
31. Morris C, Kushner GM, Tiwana PS. Facial skeletal trauma in the growing patient. Oral Maxillofac Surg Clin North Am. 2011;24:351–64.
32. Al-Moraissi EA, Ellis E. Surgical treatment of adult mandibular condylar fractures provides better outcomes than closed treatment: a systematic review and meta-analysis. J Oral Maxillofac Surg. 2015;73:482–93.
33. Hupp JR, Ellis E, Tucker MR, editors. Contemporary oral & maxillofacial surgery. St. Louis: Elsevier; 2008.
34. Smith BM, Deshmukh AM, Barber HD, Fonseca RJ. Mandibular fractures. In: Fonseca RJ, Barber HD, Powers MP, Frost DE, editors. Oral & maxillofacial trauma. 4th ed. St. Louis: Saunders/Elsevier; 2012. p. 293–330.
35. Laurentjoye M, Majoufre-Lefebvre C, Caix P, Siberchicot F, Ricard AS. Treatment of mandibular fractures with Michelet technique: manual fracture reduction without arch bars. J Oral Maxillofac Surg. 2009;67:2374–9.
36. Amaratunga NA. The effect of teeth in the line of mandibular fractures on healing. J Oral Maxillofac Surg. 1987;45:312–4.
37. Ellis E. Outcomes of patients with teeth in the line of mandibular angle fractures treated with stable internal fixation. J Oral Maxillofac Surg. 2002;60:863–5.
38. Neal DC, Wagner WF, Alpert B. Morbidity associated with teeth in the line of mandibular fractures. J Oral Surg. 1978;36:859–62.
39. Le Fort R. Étude expérimentale sur les fractures de la mâchoire supérieure. Rev Chir. 1901;23:208.
40. Golden BA, Jaskolka MS, Vescan A, Macdonald KI. Evaluation and management of frontal sinus injuries. In: Fonseca RJ, Barber HD, Powers MP, Frost DE, editors. Oral & maxillofacial trauma. 4th ed. St. Louis: Elsevier; 2012. p. 470–90.
41. Moss WJ, Kedarisetty S, Jafari A, Schaerer DE, Husseman JW. A review of hard palate fracture repair techniques. J Oral Maxillofac Surg. 2016;74:328–36.
42. Ellis E, Perez D. An algorithm for the treatment of isolated zygomatico-orbital fractures. J Oral Maxillofac Surg. 2014;72:1975–83.
43. Pitanguy I, Ramos AS. The frontal branch of the facial nerve: the importance of its variations in face lifting. Plast Reconstr Surg. 1966;38:352–6.
44. Bruns TB, Worthington JM. Using tissue adhesive for wound repair: a practical guide to dermabond. Am Fam Physician. 2000;61:1383–8.
45. Baker MD, Moore SE. Human bites in children: a six-year experience. Am J Dis Child. 1987;141:1285–90.
46. Stevens MR, Emam HA, Cunningham L. Management of human and animal bites. In: Fonseca RJ, Barber HD, Powers MP, Frost DE, editors. Oral & maxillofacial trauma. 4th ed. St. Louis: Saunders/Elsevier; 2012. p. 615–32.

# Restorative Considerations After Athletic Dental Trauma

*Douglas L. Lambert, Danette McNew, and Zainah Shaker*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_6

## 6.1 Introduction

In today's fast-paced and ever-changing society, the focus on health, wellness, and nutrition has led to continued growth in athletic participation among adults and children alike. Thus, increased levels of participation also lead to increased exposure and potential incidence of sports-related trauma and injury [1–9]. A dental trauma that occurs while playing a sport may have devastating consequences and can be a potential life-altering event if not properly addressed.

Regardless of the emphasis placed on prevention when it comes to participating in a sport, injuries can always occur no matter what the level of competition. The data and studies on the use of athletic mouthguards to diminish or prevent dental injuries are voluminous and should be a vital consideration for anyone who is active in a sport which might lend itself to physical contact or an accidental blow to the orofacial region [10–18]. Unfortunately, regardless of preventive measures taken, damage and injury to the oral cavity during participation in sports, specifically related to the anterior dentition, is a reality [19–24]. Injuries can involve both hard and soft tissues. Individuals with a Class II, division 1 occlusion are at greater risk. As the overjet increases, the frequency of injury proportionately increases. A child with a dental overjet of 3–6 mm has double the incidence of dental trauma than a child with an overjet of less than 3 mm. The reported frequency rate jumps threefold in children with an overjet in excess of 6 mm [25, 26]. Luxations, avulsions, tooth and root fractures, and bony fractures involving one or multiple teeth from an athletic dental injury can pose myriad outcome concerns for the affected individual, and the dental team must take various factors into account in selecting the methods and materials for treating the patient. Although much has been documented related to addressing the on-site and immediate needs of the individual, we must also consider the long-term restorative options.

The concept of evaluating the long-term restorative options for the athlete is not a new one, but little data exists in the literature regarding this topic. Numerous factors must be taken into consideration by the attending dental team in proposing the course of definitive treatment. In general, participation in a wide variety of sports activities can extend itself beyond the gymnasium or field to encompass many common recreational activities that can place one at risk for an injury, such as bicycling or skateboarding. Team sports, as well as individual activities, have exhibited their share of trauma. Some sports may pose a higher likelihood of trauma. Stratifying this risk may be a key concept when evaluating a patient's potential exposure to injury and reinjury, their need for protection, and subsequent treatment from sports-related trauma. Spinks and McClure completed a systematic literature review of sports-related injuries with children under the age of 16 and found that injuries in sports and recreation are well documented, but most studies have not quantified the specific injury in terms of exposure to risk [9]. Fos, Pinkham, and Ranalli formulated a sports classification matrix characterized by two dimensions: the velocity and the intensity of the sport [27]. Using the parameters of the matrix, the degree of risk can be assessed and potentially estimated based on the level of the intensity and velocity of the sport itself (Box "Classification of Sport Types" offers a summary of sport types and their level of intensity).

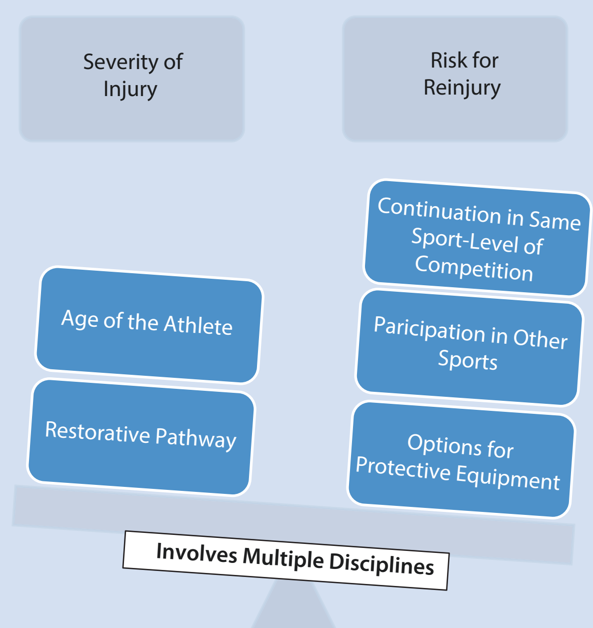
### Classification of Sport Types

1. *Noncontact*:
   - *Low Velocity*: archery, swimming, weightlifting, track, and marksmanship
   - *High Velocity*: skiing, bicycling, equestrian, skateboarding, baseball, softball
2. *Contact*:
   - Basketball, soccer, lacrosse, field hockey, and wrestling
3. *Collision*:
   - Football, ice hockey, rugby, and boxing

Fos PJ, Pinkham JR, Ranalli, DN: Prediction of sports-related dental traumatic injuries. Dental Clinics of North America 44(1): 19–33, January, 2000

As the risk variables are considered and a treatment plan formulated, the use of a decision-tree model is helpful to arrive at a practical and logical course of action. Using this type of mechanism, a systematic approach guides the practitioner, the athlete, and their family through the process in order to arrive at a path based on the science available and best-outcome scenario. Box "Factors in Restorative Treatment Planning of the Athlete" illustrates a diagramatic version of the decision process.

### Factors in Restorative Treatment Planning of the Athlete



The following list of factors should be considered:

1. *Degree or severity of the initial injury*:
   1. Hard and soft tissue involvement
   2. Loss of a tooth/teeth
   3. Bony fractures/root fractures
2. *Risk for new trauma or reinjury to the affected site*: Trauma to a previously damaged site is always a possibility. Depending on the severity of a subsequent injury, it could pose catastrophic consequences for the athlete. Proper risk vs reward assessment is prudent, especially in cases with concussions.
3. *Age of the athlete*: The younger the athlete, the higher the potential for additional trauma and/or reinjury due to the number of years remaining to participate in the sport. At-risk younger athletes will require a guarded approach to definitive or long-term restorative solutions. Use of dental implants will be governed by level of physical maturity and other risk factors and is discussed later in this chapter.
4. *Continuation in the same sport as the initial trauma/participation in other sports*: Participation in the same sport as the injury occurred or in multiple sports will influence restorative decision-making. Individuals identified as CPS (continuing to play sports) should receive special consideration when selecting both approach and materials.
5. *Level of competition*: The speed and velocity of today's athletic endeavors is rising meteorically. Athletes who continue their pursuits at the highest levels of sport may be placing themselves at risk levels above the norm, exposing themselves to higher frequency for dental trauma and possibly increased severity of the damage as a result.
6. *Option for use of protective equipment*: It has been demonstrated that the use of protective equipment can reduce the risk and degree of injury. Wearing a properly fitted mouthguard—even in a non-mandated mouthguard sport—could produce unforeseen dividends for the athlete.
7. *Multidisciplinary approach to care*: Consultation and collaboration with health professional colleagues is a vital factor for the successful long-term treatment of the athlete. Many times it becomes the role of the general dentist to be the coordinator or "quarterback" for the restorative team. This potentially includes all the dental specialties, physicians, athletic trainers, therapists, and other allies.
8. *Necessity for transitional restorations or prostheses*: The injury may lend itself to a very succinct and defined treatment, such as a fractured incisor being repaired with a direct composite. However, in more complex cases, multiple appointments with numerous specialists over an extended period of time may necessitate the use of transitional restorations for the athlete. Both removal and fixed prostheses may be considered. Some of the other factors previously listed—such as age, level of competition, and reinjury risk assessment—also play a role in the need for transitional restorative options.
9. *Materials and techniques available*: There are a multitude of direct and indirect materials and techniques for us to employ in the restoration of both soft and hard tissues. Consideration should be given to all the factors listed above when formulating a treatment plan and selecting the restorative path.

The list is not meant to be inclusive or exclusive in any manner but to simply serve as a guideline to begin the assessment of the athlete and a restorative treatment plan. It cannot be emphasized enough that any guidelines available to the profession as they relate to managing dental trauma are meant to be a template for good clinical judgment based on the specific circumstances present. Even though the practitioner may be faced with a time-sensitive treatment, it is imperative that the dentist provide the patient and/or guardian with a full disclosure of immediate and long-term outcomes so that an informed decision can be reached by the involved parties prior to initiating treatment.

## 6.2 Restorative Considerations

Once post-trauma stabilization is achieved, the next step is to complete the risk assessment for the athlete using decision-tree modeling to evaluate the path for restorative measures and develop a restorative treatment plan. The course of treatment may be classified in the one of the following ways:

- Simple—completed in one or two appointments
- Complex—requiring multiple appointments over a period of time
- Multidisciplinary—utilizing the expertise of specialist colleagues to complete the restoration

Numerous options for restoring the athlete exist as a result of advances in dental materials. Ceramics, resins, fibers, and dental implants may all play a role in the decision-making process. Working closely with dental specialists plus other healthcare members such as athletic trainers, physicians, physical therapists, and others in both the planning and treatment phases can create a path to success. The treatment plan should be formulated from the scientific literature available and sound clinical judgment based on the specific circumstances present.

Conservative modalities should be paramount in formulating the treatment plan when the athlete will continue to play sports (CPS), especially at a high level; thus, some approaches which might be considered for the mainstream patient population may not be appropriate. Use of contemporary dental materials and techniques—such as direct composite resins and adhesive bonding—is ideal for restoring many sports-related injuries. Specifically, composite resins are key because they have the ability to restore only what is missing or damaged, easily accessible, have the ability to be repaired, and are the least costly of all tooth colored materials available to the dentist. Use of the Ellis classification of tooth fractures can be helpful in quantifying the restorative path for individual teeth using direct composite resins (Box "Ellis Classification (Tooth Fractures)").

6

### Ellis Classification (Tooth Fractures)

*Ellis Class I*

- Enamel fracture: This level of injury includes crown fractures that extend through the enamel only. These teeth are usually nontender and without visible color change, but have rough edges.

*Ellis Class II*

- Enamel and dentin fracture without pulp exposure: Injuries in this category are fractures that involve the enamel as well as the dentin layer. These teeth are typically tender to the touch and to air exposure. A yellow layer of dentin may be visible on examination.

*Ellis Class III*

- Crown fracture with pulp exposure: These fractures involve the enamel, dentin, and pulp layers. These teeth are tender (similar to those in the Ellis II category) and have a visible area of pink, red, or even blood at the center of the tooth.

*Ellis Class IV*

- Traumatized tooth that has become nonvital with or without loss of tooth structure.

*Ellis Class V*

- Luxation: The effect on the tooth that tends to dislocate the tooth from the alveolus.
- Teeth loss due to trauma.

*Ellis Class VI*

- Avulsion: The complete separation of a tooth from its alveolus by traumatic injury.
- Fracture of root with or without loss of crown structure.

*Ellis Class VII*

- Displacement of a tooth without the fracture of crown or root.

*Ellis Class VIII*

- Fracture of the crown en masse and its replacement.

*Ellis Class IX*

- Fracture of deciduous teeth.

## 6.3 Case Reports

The following cases illustrate the use of composite resin materials to restore three commonly seen injuries: fractured anterior permanent tooth fragment and reattachment of the fractured segment, fractured anterior permanent tooth fragment without reattachment, and loss of a permanent incisor.

**Case Study**

### Case 1: Reattachment of a Fractured Anterior Tooth Fragment (Ellis Class I or II)

The reattachment of a fractured anterior tooth segment can be the preferred option for managing coronal tooth trauma when the fragment is available and there is minimal or no compromise of the biologic width. This technique has a long-standing success rate and can provide excellent esthetics because it maintains the tooth's original anatomic form, shade, and surface texture. In addition, the positive social and emotional effects to the patient of rebonding the fragment cannot be overlooked. Numerous reports are cited in the literature spanning four decades and justify its use in both vital and nonvital teeth [28–74]. The first reported case of a nonvital tooth reattachment was documented by Chosack et al. in 1964 and involved an endodontically treated fractured incisor and reattachment of the segment with the use of a post and core fitted to the fragment and then cemented into the tooth body [75]. Tennery was the first to employ the acid-etch technique for the reattachment of a fragment to a vital tooth in 1978 [76]. Andreasen et al. demonstrated a 25% retention rate of reattached coronal fragments after 7 years [77], while Calvalleri and German showed a 90% retention rate after 5 years [78].

Utilizing an acid-etch technique, contemporary dentin/enamel bonding agents and composite resins, the ability to bond a fractured tooth segment epitomizes the use of conservativeness and minimally invasive techniques. However, there are variables within the protocol which could influence the outcome such as preparation design, luting materials, and storage media of the fractured segment. One study compared the fracture strength of sound and restored anterior teeth using a resin composite and four reattachment techniques and concluded that fracture resistance results improved when an enamel bevel was applied prior to the adhesive system (95.8%), an internal groove was made (90.54%), or the composite was overcontoured on the facial (97.2%) in comparison to just bonding the fragment (37.09%) [79].

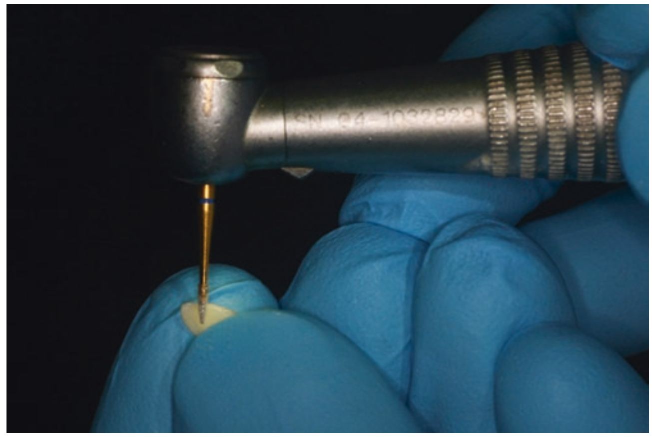
Use of various luting materials has been studied over the years, and the development of the enamel/dentin adhesives has led to their use as the system of choice for reattachment of tooth fragments. Andreasen et al. reported that the use of a dentin bonding agent with the acid-etch technique improved both fracture resistance and retention rate of the tooth fragment [36], while Farik et al. found that most fifth-generation bonding agents increased fracture resistance when used with an unfilled resin [80]. Almuammar and colleagues found that compomers produced higher bond strengths than resin-modified glass ionomer cements (RMGI), but neither could match those of composite resins when used as luting materials [81]. Use of dual-cure resin cements has shown fracture resistance lower than fragments reattached with light-cured composite resins [82]. Flowable and viscous composites with particle size classifications of hybrid, microhybird, microfilled, and nanofilled have all demonstrated the ability to serve as effective luting materials when used with adhesive systems.

Another factor in a successful reattachment is adequate hydration of the tooth fragment prior to the procedure. Limited research has been done on this topic, but proper hydration is critical to maintaining the vitality and esthetic appearance of the tooth. Due to the hydrophilic nature of many of the dentinal adhesive systems, adequate hydration allows for quality bond strength values and improves the strength of the final restoration [57]. A variety of storage media have been evaluated ranging from tap water, saline, milk, egg albumin, 50% dextrose solution, and most recently coconut water. One study found that preservation of the tooth fragment in egg albumin or 50% dextrose resulted in higher bond strengths compared to tap water [62]. Sharmin and Thomas reported that saline-stored fragments gave higher fracture resistance than those kept in milk or dry [83], and Prabhakar et al. determined that dairy milk yielded the best adhesive results and egg white the poorest [70]. While there is little doubt that hydration plays an important role in the overall attachment result, further research is needed to fully understand the merits and benefits of each media (▪ Figs. 6.1, 6.2, 6.3, 6.4, 6.5, 6.6, 6.7, 6.8, 6.9, and 6.10).

▪ **Fig. 6.1** A 12-year-old female presents with a fractured maxillary left central incisor as a result of a sports-related trauma (facial view)

▪ **Fig. 6.2** A 12-year-old female presents with a fractured maxillary left central incisor as a result of a sports-related trauma (lingual view-mirror image)

▪ **Fig. 6.3** The arch wire and bracket are removed to improve access. A 360° enamel bevel is prepared using a diamond bur

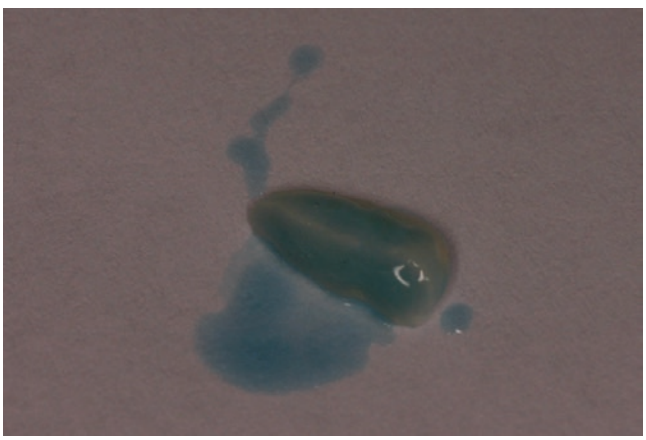


▪ **Fig. 6.4** The fractured tooth segment was kept and stored in cow's milk by the parent. The segment was rinsed with water and then carefully prepared with a similar 360° enamel bevel

▪ **Fig. 6.5** The tooth was isolated with a specialized clear matrix prior to the bonding procedure

■ **Fig. 6.6** A total-etch technique was employed to both the tooth and the fragment for 15 s and then rinsed with a water spray and gently dried with air. A universal dentin bonding agent was applied to both the tooth and the segment according to manufacturer's directions. The tooth segment was reattached to the tooth using a flowable composite and light cured with a LED source for 20 s from both the facial and lingual aspects

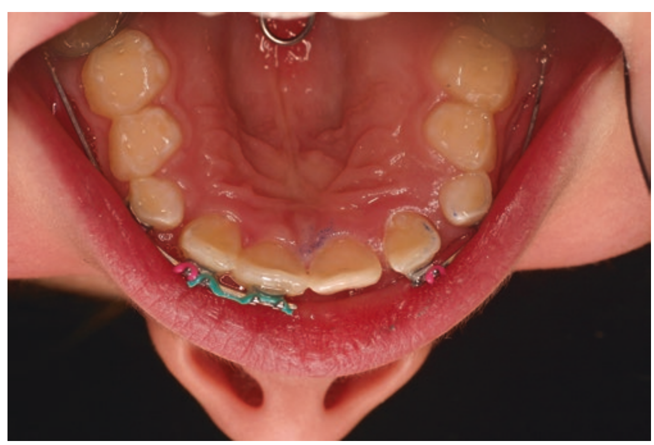




■ **Fig. 6.7** A total-etch technique was employed to both the tooth and the fragment for 15 s and then rinsed with a water spray and gently dried with air. A universal dentin bonding agent was applied to both the tooth and the segment according to manufacturer's directions. The tooth segment was reattached to the tooth using a flowable composite and light cured with a LED source for 20 s from both the facial and lingual aspects

■ **Fig. 6.8** The restored tooth was smoothed and polished with 12-fluted, spiral-bladed carbides and abrasive impregnated silicone cups and points. Occlusion and excursive movements were evaluated

■ **Fig. 6.9** Final view of the reattached tooth fragment displaying excellent shade match due to the proper hydration of the fractured segment and enamel bevel (facial view)



■ **Fig. 6.10** Final view of the reattached tooth fragment (lingual view-mirror image)

## Case Study

### Case 2: Restoration of a Fractured Anterior Tooth (Ellis Class II)

In situations where the fracture tooth fragment is not reclaimed, it presents itself in multiple pieces or is simply unusable, the dentist is faced with the challenge of rebuilding what is lost using artificial materials. Composite resins offer the dentist the best overall solution to this esthetic and functional dilemma. The advancements made since Buonocore first conceived of bonding to enamel with the introduction of acid-etching [84] and Bowen's historic work with resins [85] have been exponential. While the reattachment of the patient's own tooth segment is the ideal dental material, resins offer the next best choice for a conservative restoration especially for an active athlete who may encounter another orofacial trauma. The use of modern composites coupled with contemporary bonding agents and matrices allows for great flexibility, and its documentation in the literature as a successful direct restorative is ubiquitous. Although the severity of the fracture (Ellis Classes I–III) plays a key role in the decision-making process of choosing a restorative material, direct resins can act as both a transitional and a permanent solution after an athletic trauma in all ages (Figs. 6.11, 6.12, 6.13, and 6.14).

**Fig. 6.11** A female adult presented from the hospital emergency room after a bicycling accident. A wire trauma splint had been placed and numerous enamel/dentin fractures covered with a glass ionomer material. Endodontic therapy had been initiated on the maxillary right central incisor

**Fig. 6.13** After administration of local anesthesia, the glass ionomer material was removed with a rotary instrument and the fractured areas assessed (Ellis I and II). An enamel bevel was placed with a diamond bur and each tooth isolated with a specialize clear matrix. A total-etch technique and a universal bonding agent were used along with a microhybrid direct composite material to restore the fractured teeth

**Fig. 6.12** Numerous enamel/dentin fractures covered with a glass ionomer material. Endodontic therapy had been initiated on the maxillary right central incisor (lingual view-mirror image)

**Fig. 6.14** Final image of the restored maxillary anteriors after finishing and polishing

6

Case Study

### Case 3: Replacement of Lost/Missing Permanent Incisor in an Immature Arch

Diagnosis and timely treatment are important in saving a traumatized tooth, especially in the anterior segment of the arch. Yet with all best efforts to save and restore a damaged tooth, the concept of inevitable tooth loss is a reality. There is minimal research on the "best treatment" option for an active athlete in restoring a lost tooth or even multiple teeth. Common sense guidelines based on existing research should be evaluated to determine recommendations for our active athletes. There are a variety of replacement options for missing teeth including removable appliances (partials, flippers, or Essix tooth maintainers) and fixed appliances (resin-based, metal-based, and ceramic-based bonded bridges, cantilever, and conventional bridges). Traditional bridgework in the young athlete may be contraindicated and can result in loss of vitality of abutment teeth, while removable prostheses can lead to significant plaque accumulation and self-image concerns with esthetics [86–88]. All have been used throughout history with various levels of success and have been the only available options to our patients and patient-athletes prior to the advent of root-formed dental implants. However, one option which has demonstrated to be an excellent transitional restoration for the active athlete is the use of fiber reinforcement and composite to fabricate a bonded bridge. Fiber reinforcement has been present in dentistry for many years and appears in several forms. Uses span from splints for traumatized or periodontally involved teeth, fiber-based posts, orthodontic retainers, and reinforcement of indirect provisional restorations. Various materials lend themselves to the use in a fiber-based modality and can include polyethylene, glass, quartz, nylon, and Kevlar™ to name a few. The fabrication of a fiber-reinforced composite bridge has been successfully documented in the literature in both direct and indirect applications and extends itself to use in trauma-related cases, as well as congenitally missing teeth [89–106]. The definitive work on a direct fabrication method was reported and enhanced by Belvedere [107, 108]. The fiber-reinforced composite bridge has lent itself to be a conservative alternative for a missing anterior tooth instead of more invasive options, such as traditional fixed prostheses or dental implants. It may be an especially good choice for the athlete who is still growing (where an implant may be contraindicated) or the active athlete who may run the risk of more extensive damage should they experience a subsequent trauma to the same site (▪ Figs. 6.15, 6.16, 6.17, 6.18, 6.19, 6.20, 6.21, 6.22, 6.23, 6.24, 6.25, 6.26, 6.27, 6.28, 6.29, 6.30, 6.31, 6.32, 6.33, 6.34, 6.35, 6.36, 6.37, 6.38, 6.39, 6.40, and 6.41).

▪ **Fig. 6.15** Facial pre-op photo of missing maxillary right lateral incisor [1–2] prior to restoring with a fiber-reinforced direct composite bridge

▪ **Fig. 6.16** Pre-op photo of missing maxillary right lateral incisor [1–2] prior to restoring with a fiber-reinforced direct composite bridge (lingual view-mirror image)

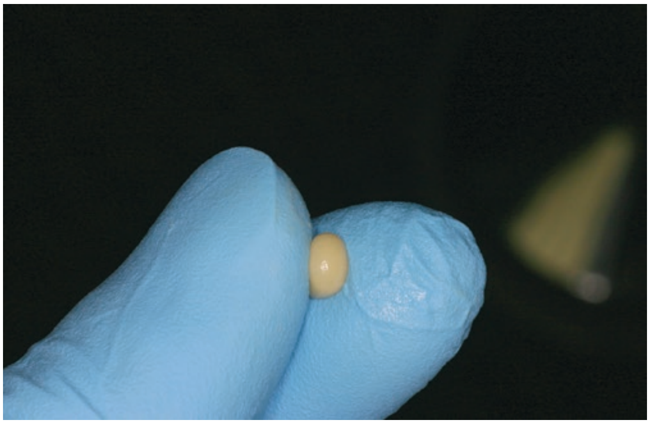


**Fig. 6.17** After shade selection, the gingival portion of the pontic is created by expressing the desired composite and rolling it into a ball

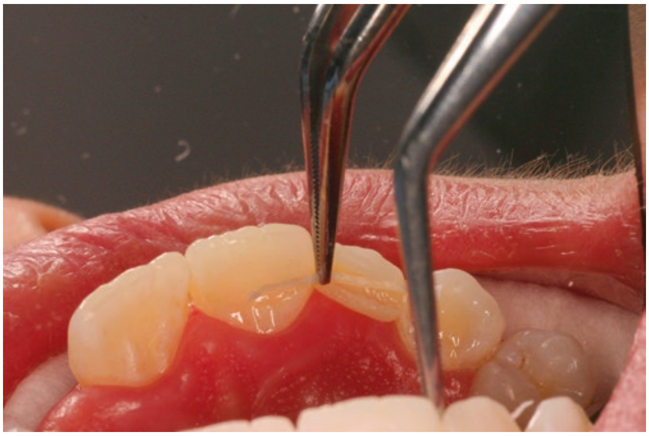
**Fig. 6.18** Gingival portion is placed and shaped with hand instruments to create a pontic on the clean tissue ridge. Cure with a LED light source for 40 s. Remove the pontic, and polish the tissue-bearing side with abrasive cups as necessary to create a smooth surface

**Fig. 6.19** Preparation of the abutment teeth is accomplished in a conservative fashion on the lingual to create a cupped-out bevel

**Fig. 6.20** Bevel interproximally in the contact areas with diamond burs

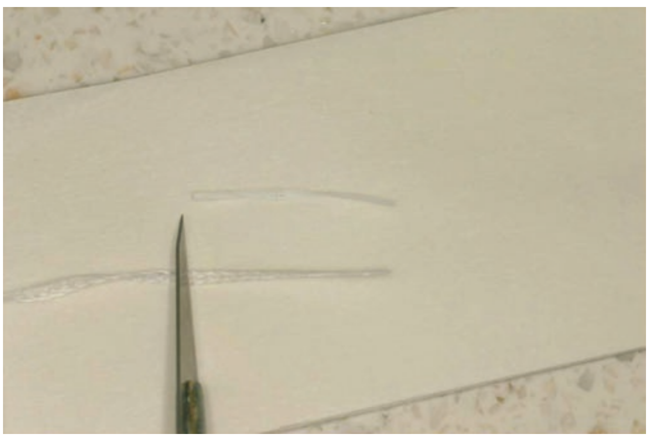
**Fig. 6.21** A total-etch technique is utilized for 15 s and then removed with a water spray and air dried. A universal bonding agent is applied over all etched surfaces according to the manufacturer's instructions and exposed to the LED curing light

**Fig. 6.22** The pontic is spot-bonded to the abutment teeth with a flowable resin after application of a universal bonding and exposed to the LED light for 20 s. Note the lingual slot which has been prepared into the composite pontic with an inverted cone diamond which will allow the fibers to be placed into the body of the pontic



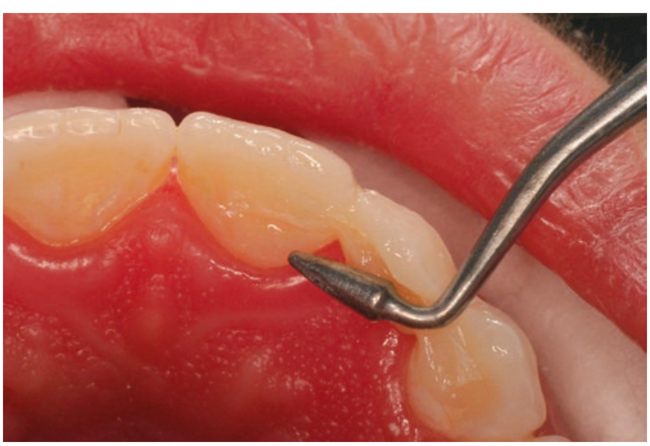
■ **Fig. 6.23** Measure the length of fibers needed for the bridge using a piece of dental floss to mimic the fiber bundle

■ **Fig. 6.26** Syringe a body composite across the lingual to fill the remaining preparations



■ **Fig. 6.24** Cut the fibers to proper length using a Bard Parker blade

■ **Fig. 6.27** Manipulate composite with an instrument to facilitate good contours prior to curing with the light source



■ **Fig. 6.25** Coat the fibers with a flowable resin composite, and place them into the lingual groove created in the abutment teeth and the pontic. Press them into the preparation with an instrument and then cured with LED light source for 40 s from the facial and lingual aspects

■ **Fig. 6.28** The final pontic portion is created by re-etching and applying the universal bonding agent prior to the remaining layer(s) of incisal shaded composite

**Fig. 6.29** Shaping of the composite is done with instruments and brushes and then exposed to the light source for an additional 40 s from the facial and lingual aspects

**Fig. 6.30** Shaping is done with instruments and brushes and then exposed to the light source for an additional 40 s from the facial and lingual aspects

**Fig. 6.31** Contouring, shaping, and polishing are completed with 12-fluted, spiral-bladed carbides and abrasive cups and points and final occlusion evaluated

**Fig. 6.32** Contouring, shaping, and polishing are completed with 12-fluted, spiral-bladed carbides and abrasive cups and points and final occlusion evaluated

**Fig. 6.33** Contouring, shaping and polishing are completed with 12-fluted, spiral-bladed carbides and abrasive cups and points and final occlusion evaluated

**Fig. 6.34** Additional example of a missing maxillary left lateral incisor (pre-op retracted view)

Fig. 6.35 Ovate pontic site ready for placement of a fiber reinforced composite bridge (pre-op lateral view)

Fig. 6.38 Pre-op lateral view of a Maryland bridge replacing a maxillary right lateral incisor which was lost after a failed endodontic treatment. Note the poor shade and contour

Fig. 6.36 Post-op lateral view of a fiber-reinforced direct composite bridge replacing a maxillary left lateral incisor

Fig. 6.39 Pre-op lingual view of the Maryland bridge (mirror image)

Fig. 6.37 Post-op frontal view of a fiber-reinforced direct composite bridge replacing a maxillary left lateral incisor

Fig. 6.40 Post-op lingual view of a fiber-reinforced composite bridge replacing the Maryland bridge on a maxillary right lateral incisor which was lost after a failed endodontic treatment (mirror image)

**Fig. 6.41** Post-op lateral view of a fiber-reinforced composite bridge replacing the Maryland bridge

## 6.4 Implants for Athletes

As previously discussed, tooth replacement prostheses range from removable appliances such as partials, flippers, or Essix tooth maintainers to fixed appliances such as resin-reinforced bridges, cantilever, or conventional bridges. All of which have been used throughout history with success. For years, these were the only available options to our patients and patient-athletes. During the past two decades, osseointegrated dental implant-supported prostheses have evolved into viable and, perhaps in some instances, the best replacement choice for our patients. The key is to determine if there is an advantage of one restorative approach over another or if there is a definitively superior choice for an athlete who will continue to play sports (CPS) after a significant orofacial trauma and subsequent tooth loss. In order to determine the best tooth replacement option(s) for our active athletes, we must consider the fact that it may not be the same option available as for our nonathletic patients. The anterior maxilla is the most traumatized region in the mouth, especially during childhood, and a repeat trauma to a restored site is a real possibility in an athlete at any age [109–113]. The resulting damage from a subsequent orofacial injury is concerning and could have unforeseen repercussions for both the individual and the restoring dental team. There is minimal research on the "best treatment" option for an active athlete in restoring a lost tooth or even multiple teeth in the anterior dentition. In addition, there is limited documentation in the literature regarding the use of dental implants as prosthetic replacements and the effects of additional injury regardless of source of the subsequent trauma [114–118].

Flanagan discussed that an impacting force could possibly fracture the bone surrounding an implant and that the interaction of the bony housing and the implant interface from a sudden external force is unknown. His case report documented secondary trauma from a bottle strike to the implant-supported crown on a maxillary left central incisor. He theorized that the cortical bone provides a protective "energy-absorbing mechanism" in the collagen polymer which helps to prevent microcracking and fracture of the bone. Vulnerable areas for damage from a blow would be the implant fixture, the implant components, and the prosthesis. He concluded that an implant encased in an adequate volume of cortical bone could successfully survive a severe sudden traumatic event of significant force [114].

Stuebinger, Hodel, and Filippi presented a case of trauma due to a fall 3 years after replacement of the maxillary central incisors with implants and porcelain fused-to-metal (PFM) crowns resulting in a bent fixation screw in one of the units. They speculated as to the type and degree of deformation which would occur in an implant as opposed to a natural tooth due to the elastic module. Their assumption was that a load delivered beyond the normal physiologic range would not "fracture the implant, but in strong tension peaks within the bone (cause) failure of the intraosseous connecting forces and, ultimately, in a fracture [115]." In another case report, the authors cited a blunt trauma to implant-retained PFM crowns on 1-1 and 2-1 and concluded that "the impact of the force applied to the implant most probably resulted in fracture of the bone surrounding the recently inserted implants. This is potentially a more serious outcome following trauma to an implant [116]."

Consequently, common sense guidelines based on existing research available should be examined and evaluated to determine recommendations for use of osseointegrated implants our active athletes.

The following concepts should be considered thoroughly during the treatment planning phase:

- The initial reason to consider an implant-supported prosthesis
- Quality of the site for the placement of the implant(s)
- Functional stresses which may be placed on the implant and the prosthesis
- Age of the athlete
- Athletic activity—continue to play sports (CPS) and level of competition
- Ancillary factors

1. *The initial reason to consider an implant-supported prosthesis*: Dental implants have become predictable, practical, and reliable replacements for non-restorable, lost, or trauma-related ankylosed teeth. Proper treatment planning should involve a multidisciplinary approach and take into consideration all aspects of the specific patient situation and what is necessary for a favorable outcome. The practitioner should weigh the advantages and disadvantages associated with the various dental restorative options.
2. *Quality of the site for the placement of the implant(s)*: Tooth loss can result in bone loss and soft tissue changes which can impact the immediacy of the implant placement. In general, the facial cortical plate surrounding the maxillary anterior teeth can

be very thin and porous. Tooth loss can result in a 40–60% decrease in bone volume from resorption during the first year, mainly on the labial aspect of the alveolar ridge. The bony ridge can then shrink or migrate to the palate in relation to the adjacent teeth [119, 120]. Additional augmentation procedures may be necessary prior to implant placement to allow for sufficient hard and soft tissue architecture and esthetic restoration of the injured site. Therefore, management and preservation of both bony and gingival contours are critical in the esthetic and functional restorative decision-making [121–123].

6

3. *Functional and traumatic stresses placed on the implant and prosthesis*: An osseointegrated implant can be at risk when subjected to chronic direct non-axial forces of occlusion [124, 125]. Even with our best efforts to control normal functional stresses on dental implants and their supporting prostheses, there remains the potential for a repeat trauma to the site [126]. Due to the limited data available regarding secondary trauma and the effect on dental implants, more scientific studies, case reports, and research are necessary. Gaining a greater understanding of the mechanisms involved and the resulting sequelae to the bone, implant, components, and prosthesis when an abrupt force is sustained is paramount.
4. *Age of the athlete*: There is a multitude of data and research dedicated to the placement of dental implants in the population based on the cessation of growth. Our young athletes are another concern. Many of our athletes have not reached an age of maturity which would allow for implants to be placed [127, 128]. Heij, Opdebeeck, van Steenberghe, Kokich, Belser, and Quirynen note that osseointegrated implants "do not participate in changes within the jawbones" and for patients, depending upon their facial profile, could create concern for the future [129]. This suggests waiting until growth ceases. Schwartz-Arad, Levin, and Ashkenazi state placement of implants during childhood is contraindicated but advise that the coordination of sequencing of any restoration(s) should occur at the time of trauma. They suggest the options for treatment are orthodontic closure of the space and reshaping teeth; tooth extraction and maintaining space with a provisional, orthodontic extrusion of root tip if untreatable or complicated root fracture with a provisional crown; tooth transplantation; intentional extraction and immediate tooth replantation; distraction osteogenesis; and decoronation—all dependent on the individual [130]. Research still needs to improve in the area of implants for children and presently leads us to be more careful and wait for implants on children and adolescents until growth ceases.
5. *Athletic activity—continue to play sports (CPS) and level of competition*: The continuation of sporting activities, regardless of the level of competition, places the individual at an increased risk for a subsequent orofacial trauma. The particular sport also plays a role in the risk assessment and potential likelihood of reinjury to the dentition as reported by Fos, Pinkham, and Ranalli [27] (Box "Classification of Sport Types").
6. *Protection*: If the decision is made to utilize osseointegrated implants as a part of the restorative process, consideration must be given to decrease the risk factors for the active athlete. This would include the proper use of all mandated protective equipment—facemasks, headgear, and shields—and a custom-fitted mouthguard. Use of a custom mouthguard for those athletes with implant-supported restorations would be highly advisable in non-mouthguard-mandated sports as well. Research on the topic of an improved mouthguard designed for athletes with anterior dental implant-supported prostheses has been reported in the literature. The use of a novel dual component material comprised of EVA (ethyl vinyl acetate) and porous rubber was tested. It displayed significantly higher shock-absorbing ability when compared to the standard EVA mouthguard. Since there is less physiologic flexibility for loading and traumatic forces with an implant-supported prosthesis compared to a normal dentition because of their rigid integration with the bone, it could potentially be an effective mouthguard material in dissipating stresses from a physical event [131].

The attached charts at the end of the chapter are provided to help in decision-making for our athletes (Charts 6.1, 6.2, 6.3, and 6.4).

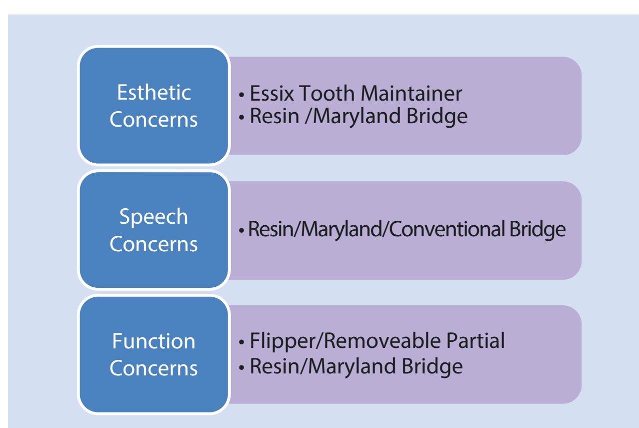



**Chart 6.1** Active athlete less than 18 years old

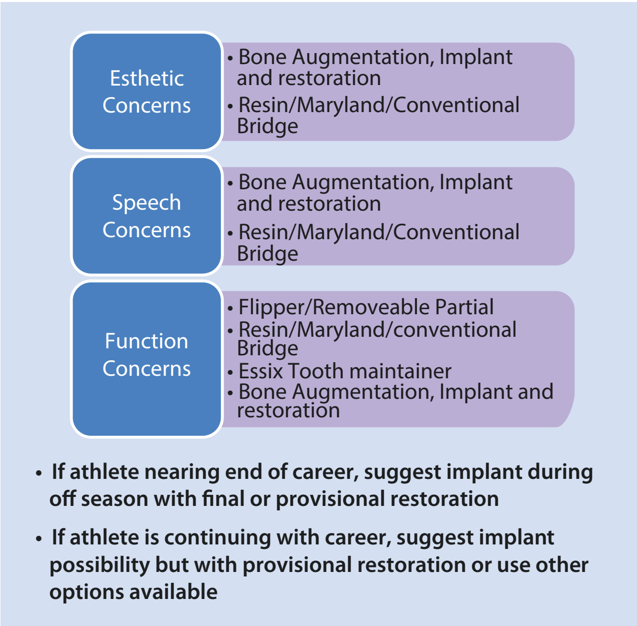



**Chart 6.2** Active athlete greater than 18 years old

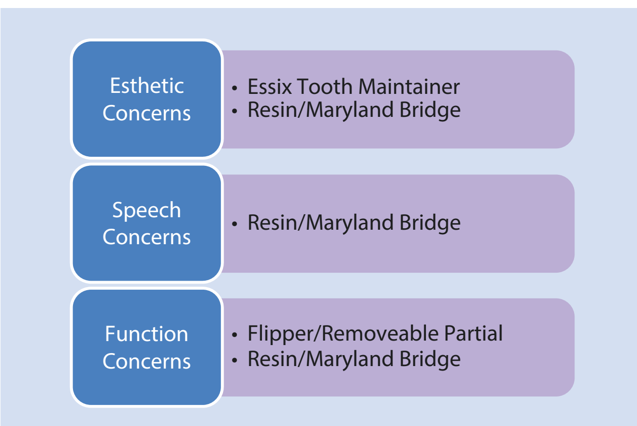



**Chart 6.3** Inactive athlete less than 18 years old

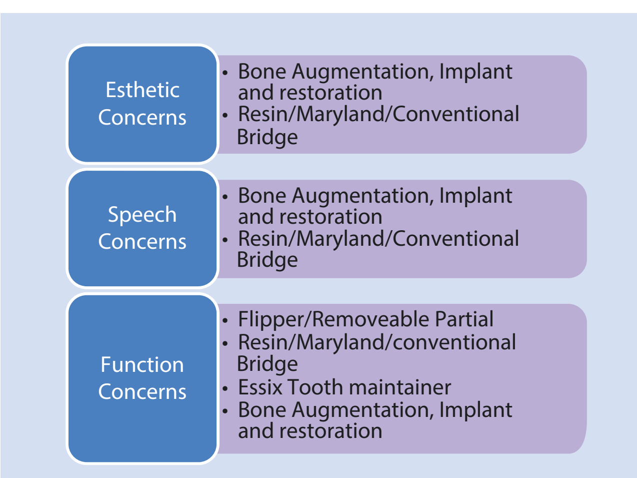



**Chart 6.4** Inactive athlete greater than 18 years old

## 6.5 Conclusions

Orofacial and dental trauma because of a sporting activity can be a life-changing event. Damage to the athlete can have an impact far beyond the initial injury, and the process by which the evaluation, treatment planning, and restorative methods are undertaken plays a significant role in the final outcome for the individual. Preventive measures, such as protective equipment and use of custom-fitted mouthguards, can be taken to decrease the potential risk or severity of a dental trauma; however, damage and injury to the oral cavity during participation in sports, especially the anterior dentition, is a reality. Guidelines exist for addressing the on-site or immediate needs after an accident, yet we must pause and consider the long-term restorative options available for the athlete.

The use of a decision-tree model can be helpful for the practitioner to evaluate the numerous factors which should be considered in the treatment planning process. They include the degree or severity of the initial injury, the risk for new trauma or reinjury to the affected site, the age of the athlete, continuing to play sports (CPS), the level of competition, option for use of protective equipment, collaborating with other health professionals, necessity for transitional restorations, as well as the definitive treatment. A melding of the dental research with a best-practices approach can help in defining a specific protocol for the athlete. Conservative and minimally invasive techniques should weigh heavily in the treatment plan, especially when the athlete will continue to play sports and at a high level where the risk for new or additional trauma is present. Reattachment of a fractured tooth segment offers the ultimate restorative procedure in terms of conservation, esthetics, and simplicity. Modern composite resins and adhesives yield infinite potential for an additive solution to dental trauma cases involving one or more fractured teeth. The incorporation of fiber reinforcement opens a multitude of transitional prostheses for the active athlete, while they are still playing or actively growing. While the use of dental implants is not recommended in the growing or immature individual, it is not contraindicated for a mature athlete. However, the limited discussion in the literature suggests that more research is needed in this area to help define a proper role for the dental implant while a person is still participating in sports at a high level. Until that time, the use of a conservative, best-practice model would suggest guarded use of dental implants in the anterior dentition to limit the potential devastating consequences of reinjury to the orofacial region.

## References

1. Young EJ, Macias CR, Stephens L. Common dental injury management in athletes. Sports Health. 2015;7(3):250–5.
2. Needleman I, Ashley P, Petrie A, et al. Oral health and impact on performance of athletes participating in the London 2012 Olympic Games: a cross-sectional study. Br J Sports Med. 2013; 47(16):1054–8.

3. Saini R. Sports dentistry. Natl J Maxillofac Surg. 2011;2(2):129–31. https://doi.org/10.4103/0975-5950.94465.
4. Dhillon BS, Sood N, Sood N, Sah N, Arora D, Mahendra A. Guarding the precious smile: incidence and prevention of injury in sports: a review. J Int Oral Health. 2014;6(4):104–7.
5. Mihalik JP, Myers JB, Sell TC, Anish EJ. Maxillofacial fractures and dental trauma in a high school soccer goalkeeper: a case report. J Athl Train. 2005;40(2):116–9.
6. Singh G, Garg S, Damle SG, Dhindsa A, Kaur A, Singla S. A study of sports related occurrence of traumatic orodental injuries and associated risk factors in high school students in North India. Asian J Sports Med. 2014;5(3):e22766. https://doi.org/10.5812/asjsm.22766.
7. Kujala UM, Taimela S, Antti-Poika I, Orava S, Tuominen R, Myllynen P. Acute injuries in soccer, ice hockey, volleyball, basketball, judo, and karate: analysis of national registry data. BMJ. 1995;311(7018):1465–8.
8. Shirani G, Kalantar Motamedi MH, Ashuri A, Eshkevari PS. Prevalence and patterns of combat sport related maxillofacial injuries. J Emerg Trauma Shock. 2010;3(4):314–7. https://doi.org/10.4103/0974-2700.70744.
9. Spinks AB, McClure RJ. Quantifying the risk of sports injury: a systematic review of activity-specific rates for children under 16 years of age. Br J Sports Med. 2007;41(9):548–57. https://doi.org/10.1136/bjsm.2006.033605.
10. ADA Council on Access, Prevention and Interprofessional Relations; ADA Council on Scientific Affairs. Using mouth guards to reduce the incidence and severity of sports-related oral injuries. JADA. 2006;137:1712–20.
11. Cohenca N, Roges RA, Roges R. The incidence and severity of dental trauma in intercollegiate athletes. J Am Dent Assoc. 2007;138:1121–6.
12. Emerich K, Kaczmarek J. First aid for dental trauma caused by sporting activities. Sports Med. 2010;40:361–6.
13. Heintz WD. Mouth protectors: a progress report. Bureau of Dental Health Education. J Am Dent Assoc. 1986;77:632–6.
14. Knapik JJ, Marshall SW, Lee RB, et al. Mouthguards in sport activities: history, physical properties and injury prevention effectiveness. Sports Med. 2007;37:117–44.
15. Labella CR, Smith BW, Sigurdsson A. Effect of mouth guards on dental injuries and concussions in college basketball. Med Sci Sports Exerc. 2002;34:41–4.
16. Luyk NH, Larsen PE. The diagnosis and treatment of the dislocated mandible. Am J Emerg Med. 1989;7:329–35.
17. Muhtarogullari M, Demiralp B, Ertan A. Non-surgical treatment of sports-related temporomandibular joint disorders in basketball players. Dent Traumatol. 2004;20:338–43.
18. Woodmansey KF. Athletic mouth guards prevent orofacial injuries: a review. Gen Dent. 1999;47:64–9.
19. Kurtz M, Camp J, Andreasen J. In: Scuderi G, et al., editors. Dental injuries in sports medicine: principles of primary care. St. Louis: Mosby; 1997. p. 149–75.
20. Kvittem B, Hardie N, Roettger M, et al. Incidence of orofacial injuries in high school sports. J Public Health Dent. 1998;58(4):288–93.
21. Gassner R, Bosch R, Tuli T, et al. Prevalence of dental trauma in 6000 patients with facial injuries: implications for prevention. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;87(1):27–33.
22. Mueller F, Marshall S, Kirby D. Injuries in little league baseball from 1987 through 1996. Phys Sportsmed. 2001;29(7):41–9.
23. Lahti H, Sane J. Dental injuries in ice hockey games and training. Med Sci Sports Exerc. 2002;34(3):400–2.
24. Gassner R, Tuli T, Hachl O, et al. Cranio-maxillofacial trauma: a 10 year review of 9543 cases with 21067 injuries. J Craniomaxillofac Surg. 2003;31(1):51–61.
25. Cameron AC, Widmer RP. Handbook of paediatric dentistry. 3rd ed. Philadelphia: Australian Academy of Paediatric Dentistry, Mosby; 2008. p. 115–67.
26. American Academy of Pediatric Dentistry. Guideline on management of acute dental trauma. Reference Manual. 2011–2012;33(6):331–32.
27. Fos PJ, Pinkham JR, Ranalli DN. Prediction of sports-related dental traumatic injuries. Dent Clin N Am. 2000;44(1):19–33.
28. Starkey PE. Reattachment of a fractured fragment to a tooth—a case report. J Indiana Dent Assoc. 1979;58(5):37–8.
29. Simonsen RJ. Restoration of a fractured central incisor using original tooth fragment. J Am Dent Assoc. 1982;105:646–8.
30. Osborne JW, Lambert RL. Reattachment of fractured incisal tooth segment. Gen Dent. 1985;33(6):516–7.
31. Ludlow JB, LaTurno SA. Traumatic fracture—one-visit endodontic treatment and dentinal bonding reattachment of coronal fragment: report of case. J Am Dent Assoc. 1985;110(3):341–3.
32. Burke FJ. Reattachment of a fractured central incisor tooth fragment. Br Dent J. 1991;170(6):223–5.
33. Baratieri LN, Monteiro S Jr, Caldeira de Andrada MA. Tooth fracture reattachment: case reports. Quintessence Int. 1990;21(4):261–70.
34. Martens LC, Beyls HM, de Craene LG, D'Hauwers RF. Reattachment of the original fragment after vertical crown fracture of a permanent central incisor. J Pedod. 1988;13(1):53–62.
35. DiAngelis AJ. Restoring traumatic coronal fractures. Advantages of a reattachment technique. Dent Teamwork. 1994;7(6):19–21.
36. Andreasen FM, Steinhardt U, Bille M, Munksgaard EC. Bonding of enamel-dentin crown fragments after crown fracture. An experimental study using bonding agents. Endod Dent Traumatol. 1993;9(3):111–4.
37. Lowey MN. Reattachment of a fractured central incisor tooth fragment. Br Dent J. 1991;170(8):285.
38. Murchison DF, Worthington RB. Incisal edge reattachment: literature review and treatment perspectives. Compend Contin Educ Dent. 1998;19(7):731–4, 736, 738.
39. Farik B, Munksgaard EC, Kreiborg S, Andreasen JO. Adhesive bonding of fragmented anterior teeth. Endod Dent Traumatol. 1998;14(3):119–23.
40. Liebenberg WH. Reattachment of coronal fragments: operative considerations for the repair of anterior teeth. Pract Periodontics Aesthet Dent. 1997;9(7):761–72.
41. Baratieri LN, Ritter AV, Monteiro Júnior S, de Mello Filho JC. Tooth fragment reattachment: an alternative for restoration of fractured anterior teeth. Pract Periodontics Aesthet Dent. 1998;10(1): 115–25.
42. Walker M. Fractured-tooth fragment reattachment. Gen Dent. 1996;44(5):434–6.
43. Wolcott J, Averbach RE. Management of complicated crown fractures: tooth fragment reattachment. Compend Contin Educ Dent. 2002;23(6):520–4, 526, 528.
44. Maia EA, Baratieri LN, de Andrada MA, Monteiro S Jr, de Araújo EM Jr. Tooth fragment reattachment: fundamentals of the technique and two case reports. Quintessence Int. 2003;34(2):99–107.
45. Sengun A, Ozer F, Unlu N, Ozturk B. Shear bond strengths of tooth fragments reattached or restored. J Oral Rehabil. 2003;30(1):82–6.
46. Cohen RG. Reattachment of a broken tooth fragment following dental trauma. Dent Today. 2000;19(10):96–9.
47. Arapostathis K, Arhakis A, Kalfas S. A modified technique on the reattachment of permanent tooth fragments following dental trauma. Case report. J Clin Pediatr Dent. 2005;30(1):29–34.
48. Reis A, Loguercio AD. Tooth fragment reattachment: current treatment concepts. Pract Proced Aesthet Dent. 2004;16(10):739–40.
49. Say EC, Altundal H, Kaptan F. Reattachment of a fractured maxillary tooth: a case report. Quintessence Int. 2004;35(8):601–4.
50. Reis A, Loguercio AD, Kraul A, Matson E. Reattachment of fractured teeth: a review of literature regarding techniques and materials. Oper Dent. 2004;29(2):226–33.
51. Stellini E, Stomaci D, Stomaci M, Petrone N, Favero L. Fracture strength of tooth fragment reattachments with postpone bevel and overcontour reconstruction. Dent Traumatol. 2008;24(3):283–8.
52. Naudi AB, Fung DE. Tooth fragment reattachment in multiple complicated permanent incisor crown-root fractures - a report of two cases. Dent Traumatol. 2008;24(2):248–52.
53. Demarco FF, de Moura FR, Tarquinio SB, Lima FG. Reattachment using a fragment from an extracted tooth to treat complicated coronal fracture. Dent Traumatol. 2008;24(2):257–61.

54. Macedo GV, Diaz PI, De O, Fernandes CA, Ritter AV. Reattachment of anterior teeth fragments: a conservative approach. J Esthet Restor Dent. 2008;20(1):5–18; discussion 19-20.
55. Pusman E, Cehreli ZC, Altay N, Unver B, Saracbasi O, Ozgun G. Fracture resistance of tooth fragment reattachment: effects of different preparation techniques and adhesive materials. Dent Traumatol. 2010;26(1):9–15.
56. Macedo GV, Ritter AV. Essentials of rebonding tooth fragments for the best functional and esthetic outcomes. Pediatr Dent. 2009;31(2): 110–6.
57. Capp CI, Roda MI, Tamaki R, Castanho GM, Camargo MA, de Cara AA. Reattachment of rehydrated dental fragment using two techniques. Dent Traumatol. 2009;25(1):95–9.
58. Chazine M, Sedda M, Ounsi HF, Paragliola R, Ferrari M, Grandini S. Evaluation of the fracture resistance of reattached incisal fragments using different materials and techniques. Dent Traumatol. 2011;27(1):15–8.
59. Bhargava M, Pandit IK, Srivastava N, Gugnani N, Gupta M. An evaluation of various materials and tooth preparation designs used for reattachment of fractured incisors. Dent Traumatol. 2010;26(5): 409–12.
60. Bruschi-Alonso RC, Alonso RC, Correr GM, Alves MC, Lewgoy HR, Sinhoreti MA, Puppin-Rontani RM, Correr-Sobrinho L. Reattachment of anterior fractured teeth: effect of materials and techniques on impact strength. Dent Traumatol. 2010;26(4):315–22.
61. Basavanna R, Kapur R, Sharma N. A single visit, reattachment of fractured crown fragment. Contemp Clin Dent. 2010;1(3):168–70.
62. Shirani F, Malekipour MR, Sakhaei Manesh V, Aghaei F. Hydration and dehydration periods of crown fragments prior to reattachment. Oper Dent. 2012;37(5):501–8.
63. Srilatha JS, Chhasatia N, Rani PJ, Mathur E. Reattachment of fractured anterior teeth-determining fracture strength using different techniques: an in vitro study. J Contemp Dent Pract. 2012;13(1):61–5.
64. de Lima MD, Martins JF, de Moura MS, Leao VL, Moura LF. Reattachment of fractured fragment of an anterior tooth: case report and nine-year follow-up. Gen Dent. 2011;59(5):e192–5.
65. Işeri U, Özkurt Z, Kazazoğlu E. Clinical management of a fractured anterior tooth with reattachment technique: a case report with an 8-year follow up. Dent Traumatol. 2011;27(5):399–403.
66. Singhal R, Pathak A. Comparison of the fracture resistance of reattached incisor tooth fragments using 4 different materials. J Indian Soc Pedod Prev Dent. 2012;30(4):310–6.
67. Lise DP, Vieira LC, Araújo É, Lopes GC. Tooth fragment reattachment: the natural restoration. Oper Dent. 2012;37(6):584–90.
68. Shetty PP, Metgud S, Jain A, Dhillon G, Astekar M. A conservative single visit reattachment of fractured crown fragment. Clin Pract. 2012;2(3):e70.
69. Shirani F, Sakhaei Manesh V, Malekipour MR. Preservation of coronal tooth fragments prior to reattachment. Aust Dent J. 2013;58(3):321–5.
70. Prabhakar AR, Yavagal CM, Limaye NS, Nadig B. Effect of storage media on fracture resistance of reattached tooth fragments using G-aenial Universal Flo. J Conserv Dent. 2016;19(3):250–3.
71. Choudhary A, Garg R, Bhalla A, Khatri RK. Tooth fragment reattachment: an esthetic, biological restoration. J Nat Sci Biol Med. 2015;6(1):205–7.
72. Jagannath-Torvi S, Kala M. Restore the natural - a review and case series report on reattachment. J Clin Exp Dent. 2014;6(5):e595–8.
73. VamsiKrishna R, Madhusudhana K, Swaroopkumarreddy A, Lavanya A, Suneelkumar C, Kiranmayi G. Shear bond strength evaluation of adhesive and tooth preparation combinations used in reattachment of fractured teeth: an ex-vivo study. J Indian Soc Pedod Prev Dent. 2015;33(1):40–3.
74. Taguchi CM, Bernardon JK, Zimmermann G, Baratieri LN. Tooth fragment reattachment: a case report. Oper Dent. 2015;40(3):227–34.
75. Chosack A, Eidelman E. Rehabilitation of a fractured incisor using patient's natural crown. Case report. J Dent Child. 1964;31:19–21.
76. Tennery NT. The fractured tooth reunited using the acid-etch bonding technique. Tex Dent J. 1978;96:16–7.
77. Andreasen FM, Noren JG, Andreasen JO. Long-term survival of fragment bonding in the treatment of fractured crowns: a multicenter clinical study. Quintessence Int. 1995;26:669–81.
78. Calvalleri G, German N. Traumatic crown fractures in permanent incisors with immature roots: a follow-up study. Endod Dent Traumatol. 1995;11:294–6.
79. Reis A, Francci C, Loguercio A, et al. Re-attachment of anterior fractured teeth: fracture strength using different techniques. Oper Dent. 2001;26(3):287–94.
80. Farik B, Munksgaard EC, Andreasen JO, Kreiborg S. Fractured teeth bonded with dentin adhesives with and without unfilled resin. Dent Traumatol. 2002;18:66–9.
81. Almuammar MF, Schulman A, Salama FS. Shear bond strength of six restorative materials. J Clin Pediatr Dent. 2001;25:221–5.
82. Reis A, Kraul A, Francci C, de Assis TG, Crivelli DD, Oda M, et al. Reattachment of anterior fractured teeth: fracture strength using different materials. Oper Dent. 2002;27:621–7.
83. Sharmin D, Thomas E. Evaluation of the effect of the storage medium on fragment reattachment. Dent Traumatol. 2013;29(2):99–102.
84. Buonocore MG. A simple method of increasing the adhesions of acrylic filling materials to enamel surfaces. J Dent Res. 1955;34: 849–53.
85. Bowen RL. Synthesis of a silica-resin direct filling material. Progress report. J Dent Res. 1958;37:90.
86. Bates JF, Addy M. Partial dentures and plaque accumulation. J Dent. 1978;6:285–93.
87. Shimura Y, Wadachi J, et al. Influence of removable partial dentures on the formation of dental plaque on abutment teeth. J Prosthodont Res. 2010;54(1):29–35.
88. Leles CR, Martins RR, et al. Discriminant analysis of patients' reasons for choosing or refusing treatments for partial edentulism. J Oral Rehabil. 2009;36:909–15.
89. Chafaie A, Portier R. Anterior fiber reinforced composite resin bridge: a case report. Pediatr Dent. 2004;26(6):530–4.
90. Rudo DN, Karbhari VM. Physical behaviors of fiber-reinforcement as applied to tooth stabilization. Dent Clin N Am. 1999;43(1):7–35.
91. Unlu N, Belli S. Three-year clinical evaluation of fiber-rein-forced composite fixed partial dentures using prefabricated pontics. J Adhes Dent. 2006;8(8):183–8.
92. Freilich MA, Meiers JC, Duncan JP, Eckorte KA, Goldberg AJ. Clinical evaluation of fiber-reinforced fixed bridges. J Am Dent Assoc. 2001;133(11):1523–34.
93. Lambert DL. Conservative aesthetic solutions for the adolescent and young adult utilizing composite resins. Dent Clin N Am. 2006;50(1):87–118.
94. Gupta A, Yelluri RK, Munshi AK. Fiber-reinforced composite resin bridge: a treatment option in children. Int J Clin Pediatr Dent. 2015;8(1):62–5.
95. Chafaie A, Dahan S, Le Gall M. Fiber-reinforced composite anterior bridge in pediatric traumatology: clinical considerations. Int Orthod. 2013;11(4):445–56.
96. Kharade P, Sharma S, Banerjee A, Gupta T. Indirect resin-bonded fiber-reinforced composite anterior bridge: a case report. Gen Dent. 2012;60(3):e170–2.
97. Lambert DL. Meeting patients' esthetic and restorative goals in a changing economy. Oral Health. 2012;102(4):87–91.
98. Van Wijlen P. A modified technique for direct, fiber-reinforced, resin-bonded bridges: clinical case reports. J Can Dent Assoc. 2000;66(7):367–71.
99. Marwaha M, Bhat M, Singh Nanda KD. Building-up a smile in a 5-year-old child: a case report. Int J Clin Pediatr Dent. 2012;5(2): 151–4.
100. Uribe F, Meiers JC, Nanda R. Fixed retention of congenitally missing lateral incisors using a prefabricated fiber-reinforced composite bridge. World J Orthod. 2008;9(4):349–54.
101. Arteaga S, Meiers JC. Technique for placement of a posterior prefabricated fiber-reinforced composite bridge. Gen Dent. 2006; 54(6):393–6.

102. Arteaga S, Meiers JC. Single-tooth replacement with a chairside prefabricated fiber-reinforced composite bridge: a case study. Gen Dent. 2004;52(6):517–9.
103. Trushkowsky R. Fiber-reinforced composite bridge and splint: replacing congenitally missing teeth. N Y State Dent J. 2004;70(5):34–8.
104. Aydin MY, Kargül B. Glass-fiber reinforced composite in management of avulsed central incisor: a case report. J Dent Child (Chic). 2004;71(1):66–8.
105. Javaheri DS. Replacement of an anterior tooth with a fiber-reinforced resin bridge. Compend Contin Educ Dent. 2001;22(1): 68–70, 72, 74.
106. Leal FR, Cobb DS, Denehy GE, Margeas RC. A conservative aesthetic solution for a single anterior edentulous space: case report and one-year follow-up. Pract Proced Aesthet Dent. 2001;13(8):635–41.
107. Belvedere PC. New method for preparation of fiber reinforced temporary crowns and bridges. Swiss Dent. 1991;12(7):17–9.
108. Belvedere PC. Single-sitting fiber-reinforced fixed bridges for the missing lateral or central incisors in adolescent patients. Dent Clin N Am. 1998;42(4):665–82.
109. Nik-Hussein NN. Traumatic injuries to anterior teeth among schoolchildren in Malaysia. Dent Traumatol. 2001;17:149–52.
110. Altay N, Gungor H. A retrospective study of dento-alveolar injuries of children in Ankara, Turkey. Dent Traumatol. 2001;17:201–4.
111. Rocha M, Cardosa M. Traumatized permanent teeth in Brazilian children assisted at the Federal University of Santa Catarina, Brazil. Dent Traumatol. 2001;17:245–9.
112. Andreasen JO, Andreasen FM. Essentials of traumatic injuries to the teeth. 2nd ed. Copenhagen: Blackwell Munksgaard; 2000. p. 7–9.
113. Levin L, Friedlander L, Geiger S. Dental and oral trauma and mouthguard use during sport activities in Israel. Dent Traumatol. 2003;19:237–42.
114. Flanagan D. External and occlusal trauma to dental implants and a case report. Dent Traumatol. 2003;19:160–4.
115. Stuebinger S, Hodel Y, Filippi A. Trauma to anterior implants. Dent Traumatol. 2004;20:169–71.
116. Allen EM, Allen PF. Trauma to an osseointegrated anterior dental implant; a case report. Dent Traumatol. 2006;22:44–7.
117. Cardoso L de C, Luvizuto ER, Trevisan CL, et al. Resolution of a titanium implant fracture after a recurrent trauma. Dent Traumatol. 2010;26:512–5.
118. den Hartog L, Meijer H, Stellingsma K, et al. Trauma to an implant-supported crown that was saved by the fixation screw: a case report. Dent Traumatol. 2010;26:366–9.
119. Cho YB, Moon SJ, Chung CH, Kim HJ. Resorption of labial bone in maxillary anterior implant. J Adv Prosthodont. 2011;3:85–9.
120. Schropp L, Wenzel A, Kostopoulos L, Karring T. Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study. Int J Periodontics Restorative Dent. 2003;223:313–23.
121. LeSage BP. Improving implant aesthetics: prosthetically generated papilla through tissue modeling with composite. Pract Proced Aesthet Dent. 2006;18:257–63.
122. Kois JC, Kan JY. Predictable peri-implant gingival aesthetics: surgical and prosthetic rationales. Pract Proced Aesthet Dent. 2001;13:691–8.
123. Esposito M, Grusovin M, Polyzos I, et al. Timing of implant placement after tooth extraction: immediate, immediate-delayed or delayed implants? A Cochrane systematic review. Eur J Oral Implantol. 2010;3:189–205.
124. Goldberg PV, Higginbottom FL, Wilson TG. Periodontal considerations in restorative and implant therapy. Periodontol 2000. 2001;25:100–9.
125. Rangart B, Renouard F. Practical guidelines based on biomechanical principles. In: Esthetic implant dentistry: soft and hard tissue management. Chicago: Quintessence; 2001. p. 58.
126. Alani A, Austin R, Djemal S. Contemporary management of tooth replacement in the traumatized dentition. Dent Traumatol. 2012;28(3):183–92.
127. Zachrisson BU. Planning esthetic treatment after avulsion of maxillary incisors. J Am Dent Assoc. 2008;139:1484–90.
128. Sarver DM. Growth maturation aging: how the dental team enhances facial and dental esthetics for a lifetime. Compend Contin Educ Dent. 2010;31(4):274–80, 282-3.
129. Heij DG, Opdebeeck H, van Steenberghe D, Kokich VG, Belser U, Quirynen M. Facial development, continuous tooth eruption, and mesial drift as compromising factors for implant placement. Int J Oral Maxillofac Implants. 2006;21(6):867–78.
130. Schwartz-Arad D, Levin L, Ashkenazi M. Treatment options of untreatable traumatized anterior maxillary teeth for future use of dental implantation. Implant Dent. 2004;13(2):120–8.
131. Miyahara T, Dahlin C, Galli S, et al. A novel dual mouthguard material for patients with dental implants. Dent Traumatol. 2013;29:303–6.

Check for updates

# Prevention of Athletic Dental Injuries: The Mouthguard

*Steve Mills and Emilio Canal*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_7

## 7.1 Introduction

Orofacial and dental sports injuries have been reported to be very common, and but they have also been termed a "rare" sports injury [1–4]. In spite of this apparent contradiction, several things have been generally agreed upon; orofacial injuries do occur in sports activities, and many are preventable. The prevention of both the incidence and severity of orofacial injuries could potentially eliminate life-changing dental injuries, save significant costs to athletes, and keep players from missing playing time. Facial protection with face shields and helmets in various sports helps greatly with this, but the single most important piece of protective equipment for today's athlete in sports that carry the risk of injury is the athletic mouthguard.

The ideal piece of protective sports gear should be characterized by several features. First and foremost to the athlete, the device should not interfere with the player's ability to perform his or her activity to the optimum of their capability. It should not be a distraction and it should be comfortable. It cannot interfere with the athlete's ability to communicate with teammates and should not interfere with his or her perception of their ability to breathe. It should not impact the game in any way such as the potential for a football helmet to be used as a striking device in American-style football. It should be able to show conclusively that it is effective. And finally, it should not interfere with the player's enjoyment of the sport or his or her participation in it. The properly designed, fabricated, and fitted mouthguard has the ability to achieve all of these goals.

An athletic mouthguard or "mouth protector" is defined by the American Society for the Testing of Materials (currently known as ASTM international) as "a resilient device or appliance placed inside the mouth (or inside and outside), to reduce mouth injuries, particularly to teeth and surrounding structures" [5]. In spite of this relatively simple definition, no single mouthguard design has been designated "the best," and no one material is believed to be a "perfect" material. A good statement of what constitutes an acceptable mouthguard has been produced by the Academy for Sports Dentistry (Fig. 7.1) [6].

The athletic mouthguard and the treatment of orofacial trauma are undeniably the most important responsibilities the dental professional has to athletes. It is important to try to be informed about mouth protectors, as dental professionals are expected to be the experts on this device. The history of the appliance, the technical features of guards, the functions and the proven effectiveness of them, and the different types

**A Properly Fitted Mouthguard**
**An athletic mouthguard is a resilient device or appliance placed inside the mouth to reduce injuries particularly to the teeth and surrounding** structures.
For optimal safety and well-being of athletes competing in the 21st Century, the Academy for Sports Dentistry has adopted the position that the single word "mouthguard" must be replaced by the term "a properly fitted mouthguard".
In contact sports, it is critical that the mouthguard provides protection from direct and indirect impact.
It must fit accurately, stay in position during impact, and redistribute the impact's energy. The criteria for the fabrication or adaptation of a properly fitted mouthguard must include the following considerations:

1. Pertinent Medical History
2. Dental Status that considers:
   1. Dental Caries
   2. Periodontal Status
   3. Developmental Occlusion
   4. Orthodontic or Prosthodontic Appliances
   5. Congenital/Pathological Conditions
   6. Jaw Relationships
3. Demographic Factors
4. Type of Sport Played

The fitting of a mouthguard is best accomplished under the supervision or direction of a dentist. The athlete and/or parents should always be advised of the special design for the "properly fitted mouthguard" and the end product should have the following properties and considerations:
It should be fabricated to adequately cover and protect both the teeth in the arch, and the surrounding tissues.
It should be fabricated on a stone model taken from an impression of the athlete.
Adequate thickness in all areas to provide for the reduction of impact forces. In particular, a minimum of 3 mm thickness in the occlusal/labial area.
It should have a seated equilibrated occlusion that is balanced for even occlusal contact. This helps to provide for the ideal absorption of impact energy.
A fit that is retentive and not dislodged on impact.
Speech considerations equal to the demands of the playing status of the athlete.
A material that meets FDA approval.
The properly fitted mouthguard should be routinely and professionally examined for fit and function. Frequency of routine inspection is dependent on factors such as the athlete's age, the demand of the sport that the athlete is engaged in, and the willingness for the athlete to properly care for the appliance. The frequency of the inspection should be determined by the dental professional for each individual situation and athlete.
Approved by the Academy for Sports Dentistry Board of Directors 04/05/2010

**Fig. 7.1** The Academy for Sports Dentistry's Position Statement on the Properly Fitted Mouthguard, 2010 of mouthguards all should be basic knowledge for sports dentists. The custom-fitted mouthguard is the most recommended type and is something that any dentist has the capability to make for their players. However, while many dentists choose not to make their own appliances, they should still know how an effective mouthguard is fabricated. These are the subjects that will be covered in this chapter.

## 7.2 The History of the Athletic Mouthguard

The history of the mouthguard is not easily defined, but it can be assumed that both athletes and warriors over the centuries have tried to protect themselves from facial and oral injury. Medieval helmets and ancient Samurai headgear indicate features that protected specific areas of the head and face. But as to the protection of the mouth specifically in both war and sport, little has been identified from the distant past.

The use of mouth protection in sports usually focuses on the sport of boxing starting in the late nineteenth century. However the first documentable mouth protector found was a simple latex device used in baseball in the United States in the 1870s [7]. George Wright, a star player on the Cincinnati Red Stockings, produced a 50 cent rubber guard which was clenched between the teeth. The Red Stockings catcher, Doug Allison, who also was one of the first catchers to use gloves to catch the ball, was possibly the first who used it. A catcher in those days was almost entirely unprotected and generally stood far behind the batter and caught the ball on the first bounce. As catchers inched closer to the batter, it became more important for them to protect themselves from injury. The mouthguard was used to some degree until the invention of the catcher's mask in 1877 (patented in 1878) by the coach of the Harvard Law School baseball team, Winthrop Thayer, who created it to convince outfielder James Tyng to become his catcher. George Wright then developed the first commercial catcher's mask. Thayer received a royalty for each one purchased [7].

The sport of boxing is where the development of a mouthguard can be more clearly documented. The use of mouth protectors was common in the late nineteenth century, but these "devices" were often "cotton, tape, sponge, and even pieces of wood" [8]. A wonderful paper in 1994 by Robert Reed gave several firsthand testimonies of the development of the first true mouthguards [9]. A London dentist, Woolf Krause, fashioned the first true mouthguard or "gum shield" in 1890 out of gutta-percha to protect the boxers primarily from lip lacerations. The participant needed to clench his teeth together to hold the guard in place.

Krause's son Philip improved on this design by creating a guard out of vellum rubber. A professional boxer named Ted "Kid" Lewis first used Phillip's mouthguard in 1913. The following is, in his own words, how he came to use a mouth protector:

> It's quite correct about me being the first one in the U S to use this protector. I was also the first to use it in England, it happened by fluke really. I use to suffer with cut lips, in fact my lips used to cut very easily at this particular time. I use to pal around with an assistant dentist, he is a full fledged dentist now. his name was Phil Krause we were school chums. After one of my fights, my lip cut as usual, we were celebrating one of my victories at home I could hardly eat, when my pal Phil asked me to his office. the next day I arrive at his office, he puts me in the chair and takes an impression of my mouth in a few hours. Later he arrives at my house with his new gadget. I put it in my mouth and found it very awkward. But he told I would get used to it. I then tried it out in training. And found it very useful. That was how the gum shield was born. From that day to the day I finished boxing I always got through my bouts minus a cut lip [9].

"The earliest recording of a U.S. mouthguard-type device was in 1916 when Thomas Carlos, a Chicago Dentist, designed a mouthpiece for U.S. Olympian Dinnie O'Keefe" [8]. Mouthguards become prevalent following a 1927 boxing match between Mike McTigue and Jack Sharkey. McTigue was clearly winning the fight; however, a chipped tooth severely cut his lip and forced him to forfeit the match. From then on, mouthguards become commonplace for boxers and also opened the possibilities for mouthguard use to flourish [8].

Three years following the infamous McTigue/Sharkey fight, mouthguards found their way into dental literature. Dr. Clarence Mayer, who was a dentist and also a boxing inspector, wrote about how custom mouthguards could be created from impressions using wax and rubber. He also suggested using steel springs to reinforce the materials [8].

In 1947, a major breakthrough was made when Los Angeles dentist Rodney O. Lilyquist used transparent acrylic resin to form the first acrylic splint. This mouthguard was molded to fit over the upper and lower teeth and made for a much more unobtrusive object. During this time, dental injuries were responsible for around 24–50% of all American football injuries. The *Journal of American Dental Association* picked up Lilyquist's technique, which led to nationwide recognition for him, and he became known as the father of the modern mouthguard [10, 11]. Dick Perry, a UCLA basketball player, was the first known athlete to use an acrylic mouthguard. Later on Frankie Albert, the quarterback for the San Francisco 49ers, was the first known professional non-boxing athlete to wear this type of mouthguard [12].

In the 1950s, latex rubber was the material most commonly used for athletic mouthguards, but William Godwin was also perfecting a vacuum forming process on casts of athletes' teeth using ethylene vinyl acetate (EVA) originally obtained through the automobile manufacturing industry [13, 14]. In the late 1950s, Dr. A.G. Jacobs pioneered the use of a silicone lining material to allow athletes to make their own mouthguards. In the early 1960s, Dr. Jacobs developed the first "boil and bite" mouthguard of EVA that athletes could easily form for themselves [15]. This innovation was groundbreaking as it made a simple fitted mouthguard much more accessible to athletes and added greatly to the success of rule changes first to American-style football and then to other sports.

In the early 1950s, the American Dental Association (ADA) realized that there was a significant injury problem in American-style football. The helmet was first introduced in the 1920s to protect the skull from catastrophic injury. This early helmet afforded no facial protection and only with the development of a plastic helmet was the introduction of a face shield made a reality. The rigid plastic construction of the helmet made the physical attachment of rigid face shield possible. The ADA along with the American Association for Health, Physical Education, and Recreation issued a report in 1960 that found that when no face shield or mouthguard was worn, 50% of all high school football injuries occurred in or around the mouth [16].

As a result of this information, the National Alliance Football Rules Committee composed of the The National Federation of State High School Associations, the National Junior College Athletic Association, and the National Association of Intercollegiate Athletics, in 1962, mandated that players must wear a facemask and a mouthguard. Interestingly, the National Collegiate Athletic Association (NCAA) did not immediately adopt this rule, but after 11 years, in 1973, they also instituted the mandate. These initial rule changes resulted in dramatic improvements in injury rates within a very short time. It was reported that prior to the rule changes, there was an incidence of 2.26% dental injuries per every 100 football players. This number was reduced to 0.3% in 1966 [16].

Dental protection in ice hockey followed a similar pathway, albeit later, with a similar development of helmet introduction followed by face shields followed by dental protection [17]. The NCAA mandated mouthguard use for ice hockey in 1975, as did many other governing bodies of youth and amateur hockey. For the 2017–2018 ice hockey season the NCAA rescinded the mandatory mouthguard rule. In both ice hockey and American-style football, professional players are not required to wear mouthguards. Currently boxing is the only professional sports to require the use of mouthguards.

As of 2016 the NCAA requires mouth protection in football, men and women's ice hockey (rescinded in 2017), men and women's lacrosse, and women's field hockey [18]. The NCAA is phasing in women's rugby, and this sport, in all likelihood, will require mouthguards as well. The National Federation of State High School Associations recommends mandated use of mouthguards in those sports as well as for wrestling participants who are wearing orthodontic appliances [19]. The American Dental Association has "endorsed the preventive value of orofacial protectors, including helmets, faceguards and mouth protectors, for use by participants in sporting and recreational activities with some degree of injury risk and at all levels of competition." In addition to the more common sporting activities such as basketball or baseball, the ADA's list of sports for which they recommend mouth protection names many sports including activities such as skateboarding and nontraditional sports such as cheerleading [20]. Many other sports that are played internationally such as rugby, hurling, field hockey, martial arts, team handball, and many more have various mandates depending on the region and country in which the sports are played.

**3. Classification Mouth Protectors**
3.1 Mouth protectors covered by this practice shall be of the following types and classes:
3.1.1 *Type I—Thermoplastic Type:*
3.1.1.1 *Class 1*—Vacuum-formed.
3.1.1.2 *Class 2*—Mouth-formed.
3.1.2 *Type II—Thermosetting Type:*
3.1.2.1 *Class 1*—Mouth-formed.
3.1.3 *Type III*—Stock type.

**Fig. 7.2** ASTM International classification of mouth protectors, 2006

## 7.3 Types of Mouthguards

The athletic mouthguard can be characterized by type, material, and physical properties. Different sports and different athletes demand different particular characteristics of an adequate piece of safety equipment. In this section we will describe the mouthguard in various ways to enable dentists and athletes a way to decide which device is best for any specific circumstance.

The ASTM International is an organization whose mission is to develop and create voluntary "consensus standards" for industry and consumers. Its subcommittee F08 has written in standard F 697-00 (approved in 2006) that there are three types of "mouth protectors," Type I, II, and III, with several subgroups (Fig. 7.2) [5]. This classification is the most common and straightforward way to categorize mouthguards. These will be discussed in reverse order from the standard to explain them from simplest to more sophisticated.

### 7.3.1 Stock Type

This is by far the simplest type of mouth protector. It is usually a device that is supposed to be used directly from the package with no attempt made at fitting for the individual. The use of this type of device results in a very loose-fitting appliance where the athlete must clench his or her teeth together to keep the device in place. This obviously leads to unintelligible speech and difficulty in breathing through the mouth. It is also much more likely that the guard will be dislodged when the player is contacted during play (Fig. 7.3).

These guards are usually designed for the upper arch, but often they are configured to cover both the upper and lower teeth. There are quite a few styles and shapes available and at best should be used only when an athlete can stop his activities often and when ongoing verbal communication is not important.

Most sports dentists do not recommend these devices, but supporters note a few benefits. They are convenient and can be used immediately without fitting. Orthodontists sometimes recommend these, as they do not restrict the ongoing movement of teeth during orthodontic treatment. They are generally inexpensive. They make easy "spare

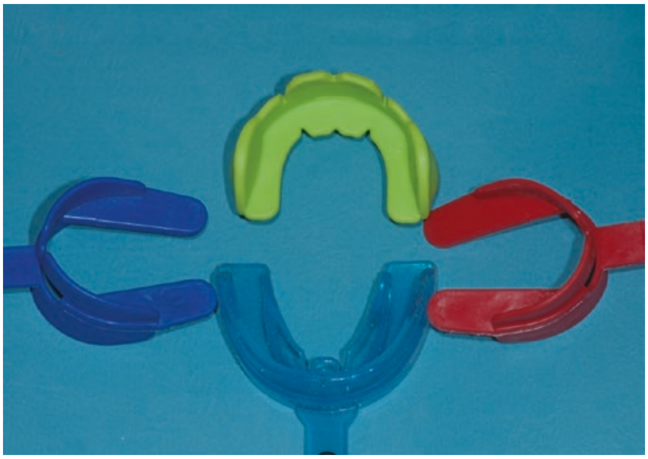


Fig. 7.3 Stock-type mouthguards

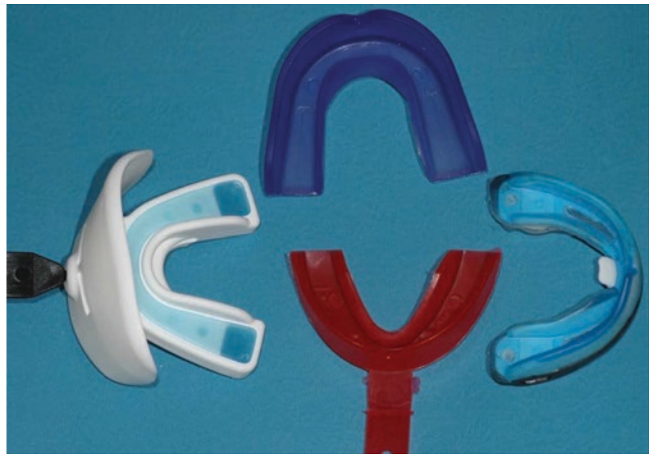


Fig. 7.4 Mouth-formed mouthguards

mouthguards" to be kept on hand in case someone has lost or destroyed their own guard.

Stock mouthguards should be used only in very specific circumstances and should not be thought to be an acceptable alternative for general use. They do not meet the basic requirements of a "properly fitted mouthguard" (Fig. 7.1).

### 7.3.2 Mouth-Formed

This type is the most widely used mouthguard. They are accessible and cost-effective and come in a large variety of styles and designs to supposedly fill all needs. As it is defined, it is to be molded in the mouth and has two basic types: the shell-lined or the thermoformed type. The shell-lined type is one where a resilient material is mixed and placed in a dental arch-shaped shell and then it is placed in the mouth to set. This type has largely disappeared from use.

The more common type is the thermally formed group usually termed the "boil and bite" group, as that is how it is most often fitted. This type is readily accessible, and in recent years an almost limitless number of varieties have been marketed. They are generally affordable, and the creativity in their design and materials has led this to become a very significant industry in the sporting goods world. Athletes often gravitate toward these as they are conveniently obtained and can be fitted without the intervention of a dental professional. Boil and bite mouthguards come in a wide range of prices depending on the features of a particular guard with a price to literally match every budget.

The mouthguard is individually fitted usually by heating the device in hot water (or in a microwave oven) and then placing inside the mouth to form it to the teeth. Once it is in the mouth, the participant molds in with his or her fingers to mold the outer side of the guard by pushing on the cheek and lips, by sucking hard on it while placing the tongue firmly against it, and by biting on it to fit it to the lower teeth.

The huge variety of these types of mouthguards makes it hard to assess whether accomplishing a good fit is possible or not. The fit largely depends on the individual doing the fitting. Dentists know how difficult it is to learn the skill of taking a good dental impression, and this fitting of a mouth-formed guard can be compared to that. Therefore it is difficult to categorically state whether or not this type of mouthguard is acceptable (Fig. 7.4).

Mouth-formed mouthguards may vary, but one particular mouthguard made of a unique material (a polyolefin polymer trade name Vistamaxx) applied for and received, in 2016, the American Dental Association's Seal of Acceptance. At the time of this writing, this mouthguard is the only one to have ever been awarded this designation.

### 7.3.3 Custom-Fitted Mouthguards

These mouthguards are created by dental professionals fabricating specific materials over a cast of an athlete's teeth. These guards are usually made for the maxillary teeth except in prognathic Class III-type occlusions. The internal adaptation achieved and the ability to create comfortable borders make these mouthguards the most comfortable and intimately fitted. Studies have shown that these guards are the most desirable to players [21] (Fig. 7.5)

This type of mouthguard, most commonly, uses some type of ethylene vinyl acetate (EVA) that is heated and then formed over the dental cast. The two most common ways of forming these guards are to use a vacuum forming technique or a heat and pressure technique.

The advantages of the custom-fitted guard beyond the intimate fit are that (a) the guards are specific to the athlete, (b) they can be customized to suit the sport played and the athlete's requirements, and (c) the device is not easily dislodged during play. Also, because a custom guard is tailored to the athlete, speech considerations and the ability to breathe without detriment are not affected in any critical way. A significant number of studies have been conducted measuring VO2 max levels of an athlete wearing a mouthguard versus one without. VO2 max refers to the maximum amount of oxygen an individual can utilize during intense or maximal exercise; the result has found that there is no physiological effect on breathing with a mouthguard in place and


**Fig. 7.5** Custom-fitted mouthguards



that any complaints by the athlete are probably psychological. However there are studies done with stock mouthguards that do show a reduction in VO2 max [22–24]. For sports that are defined by continuous play, that are highly aerobic, and those sports in which athletes play without break for extended periods of time, this type of guard is by far the most suitable. They are also the type usually preferred by athletes [21].

The major disadvantages of this type of guard are cost and accessibility. They tend to be significantly more expensive than retail guards. An impression of the athlete's teeth needs to be taken. This is most commonly achieved at a dentist's office, but there are products available which allow the purchaser to fabricate their own impression. This impression is then used by the dental professional to fabricate the guard, or it is sent to a professional dental laboratory for the fabrication of the mouthguard.

#### 7.3.3.1 Vacuum-Formed Mouthguards

The vacuum-formed type of custom guard has been used since first investigated in the early 1960s [13, 14]. A sheet of mouthguard material was heated and placed over a dental cast. A vacuum then pulls the material down over the cast. Currently a more modern version of this technique is still used with more sophisticated machines. This technique can be used to create a well-fitted mouthguard, and the machine that is used in its fabrication tends to be less expensive than the machine used in the heat and pressure technique. While this technique can create a very acceptable mouthguard, it is difficult to laminate multiple sheets of mouthguard material in order to add strength and longevity. In addition, if only one sheet of material is used, as is the common practice, the guard tends to lose its shape after a certain period of time.

#### 7.3.3.2 Heat-Pressure Laminated Mouthguards

The heat-pressure lamination technique takes advantage of a specific type of machine that heats a sheet of mouthguard material and then, with a special pressure chamber, *pushes* rather than pulls the sheet onto the cast. This creates a very exact and detailed impression of the cast onto the internal aspect of the guard. Because of this detail, the fit of this guard is very snug and intimate. In addition, this technique allows for the addition of multiple layers of material onto one another. Each layer is directly fused or *laminated* to the previous one. Customization of thickness in different parts of the guard is a distinct advantage. This lamination technique also gives the dental professional the ability to laminate a clear layer as the final layer. This clear layer allows mouthguards to be decorated and personalized with logos, names, and decorations. In the authors' experience, it is not uncommon to see a laminated mouthguard last multiple seasons as compared to vacuum and boil and bite types that in most cases do not make it through one season of play.

## 7.4 Materials and Physical Properties of Mouthguards

The athletic mouthguard works to protect teeth and their supporting structures by both absorbing and distributing injurious forces that impact the mouth. Mouthguards have been and continue to be made of a large number of different materials all of which possess varying abilities to fulfill its protective function. The early use of gutta-percha and several different forms of rubber has been replaced by different plastics and newer materials of varying physical characteristics. Creative new materials are being developed every year as the mouthguard industry tries to stay innovative and to further meet the needs of athletes.

The most commonly used materials currently are some form of ethylene vinyl acetate (EVA) which has desirable handling characteristics as well as the proven ability to absorb and distribute injurious forces to the teeth and the supporting structures. This material is also the most widely studied material in the dental literature. In addition to this however, polyvinyl chloride, acrylic resin, polyurethane, polyolefin, latex rubber, and other polymeric and thermoformable materials have been used [25].

The properties a mouthguard must possess are a combination of measurable material features plus the ability to be fabricated easily. This must result in a comfortable piece of athletic equipment. The vast majority of studies that have evaluated mouthguards have done so by testing one or more of their physical characteristics in an in vitro setting. The most common tests have been impact tests of guards or the materials testing the absorption of impact forces as well as the distribution of forces [25].

Unfortunately, comfort, fit, and clinical efficacy cannot be evaluated in this way. There are no considerations in these impact tests to evaluate the ability of a mouthguard to stay in place at time of contact in order to protect the athlete. In addition, the comfort to the athlete of different mouthguards to one another cannot be measured in a laboratory setting.

### 7.4.1 Physical Properties

In his excellent systematic review of the literature of mouthguards, Knapik et al. list six properties that combine to define any particular mouthguard. These are shock-absorbing capability, hardness, stiffness, tear strength, tensile strength, and water absorption [25]. These are strictly material measurements and do not fully define the effectiveness of mouthguards.

Shock absorption is the ability of a material or a device to reduce the impact energy or force transmitted to the surface below the tested item. Shock absorption measurements have been achieved in two ways: A device can drop a load onto the material, or it can use a pendulum to swing an impacting force onto the material. The acceleration of the impacting force can be measured. The mass of the object can also be recorded, and then the force transmitted can be calculated.

The force absorption can then be measured in one of the two ways. Firstly, the rebound of the impacting object can be measured to see how much energy has been dissipated. Secondly and more directly, a force transducer can be placed under the tested device, and the force transmitted can be registered. Other methods such as material compression or the use of strain gauges have been used.

Hardness is defined as the "resistance of a material to penetration with a load applied." This is measured with a device called a durometer, and the resulting measurement carries the name of the commercial durometer used (e.g., Shore A or Rex A). The values for hardness are rated from 0 to 100. A measure of 0 means the material was penetrated completely, and 100 means no penetration at all.

"Stiffness is related to hardness and as hardness increases so does stiffness" [25]. Low stiffness materials tend to deform with applied force, allowing most of the force to be centered under the impact. High stiffness materials tend to distribute forces over a larger area.

Tear strength indicates the ability of a material to resist tearing forces. A notched material of known thickness is pulled until it tears. It was stated that studies varied in this measurement due largely to the method used, so only those comparisons within individual studies should be used.

Tensile strength is the "pull force required to break a material of known size" [25]. Again a notched piece of material is pulled apart until it breaks.

Water absorption is the amount of water taken up by a material. These measurements are done by measuring the amount of water taken up after a material is submerged or by measuring the weight change of a material in water after a specific time and temperature.

Shock absorption, hardness, and stiffness have been used to indicate the potential protectiveness of a given mouthguard or material. Tear strength, tensile strength, and water absorption generally designate the durability of mouthguards. The last three are generally not investigated, but they are critical as many athletes chew or otherwise abuse their guards. Also water absorption can be an indication of the ability of a mouthguard to absorb saliva and thereby to absorb microorganisms [25].

## 7.5 Functions and Efficacy of the Athletic Mouthguard

The athletic mouthguard is designed to protect the player from a variety of orofacial injuries. In addition to the prevention of dental injuries, the mouthguard has also been said to have other benefits to present to the wearer. This section will list some of these functions and critically evaluate the mouthguard's ability to achieve these goals.

### 7.5.1 Dental Protection

The athletic mouthguard's main function is undoubtedly the protection of the teeth. This is also the one area where it can be said that there is reliable evidence to back up this claim. Knapik in his systematic review states that "risk ratios ranged from 1.6 to 1.9 for the different groups of studies examined" when looking at studies comparing mouthguard wearers versus no wearers [25]. This protective benefit is not overwhelming and does not match the dramatic effects of mouthguard wear that was reported in the early 1960s with football use, but it is an indication that, when evaluated critically, mouthguards do work to protect teeth.

Sigurdsson in a paper critically evaluating the literature behind claims of the effectiveness of mouthguards noted one study by LaBella et al. which "attempted to investigate prospectively or in real time" using college basketball players to compare wearers of mouthguards versus nonwearers [26]. This study looked at athletic exposures (either games or practices) of mouthguard wearers and showed a 0.12 per 1000 athletic exposure dental injury rate as compared to 0.67 per 1000 athletic exposures for nonwearers [27].

A common discussion is whether custom-fitted mouthguards are more protective than retail or boil and bite mouthguards. Knapik points out that while in vitro studies indicate a clear superiority of custom-fitted mouthguards, there are no clinical studies which support this claim [25]. At least one author has claimed that there is no difference between using a boil and bite mouthguard versus wearing no mouthguard at all, although these claims are based on questionable studies and questionable reading of the results [28].

One very large study from the US Army has shown that its introduction of boil and bite mouthguards into its basic training significantly reduced the number of dental injuries seen [29]. This paper reported a 1999 study at Fort Leonard Wood that showed a 74% reduction in dental injuries "during military combat training activities." This led to the adoption of the use of mouth-formed mouthguards for basic trainees engaged in this training activity and to Army Regulation 600-63 which requires mouthguards during army military combat training. Therefore it does seem that boil and bite mouthguards do help decrease dental injuries. However due to the impracticality of fabricating custom-fitted mouthguards to large numbers of trainees, this study does not compare custom-fitted guards to retail products [29].

In a large-scale study using the HS RIO (High School Reporting Information Online) data collection system, Collins et al. showed that 22 players in this large collection of athletic exposures were injured when wearing a mouthguard. Within this group, 21 of the 22 were wearing store-bought mouthguards, and only 1 was wearing a custom-fitted mouthguard [3]. This finding is interesting, but it was not a purpose of the study and should be read with caution. It does however imply some superiority of custom guards to retail ones.

### 7.5.2 Soft Tissue Injuries



A good number of studies look at soft tissue injuries as a measurable orofacial injury. However, few studies have evaluated soft tissue injuries as an individual entity. Therefore there is no comprehensive data that specifically addresses the ability of mouthguards to protect soft tissue.

Historically and anecdotally boxers used the first athletic mouthguards to primarily avoid intraoral and perioral cuts and tears to lips and mucosa. As was previously mentioned, this was the primary reason that Ted “Kid” Lewis stated as the reason he both started and continued using his device. It was also the reason for the legalization of the use of mouthguards in boxing when Jack Sharkey unfairly lost a fight that he was winning as a result of a soft tissue cut which caused an excessive amount of bleeding.

In the study mentioned previously by LaBella et al., oral soft tissue injuries were specifically addressed. The findings showed a 0.69 versus 1.06 soft tissue injures per 1000 athletic exposures, but this difference was not significant [27].

It does appear that the protection against soft tissue injuries is a realistic expectation when using an athletic mouthguard, especially if undergoing traditional orthodontic treatment. This, however, is not a proven fact based on the dental literature.

### 7.5.3 Concussion Protection

Since the 1960s with the publication of the findings of Jack Stenger, there has been a widely held belief that the use of an athletic mouthguard can play a role in the reduction in the incidence and severity of concussions in sports [30]. This topic is covered elsewhere in this volume, and currently it is believed that there is a weak, if any, connection with mouthguards and concussions. Anecdotal, small studies and at least one clinical trial continue to implicate the mouthguard as a protective piece of equipment in the fight against concussions, but realistic proof has not been found.

### 7.5.4 Bone Protection

There are two papers that deal with the ability of mouthguards to protect against injuries to the bone. Following Stenger’s groundbreaking “case reports” of concussion protection, Hickey et al. looked to investigate this with cadavers and pressure sensors. In addition, he also examined bone deformation on the mandible of these cadavers when stuck with and without mouthguards. He showed a definite diminution of bone deformation when a mouthguard was in place [31].

Takeda et al. reproduced this work with an artificial bone system and found similar diminution of energy to the bone [32]. These two studies are both in vitro, and obviously a clinical study would be extremely difficult to accomplish. It does however appear that a resilient mouthguard *should* dampen the effect of a blow to the jaws if this specific force was received.

### 7.5.5 Temporomandibular Joint Protection

The Academy for Sports Dentistry makes a slide series available to its members from which to teach others about sports dentistry. In this series are a well-known two photograph set of a dry skull without a mouthguard in place and one with a guard [33]. These pictures are meant to demonstrate that with a guard the mandibular condyle is displaced downward and out of the glenoid fossa. This example has been used for years to imply that mouthguards have a protective effect on the temporomandibular joint.

Unfortunately the truth is not that simple. At least one expert in the treatment of TMD (temporomandibular joint dysfunction) has stated that the most stable positions for the joint “are when the condyles are in their most superoanterior position in the articular fossa, resting against the posterior slopes of the articular with the discs properly interposed. The condyles assume this position when the elevator muscles are activated with no occlusal influences” [34], in other words, a fully occluded position. This would lead one to believe that a mouthguard that allows for a solid clenched “bite” might protect the TMJ but not simply by altering the position of the condyle.

Mark Roettger of the University of Minnesota, using a magnetic resonance imaging (MRI) system, evaluated the actual position of the condyle on a living volunteer (Fig. 7.6a–c). He imaged the condyle with the subject fully occluded with no mouthguard, one that was 3 mm thick between the molars and one which was 5 mm thick. With the use of a reference line, one can see that the condyle actually seems to change very little. Dr. Roettger notes that “there is an increased recoil space and that the disc stays in its proper position.” He has also cautioned that this example is only one individual [49].

### 7.5.6 Enhancement of Athletic Performance

The possibility that an intraoral device may impact the performance of an athlete has been studied for quite some time. A fair number of retail products as well as a fairly widespread literature have implied that a specifically designed appliance worn by an individual with certain characteristics, especially related to occlusion, might impact performance characteristics

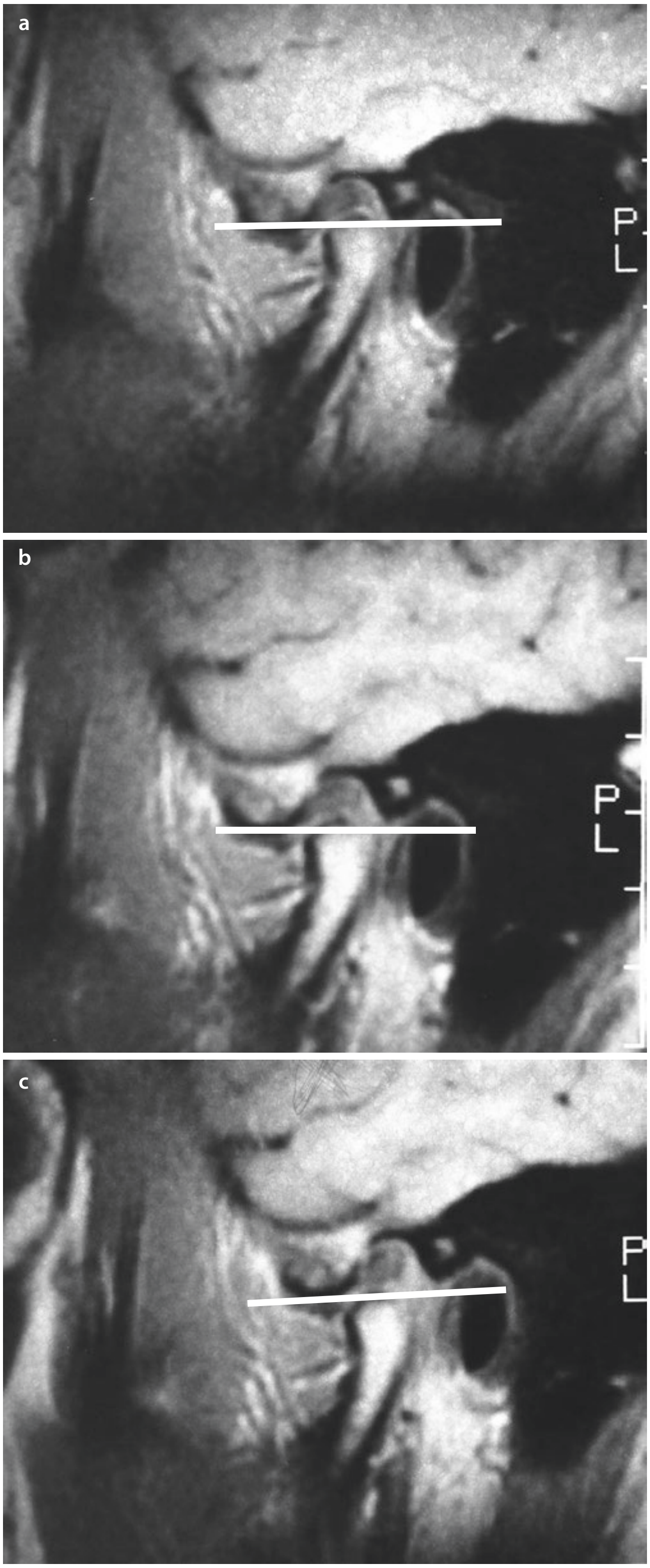



**Fig. 7.6** **a** Condyle while fully occluded and no mouthguard. **b** Condyle with a 3-mm-thick mouthguard while fully occluded. **c** Condyle with a 5-mm-thick mouthguard fully occluded

such as strength, endurance, and hormonal changes. This is covered elsewhere in this volume, and for now, this question, like concussion protection, is unproven but is still actively being researched all over the world.

In addition to the potential functions named above, Johnson and Winters, in 2000, listed the following advantages of mouthguard use: athletic confidence, savings on the cost of treating orofacial injuries, and that the athletic mouthguard is an opportunity for the dental profession to be an advocate and direct aid to athletes. These advantages are obviously not measurable, but they do indicate that the benefits of oral protection can go beyond just avoiding injuries [36].

## 7.6 Fabrication Techniques

The two types of custom-fitted mouthguards are characterized by the involvement of a dental professional, whether a dentist, dental technician, dental hygienist, or some other member of the dental team. The dental literature is relatively full of articles offering variations on the basic techniques of both vacuum forming and heat-pressure forming mouthguards [28, 37, 38]. While the various techniques may all have their own advantages, none has been clinically evaluated from the standpoint of the technique itself. While mouthguards fabricated by one or the other technique may perform differently in in vitro situations, the differences have not proven to hold any advantage in vivo.

In this section we will discuss the basic techniques for both vacuum forming and heat-pressure lamination techniques.

### 7.6.1 Vacuum-Formed Mouthguard Technique

The basic concept to create a vacuum-formed mouthguard is to use a vacuum forming machine to heat a sheet of material, usually ethylene vinyl acetate, and then to draw it down over a cast of an athlete's teeth. The vacuum pulls the material down and removes the air between the cast and the material, and this forces the material to conform to the cast. The sheet of material is then cut to the appropriate borders to create the finished guard, or second layer of material can be added. The steps of this process are:

1. Creation of the dental cast: An impression is taken of an athlete's teeth. This is probably the most important step in the mouthguard process. The impression is usually of the maxillary arch unless the athlete has a prognathic occlusion. This upper arch is used as the literature is clear that the vast majority of the dental injuries involve the maxillary incisors. If the mandibular incisors are forward of the maxillary ones in centric occlusion, then a lower impression is recommended. The impression can be taken with any quality impression material, but alginate is the most commonly used impression material. It should be "poured up" in a good quality stone that is strong enough to hold up to multiple mouthguard formations. The impression must have adequate extensions to insure that the mouthguard covers all of the most important structures (Fig. 7.7).

**Fig. 7.7** Typical casts for a custom-fitted mouthguards with adequate extension. The cast can be finished with the palate intact to retain model strength, but then it must be vented with a hole in the palate to ensure optimal vacuum forming details

**Fig. 7.8** Applying separating medium

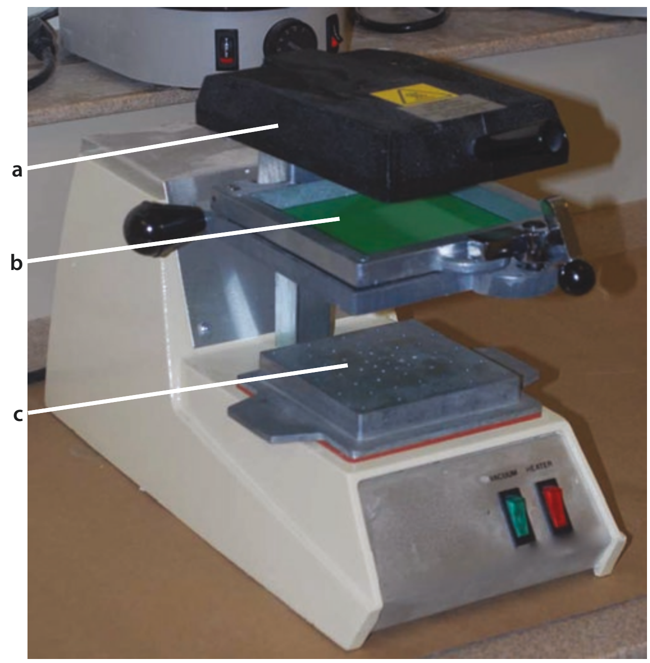



**Fig. 7.9** Typical vacuum forming machine. **a** Heating element, **b** sheet of EVA secured in the holding component, **c** vacuum stage

The cast is trimmed to remove all of the “land area” so that the material can be pulled down evenly. The palate area can be left intact, but as the guard will not be covered by the mouthguard, it is not necessary. Leaving the palate as part of the cast will make for a more durable cast, but if it is left, a hole must be placed in the palate to allow for adequate vacuum forming. The facial surface of the incisors should be approximately 90° to the platform. The cast must then be treated with a die separating medium to allow for easy removal of the mouthguard material (Fig. 7.8).

2. One sheet of EVA is placed in the forming machine, and the cast is placed in the center of the vacuum stage of the device (Fig. 7.9). The EVA sheets can be obtained in a variety of thicknesses depending on what is trying to be accomplished. A common thickness is 4 millimeters (0.016 in.). This thickness is generally used when the mouthguard is to be made with just one layer. One manufacturer creates a “laminated” 4 mm sheet for just this purpose.

In this section, a laminated mouthguard is being created, so the first sheet of material used will be 3 mm thick (0.012 in.). In the vacuum forming process, the thickness of the formed sheet reduces approximately by half and in some areas, such as the incisal edge of the anterior teeth, thins even more [39].

The sheet is heated until it sags approximately 20 mm from its original position, the vacuum is turned on, and the material is lowered over the cast. This amount of

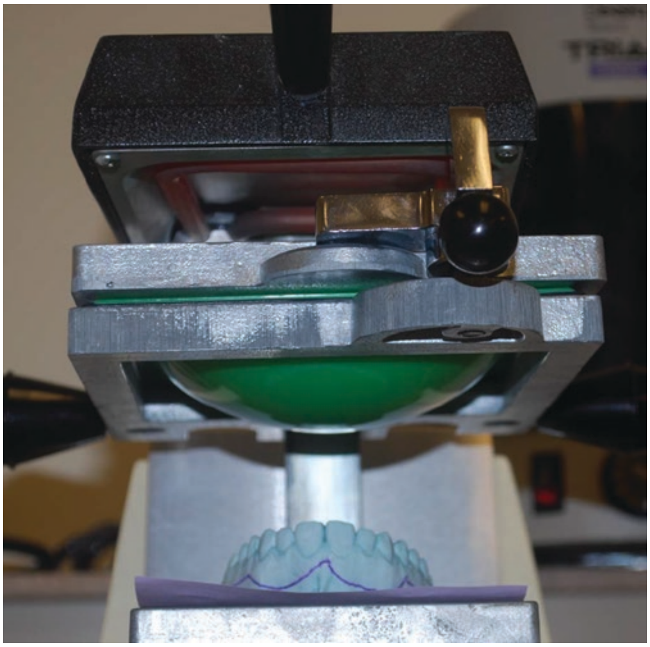


Fig. 7.10 EVA sheet heating and sagging 1–2 in.

sagging will allow the material to achieve the temperature which is most recommended for vacuum forming [40]. It has been demonstrated in one study to leave the vacuum on for approximately 90 s [41]. With moistened fingers or a moistened paper towel, the material can be digitally molded further as it cools (Fig. 7.10).

3. Once thoroughly cooled, the cast and the EVA are trimmed to the proper contours for a mouthguard as marked on the original cast. According to one clinical handbook from Australia, the facial extensions in the anterior region ideally should be within 2 mm of the vestibular reflection, and the frenum attachments should be relieved [37]. The palatal borders are recommended to be approximately 10 mm above the gingival margin [37]. This last recommendation however is not usually followed, and the most common trimming of the palatal extension is with 1–2 mm to the gingival margin. Trimming can be accomplished with scissors, with a heated knife or scalpel, or with an electric hot knife.
4. At this time the first layer can be decorated or personalized with logos and the athlete's name. Decals are available easily or can be created individually. Any decoration on the first layer must be made of vinyl or must not reach the borders of the guard as this decoration may not laminate completely (Fig. 7.11).
5. A useful step if trying to laminate with a vacuum forming technique is to take the first layer and vent any areas that might trap air when the second layer is placed. Each depression is punctured with a sharp-tipped pin or bur (Fig. 7.12).

Fig. 7.11 Decorations and name on first layer. Paper logos must not touch the edges of the guard to ensure sealing it into the lamination

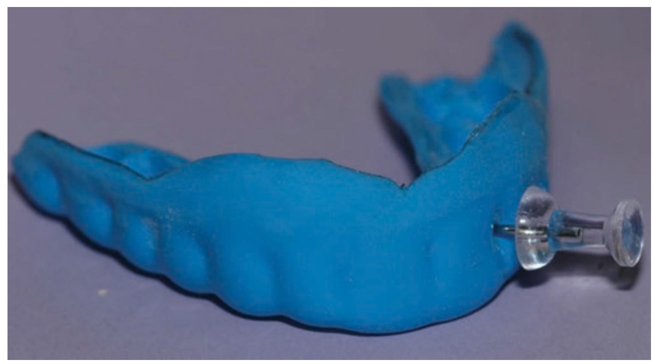


Fig. 7.12 Puncturing any potential areas and indentations that might trap air

6. A second and final layer of EVA is then heated and placed over the preliminary guard as described in step 2. Prior to this step, the first layer must be cleaned and made free of any oils from handling it to allow for complete lamination. In fact, it is often helpful to slightly roughen the surface to remove the "gloss" (Fig. 7.13).
7. After this last step, allow the guard to cool, and then the final guard is trimmed to the extensions established in step 3. The facial and buccal borders are left somewhat rounded, and the palatal borders are finished to a thin feathered edge. The final thickness of the guard is usually recommended to be approximately 3 mm at least over the incisal surfaces of the anterior teeth and the occlusal surfaces of the posterior teeth [28].
8. The finished edges are buffed with vinyl buffing wheels in a hand tool, and then the mouthguard is flamed gently to achieve a glossy finish (Fig. 7.14).
9. The final guard is well laminated and has excellent internal definition (Fig. 7.15).

Fig. 7.13 Second clear layer laminated over the colored and decorated first layer

Fig. 7.14 Buffing and flaming the extensions to finish

10. The occlusion will be finished specifically for the athlete either chairside or with an opposing mandibular model. This can be done simply by heating the posterior occlusal surfaces of the guard and then putting the guard in the athlete's mouth and having them bite gently to balance the occlusion. This could be accomplished extraorally by mounting the opposing arch (usually the mandibular arch) on a simple articulator and establishing a depth of bite. The guard is then heated and then occluded with the opposing cast to produce the occlusion.

    Takeda et al. have consistently championed the concept of the totally balanced occlusion for both the protection of all of the teeth and the stability of the appliance [42]. While this concept makes sense, the reality seems to be that a solid occlusion of the posterior teeth is the most commonly employed.

    This vacuum method creates a very excellent and serviceable mouthguard. It should be noted however that this technique uses lower heat and results in a slightly inconsistent lamination. There are also wide variations between various vacuum forming machines. The vacuum forming technique historically has been used for one sheet mouthguard fabrication as opposed to lamination. Because of this, some machines are better suited to this technique than others. It is essential to this technique to seal all of the margins with heat to make sure that the mouthguard stays together and remains fully laminated.

### 7.6.2 Heat-Pressure Lamination Technique

This technique became popular in the United States in the1990s while already being used elsewhere in the world. It is based on the use of special machines that are designed to push rather than pull a heated sheet of mouthguard material over an athlete's cast. It allows for a very precise internal adaptation of the material to the cast, and the pressure enables one to achieve excellent lamination consistently. There are four or five types of pressure machines currently available, and each one is slightly different, but the basic premises are the same: Multiple sheets of material are laminated to one another to achieve an excellent fitted and personalized mouthguard.

For this section the machine used is the MiniStar from Great Lakes Orthodontics (Fig. 7.16). This machine heats the sheet, and then this is flipped over on top of the cast. The portion of the machine that holds the material is the pressure


**Fig. 7.15** The finished vacuum-formed laminated mouthguard

chamber. Other types of heat and pressure machines use a pressure chamber that forces the heated material down onto the cast. Both types of machine work by the same principle but are mechanically different.

1. The impression and the cast should have the same characteristics as with the vacuum forming technique, but it is not as critical as the material is uniformly pushed onto the cast (Fig. 7.17). No allowance has to be made for proper suction. The palate can be left untouched but is often removed to

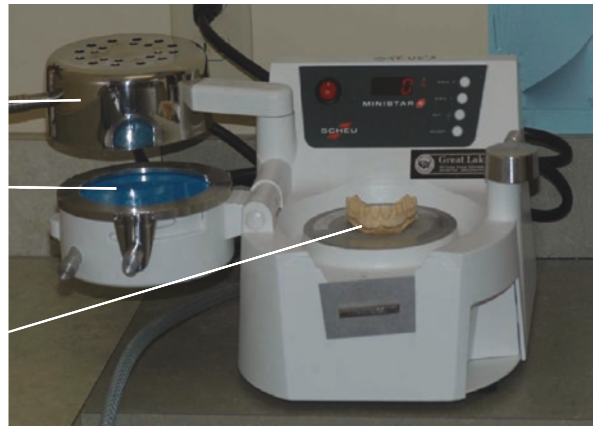



**Fig. 7.16** Heat-pressure lamination machine. **a** Heating element, **b** sheet of EVA secured in the pressure section, **c** stage with cast. The stage can be replaced with metal beads

make it easier to form or trim or to remove the mouthguard. The cast is once again treated with die separating medium.

2. The round sheet of material (0.012 in. of EVA) is locked into the pressure unit, and the heating element is swung over. For most of the current devices used, the machine is programmed for each type of material, color of material, and thickness of the material. Setting the machine to time the heating of the sheet is done with a scanner and a bar code recognition system (Fig. 7.18).
3. After the appropriate heating time, the chamber is flipped over onto the cast and locked in place. The action of locking the chamber initiates the pressure, and the machine automatically counts down a specified time for cooling. The cooling of the material before trimming is critical to avoid distortion of the guard (Fig. 7.19).
4. After cooling the first layer is trimmed in a similar manner to that of the vacuum forming section (Fig. 7.20). Some experts recommend trimming this section without removing the mouthguard from the cast. The thinking is that this will avoid distorting the first layer. It is possible however to remove this first layer once it is thoroughly cooled, and this makes it easier to trim. This is also a good time to thin or remove any areas of the mouthguard which are not essential for protection, such as the palatal and buccal sections of the posterior molar areas. Once again it is advisable to clean this layer of all contaminants or oils and perhaps to buff it slightly to remove the gloss of the first layer.
5. The first layer is then customized, and a second layer of 0.012 in. (3 mm) clear EVA is placed over the first. Again, as with the vacuum forming technique, a sharp tack or instrument can be used to puncture the

**Fig. 7.17** Trimmed casts are essentially the same as for vacuum forming, but for pressure forming, the palate does not need to be removed or vented. It is done so here for the ease of trimming and working with the mouthguard


**Fig. 7.18** The heating element in place. Note the timer counting down the appropriate time for the thickness and color of the material


**Fig. 7.19** The pressure element with the material has been turned over onto the cast and locked in place. The machine then counts down until adequately cooled

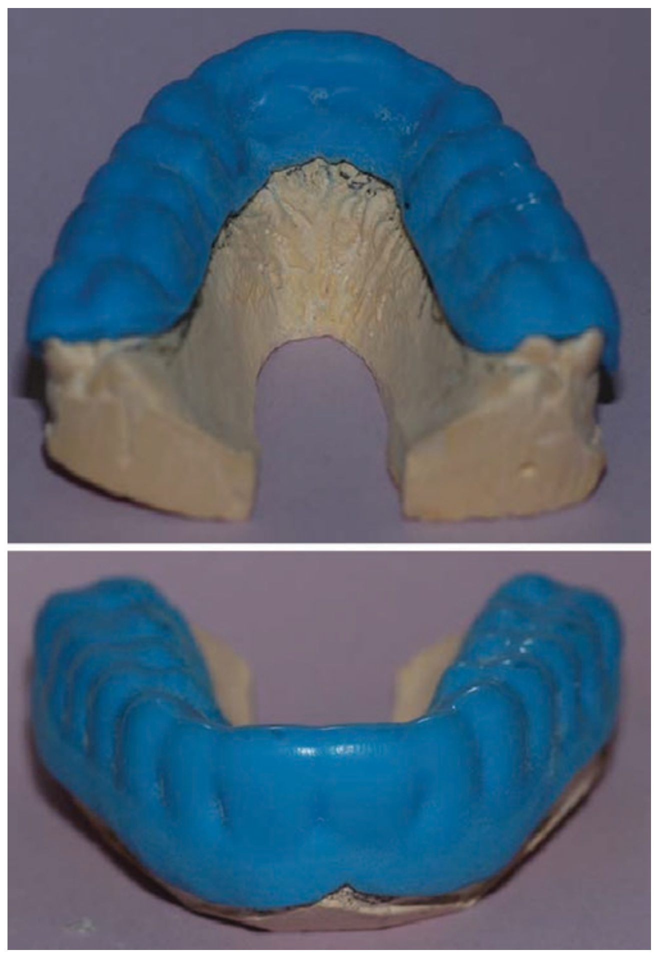


**Fig. 7.20** The first layer trimmed and corrected to margins. It is advisable to thin the margins of the palatal extensions to a feather-edge. The buccal and facial margins are thinned but left slightly rounded

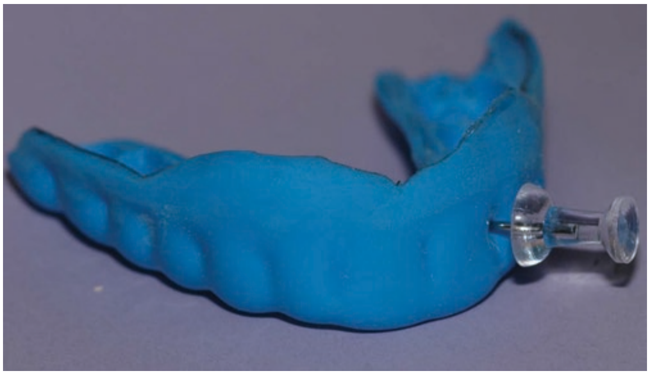


**Fig. 7.21** Puncturing with sharp object in areas of deep indentations will avoid air entrapment and incomplete lamination

**Fig. 7.22** Customized with name, paper logo, and vinyl tape stars

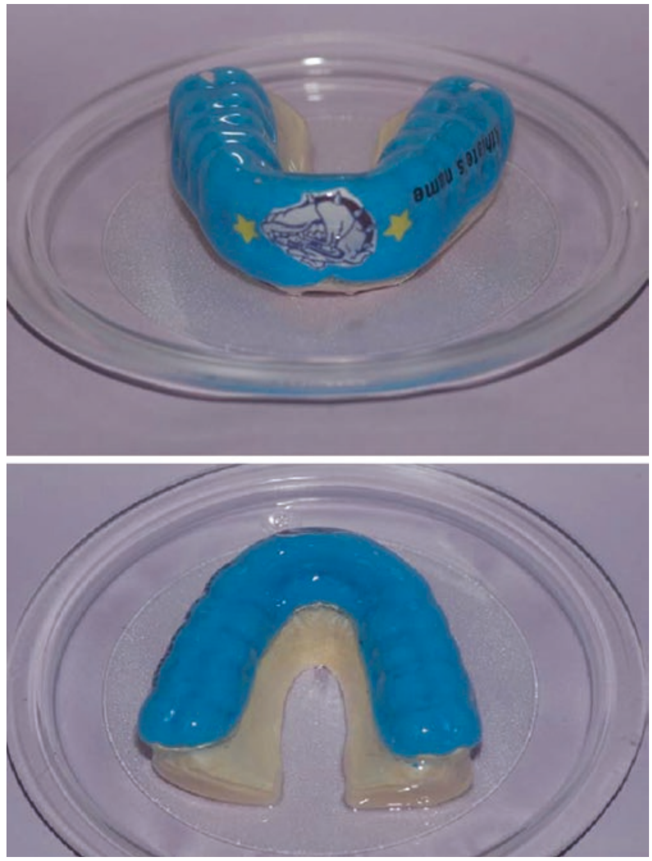


**Fig. 7.23** Clear layer laminated over the first. Note the consistency of the clear layer filling the indentations as a result of the punctures of the first

first pull at any indentations that may trap air between the layers. Also, even though it is more likely to get consistent lamination with this technique, it is still prudent to make sure that the first layer is clean and free of contaminants and oils and perhaps even de-glossed prior to lamination (Figs. 7.21, 7.22, and 7.23).

6. After cooling, the mouthguard is trimmed to the proper extensions. The facial and buccal borders are left rounded, and the palatal borders are feather edged. The finished mouthguard is polished with different buffing wheels and then detailed with a butane flame (Fig. 7.24).
7. The occlusion is corrected either intraorally or on an articulator as described in the section on vacuum forming techniques. With the heat-pressure lamination technique, each layer still thins to approximately half of its initial thickness, but with the pressure machine, the thickness over the entire mouthguard is slightly more consistent than the vacuum-formed guards that pull thin spots more often. The guard created here used two layers of 3 mm EVA and has resulted in a guard that is 3–4 millimeters thick in the critical anterior facial area and the occlusal regions of the posterior teeth (Fig. 7.25).

   The heat-pressure lamination technique produces a mouthguard with consistently precise and sharp internal adaptation (Fig. 7.26). Intimate adaptation is possible with a vacuum forming technique, but it is more consistently and easily achieved with the pressure technique. This results in a very snug and secure fit.
8. Using tools available by at least one manufacturer may use a pressure lamination technique to create custom mouthguard material.

Fig. 7.24 Finished heat-pressure laminated mouthguard

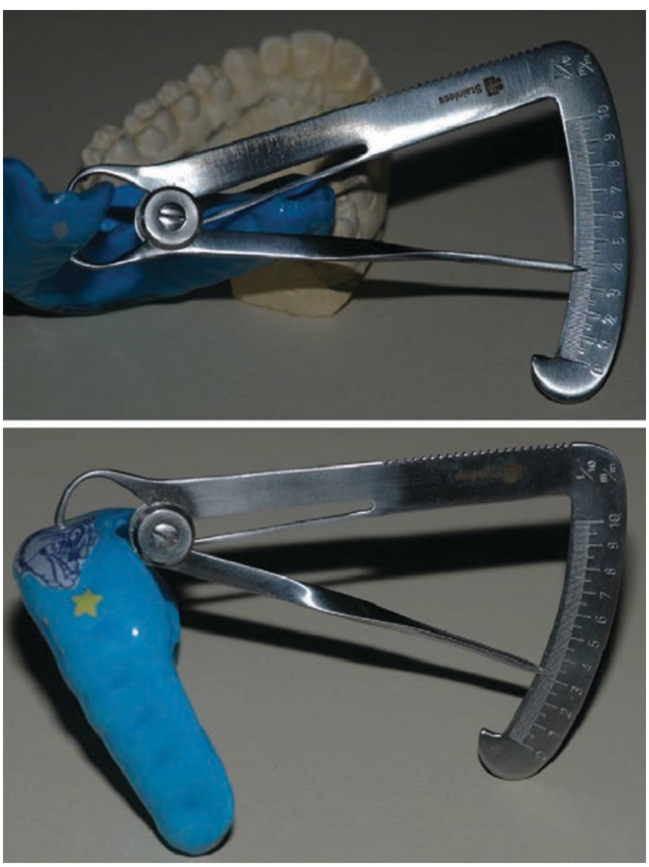


Fig. 7.25 The thickness of the guard made of two layers of 3-mm-thick EVA results in a guard of 3–4 mm thickness

Fig. 7.26 The internal detail achievable with the pressure technique is clear and very precise This results in a secure and snug-fitting guard

### 7.6.3 Straps for Custom-Fitted Mouthguards

Athletes who play sports that have helmets with face shields as part of the standard equipment (e.g., American football, youth and collegiate ice hockey, and men's lacrosse) often request to be given a mouthguard that has a strap or a tether which can attach the mouthguard to the face shield. Most sports dentists would prefer that a properly fitted mouthguard not have this strap as it impairs speech and lessens the comfort of an otherwise excellent appliance. Straps do have the advantage of lessening a player's likelihood of losing his mouthguard during or between practices or games. It has also been suggested that having an attachment would be a safety feature that would make it easier to remove a dislodged guard from someone's mouth if needed. In reality, the likelihood of this happening with a properly fitted mouthguard is extremely small.

Many retail mouthguards can be purchased with a strap attachment either as part of the guard or with one that can be easily attached. Attaching a strap to a custom-fitted mouthguard is not difficult, but there is no one best method for doing this. In fact many techniques have been used with varying degrees of success. Two common and generally reliable methods will be described.

One manufacturer makes a simple tab that can be attached to a mouthguard (Fig. 7.27). The tab is attached by heating the tab and the mouthguard. The two are then pressed together. Placing a layer of hot glue between the two items can enhance this adhesion. The mouthguard and tab can then be shaped and polished to make the appliance comfortable (Fig. 7.28).

Another slightly more durable technique utilizes the lamination technique. A small dowel such as the shaft of a cotton-tipped applicator is tacked to the incisal edge area of the mouthguard with a small amount of hot glue (Fig. 7.29). The dowel should be lightly lubricated taking care not to get lubricant on the mouthguard that is to be laminated. A clear layer is then placed over the dowel and the mouthguard (Fig. 7.30). The dowel is removed (Fig. 7.31), and an appropriate diameter piece of surgical tubing is threaded through the created channel (Fig. 7.32). The two ends of the tube are attached by placing a small dowel into the two ends. The center of the tube loop is kept together with a small amount of vinyl tape. As shown in the photograph (Fig. 7.32), the circle of tubing can be looped around a bar of a face shield.

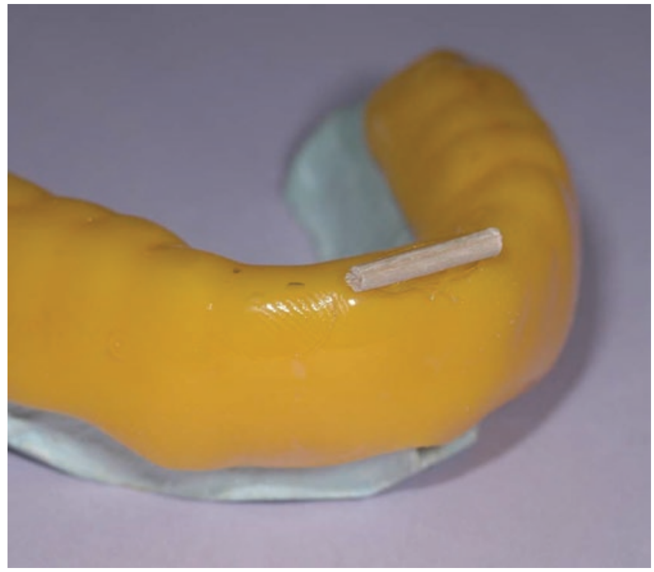


**Fig. 7.29** Small dowel tacked to mouthguard

**Fig. 7.27** Mouthguard, strap attachment tab, and hot glue stick

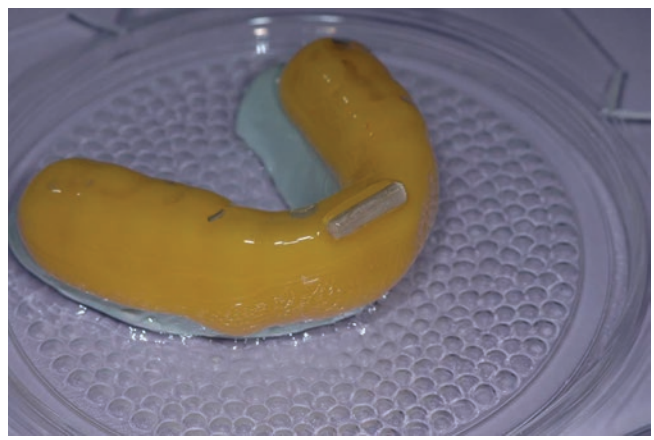


**Fig. 7.30** Clear layer over dowel

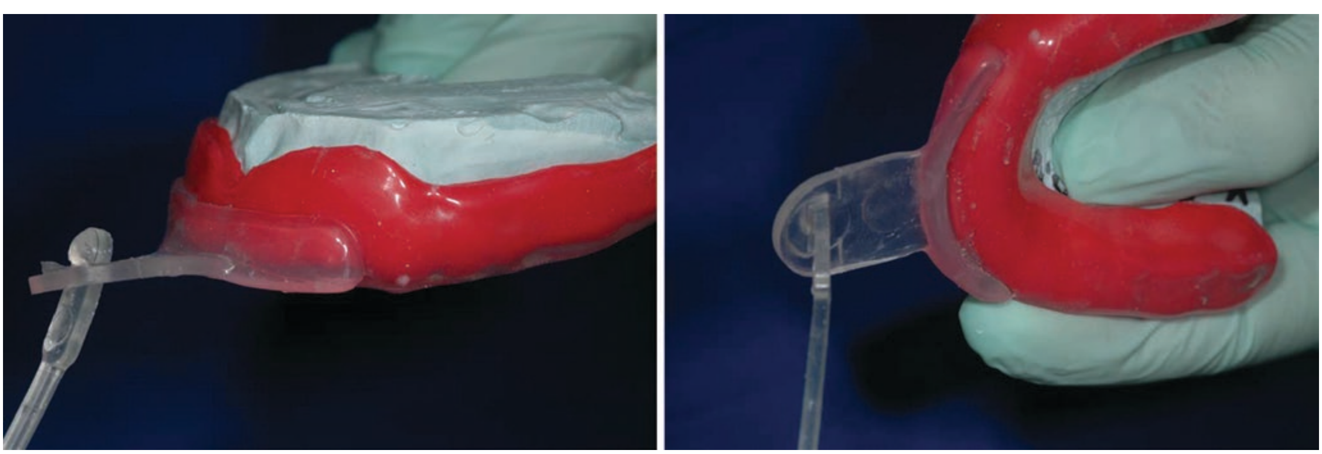


**Fig. 7.28** Attached tab and tether to mouthguard

### 7.6.4 Variations on the Basic Custom Mouthguard Technique

The basic one- or two-layer vacuum-formed mouthguard and the basic two-layer heat-pressure laminated mouthguard are designs that seem to work well for the majority of athletes. However many variations of these basic concepts have arisen and may or may not increase the effectiveness of the mouthguard for various athletes in various sports. At least one commercial supplier of custom mouthguards offers a variety of thicknesses for what they describe as special for different ages and different needs and sports. On the surface, it seems only common sense that a thicker mouthguard will promote greater safety, but the clinical evidence for this is lacking.



Most of the variations on the common theme are tested in in vitro situations that may or may not translate to clinical effectiveness. Perhaps the most common variation is to add layers to increase the thickness of EVA over the teeth. An often-cited study by Westerman indicated that after 4 millimeters of thickness, the shock absorption tends to plateau [43]. Takeda et al. in their textbook give a common sense rationale for recommending a 3-mm-thick final dimension of their mouthguards. This has to do with the plateau of protective ability based on force absorption at approximately 4 mm coupled with the generally observed finding that most athletes find the 4 mm mouthguard a bit too thick to be comfortable [42].

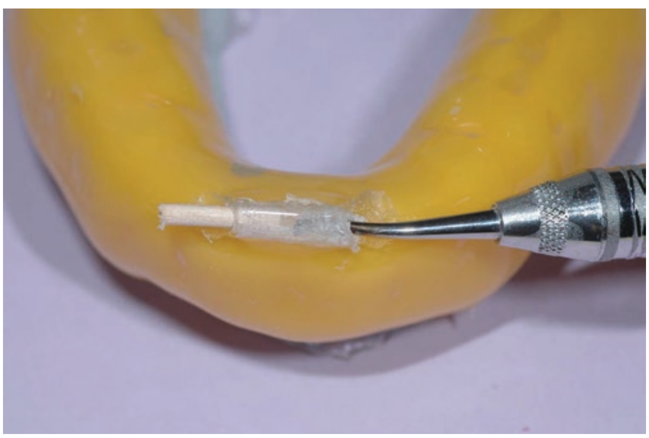


Fig. 7.31 Removal of the dowel

Another study has shown that the thickness of EVA continues to increase protection until approximately 9 millimeters. This is interesting but is clinically unrealistic. So, for most athletes where the risk of injury is possible but not considered common, 3 millimeters is appropriate and is the ASD recommended thickness on their position paper on properly fitted mouthguards. For those athletes, like boxers and mixed martial arts competitors, where injury is expected, they might willingly accept a thicker guard.

It is often attempted to combine the shock absorption of the most common hardness of EVA with a layer of something harder (like biocryl or titanium). This concept tries to combine the shock absorption of EVA with a harder material's ability to distribute forces. This again makes a great deal of common sense, but laboratory tests have indicated that merely introducing a more rigid layer of hard material between two layers of soft material does little to change the benefits of the guard.

It has also been speculated that a space of air or a void between the mouthguard and the teeth would aid in dental protection. This actually has been seen to improve some characteristics in potential design.

Takeda et al. have proposed a composite design that incorporates both a hard insert as well as a space over the facial and incisal surface of the incisors. He used in vitro testing of four conditions of (1) no mouthguard (the control), (2) a dual-laminated mouthguard made with two sheets of 3 millimeter ethylene vinyl acetate, (3) a dual-laminate guard with a hard resin inner layer, and (4) a dual-laminated mouthguard with a space over the incisors and a hard acrylic insert. They found that the combination of space and hard insert tested best in their experimental design [35].

The level of sophistication and time that this takes to fabricate will make this design both expensive and uncommon, but for those circumstances where the maximum in protection is desired, it may be the most complete design. However,

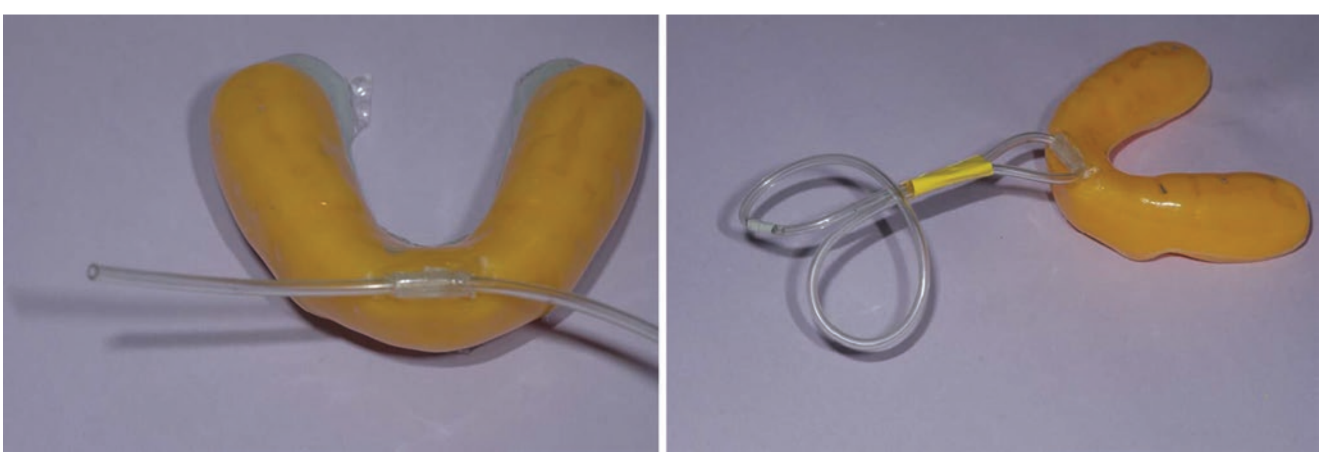


Fig. 7.32 Surgical tubing inserted through the channel and the completed strap

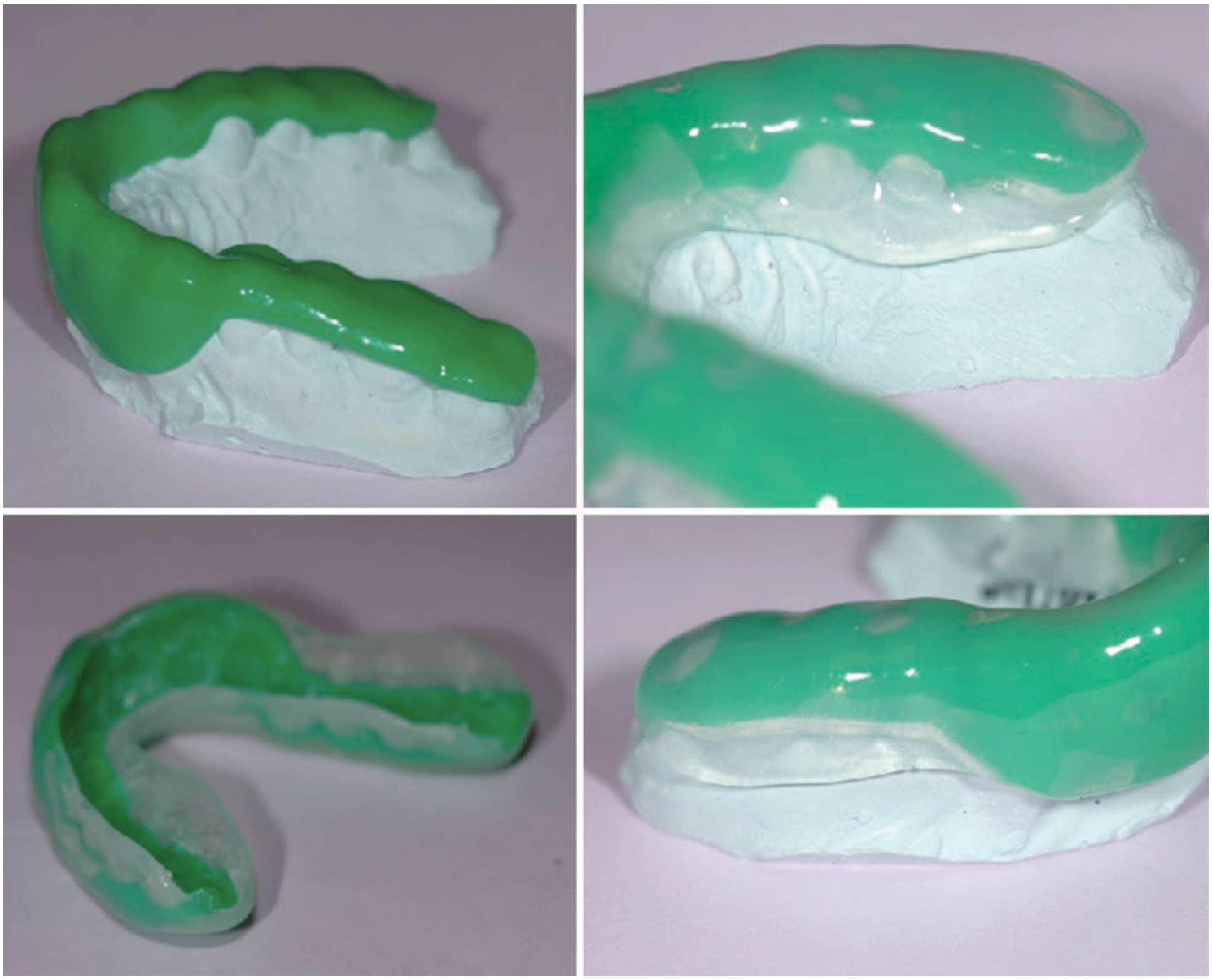


Fig. 7.33 A laminated mouthguard where the key areas are laminated, and one layer is used only for areas that do not need as much protection

like most innovative designs, the in vitro results cannot automatically be extrapolated to equate to clinical situations.

Finally, most athletes will desire the most comfortable design regardless of protection. This means a combination of fit and imperceptibility. The "thinness" of a mouthguard generally translates into the lack of perceptibility of the guard in the mouth, and most athletes want a guard that is as thin as it can be. This can be achieved by trimming a first layer of EVA to cover only the incisors and the occlusal surfaces of the occluding teeth. Then the second layer is pulled directly over much of the cast and only laminates to the separate segments. This can easily be accomplished with both the vacuum forming and the heat-pressure laminating techniques. The mouthguard will then be acceptably thick where it needs to be and as thin as possible for the rest. This is a good combination of protection and comfort (Fig. 7.33).

Other newer materials such as the rigid material used in the "SISU" mouthguard, or the softer EVA derivative, "Polyshok," or an interesting polyolefin material known as Vistamaxx which recently received the American Dental Association Seal of Acceptance in a boil and bite guard, are some new materials which have been used. Also various hardnesses of EVA from various manufactures are available. They all hold promise for different circumstances but have no more clinical proof of protection than any others.

### 7.6.5 Mixed Dentition and Orthodontic Patients

Athletes undergoing orthodontic treatment and young athletes who are in the midst of a transitional mixed dentition still participate in sports [38]. The dentitions of these players are characterized by changes in the position of the teeth. These situations make a very intimately fitted mouthguard to all of the teeth unrealistic.

The basic goal of fabricating a mouthguard for a changing dentition is to create an excellent mouthguard for the length


Fig. 7.34 Blocked out mixed dentition cast to allow for changes which might occur over a 4-month season

of one season. The dental professional, especially the dentist, should envision the changes that will occur over the course of one season to be able to fabricate a mouthguard.

The most important feature of making a mouthguard for the mixed dentition is to evaluate the natural changes in the dentition that will inevitably occur. Primary tooth exfoliation, permanent tooth eruption, space closure, and continued eruption of existing permanent teeth are all important features when projecting what will happen in the immediate future. These changes will be addressed with the use of appropriate blockout materials to allow for the changes while maintaining a snug-fitted mouthguard (Fig. 7.34).

Teeth with short clinical crowns characterize a dentition that contains a significant number of partially erupted permanent teeth as well as primary teeth. The retention of an athletic mouthguard is based almost entirely on the intimate fit of the guard to the teeth. It is therefore very important that any teeth that will remain in one place during the course of the use of the guard must be very intimately fitted to create a well-retained mouthguard. These guards tend to be loose compared to older athletes. A custom-fitted guard is much more likely to be well tolerated by these players.

Orthodontic patient's teeth are continuously changing throughout the course of their treatment. Patients who are early in this process tend to see significant changes in tooth position quite quickly. In addition to this, the actual orthodontic appliances such as bands, brackets, and wires, as well as auxiliary appliances, often create impediments to wearing an appliance that can easily be placed in the mouth over the devices.

One of the most challenging and crucial aspects of making a custom-fitted mouthguard for an orthodontic patient is the ability to obtain an acceptable impression. The impression tray needs to be large enough to be able to include all parts of the intraoral appliances. The impression material must not tear or distort when being removed from the mouth.

If it is practical, it is best to remove the orthodontic arch wire from the patient's brackets and to impression the teeth with no wire in place. The brackets themselves and any bands with orthodontic features will create small tears in the impression material, but these will be corrected for prior to the mouthguard forming technique.

If it is not possible or practical to remove the arch wire, some type of soft wax (rope wax, orthodontic relief wax, etc.) is often placed over the brackets and wire. This will allow the impression material to be removed more easily. It is important not to use too thick a layer of wax, as this will make the resulting mouthguard feel too prominent. It would also make it difficult to remove some stone from the cast to thin the prominence, as it would be necessary to guess how much to remove.

Another innovative way to take this impression is to stretch an elastic sheet over the brackets. A rectangular piece of rubber dam or similar elastic material can be cut, and a small hole can be placed at each end. Other holes can be placed for individual teeth so that the elastic sheet does not slide over the dentition. The posterior holes can then be hooked over the most distal feature of the set of brackets on one side, and the other end can be stretched around and hooked over the same feature on the other side. The auxiliary holes are placed over the brackets that it approximates (Fig. 7.35). The elastic component neatly covers the brackets and conforms to the teeth above and below the brackets. A commercial version of this is available if elastic material cannot be fabricated.

Once an acceptable cast has been created, the situation is similar to that described for the mixed dentition. It has to be determined what changes will be most likely to occur during the course of the particular season. Space manipulation, rotations of teeth, and arch width and length changes as well as any eruption changes are the most common features of orthodontic care that must be accounted for. In addition, the mouthguard must be fabricated so that it can easily be inserted and removed while staying securely in place.

Again, blockout material can be used to create spaces where dental changes can be accommodated. Unlike the mixed dentition cases, there are usually just a few teeth that will be able to be intimately fitted to the mouthguard as most are being moved. The appliances themselves often help hold the mouthguard in place but the overall fit must, by necessity, be somewhat loose. The soft tissue extensions in the orthodontic patient often aid in accomplishing a good fit.

**Fig. 7.35** Using a rubber sheet to cover orthodontic brackets

The use of custom-fitted mouthguards in orthodontic patients is especially significant with high school wrestlers in the United States. Wrestling is a very intimate, intense, and aggressive sport. It is essential that any intraoral device must be secure so that it does not come loosen during competition. The athlete must be able to breathe easily and occlude well. High school wrestlers are required to wear guards to fully cover orthodontic appliances (maxillary and/or mandibular) to protect their opponents as well as to reduce their own intraoral injuries. Therefore, fitting mouthguards to these orthodontic patients is critical.

## 7.7 Contamination, Sanitization, and Replacement of Athletic Mouthguards

The athletic mouthguard is undeniably an effective and valuable piece of sports safety equipment. A single mouthguard does not however last forever. The athlete should be instructed how to care for his guard and when to replace it. Mouthguards often become chewed, worn, contaminated, and torn, and the edges can become rough and sharp. This leads to a poorly fitted, poorly configured, and ineffective protective mouthguard.

One of the true benefits of a properly fitted mouthguard is that it is less likely to be worn loosely or improperly. A loosely fitted mouthguard is often moved about considerably during sports activities and can be chewed out of shape. It can be wedged into a face shield or helmet. Between contests and practices, a mouthguard can be improperly stored, and the guard then can be contaminated with contaminants and can be distorted. A properly fitted custom-fitted guard or an excellent mouth-formed guard can minimize these possibilities.

All intraoral appliances and mouthguards are no exception and must be regularly cleaned. All dental appliances can and do accumulate a significant microbial load. Studies by one particular group of investigators have demonstrated that mouthguards have been significantly contaminated in hockey players, football players, and dental students [44–46]. This group also has published case reports of the contamination of mouthguards leading to illness or impairment in several situations [47].

The athlete must attempt to keep the mouthguard clean. This usually implies regularly cleaning with a toothbrush and allowing the guard to dry between uses. In addition several commercial cleaners and disinfectants are available. It is important that any cleaning agent or disinfectant does not include any agent such as alcohol that might degrade the mouthguard material.

Mouthguards that are severely chewed, worn, misshapen, rough, or sharp edged can induce intraoral injuries. This, coupled with the potential microbial contamination of the appliance, is potentially an unhealthy situation.

Mouthguards are ideally used for one sports season. In reality, properly fitted mouthguards can be useful for longer than that if cared for correctly. Mouthguards should be kept clean and disinfected, and it should be stored in a properly vented case that allows it to dry. All effort must be made to not chew or abuse the mouthguard. The mouthguard must retain its secure fit, and it should retain its structural integrity. If it becomes worn, torn, ragged, or sharp edged, it should no longer be used, and it should be replaced.

In 2009 three clinical recommendations were published using the SORT or Strength of Recommendation Taxonomy on the care of mouthguards. Strengths for these recommendations are assigned grades of A, B, and C [48] (Fig. 7.36).

There is no reason to believe that there is any danger in using a properly cared for athletic mouthguard. It must be clean, it should be disinfected as much as possible, and it must maintain its structural integrity. It must not initiate any intraoral injury or irritation. And finally it must be replaced if it in any way does not fit the ideals of a properly fitted mouthguard.

## Clinical Recommendations

**SORT: Strength of Recommendation Taxonomy**
**A:** consistent, good-quality patient-oriented evidence
**B:** inconsistent or limited-quality patient-oriented evidence
**C:** consensus, disease-oriented evidence, usual practice, expert opinion, or case series

| Clinical Recommendation | SORT Evidence Rating |
|---|---|
| Sanitize the protective athletic mouthguard daily. | C |
| Replace the protective athletic mouthguard regularly. | A |
| Replace the protective athletic mouthguard when it becomes sharp or jagged whenever there is an oral irritation or ulceration. | B |

For more information about the SORT evidence rating system, see www.aafp.org/afpsort.xml and Ebell MH, Siwek J, Weiss BD, et al. Strength of Recommendation Taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician*. 2004:69:549–557.

**Fig. 7.36** Clinical Recommendations using the Strength of Recommendation Taxonomy (SORT)

## 7.8 Summary

The athletic mouthguard and the treatment of the injured athlete are the most important connections of the sports dentist to the sports medicine team. Athletic mouthguards are effective devices to lessen the severity and incidence of dental injuries. Mouthguards do not negatively affect performance and can be made comfortable and practical to athletes in sports that carry the risk of injury.

Today's sports dentist must be knowledgeable about the characteristics of the ideal mouthguard and how to fabricate an excellent mouthguard and must be creative enough to customize each mouthguard to fit the needs of his or her player/patients. Dentists must skillfully navigate the many different options for mouthguards for athletes and help players and their families obtain the best appliance for their activity and sport.

There is no doubt that an athletic mouthguard is one of the most effective pieces of sports safety equipment. It is imperative that sports dentists know everything about the modern mouthguard.

## References


1. Kumamoto DP. Sports dentistry in the United States. Int J Sports Dent. 2008;1(1):7–21.
2. Kvittem B, Hardie NA, Roettger M, Conry J. Incidence of orofacial injuries in high school sports. J Public Health Dent. 1998;58(4):288–93.
3. Collins CL, MacKenzie LB, Ferketich AK, Andridge R, Xiang H, Comstock RD. Dental injuries sustained by high school athletes in the United States, from 2008/2009 through 2013/2014 academic years. Dent Traumatol. 2016;32:121.
4. Huffman EA, Yard EE, Fields SK, Collins CL, Comstock RD. Epidemiology of rare injuries and conditions among United States high school athletes during the 2005-2006 and 2006-2007 school years. J Athl Train. 2008;43(6):624–30.
5. ASTM 2006 Mouthguard Standard ASTM 697 - 00(2006). Standard practice for the care and use of athletic mouth protectors.
6. A Properly Fitted Mouthguard. Position statements of the Academy for Sports Dentistry. 2010. http://www.academyforsportsdentistry.org/position-statement. Accessed 16 Jan 2016.
7. Gutman D. Banana bats & ding-dong balls: a century of unique baseball inventions. New York: MacMillan; 1995.
8. The history of the athletic mouthguard. Keystone Industries, U.S. Dental Manufacturer. https://keystoneind.wordpress.com/2013/09/19/the-history-of-athletic-mouthguards/. Accessed 16 Jan 2016.
9. Reed RV Jr. Origin and early history of the dental mouthpiece. Br Dent J. 1994;176(12):478–80.
10. Acrylic splints for athletes: transparent slip casings for the teeth as a protection from blows. J Am Dent Assoc. 1948;36(1):109–10.
11. Protecting athletes' teeth. Pittsburgh Post-Gazette. 1948;21:162–18.
12. Mouthguards. Wikipedia. https://en.wikipedia.org/wiki/Mouthguard. Accessed 16 Jan 2016.
13. Godwin WC. A simplified mouth protector technique. J Mich State Dent Assoc. 1962;44:132–3.
14. Godwin WC. A simplified mouth protector technique (part II). J Mich State Dent Assoc. 1962;44:227–34.
15. SafeTGardCorporation. Zoominfo. http://www.zoominfo.com/s/#!search/profile/company?companyId=88949479&targetid=profile. Accessed 16 Jan 2016.
16. Ranalli DR. Prevention of craniofacial injuries in football. Dent Clin N Am. 1991;35(1):627–45.
17. Castaldi CR. Prevention of craniofacial injuries in ice hockey. Dent Clin N Am. 1991;35:647–57.
18. Guideline 3C. Mouthguards, Jan 1986. Revised August 2009. 2013-2014 NCAA sports medicine handbook. p. 113–14. Accessed online at http://www.ncaa.org/sites/default/files/2013-14%20Sports%20Medicine%20Handbook.pdf.
19. National Federation of State High School Associations. Position statement and recommendations for mouthguard use in sports. Revised and approved October 2014. Available at http://www.nfhs.org/media/1014750/mouthguard-nfhs-smac-position-statement-october-2014.pdf Accessed 15 May 2015.

20. American Dental Association Council on Access. Prevention and mouthguards to reduce the incidence and severity of sports-related oral injuries. JADA. 2006;137(12):1712–20.
21. DeYoung AK, Robinson E, Godwin WC. Comparing comfort and wearability: custom-made vs. self-adapted mouthguards. JADA. 1994;125(8):1112–8.
22. Collares K, Correa MB, Mohnsam da Silva IC, Hallal PC, Demarco FF. Effect of wearing mouthguards on the physical performance of soccer and futsal players: a randomized cross-over study. Dent Traumatol. 2014;30(1):55–9.
23. Queiróz AF, de Brito RB Jr, Ramacciato JC, Motta RH, Flório FM. Influence of mouthguards on the physical performance of soccer players. Dent Traumatol. 2013;29(6):450–4.
24. Bourdin M, Brunet-Patru I, Hager PE, Allard Y, Hager JP, Lacour JR, Moyen B. Influence of maxillary mouthguards on physiological parameters. Med Sci Sports Exerc. 2006;38(8):1500–4.
25. Knapik JJ, Marshall SW, Lee RB, Darakjy SS, Jones SB, Mitchener TA, dela Cruz GG. Mouthguards in sport activities: history, physical properties and injury prevention effectiveness. Sports Med. 2007;37:117–44.
26. Sigurdsson A. Evidence-based review of prevention of dental injuries. Pediatr Dent. 2013;35(2):184–90.
27. LaBella CR, Smith BW, Sigurdsson A. Effect of mouthguards on dental injuries and concussions in college basketball. Med Sci Sports Exerc. 2002;34(1):41–4.
28. Padilla RR. A technique for fabricating modern athletic mouthguards. J Calif Dent Assoc. 2005;33(5):399–408.
29. dela Cruz GG, Knapik JJ, Birk MG. Evaluation of mouthguards for the prevention of of orofacial injuries during United States Army basic military training. Dent Traumatol. 2008;24(1):86–90.
30. Stenger JM, Lawson EA, Wright JM, Ricketts J. Mouth guards: protection against shock to head neck and teeth. JADA. 1964;69:273–81.
31. Hickey JC, Morris AL, Carlson LD, Seward TE. The relation of mouth protectors to cranial pressure and deformation. JADA. 1967;74(4):735–40.
32. Takeda T, Ishigami K, Hoshina S, Ogawa T, Handa J, Nakajima K, Shimada A, Nakajima T, Regner CW. Can mouthguards prevent mandibular bone fractures and concussions? A laboratory study with an artificial skull model. Dent Traumatol. 2005;21:134–40.
33. 2015 ASD Sports Dentistry for today's athlete members purchasers only. Copyright 2015. Academy for Sports Dentistry. www.academyforsportsdentistry.org.
34. Okeson JP, editor. Management of temporomandibular disorders and occlusion. 6th ed. St Louis: Mosby/Elsevier; 2008.
35. Takeda T, Ishigami K, Handa J, Naito K, Kurokawa K, Shibusawa M, Nakajima K, Kawamura S. Does hard insertion and space improve shock absorption ability of mouthguard? Dent Traumatol. 2006;22(2):77–82.
36. Johnsen DC, Winters JE. Prevention of intraoral trauma in sports. Dent Clin N Am. 1991;35(4):657–66.
37. The fabrication, use and maintenance of sports mouthguards. Revised ed. Australian Dental Association; 2013.
38. Croll T, Castaldi CR. Custom sports mouthguard modified for orthodontic patients and children in the transitional dentition. Pediatr Dent. 2004;26(5):417–20.
39. Tunc ES, Ozdemir TE, Arici S. Postfabrication thickness of single- and double-layered pressure formed mouthguards. Dent Traumatol. 2013;29(5):378–82.
40. Maeda Y, Yasui T, Yonehata Y. Fabricating manual for mouthguard – invitation for sports dentistry. Tokyo: Quintessence; 2001. (in Japanese)
41. Mizuhashi F, Koide K, Takahashi M. Mouthguard thickness varies with vacuum application time in the vacuum-forming method. Int J Sports Dent. 2015;8(1):37–44.
42. Takeda T, Ishigami K, Nakajima K, Kurokawa K. Mouthguards: the effects and the solutions for underlying problems. New York: Nova Science; 2011.
43. Westerman B, Stringfellow PM. Forces transmitted through EVA mouthguard materials of different types and thickness. Aust Dent J. 1995;40:389–91.
44. Glass RT, Bullard JW, Conrad RS. The contamination of protective mouth-guards: a characterization of the microbiota found in football players' protective mouth-guards as compared to the oral microbiota found in first-year medical students. Am Dent Inst Cont Educ J. 2006;93:23–38.
45. Glass R, Bullard J, Goodson L, Conrad R. Microbial contamination of protective mouth-guards in hockey players: an in vivo study. Compend Contin Educ Dent. 2001;22:1093–108.
46. Glass RT, Conrad RS, Kohler GA, Warren AJ, Bullard MS. Microbiota found in protective athletic mouthguards. Sports Health. 2011;3(3):244–8.
47. Glass R, Wood C, Bullard J, Conrad S. Possible disease transmission by contaminated mouth-guards in two young football players. Gen Dent. 2007;55:436–40.
48. Glass RT, Conrad RS, Wood CR, Warren AJ, Kohler GA, Bullard JW, Benson G, Gulden JM. Protective athletic mouthguards: do they cause harm? Sports Health. 2009;1:411.
49. Roettger M. Personal correspondence by email. Accessed 14 Feb 2016.

# Sports Dentistry and Public Health: Rules, Policy, and Politics

*Todd Thierer*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_8

## 8.1 Public Policy and Mouthguard Use

The Institute of Medicine has defined public health as "what we, as a society, do collectively to ensure the conditions in which people can be healthy" [1]. While it is hard to pinpoint the origin of the first public health measures, the ancient Romans developed the technology to implement the first large-scale projects in sanitary engineering [2]. There is also evidence that ancient cities in India had sewer and sanitary systems [3]. These measures were enacted using a public policy based primarily on the belief system of those in power interfacing with the political and social systems in which these communities existed.

The *Merriam-Webster Dictionary* [4] defines public policy as:

1. The governing policy within a community as embodied in its legislative and judicial enactments which serve as a basis for determining what acts are to be regarded as contrary to the public good
2. The principle of law by virtue of which acts contrary to the public good are held invalid

Therefore, public policy should provide the basis for statutes, rules, and other regulatory requirements. It should also allow for the removal of statutes, rules, and other regulatory requirements that are inconsistent with public policy. Unfortunately, there is no one source for public policy. Rather, there are combinations of influences that generate public policy. It has been suggested that the history of public health policy can be divided into three phases [5] "the dominance of medicine (the so-called clinical gaze, which Michel Foucault discussed in his classic 1963 book The Birth of the Clinic) to epidemiology (the community gaze which David Armstrong discussed in his 1983 book Political Anatomy of the Body), and finally to economics." However, all of these elements still play a role in determining public health policy. Each element exists, not in a vacuum, but within a milieu containing all the other elements (Fig. 8.1).

### 8.1.1 Scientific Evidence

Scientific consensus is just one of the influencing factors in creating a public policy regarding mouthguard use. The literature is generally supportive of the role of mouthguards in preventing orofacial injury. In a review of the literature ([6], pp. 270–280; quiz 281), the authors concluded that "Mouthguards have been shown to reduce the number of dental injuries." In another comprehensive review of mouthguard effectiveness


**Fig. 8.1** Impacts on mouthguard policy

([7], pp. 117–144), the authors stated that "Meta-analysis indicates that the overall risk of an orofacial injury is 1.6–1.9 times higher when a mouthguard is not worn." However, in a subsequent evidence-based dental review of this article, it was noted that "A major limitation of the current study is that although the authors performed a thorough systematic review and meta-analysis, no diagnostics were performed for publication bias and evaluating for heterogeneity. Not addressing publication bias leads to question the validity of the meta-analysis findings." Similarly, the Task Force on Community Preventive Services, an independent, nonfederal group of national, regional, and local public health and prevention services experts ([8], pp. 55–80), stated that "For the other interventions reviewed in this report… population-based interventions to encourage use of craniofacial protective equipment in contact sports—all systematic reviews found insufficient evidence of effectiveness or ineffectiveness." They go on to say that "It should be noted again that a finding of insufficient evidence is not a recommendation for or against use of an intervention, but rather a reflection both of the lack of qualifying studies on which to base a recommendation and of the need for more and better research on intervention effectiveness. In the absence of evidence that meets current standards for effectiveness, some organizations based recommendations for action on other factors, described briefly in this report. Until research findings become available, state and local health departments, funders of public health programs, and policymakers and other decision makers can judge the usefulness of these interventions based on other criteria and approaches."

This illustrates one of the underlying conundrums in advancing public policy in the area of mouthguard use. While there are many articles which demonstrate the effectiveness of mouthguards in preventing orofacial injury, either the quality of the evidence or the focus of the article does not conform to the standards necessary for government and other groups who advise decision-makers to recommend implementation of mandatory use. This appears to be a broader problem as Klugl et al. noted in their paper ([9], pp. 407–412): "less than 2% of the studies over the past 3 years examined the effectiveness of prevention programs in a real-world context. Although this study was not designed to determine why this is so, it is clear that these intervention studies are very difficult to perform. This difficulty, however, should not deter researchers from seeking the evidence to prevent injuries in real-life situations. Research in the area of regulatory change is under-represented, yet numerous studies have shown that it might represent one of the greatest opportunities to prevent injury." In an effort to address these issues, the International Association of Dental Traumatology (IADT) is working on a core outcome set ([10], pp. 1–8) for traumatic dental injuries in children and adults. On their website the IADT state that:

> Dental trauma is common and can occur throughout life. Numerous treatment options and interventions are available, depending on the specific traumatic injury sustained. Evidence-based comparisons of treatments and interventions can be challenging because of the diversity of outcomes reported in clinical studies. Outcomes need to be relevant to patients, clinicians and policy makers if the findings of research are to influence practice and future research. Furthermore, there is evidence to show that clinical researchers may favour reporting of outcomes that enhance results – this is known as outcome reporting bias. These issues could be addressed through the development and use of an agreed standardised collection of outcomes, known as a core outcome set (COS). The reporting of core outcomes in published studies allows researchers to compare outcomes from different studies and where appropriate, using meta-analysis techniques, to combine the results from studies in a particular area.

If adopted widely, this has the potential to strengthen the scientific basis for public policy regarding the use of mouthguards and other orofacial protective devices.

## 8.2 Professional Organizations/ Governing Bodies

### 8.2.1 Professional Organizations

Issues with the variability in the quality of the literature have not prevented national and international organizations that represent members of the scientific and practicing communities from putting out official policy recommendations. The American Academy of Pediatric Dentistry (AAPD) has had its "Policy on Prevention of Sports-related Orofacial Injuries" from at least 1991 ([11], p. 67). In the most recent update, they state that "Popular sports such as baseball, basketball, soccer, softball, wrestling, volleyball, and gymnastics lag far behind in injury protection for girls and boys." They go on to recommend "Mandating the use of properly-fitted mouthguards in other organized sporting activities that carry risk of oro-facial injury."

The House of Delegates of the American Dental Association (ADA) [12] voted in 1994 for the following resolution which is still listed in the current policies of the ADA:

> Resolved, that the American Dental Association recognizes the preventive value of orofacial protectors and endorses the use of orofacial protectors by all participants in recreational and sports activities with a significant risk of injury at all levels of competition including practice sessions, physical education and intramural programs, and be it further
>
> Resolved, that constituent and component dental societies be urged to adopt formal policies and programs aimed at encouraging the widespread use of properly fitting orofacial protectors (such as mouthguards, face shields and helmets) by athletes in their communities, and be it further
>
> Resolved, that the ADA work actively with international and national sports conferences, sanctioning bodies, school federations and others to mandate the use of orofacial protectors, and be it further
>
> Resolved, that the appropriate Association agency make the implementation of this policy a priority item.

However, on their current website [13], they do not mention the mandatory or required use of mouthguards, yet they state that "Reasons given for why mouthguards are not used include awareness, cost, and lack of requirement for their use."

The Academy for Sports Dentistry [14] in their online position statements say that "The ASD strongly supports and encourages a mandate for use of a properly fitted mouthguard in all collision and contact sports."

The American Association of Endodontists (AAE) in their position paper [15] states:

> The American Association of Endodontists recommends the use of mouth guards during participation in sports as their use may minimize the effect of impact injuries on the dentition and supporting structures. Participants in sporting events are encouraged to contact their dentist for fabrication of a custom mouth guard. In addition, organizers and coaches of children's sports are encouraged to recommend and/or require the use of mouth guards for all of their participants.



The American Academy of Pediatrics states on their website [16] that "Mouthguards can help protect your child from a dental emergency. They should be worn whenever your child is participating in sports and recreational activities."

The Canadian Dental Hygienists Association ([17], pp. 1–18) states in their position paper "The CDHA therefore strongly recommends that dental hygienists play an integral role in the prevention of orofacial injury in sports and promote properly fitted mouthguards as an essential piece of protective equipment, in sports that present a risk of orofacial injury at the recreational and competitive level, in both practices and games."

The American Public Health Association, in their 1995 position paper [18] stated:

> Noting the efficiency and effectiveness of mouthguards in preventing oro-facial injury and
>
> Recognizing that mouthguards are often required in men's but not women's contact sports and
>
> Aware that few insurance plans or state Medicaid programs offer coverage for preventive, quality fitted mouthguards, while often including treatment costs for facial and dental injuries; therefore
>
> Recommends to schools and other sponsoring organizations that all participants involved in contact sports be required to wear quality fitted protective mouthguards;
>
> Urges that the health insurance industry cover the cost of oro-facial injury prevention quality fitted mouthguards in health plans, as well as specifically in dental insurance managed care organizations, e.g., school insurance plans;
>
> Recommends that health insurance companies and managed care organizations promote quality fitted mouthguards as established devices for preventing oro-facial injuries;
>
> Recommends that the Health Care Financing Administration, Congress, and the states take steps to assure that Medicaid recipients and the uninsured population have quality fitted mouthguards when indicated for contact sports;
>
> Urges that studies of cost-effectiveness of mouthguards be conducted; and
>
> Recommends that schools and other sponsoring organizations teach the value of quality fitted mouthguards to all athletes in health education/health promotion or physical education classes.

Other organizations have mouthguard-related information on their websites. For example, the American Association of Orthodontists has information related to "National Facial Protection Month" and features information for consumers and professionals about mouthguard use. The National Facial Protection Month is also sponsored by the American Association of Oral and Maxillofacial Surgeons, the American Academy of Pediatric Dentistry, the Academy for Sports Dentistry, and the American Dental Association. Those organizations have similar materials on their respective websites.

### 8.2.2 Sports Governing Bodies

There is a great deal of sporting activity that goes on in the United States and in the world, which is not regulated by a larger body. In addition to team sports that are played in less formal settings (flag football, pickup basketball games etc.), recreational activities such as skateboarding, bicycling, and inline skating also have a significant risk of orofacial injury. In the United States, the main regulatory bodies for sporting activities are at the high school, college, amateur, and professional levels. Most sports have at least one governing body which typically is sanctioned by the United States Olympic Committee. For sports that are played at the high school level, the National Federation of State High School Associations (NFHS) is the governing body for member schools. Collegiate sports are governed primarily by the National Collegiate Athletic Association (NCAA). Professional sports are generally governed by their professional league. Other sports, for example, sports played at the X games [19] are governed by the sponsor of the event, in this case ESPN.

Tuna and Ozel ([20], pp. 777–783) noted that "According to the American Dental Association and the International Academy of Sports Dentistry, mouthguards should be used in the following 29 sports or exercise activities: acrobatics, basketball, bicycling, boxing, equestrian events, extreme sports, field events, field hockey, football, gymnastics, handball, ice hockey, inline skating, lacrosse, martial arts, racquetball, rugby, shot putting, skateboarding, skiing, skydiving, soccer, softball, squash, surfing, volleyball, water polo, weight lifting, and wrestling." Table 8.1 shows selected sports and their governing bodies.

**Table 8.1** Selected sports and their governing bodies

| Sport | US governing bodies |
|---|---|
| Acrobatics | National Collegiate Acrobatics and Tumbling Association |
| | USA Cheer |
| Basketball | USA Basketball |
| | National Basketball Association |
| Bicycling | USA Cycling |
| Boxing | USA Boxing |
| | World Boxing Association |
| | Association of Boxing Commissions |
| | World Boxing Federation |
| Equestrian events | United States Equestrian Federation |
| Field events | USA Track & Field |
| Field hockey | USA Field Hockey |
| Football | USA Football |
| | National Football League |
| Gymnastics | USA Gymnastics |
| Handball | United States Handball Association |
| Ice hockey | USA Hockey |
| | National Hockey League |
| Inline skating | USA Roller Sports |
| Lacrosse | US Lacrosse |
| | Major League Lacrosse |
| Martial arts | USA Karate |
| | USA Taekwondo |
| | Association of Boxing Commissions (Mixed Martial Arts) |
| Racquetball | USA Racquetball |
| Rugby | USA Rugby |
| Shot putting | USA Track & Field |
| Skateboarding | USA Roller Sports (some dispute) |
| Skiing | US Ski and Snowboard Association |
| Skydiving | US Parachute Association |
| Soccer | US Soccer Federation |
| | Major League Soccer |
| Softball | Amateur Softball Association/USA Softball |
| Squash | US Squash |
| Surfing | Surfing America |
| Volleyball | USA Volleyball |
| Water polo | USA Water Polo |
| Weight lifting | USA Weightlifting |
| Wrestling | USA Wrestling |
| High school athletics | National Federation of State High School Associations |
| College athletics | National Collegiate Athletic Association |

#### 8.2.2.1 High School Athletics

The National Federation of State High School Associations (NFHS) writes the rules of competition in 16 high school sports for girls' and boys' competition [21]. Their official position statement [22] states that the "NFHS currently mandates the use of mouthguards in football, field hockey, ice hockey, lacrosse and wrestling (for wrestlers wearing braces). The Sports Medicine Advisory Committee (SMAC) of the NFHS recommends that athletes consider the use of a properly fitted, unaltered mouthguard for participation in any sport that has the potential for oral-facial injury from body or playing apparatus (stick, bat, ball, etc) contact." The NFHS also adds detail to the requirements: "All goalkeepers shall wear tooth protectors, which may be attached to the face-mask/helmet. A tooth protector shall be of any readily visible color, other than white or clear. A tooth and mouth protector (intraoral) which shall include an occlusal (protecting and separating the biting surfaces) and a labial (protecting the teeth and supporting structures) portion and covers the posterior teeth with adequate thickness. It is recommended the protector be properly fitted: (1) Constructed from a model made from an impression of the individual's teeth or (2) Constructed and fitted to the individual by impressing the teeth into the tooth and mouth protector itself." For ice hockey, their rules "Requires that all players, including goal-keepers, wear an internal/external mouth protector which should cover all the remaining teeth or one jaw." For wrestling "each contestant who has braces or has a special orthodontic device on their teeth, shall now be required to wear a tooth and mouth protector that covers the teeth and all areas of the braces or special orthodontic device with adequate thickness."

Each state high school athletic association belongs to the NFHS. However, not all schools in each state belong to the state association. In some states, some private schools might not belong to the state association. This is important because while the NFHS offers guidance and recommendations on the use of orofacial protective equipment, it only governs member schools.

A recent article ([23], pp. 35–40) reviewed mandatory mouthguard rules for high school athletes in the United States. The author noted three state high school athletic associations that mandated mouthguard use in at least one sport beyond what the NFHS requires. The New Hampshire Interscholastic Athletic Association [24] states that mouthguards are required in:

- Soccer
- Field hockey
- Football
- Basketball
- Ice hockey
- Lacrosse
- Wrestlers with braces

The Maine Principals' Association [25] states in their soccer bulletin that:

> All players shall wear a mouth guard that is made of a readily visible color (may be multicolored but not clear or white) that must cover all upper molars, as well as upper front teeth. Shock mouth guards are acceptable. Mouth guards must be worn in all practices and competitions (including during pregame warm-ups) and are required of all participants at all levels (e.g. freshman, junior varsity, and varsity for boys and girls).
> Play should not be stopped immediately for infringement of this rule.

1. Coaches assume the responsibility to make certain that each player is wearing a legal mouth guard.

8

The Massachusetts Interscholastic Athletic Association [26] in their handbook states that for lacrosse "All participants must wear mouth guards." They also state that "Mouth guards are recommended for all baseball players while on the field," "Mouth guards are highly recommended for all basketball players while on the court," and "Mouth guards are highly recommended for all soccer players while on the field."

Additionally, in 2013, the Connecticut Interscholastic Athletic Association's (CIAA) board endorsed the requirement for all boys' and girls' high school soccer goalies to wear a mouthguard. The CIAA [27] also requires mouthguards in middle school for baseball, ice and field hockey, football, lacrosse, soccer, and softball.

Also, the New York State Public High School Athletic Association [28] states "a mouthpiece shall be worn by the soccer goalie for protective purposes."

Although the NFHS requires mouthguards for both ice and field hockey, the Rhode Island Interscholastic League [29] gives an additional rationale for its use:

"In order to avoid any liability for dental injuries as well as to ensure no unfair competitive advantage to teams not observing the rule, 'All players, including goalkeepers, shall wear and have properly inserted into their mouth during the course of play a properly fitted tooth and mouth protector.'" It is interesting that they felt it necessary to reinforce an already existing rule. This speaks to the public perception realm of mouthguard use that will be discussed later in the chapter.

In their 2014–2015 Athletic Trainers Packet, the West Virginia Secondary Schools Activities Commission [30] states: "Mouth Guard Policy for Basketball, Soccer, and Wrestling – The use of mouth guards is recommended for all practices and matches. Please note the mandatory requirement has been replaced by a recommendation."

While the NFHS mandates the use of a mouthguard for wrestlers with braces, the Wisconsin Interscholastic Athletic Association [31] states in their October 2013 newsletter that "A wax substance that covers the braces of a wrestler may meet the requirements of a suitable mouth guard. It will have to be inspected and approved by of the referee. This was an interpretation made last season."

#### 8.2.2.2 College Athletics

The NCAA [32] currently requires mouthguards in ice hockey, field hockey, football, and lacrosse. For basketball (men and women), the rulebook states "mouth guard protectors are appropriate equipment when they meet the qualifications outlined in this rule." The wrestling rulebook states that "It is recommended that all wrestlers wear a protective mouth guard." There are no other recommendations in other published NCAA rulebooks regarding mouthguards.

#### 8.2.2.3 Amateur Athletics

The US Olympic Committee [33], through its component organizations, currently requires mouthguard use in multiple sports:

1. *Boxing*: "Gumshields (mouth piece) must be worn by Boxers during all Bouts." "Boxers who wish to compete with braces are required to have attached to their passbooks a completed Release To Compete With Braces form (Appendix). This form requires the written approval of their dentist, parents, and/or guardian (if under 18 years of age) and a dentist-molded mouthpiece. This includes upper and/or lower braces. Boxers competing with braces waive the right to dental coverage under the USA Boxing insurance program."
2. *Field hockey*: "Mouth guards and shin guards are required in all divisions for players and goalkeepers fewer than 19 years of age." "Field players: – are recommended to wear shin, ankle, and mouth protection."
3. *Football*: "Every player is required to wear a tooth and mouth protector (intraoral) that shall include an occlusal (protecting and separating the biting surfaces) and a labial (protecting the teeth and supporting structures) portion and covers the posterior teeth with adequate thickness. It is recommended the protector be properly fitted and constructed from a model made from an impression of the individual's teeth and constructed and fitted to the individual by impressing the teeth into the tooth and mouth protector itself. The tooth and mouth protector shall be of any readily visible color, other than completely white or completely clear. The tooth and mouth protector need not be attached to the helmet or face mask. A tooth and mouth protector with a breakaway tether is legal."
4. *Ice hockey*: "All players, including goalkeepers, in the 12 & under (Youth and Girls') through Youth 18 & under (including High School) and Girls' 19 & under age classifications are required to wear a colored (non-clear) internal

mouthpiece that covers all the remaining teeth of one jaw, customarily the upper. It is strongly recommended, in all classifications, that all players wear a mouthpiece form fitted by a dentist." "USA Hockey strongly recommends that all players and goalkeepers in all age classifications properly wear an internal mouthpiece."

5. *Roller derby*: "Players must additionally wear mouth guards." "Mouth guard may not be removed except when a player is at the Team Bench or in the penalty box."
6. *Inline hockey*: "Mandatory protective equipment for players under the age of 18 (and 18 year olds playing in the 18-and-under age division) includes: HECC approved helmet with helmet- and chin-straps properly fastened, HECC approved full facemask with a chin cup, colored (non-clear) internal mouth guard."
7. *Women's lacrosse*: "All players must properly wear a professionally manufactured intra-oral mouthpiece that fully covers the teeth. The mouthpiece shall be of any readily visible color other than clear or white. It shall include occlusal (protecting and separating the biting surfaces) and labial (protecting the teeth and supporting structures) portions and shall cover the posterior teeth with adequate thickness. There may be no protruding tabs for field players.
   It is recommended that the mouthpiece be:
   1. Properly fitted, protecting the anterior (leading) dental arch
   2. Constructed from a model made from an impression of the individual's teeth
   3. Constructed and fitted for the individual by impressing the teeth into the mouthpiece
   4. Provided by a dental professional"

8. *Men's lacrosse*: "Equipment is to be in accordance with NCAA Rules, except as noted. Mouth Guards, Arm Pads, Gloves, and NOCSAE Helmets are required."
9. *Taekwondo*: "A contestant shall wear a USAT-approved uniform, trunk protector, groin guard, forearm guards, shin guards, hand protector, sensing socks (in the case of using PSS) and be equipped with a mouth piece before entering the Field of Play."
10. *Rugby*: "Mouth Guards should be worn by every player, every time they play contact rugby. There are several mouth guards on the market that can work for rugby; comfort and protection should be key in choosing a mouth guard."

#### 8.2.2.4 Professional Athletics

The National Football League does not require mouthguards, nor are they mentioned at all in their rulebook [34].

The Major League Baseball does not require mouthguards, and their use is not referenced in the official rulebook [35].

The National Hockey League also does not reference mouthguards in their rulebook [36]; however they do state that "A mask or protector of a design approved by the League may be worn by a player who has sustained a facial injury. In the first instance, the injured player shall be entitled to wear any protective device prescribed by the Club doctor."

The National Basketball Association rulebook [37] makes no mention of mouthguards but does state "The officials shall not permit any player to wear equipment which, in their judgment, is dangerous to other players. Any equipment which is of hard substance (casts, splints, guards and braces) must be padded or foam covered and have no exposed sharp or cutting edge. All the face masks and eye or nose protectors must conform to the contour of the face and have no sharp or protruding edges. Approval is on a game-to-game basis."

Major League Lacrosse does not publish a readily available rulebook, but the National Lacrosse League states the following in its rulebook [38] "All players will be required to conform to league Uniform Standards. The following items shall be inspected when the League or referees conduct a random equipment check:

1. Helmet, Facemask, Chinstrap.
2. Gloves
3. Shoulder/Vest and Rib Pads
4. Goalie Pants/Pads
5. Goalie Shin Pads
6. Any other equipment worn by the player

The following other pieces are recommended to be worn: intra oral mouth piece, arm pads, elbow pads, knee pads and athletic support."

The Major League Soccer follows the rules of the Fédération Internationale de Football Association [39] which does not reference the use of mouthguards at all.

The World Boxing Association [40] mandates the use of a mouthguard: "All boxers are required to wear a mouthpiece during competition. A second mouthpiece must be available from a boxer's second. If a mouthpiece is dislodged during competition, the referee will call time and replace the mouthpiece at the first opportune moment, without interfering with the immediate action. Points may be deducted by the referee, if he feels the mouthpiece is being purposely spit out." The World Boxing Federation has a similar mouthguard mandate [41]. The Association of Boxing Commissions also requires a mouthguard [42].

#### 8.2.2.5 Other Organizations

In 2004 the Military Training Task Force of the Defense Safety Oversight Council chartered a Joint Services Physical Training Injury Prevention Working Group to take an evidence-based approach and develop recommendations for the reduction of injury in the United States military ([43], pp. S156–S181). They recommended that "all Services provide mouthguards for all Service members participating in activities with a high risk for orofacial injuries. The working group found good evidence that mouthguards reduce orofacial injuries when worn during activities with high orofacial injury risk. Examples of potential high-risk activities listed by the working group include combatives, obstacle and confidence courses, rifle/bayonet training, etc., and contact sports such as basketball, football, etc."

As a result, the United States Army has mandated the use of mouthguards as per their regulations [44]: "Unit commanders will require and enforce mouth guard use during the following training: pugil stick, bayonet and/or rifle, obstacle and/or confidence course, and hand-to-hand combat. Commanders will require mouth guard use during physical training or unit sports activities that may involve injury to the face or mouth as a result of head-to-head contact, falls, tooth clenching, or blows to the mouth." Other United States Armed Forces branches have yet to implement similar requirements.

## 8.3 Legal Considerations



The use of protective equipment is important in reducing risk of injury and reducing severity of injury during physical activities. Its use, however, also is affected by local, state, and federal rules, regulations, statutes, and laws. It is not simply whether or not there is a requirement for use of protective equipment. Tort law, case law, civil codes, and community standards are all important in determining who is at risk. For example, if a student athlete gets injured during a football practice because he was not wearing his mouthguard, who is liable for that injury?

This is a very complicated question with many moving parts. In order to understand the issue, we should define some terms. Spillane ([45], pp. 21–24, 65) defines the following (author added italics):

> *Liability* is being at fault for causing injury or loss either by means of an intentional act or because the wrongdoer was negligent. These "wrongs," which one person inflicts on another, are referred to in the law as "*torts*."
> *Damages* include, but are not limited to, current and future medical expenses, loss of income, loss of the function of a body part, disfigurement, pain and suffering, and the possible psychological effects resulting from a negligent or wrongful act.
> *Negligence* is failure to provide for a "duty of care" owed to someone, with resulting, causally related damages.

In order to bring a tort case, there must be both damages and liability. Damages are usually pretty easy to establish, although the extent of the damages is typically in dispute. Liability (and negligence) is harder to establish and depends on the specifics of the case. Let us look at an example to illustrate the legal considerations that came into play in an actual case, Sonetti V. Huntington Beach Union School District [46].

In 2011, Mark Sonetti was a senior on the varsity football team. He was practicing kickoff drills and was hit from behind, collided with another player, and fractured his mandible. He was not wearing a mouthguard at the time. The kickoff drills did not involve tackling but did involve physical contact (blocking). Although helmets and mouthguards are required in actual games, it is not clear what the requirement is in practice. There is a concept in tort law known as assumption of the risk. "When assumption of risk applies, the defendant will not be liable for injuries sustained if the injury was the result of the 'inherent risks' of the activity, but instead must only avoid intentional injury or conduct that is so reckless as to fall outside of the ordinary risks of the sport ([47], pp. 41–43)." Therefore, by agreeing to take part in an activity, the participant is accepting the normal risks of injury associated with the sport. However, if there are ways to mitigate that injury, which are either mandated or standard practice, others may be held liable if they do not provide the proper equipment or training, or do not verify that a person has the proper training or equipment. The school had asked the court, and had initially been granted, a dismissal of the case based on the assumption of the risk. The case then went to appeals court. The appeals court reversed the decision of the trials court and said that they had erred in dismissing the case.

During the pretrial depositions, the student testified that spring kickoff drills typically did not involve the use of helmets, but the fall drills did, mostly to get the students used to playing with them. The coach agreed that this was the case. The student and the coach both agreed that the use of mouthguards was a standard practice for all kickoff drills. The appeals court concluded that because the use of mouthguards was considered a standard practice, that if the school had not handed out mouthguards, they were potentially liable for the student's injuries. The appeals court [46] goes on to say "A trier of fact could reasonably conclude that helmets and/or mouthguards were standard practice and that had the district passed out helmets or mouthguards, Sonetti's injuries would have been prevented or would have been less severe."

There are a variety of interesting and important points that this case illustrates. Although there is mandated use of mouthguards during football games, there is a fuzzy line as to what is required during practices. In the absence of a clear mandate, the court was focused on what the standard practice was. If the standard practice was not clear, the court would have likely dismissed the case.

Another area of potential liability is negligence on the part of the dentist. When patients are participating in activities where a mouthguard may reduce the risk of injury, dentists could be assuming liability in the following areas ([48], pp. 189–207, viii):

- Informed consent
- Instructions regarding mouthguard use
- Type of mouthguard recommended
- Illness related to mouthguard use
- Mouthguard defects

While the risk in each of these areas is small, it is not insignificant. Dentists who fail to discuss the use of a mouthguard with patients/guardian they know (or suspect) are participating in a sport where a mouthguard could reduce the risk of injury could be liable if the patient sustains an injury that might have been prevented (or severity reduced) if they were wearing a mouthguard. Any discussion regarding mouthguard use should be documented in the patient's record.

Even if the dentist does discuss mouthguard use with their patient/guardian, if the patient sustains an injury because they used the mouthguard improperly, the dentist may also be at risk. Therefore, it is probably best to use a standard instruction sheet based on information from a reputable organization (ADA, AAPD, ASD, etc.). This sheet should be included in the patient's record.

There are multiple types of mouthguards, and not all of them may equally protect against injury. It is the dentist's responsibility to be aware of what the appropriate mouthguard is to reduce the risk of injury. For example, if a dentist recommends the use of a mouthguard without specifying which type to use or reviewing the risks and benefits of each, he/she may be liable if the patient gets injured while using a mouthguard. Again, standardization and having a preprinted information sheet which reviews the types and uses of different mouthguards could be helpful.

Mouthguards that are not properly sanitized between uses can be a source of microbial infection. In their study looking at microbiota in protective athletic mouthguards (PAM), Glass et al. ([49], pp. 244–248) stated that "Previous studies of this subject pool evaluated the clinical status of the players' oral cavities and found an increase in both the oral lesions and the severity as the season progressed. The large number of bacteria and yeasts found in the present study support a relationship between the microbial-contaminated PAM and the oral lesions." Dentists can also be at risk regarding any infections or other pathogenic sequelae related to microbial contamination of a mouthguard. Documenting discussion of proper sanitization of mouthguards is important in reducing this risk.

Finally, if a mouthguard fails and a patient is injured, as a result, the dentist may be liable if they fabricated the mouthguard themselves or modified a mouthguard that was prefabricated or fabricated by a dental laboratory. As always, documentation of a discussion of risks associated with mouthguard use is important in reducing liability risk.

## 8.4 Economics

The mouthguard industry was estimated to be at about $81.4 million in the United States in 2014 [50], and the larger protective sports equipment industry is projected to grow to about $2.1 billion in 2018 [51]. Cost for mouthguards can range up to $1500, although the boxer Floyd Mayweather reportedly paid $25,000 for his mouthguard [52]. While it is difficult to exactly determine the impact of economic issues on mouthguard use, they undoubtedly influence the process. The most obvious impact is based on the cost of mouthguards. In a 2004 survey ([53], pp. 159–162), the authors note "fitting athletes with custom mouthguards requires a visit to the dentist and can be relatively expensive (80–100 US$). Although cost was not a major barrier in our study, it may be a barrier in a different population or if parents were asked about the cost of mouthguards over their child's playing career."

Additionally, with so much money at stake, the mouthguard industry is very competitive. There is a potential financial benefit to dentists and mouthguard manufacturers with increased use of mouthguards. While the literature is very clear about the benefits of mouthguard use, the public at large may be skeptical about these claims because of the perceived economic benefits to dentists and the industry at large.

## 8.5 Politics/Public Perception/Media

Mandates are one way to promote mouthguard use; however, other methods might eventually prove more successful in encouraging good preventive behavior. While the NBA does not mandate the use of mouthguards, the images of popular players using mouthguards have become ubiquitous. Recently, Stephen Curry, point guard for the Golden State Warrior, discussed his mouthguard use on late-night television [54]. Even his avatar in the video game NBA2K16 is shown using a mouthguard. The *New York Times* [55] recently stated that "during the past decade, the mouth guards have undergone sleek design improvements, and research during that time has increasingly shown that the benefits of wearing one may go beyond simply shielding one's teeth." They go on to say that "The league regulates mouth guards largely the same way it does other supplementary equipment, like compression sleeves or rubber wristbands."

In April of 2014, the American Association of Orthodontists (AAO) partnered with Patrick Corbin, pitcher for the Arizona Diamondbacks, to promote the National Facial Protection Month.

In 2011, Michael Vick, then quarterback for the Philadelphia Eagles, sustained a concussion and lacerated lip during a game. Alex Marvez of FoxSports.com wrote "A FOXSports.com video analysis of the game telecast shows Vick wasn't wearing a mouth guard while playing against the Falcons, including during the accidental collision with Eagles right tackle Todd Herremans that knocked him from the game. The impact left Vick with a concussion and lacerated tongue—both of which may not have occurred had he been using that piece of protective equipment. The Eagles declined comment through a team spokesman and Vick wasn't available Tuesday to comment on whether he was wearing a mouth guard." This story garnered much interest, and most comments were related to the poor judgment that Michael Vick displayed.

Unfortunately, sometimes athletes will send the opposite message to the public. As the *New York Times* noted in 2014 [56] "in this year's N.C.A.A. tournament, while players layer up with kneepads and ankle braces and micromanage their diets and their sleep, the vast majority still leave their teeth unprotected." The same article references a 2007 *Journal of the American Dental Association* paper ([57], pp. 1121–1126) which looked at dental injuries reported to the University of Southern California athletic department and concluded that "Given the relatively high incidence of dental injury in basketball and the potential of mouthguard use to reduce the incidence and severity of the trauma, mandatory use of mouthguards among collegiate basketball players should be considered." While the *NY Times* article seemed to be promoting the use of mouthguards, millions of people were watching the stars of the NCAA tournament playing basketball without using proper orofacial protection. It is no surprise that a 2008 Sports Medicine paper ([58], pp. 795–805) noted that "building on experiences from other health policy areas, influence from the media coverage of certain sports and sportspersons can neutralize scientific knowledge in decisions regarding safety policies."

As previously discussed, these safety policies have many authors and a confusing process of implementation and enforcement. There are multiple governing bodies, depending on the activity and the level at which it is being played. Additionally, the US government, on the local, state, and national levels, has generally taken a hands-off approach to regulating sporting activities. There have been some exceptions which have highlighted the tension between sports governing bodies and the legislature. One example is what happened in Minnesota in 1992 [59]. The Minnesota State High School League (MSHSL) decided that, in addition to the NFHS mandated mouthguard use, they would add mandates in soccer, volleyball, softball, wrestling, basketball, and baseball. This touched off a fierce debate which ultimately ended with the state legislature getting involved. The legislature told the MSHSL that the mandates seemed to have the "force of law" and that only the state legislature could enact new laws. Rather than risk their ability to be the governing body for high school sports in Minnesota, the MSHSL backed down and rescinded the mandates. "That winter, the state legislature told us to get rid of the rule," recalls Skip Peltier, Associate Director of the MSHSL. "And there was no way politically we could hang onto it, because if we didn't get rid of it, they would. But I still believe it was the right decision to put it in place" [60].

## 8.6 Mouthguards and the International Community

While each government has its own way of governing sports in their country, the most popular sports have international organizations that publish standard rules of play. Some of these organizations have rules that have been adopted in the United States as described above. Most countries follow the rules of these international organizations. Very few of these organizations require mouthguard use. The Federation Of International Lacrosse rules [61] require mouthguard use for both men and women. For women "All players, including the goalkeeper, must properly wear a professionally manufactured intra-oral mouth guard that fully covers the upper jaw teeth." For men the rules note additionally that "The mouth guard will be mandatory personal equipment for FIL events starting with the 2016 FIL under 19 world championship."

The International Ice Hockey Federation rules [62] state that "All skaters in the age category Under-20 must wear a mouth guard." As previously mentioned, both the World Boxing Association and World Boxing Federation require mouthguard use.

Some countries have added additional requirements above and beyond what the international organizations have required. For example, the international rugby group, World Rugby, does not require mouthguard use. However, "In New Zealand a 'domestic safety law variation' was introduced over the 1997–1998 seasons to require all players to wear mouthguards during matches. In 1997, mouthguard use became mandatory for all players at under 19 level and below, and in 1998 this was extended to players of all grades (levels of play)" ([63], pp. 650–651).

## 8.7 Conclusion

Although mouthguard use has extensive support in the literature for the reduction of risk of orofacial history, its use in sports is limited by social, political, economic, and legal factors as described in this chapter.

It is clear that the way to reduce orofacial injuries is not simply to mandate mouthguard use for all sports. Efforts to do this have been met with significant resistance and created an antagonistic environment between those who support the mandate and those who don't. While mandates may be appropriate for some sports, especially those where there is currently a consensus among most stakeholders of the necessity of mouthguard use, education and the use of marketing and social media will likely be more effective in achieving the desired outcome. Therefore, I would make the following recommendations:

1. Work with as many groups as possible (parent groups, coaches, student groups, regulatory groups, etc.) to educate them on the benefits of mouthguard use.
2. Target popular sport figures to promote the use of mouthguards. Having spokespeople is a start, but these players must "walk the talk" as well. That is, they should be seen using mouthguards, while they play and during media opportunities, photo ops, etc.
3. Work with video game developers to incorporate the use of mouthguards into the characters in their games. If all of the players in a popular basketball video game are using mouthguards, that will send a strong message to the kids (and adults) that play the game.
4. Work locally to increase awareness of the benefits of mouthguards. Top-down efforts, either at the state or national level, will be less effective.
5. Work with social media outlets to promote mouthguard use. See if mouthguard education can be "embedded" in related stories, rather than a story itself.
6. Control the narrative. A story in the news (TV, social media, newspaper, etc.) about an injury that could have been avoided with the use of a mouthguard is more powerful than a research article. These stories should be repeated until consumers demand a change.

By focusing on creating a culture among parents, students, coaches, and the general public that mouthguard use is part of participating in sports, the use of orofacial protective devices will increase and hopefully become the norm rather than the exception.

8

## Appendix

**USA Boxing, Inc.**

____________________________________

**NAME OF EVENT**

**Release to Compete With Braces**

USA Boxing Technical Rule 5.1.3.1. Boxers who wish to compete with braces are required to have attached to their passbooks a completed Release To Compete With Braces form. This form requires the written approval of their dentist, parents and/or guardian (if under 18 years of age) and a dentist-molded mouthpiece. This includes upper and/or lower braces. Boxers competing with braces waive the right to dental coverage under the USA Boxing insurance program.

I understand the above rule and give my permission for ______________________________ (Boxer's name)
to compete in amateur boxing.

Dentist Approval:

| | |
|---|---|
| ________________ Print Name | ________________ Signature |
| ________________ Date | ________________ State License Number |

Parent or Legal Guardian Approval (if boxer is a minor):

| | | |
|---|---|---|
| ________________ Print Name | ________________ Signature | ________ Date |

Athlete:

| | | |
|---|---|---|
| ________________ Print Name | ________________ Signature | ________ Date |

## References


1. Institute of Medicine (US). Committee for the study of the future of public health. In: The future of public health, vol. 88. Washington, DC: National Academy Press; 1988.
2. Porter D. Health, civilization, and the state: a history of public health from ancient to modern times. London: Routledge; 1999.
3. Tulchinsky TH, Varavikova EA. The new public health: an introduction for the 21st century. San Diego: Academic Press; 2014.
4. Public Policy. December 9, 2015. http://www.merriam-webster.com/dictionary/public%20policy.
5. Sridhar D. Health policy: from the clinical to the economic gaze. Lancet. 2011;378(9807):1909.
6. Kumamoto DP, Maeda Y. A literature review of sports-related orofacial trauma. Gen Dent. 2004;52(3):270–80; quiz 281.
7. Knapik JJ, et al. Mouthguards in sport activities history, physical properties and injury prevention effectiveness. Sports Med. 2007;37(2):117–44.

8. Gooch BF, et al. A comparison of selected evidence reviews and recommendations on interventions to prevent dental caries, oral and pharyngeal cancers, and sports-related craniofacial injuries. Am J Prev Med. 2002;23(1):55–80.
9. Klugl M, et al. The prevention of sport injury: an analysis of 12,000 published manuscripts. Clin J Sport Med. 2010;20(6):407–12.
10. Williamson PR, et al. Developing core outcome sets for clinical trials: issues to consider. Trials. 2012;13:132.
11. Anonymous. Policy on prevention of sports-related orofacial injuries. Pediatr Dent. 2013;35(6):67. Print.
12. American Dental Association. Current policies. 2014. https://www.ada.org/~/media/ADA/Member%20Center/Members/current_policies.ashx.
13. American Dental Association. Oral health topics: mouthguards. 2015. http://www.ada.org/en/member-center/oral-health-topics/mouthguards.
14. Academy for Sports Dentistry. Position statement. 2015. http://www.academyforsportsdentistry.org/position-statement.
15. American Association of Endodontists. Position statement: use of mouthguard to prevent injury. 2015. https://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/useofmouthguardstopreventinjury.pdf.
16. American Academy of Pediatrics. Mouthguards: information for parents. 2015. https://www.healthychildren.org/English/healthy-living/oral-health/Pages/Mouthguards.aspx.
17. Canadian Dental Hygienists Association. CDHA position paper on sports mouthguards, putting more bite into injury prevention. Can J Dent Hyg. 2005;39(6):1–18.
18. American Public Health Association. Promotion of quality-fitted mouthguards for oro-facial injury prevention. Policy number 9506 ed. Washington, DC: APHA, 1995. Print.
19. Anonymous. ESPN X-games. 2016. http://xgames.espn.go.com/xgames/.
20. Tuna EB, Ozel E. Factors affecting sports-related orofacial injuries and the importance of mouthguards. Sports Med. 2014;44(6):777–83.
21. Anonymous. National Federation of State High School Associations, about us. 2016. https://www.nfhs.org/who-we-are/aboutus.
22. National Federation of State High School Associations. Position statement and recommendations for mouthguard use in sports. 2014. http://www.nfhs.org/sports-resource-content/position-statement-and-recommendations-for-mouthguard-use-in-sports/.
23. Mills SC. Mandatory mouthguard rules for high school athletes in the United States. Gen Dent. 2015;63(6):35–40.
24. New Hampshire Interscholastic Athletic Association Handbook. By-Law Article III - sports medicine. 2016. http://nhiaa.org/PDFs/5510/5ByLawArticleIIISportsMedicine.pdf.
25. Maine Principals Association. 2015 MPA Soccer Bulletin. 2015. http://www.mpa.cc/images/bulletins/bulletin_soccer.pdf.
26. Massachusetts Interscholastic Athletic Association. Handbook. 2015. http://www.miaa.net/gen/miaa_generated_bin/documents/basic_module/MIAAHandbook1517.pdf.
27. Connecticut Interscholastic Athletic Association. Guidelines for connecticut middle athletic program. 2016. http://www.casciac.org/pdfs/MiddleLevelAthleticGuidelinesFinalOctober25,2010.pdf.
28. New York State Public High School Athletic Association. Handbook. 2016. http://www.nysphsaa.org/Portals/0/PDF/Handbook/2015-16%20School%20Year/December%202015/Complete%202014-2015%20Handbook%2012-21-15.pdf.
29. Rhode Island Interscholastic League. Rules, Article 22. 2016. http://www.riil.org/files/2514/3861/5367/ARTICLE_22_HOCKEY_2015.pdf.
30. West Virginia Secondary Schools Activities Commission. Athletic trainers packet. 2016. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0ahUKEwjZ68PYsZvKAhUBtRQKHV4eBSsQFggdMAA&url=http%3A%2F%2Fwww.wvssac.org%2Fwp-content%2Fuploads%2F2014%2F10%2FAthletic_Trainers_Packet.pdf&usg=AFQjCNHww5_D1t_Inp1is-E1HZ7Ovy-34A.
31. Wisconsin Interscholastic Athletic Association. October 2013 Newsletter. 2013. https://www.wiaawi.org/Portals/0/PDF/News/Bulletin/bulletin2013_10-25.pdf.
32. NCAA. NCAA publications. 2016. https://www.ncaapublications.com/.
33. United States Olympic Committee. USOC. 2016. http://www.teamusa.org/.
34. National Football League. NFL rulebook. 2016. http://uaasnfl.blob.core.windows.net/live/1807/2015_nfl_rule_book_final.pdf.
35. Major League Baseball. Official rules. 2016. http://mlb.mlb.com/mlb/downloads/y2015/official_baseball_rules.pdf.
36. National Hockey League. Official rules. 2016. http://www.nhl.com/nhl/en/v3/ext/rules/2015-2016-Interactive-rulebook.pdf.
37. National Basketball Association. Official rules. 2016. https://turner-nbahangtime.files.wordpress.com/2015/11/official-nba-rule-book-2015-16.pdf.
38. Pointstreaksites.com. National Lacrosse League rules. 2016. http://pointstreaksites.com/files/uploaded_documents/2715/2015_NLL_Rulebook.pdf.
39. Fédération Internationale de Football Association. Laws of the game. 2016. http://www.fifa.com/mm/Document/FootballDevelopment/Refereeing/02/36/01/11/LawsofthegamewebEN_Neutral.pdf.
40. World Boxing Association. Rules of World Boxing Association. 2015. http://www.wbanews.com/wp-content/uploads/2015/06/WBA-Rules-adopted-in-Bulgaria-6-11-15.pdf.
41. World Boxing Federation. Rules and regulations. 2015. http://www.worldboxingfederation.net/wbfrulesandregulations.htm.
42. Association of Boxing Commissions. Uniform rules of boxing. 2015. http://abcboxing.com/ABC_Uniform_Championship_Rules.pdf.
43. Bullock SH, et al. Prevention of physical training–related injuries: recommendations for the military and other active populations based on expedited systematic reviews. Am J Prev Med. 2010;38(1):S156–81.
44. United States Army. Army Regulation 600-63. 2016. http://www.apd.army.mil/pdffiles/r600_63.pdf.
45. Spillane DM. Dentists, mouthguards, and the law. J Mass Dent Soc. 1994;43(4):21–4, 65.
46. Sonetti V. Huntington Beach Union High School Dist. No. G049198. 2014.
47. Murphy KL, Beh HG. The standard of care and the assumption of risk defense in a negligent injury case in a physical education class. J Phys Educ Recreat Dance. 2014;85(8):41–3.
48. Machen DE, Machen HA. Legal considerations in sports dentistry. Dent Clin N Am. 2000;44(1):189–207, viii.
49. Glass RT, et al. Microbiota found in protective athletic mouthguards. Sports Health. 2011;3(3):244–8.
50. Statistica.com. Consumer spending on protective mouth guards in the U.S. from 2010 to 2014 (in million U.S. dollars). 2016. http://www.statista.com/statistics/241721/consumer-purchases-of-protective-mouth-guards-in-the-us/.
51. Prweb.com. U.S. market for protective sports equipment to reach $2.1 billion in 2018: BCC research. 2013. http://www.prweb.com/releases/2013/8/prweb11009196.htm.
52. Forbes Magazine. Floyd Mayweather's $25k mouth guard made with $100 bills. 2016. http://www.forbes.com/sites/hunteratkins/2015/04/08/floyd-mayweathers-25k-mouth-guard-made-with-100-bills/.
53. Pribble JM, Maio RF, Freed GL. Parental perceptions regarding mandatory mouthguard use in competitive youth soccer. Inj Prev. 2004;10(3):159–62.
54. Jimmy Kimmel Live. Stephen Curry's mouth guard chewing habit. 2016. https://www.youtube.com/watch?v=eSYdZFwjwqw.
55. NY Times Online. In a league of flying elbows, the use of mouth guards soars. 2015. http://www.nytimes.com/2015/02/11/sports/basketball/in-a-league-of-flying-elbows-the-use-of-mouth-guards-has-soared.html?_r=0.
56. NY Times Online. N.C.A.A. players ignore advice on mouth guards, often at their own peril. 2014. http://www.nytimes.com/2014/03/30/

sports/ncaabasketball/ncaa-players-ignore-advice-on-mouth-guards-often-at-their-own-peril.html.
57. Cohenca N, Roges RA, Roges R. The incidence and severity of dental trauma in intercollegiate athletes. J Am Dent Assoc. 2007;138(8):1121–6.
58. Timpka T, et al. Meeting the global demand of sports safety. Sports Med. 2008;38(10):795–805.
59. Roettger M. Minnesota mouthguard mandate: A ten-year retrospective. Dent Traumatol. 2002;18(4). Print
60. Catalano J. Guarding the mouth. 1997. http://www.momentummedia.com/articles/cm/cm0505/gdmouth.htm.
61. Federation of International Lacrosse. Federation of International Lacrosse. 2016. http://filacrosse.com/.
62. International Ice Hockey Federation. Rulebook. 2016. http://www.iihf.com/fileadmin/user_upload/PDF/Sport/IIHF_Official_Rule_Book_2014-18_Web_V6.pdf.
63. Quarrie KL, et al. An evaluation of mouthguard requirements and dental injuries in New Zealand rugby union. Br J Sports Med. 2005;39(9):650–1.

# Concussion in Sport: Role of Dentistry

*Jennifer Oberstar*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_9

## 9.1 Definition of Concussion

According to McCrory et al., the "broadest clinical definition of sports related concussion (SRC) is immediate and transient symptoms of traumatic brain injury (TBI)." This definition is not adequate in that it does not explain processes through which the brain is impaired or allow one to ascertain severity or analyze persistent symptoms or abnormalities. Sometimes TBI is used in place of the definition of concussion, and different researchers use different definitions making comparisons among studies challenging. Therefore, the 2016 Berlin Fifth International Conference on Concussion in Sport (CIS) expert panel has modified previous language regarding the definition of concussion. They contend SRC is a "traumatic brain injury induced by biomechanical forces" [1]. Common features were also delineated in the following:

- SRC may be caused by a blow to the head, face, neck, or elsewhere with an impulsive force.
- Short-lived impairment (minutes to hours) of neurological function that resolves spontaneously is common.
- Neuropathological changes or functional disturbance instead of structural injury presents with no abnormalities when standard neuroimaging is displayed.
- Clinical signs and symptoms may or may not result in loss of consciousness.
- Symptoms may follow a sequential course or may be prolonged.



### 9.1.1 Consensus Statement

The 2001 Vienna First International Conference on CIS focused on improving safety and the health of post-concussed athletes. The outcomes of this meeting included a single concussion in sports definition and the creation of stepwise return-to-play decision-making guideline. The initial Sport Concussion Assessment Tool (SCAT) Card was published after the second meeting in Prague in 2004. The third meeting held in Zurich in 2008 addressed a systematic review of the current literature drafting the first consensus statement. The SCAT was revised leading to the SCAT2 and pocket version. The fourth CIS Conference was in Zurich in 2012 using the NIH style of consensus development, and further scientific reviews were utilized to produce a consensus statement with a revised SCAT3, pocket version, and Child SCAT3. The fifth CIS Conference took place in Berlin in 2016 following a process that would identify 12 important questions. Expert panels used systematic reviews to answer each of the identified important topics. The consensus statement was written by co-chairs on the scientific committee followed by editing, peer review, and approval by the expert panel [2]. The updated concussion statement is published in the *British Journal of Sports Medicine* along with updated versions of the SCAT5, Child SCAT5, and sideline SCAT5.

## 9.2 Neurometabolic Cascade of Concussion

Acceleration, deceleration, and rotational forces being transmitted to the brain can result in concussion. A neurometabolic cascade is initiated that induces cell membrane and axonal stretching leading to an unregulated efflux of ions through previously regulated ion channels [3]. Excitatory amino acids such as glutamate are released due to rapid depolarization. Energy transported by adenosine triphosphate (ATP) is required to reestablish ionic balance. As energy is depleted, there are also decreased cerebral blood flow and mitochondrial dysfunction [4]. After biomechanical injury, deformation of neuronal membranes leads to excessive potassium efflux into the extracellular space. In the acute phase of oxidative (mitochondrial) dysfunction, a significant influx of calcium accumulates in the mitochondria. This oxidative dysfunction decreases over 10 days after injury. Energy sources to the post-concussive brain are modified when compared to the healthy brain. Glucose metabolism is decreased; creatine and creatine phosphate are no longer available as fuel sources post-concussion, and ketone bodies appear to have some neuroprotective implications in animal studies [3]. This deregulation leads to an imbalance between energy demand and available alternative energy sources (Fig. 9.1).

### 9.2.1 Description

Functional versus structural injury**:** In SRC, neurological impairment is short-lived and resolves spontaneously. Clinical signs and symptoms largely reflect the result of direct impact resulting in acute acceleration or deceleration of the body with the brain moving within the cranium, which leads to injury. Cumulative exposures can have clinical consequence, and some studies report cognitive deterioration is directly related to exposure burden, whereas other studies have failed to reproduce this result [5].

## 9.3 Concussion Evaluation

SRC is an evolving injury in the acute phase and there are quickly changing clinical signs and symptoms. SRC is considered one of the most complex injuries in sports medicine to diagnose, assess, and manage. Complicating this, often there is no loss of consciousness or delineating neurologic signs. There is also no perfect diagnostic test. So, SRC cannot be ruled out when injury occurs. If SRC is suspected, the individual should be removed from the playing field and assessed by a physician.

Sideline evaluation of cognitive function is essential in the assessment of injury. Neuropsychological (NP) test batteries assessing attention and memory function are effective. Included in this is the SCAT5, which contains Maddocks questions (Maddocks) and standardized assessment of concussion (SAC) [6]. Orientation questions like person, place, and time are unreliable compared to memory assessment. These sideline

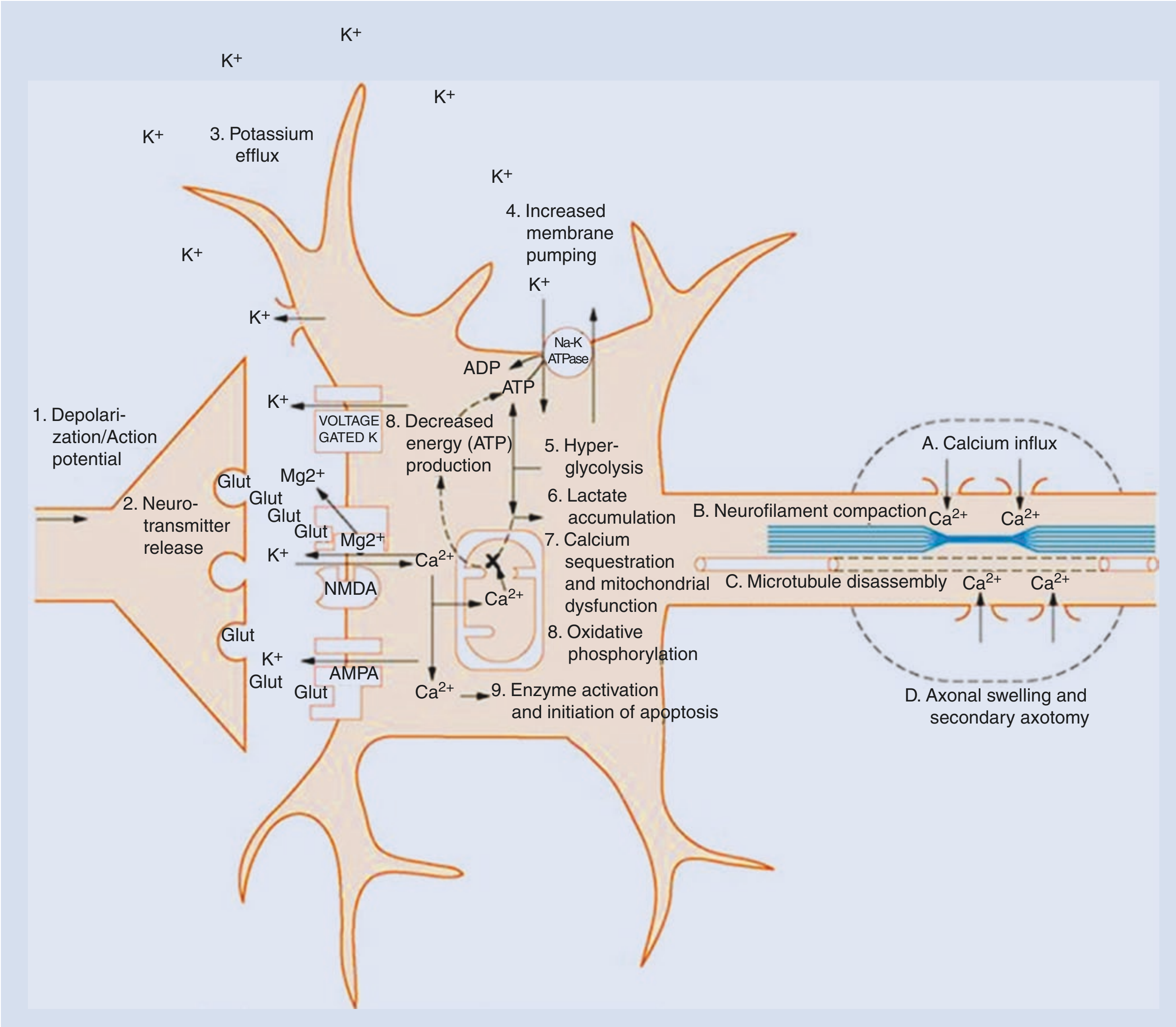



**Fig. 9.1** Neurometabolic cascade after traumatic injury. Cellular events: 1. Nonspecific depolarization and initiation of action potentials. 2. Release of excitatory neurotransmitters. 3. Massive efflux of potassium. 4. Increased activity of membrane ionic pumps to restore homeostasis. 5. Hyperglycolysis to generate more adenosine triphosphate (ATP). 6. Lactate accumulation. 7. Calcium influx and sequestration in mitochondria, leading to impaired oxidative metabolism. 8. Decreased energy (ATP) production. 9. Calpain activation and initiation of apoptosis. Axonal events: A. Axolemmal disruption and calcium influx; B. Neurofilament compaction via phosphorylation or sidearm cleavage. C. Microtubule disassembly and accumulation of axonally transported organelles; D. Axonal swelling and eventual axotomy. *ADP* adenosine diphosphate, *AMPA* alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid, *Glut* glutamate, *NMDA N*-methyl-D-aspartate. (From Giza CC, Hovda DA. The neurometabolic cascade of concussion. J Athl Train 2001;36:230)

screening tests are not meant to replace neurological evaluation or as an ongoing management tool for SRC.

The purpose of the rapid screening for SRC is not to make a definitive diagnosis of head injury. If clear on-field signs of SRC (e.g., loss of consciousness, tonic posturing, balance disturbance) are apparent, the individual should be immediately suspended from play. Sideline (SCAT5) and a more thorough diagnostic evaluation should be performed in a distraction-free environment. Sideline assessment, including information from the athlete and assessment or inspection of videotape of the incident, can be used to determine if concussion is no longer suspected. The physician determines the timing of return to play.

Contact sports have a tendency to be fast-paced, in disorganized environments, and field of play is often obscured. Therefore, these present challenges in diagnosing SRC. Because of this, suspected SRC should be approached with multidimensional testing and well-established instruments for sideline assessment. Both the SCAT5 and Child SCAT5 are suggested in the evaluation of SRC. These sideline assessments help in distinguishing if an athlete is concussed or non-concussed, but the tests' usefulness decreases significantly after 3–5 days of the injury. The symptom checklist has more utility in tracking recovery. Replicating baseline testing conditions should be similar. Additional areas that may be assessed are clinical reaction time, gait/balance assessment, video observation, and oculomotor screening. Video review appears to be a promising mode of identifying and evaluating SRC.

### 9.3.1 Signs and Symptoms: Physical, Cognitive, Emotional, Sleep [4]

The healthcare provider has the challenge of recognizing and evaluating SRC on the field. Rapid assessment is often required during competition with time constraints and with coaches and athletes wanting to return to play. It is critical that more serious injuries are excluded and proper disposition of the athlete is decided. The SCAT5 is often difficult to perform within 10 min. Sideline evaluations are based on injury recognition, assessment of symptoms, balance, and cognitive and neurologic function. Because SRC is an evolving injury, serial assessments are often required. It is important to err on the side of caution and restrict an athlete's participation if injury is suspected. After injury, a detailed concussion history is imperative. The 2016 Berlin expert panel have reported that if a SRC is suspected, one or more of the following will be present:

**Clinical symptoms** - somatic (e.g., headache), cognitive (e.g., feeling like in a fog), and/or emotional symptoms (e.g., lability)

**Physical signs** - e.g., loss of consciousness, amnesia, neurological deficit

**Balance impairment** - e.g., gait unsteadiness

**Behavioral impairment** - e.g., irritability

**Cognitive impairment** - e.g., slowed reaction times

**Sleep/wake disturbance** - e.g., somnolence, drowsiness

These signs and symptoms may be present with non-brain-related injury. Therefore, concussion is added to the differential diagnosis for further investigation, but concussion cannot be diagnosed based on the symptoms present. The majority of athletes (80–90%) have symptom resolution after a week, but younger children under age 12 may take a month before fewer symptoms are present. Symptom resolution does not guarantee complete cognitive recovery [4].

#### 9.3.1.1 Concussion Evaluation Tools

When an athlete aged 13 and older is suspected of having sustained an SRC, the SCAT5 is a tool used by sideline physicians to assess for the presence of concussion and initiate management based on clinical findings. If the athlete falls into the ages of 5–12 years old, the Child SCAT5 will be utilized. The athlete with a possible concussion should be immediately removed from play and evaluated by medical personnel for any red flag signs.

**Red flags SCAT5**

| | |
|---|---|
| – Neck pain or tenderness | – Seizure or convulsions |
| – Double vision | – Loss of consciousness |
| – Weakness or tingling/burning in arms or legs | – Deteriorating conscious state |
| – Severe or increasing headache | – Vomiting |
| | – Increasingly restless, agitated, or combative |

Observable signs including lack of movement, disorientation, vacant stare, and orofacial injury after head trauma should be assessed. The Glasgow Coma Scale (GCS) can be performed serially to assess for changes in eye, verbal, and motor response. Maddocks questions for memory assessment and cervical spine examination are critical in the initial assessment. A cervical injury should be assumed until proven otherwise [7, 8]. Decision to transport an athlete should be made in a timely fashion. The athlete background and symptom evaluation should be performed in a distraction-free environment. The cognitive screening includes the following: orientation, immediate memory from lists of words, and concentration assessed by digits backwards and months in reverse order. Neurological screening includes the ability to read without difficulties, full pain-free active neck movements, movement of the head or neck without double vision, coordination, and tandem gait testing. Delayed recall should include words from the list read earlier. When differentiating concussed from non-concussed athletes, the graded symptom checklist, Standardized Assessment of Concussion (SAC), and Balance Error Scoring System (BESS)/modified Balance Error Scoring System (mBESS) were found to be the most useful post-injury [9]. When assessing for concussion, a single scoring system should not determine the diagnosis. Although tools such as the SCAT5 are utilized, more research is required to determine the efficacy of these tools in improving SRC identification and management.

**Components of SCAT5**

1. Red flags
2. Observable signs
3. Maddocks questions
4. Glasgow Coma Scale
   - Cervical spine evaluation
5. Athlete background and symptom evaluation
6. Cognitive screening
7. Neurological screening
8. Delayed recall
9. Decision

### 9.3.2 Neuropsychology Evaluation

Most concussion symptoms will resolve within 7–10 days. Neuropsychological (NP) testing can be useful for those athletes with prolonged symptoms and recovery. This testing can be used as part of a comprehensive investigation and should not be used alone to guide management. "Both paper and pencil and computerized testing have significant individual variability with regard to domains measured and performance measures such as validity, sensitivity, specificity, reliable change index and baseline variability" [4]. Computerized NP baseline testing can be useful in comparing post-injury results, but this can result in expenses that may not be affordable for many school systems. NP testing performed when the athlete is asymptomatic may be useful when considering return-to-school and return-to-play decisions. Although neuropsychologists have the background to administer and interpret test results, the ultimate return-to-play decision should remain with the medical team [10].

#### 9.3.2.1 Pencil and Paper

Neuropsychological testing is a tool used to assess cognitive impairment after an athlete sustains a concussion and to document an athlete's recovery. Testing is administered and interpreted by a neuropsychologist; therefore, this method is expensive and more time-consuming. The testing typically focuses on memory, cognitive processing speed, and reaction time [4].

#### 9.3.2.2 Computer-Based Testing

Computerized neuropsychological testing has not been validated as a diagnostic tool [29]. Advantages of computerized testing include less time to administer, more cost-effectiveness, and more precision reporting on reaction time.

### 9.3.3 Vestibular/Ocular-Motor System (VOMS) Evaluation

Vestibular/ocular-motor screening can assist with proper assessment and subsequent referral placement. Prolonged symptoms along with vestibular and oculomotor impairment may be associated with delayed recovery after SRC. Examples of vestibular symptoms may include the following: blurry vision, dizziness, nausea, and vertigo. Oculomotor symptoms may include blurry vision, nausea, difficulty scanning, convergence insufficiency, and difficulty reading [25, 26]. These symptoms can impact academic performance, reading, and ultimately overall performance.

#### 9.3.3.1 King-Devick Test

The King-Devick (KD) test is a vision-based test consisting of numbers that the athlete must track with her eyes and read aloud. This test captures the saccadic eye movements and periods of fixation, and results may vary with changes in cognition [24, 32]. The administrator of the test tracks the testing time and errors, while the athlete performs the test. After the athlete performs a strenuous workout, the test times are not prolonged. However, if the athlete is concussed, the time to completion is prolonged. The mechanisms by which the KD test results are prolonged are postulated to be related to saccadic slowing, pauses between saccades, inaccurate saccades, and abnormal eye movements [11]. The KD test demonstrates promise as a sideline concussion screening tool, but further studies are needed [12] (◘ Fig. 9.2).

### 9.3.4 Balance Assessment

Following SRC, acute postural instability may persist for up to 72 h [5]. Studies report that changes in balance and postural stability, when compared with preseason baselines, are sensitive for the diagnosis of concussion. The Balance Error Scoring System (BESS) is a standardized sideline assessment tool. This test can be administered in 5–7 min and is cost-effective. Three stances (double-leg stance, single-leg stance, and tandem stance) are performed on two surfaces (firm surface/floor or medium density foam). The athlete stands during each stance with hands on hips and eyes closed for 20 s. Points are deducted from a maximum of 60 when using two surfaces, if the athlete lifts her hands off the hips, stumbles, steps away, opens her eyes, or falls. BESS performance returns to preseason baseline levels by 3–7 days post-injury for most athletes. Balance testing is limited by fatigue, exercise, serial assessments, and reliability. According to McCrea, sensitivity of balance testing is best within 24 h of injury (0.34), and specificity was 0.91 and 0.96 between days 1 and 7 post-injury. Completing the BESS on the sideline can be challenging, so the modified BESS is often used at halftime or in the locker room after practice or game. The high specificity combined with other assessment tools reinforces the use in the sideline evaluation.

### 9.3.5 No RTS Same Day

There is a no return to sport on same-day policy for those diagnosed with concussion. In 2009, legislation began in Washington state with the Lystedt law. The legislation mandates any athlete younger than 18 must be removed from play and is unable to return on the day of injury. Many states have concussion laws that oppose a same-day return. Signs and symptoms may evolve over time; therefore, reassessment of the athlete's condition and erring on the side of caution are reasonable actions.

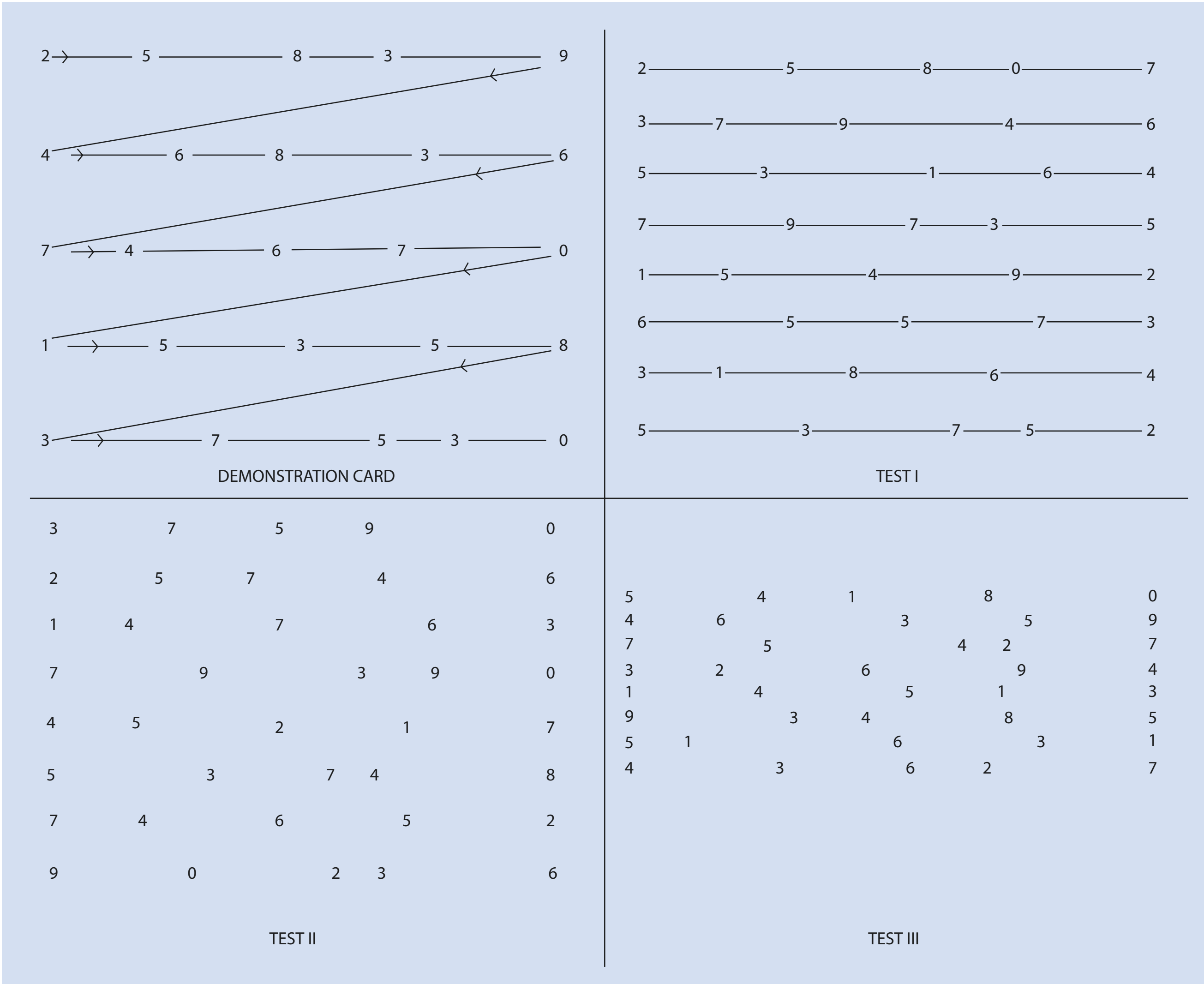



**Fig. 9.2** The King-Devick test

## 9.4 Concussion Management

If the athlete demonstrates signs and symptoms of cervical spine injury, intracranial bleeding, or skull fracture, the emergency action plan should be activated, and the athlete will be transported to an appropriate medical facility [13]. The sideline examination should include documentation of the injured athlete's symptoms and neurologic examination including cognition, cranial nerve function, and balance. Visual tracking can provide further information. Symptom scores and cognitive assessment are the most sensitive and specific within 48 h of injury. Comparing post-injury scores to the athlete's baseline is also useful. This does not replace the benefits of knowing the athlete prior to injury and using clinical judgment despite the athlete passing a sideline screening. All athletes, regardless of level of competition, should be managed using the same principles.

### 9.4.1 Management Strategies

#### 9.4.1.1 Stepwise RTS Progression (Table 9.1 [10])

After an initial rest period, if deemed necessary, activity can begin if it stays below a threshold that will exacerbate physical and cognitive symptoms. Once the athlete's concussion symptoms have resolved, the stepwise RTS progression begins. The athlete should be asymptomatic at each level. The RTS progression will take roughly a week to complete. If the athlete is symptomatic, the athlete should return to the previous asymptomatic level. The athlete is able to advance to the next level if the athlete meets all criteria without having symptoms reoccur. The time to RTS may vary due to a myriad of factors including the athlete's age, level of sport, and history. The table from the Berlin Consensus Statement demonstrates the stepwise process to

**Table 9.1** Graduated return-to-sport (RTS) strategy

| Stage | Aim | Activity | Goal of each step |
|---|---|---|---|
| 1 | Symptom-limited activity | Daily activities that do not provoke symptoms | Gradual reintroduction of work/school activities |
| 2 | Light aerobic exercise | Walking or stationary cycling at slow to medium pace. No resistance training | Increase heart rate |
| 3 | Sport-specific exercise | Running or skating drills. No head impact activities | Add movement |
| 4 | Noncontact training drills | Harder training drills, e.g., passing drills. May start progressive resistance training | Exercise, coordination, and increased thinking |
| 5 | Full-contact practice | Following medical clearance, participate in normal training activities | Restore confidence and assess functional skills by coaching staff |
| 6 | Return to sport | Normal game play | |

NOTE: An initial period of 24–48 h of both relative physical rest and cognitive rest is recommended before beginning the RTS progression
There should be at least 24 h (or longer) for each step of the progression. If any symptoms worsen during exercise, the athlete should go back to the previous step. Resistance training should be added only in the later stages (stage 3 or 4 at the earliest). If symptoms are persistent (e.g., more than 10–14 days in adults or more than 1 month in children), the athlete should be referred to a healthcare professional who is an expert in the management of concussion
Graduated return to sport. The process of returning to sport participation after a SRC follows a graduated step-wise rehabilitation process as published in the Berlin Consensus Statement

RTS after sustaining a SRC. RTS must be individualized, and further evaluation is required for those athletes, who have a prolonged recovery course.

#### 9.4.1.2 Stepwise Return-to-Learn Progression (Table 9.2 [1])

An athlete's primary responsibility is to return to the classroom after SRC. If the athlete is unable to return to the classroom for full days, the daily schedule should be modified [27]. Areas that can impact academic performance include deficits in oculomotor function, attention, vestibular function,

**Table 9.2** Graduated return-to-school strategy

| Stage | Aim | Activity | Goal of each step |
|---|---|---|---|
| 1 | Daily activities at home that do not give the child symptoms | Typical activities of the child during the day as long as they do not increase symptoms (e.g., reading, texting, screen time). Start with 5–15 min at a time and gradually build up | Gradual return to typical activities |
| 2 | School activities | Homework, reading, or other cognitive activities outside of the classroom | Increase tolerance to cognitive work |
| 3 | Return-to-school part time | Gradual introduction of schoolwork. May need to start with a partial school day or with increased breaks during the day | Increase academic activities |
| 4 | Return-to-school full time | Gradually progress school activities until a full day can be tolerated | Return to full academic activities and catch up on missed work |

and cognitive stamina [14]. Academic accommodations may include decreased computer screen time, larger font size for reading, a quiet room, increased testing time, pre-printed notes for minimal eye tracking, and breaks for fatigue. A multidisciplinary approach including the athlete, athlete's family, school, physician, and coaching staff is recommended to optimize the athlete's return to the classroom.

## 9.5 Sequelae of Concussion

Unfortunately, it is not always apparent which athletes will suffer long-term consequences from concussion. Prolonged symptoms do occur in a minority of cases. Risk factors that may make athletes more vulnerable to injury include the following: previous concussions including number, severity, and time to recovery, age, sex, style of play, genetics, mood disorders, learning disabilities and attention disorders, history of preexisting migraines, and player position [4]. The neurological effects of SRC are typically transient, but a small percentage of athletes continue to demonstrate symptoms months after the initial injury. The concussed brain appears less responsive to neural activation. It is important to understand preexisting psychological or cognitive disorders to provide optimal medical therapy post-concussion.

### 9.5.1 Second-Impact Syndrome

Although post-concussive symptoms are important to identify and monitor, another reason to delay return to play is second-impact syndrome (SIS). The original term "second-impact syndrome" was described by Saunders and Harbaugh in 1984 (Barkhoudarian et al. [30]). SIS is often described as a severe brain injury affecting adolescent and young adult males that is the result of repeated TBI in the setting of cellular ionic disturbance, decreased cerebral blood flow, and glucose metabolic dysfunction [3]. This is a hypothetical brain disturbance in which the athlete receives a second head injury prior to recovery from the first insult. The athlete often appears stunned without loss of consciousness and can independently leave the field before rapidly deteriorating. It has been suggested that diagnostic imaging reveals massive cerebral swelling. This leads to increasing intracranial pressures that result in brainstem herniation. Outcomes are extremely poor leading to a mortality rate of 50% and morbidity of 100% [15]. The current literature remains controversial debating the mere existence of this condition. According to McCrory, out of 17 cases, only 5/17 were probable cases of diagnosed SIS. Thirteen out of the seventeen cases reported sports-related catastrophic brain injury with massive cerebral swelling without association with a second impact [16]. American football is the most common sport associated with SIS. Other case reports have occurred in boxing, rugby, and karate. At this time, there is not high-quality literature available supporting definitive cases of SIS.

### 9.5.2 Chronic Encephalopathy (CE)

Over the past several years, there has been much deliberation regarding long-term effects of concussion. There are several reported case studies describing aggressive behavior, loss of control, impaired attention, depression, memory loss, and disturbance in executive function in athletes. These clinical symptoms were first described in 1928 by Harrison Martland, who noted changes in experienced boxers [3, 17]. Emerging research has identified clinical and neuropathologic evidence of CE with a variety of sports, including the following: American football, boxing, wrestling, ice hockey, baseball, and soccer. CE has also been associated with other non-sporting-related activities such as military service, epileptic seizures, and physical abuse. After years of repetitive brain trauma, clinical symptoms develop slowly over years. The clinical presentation may affect mood, behavior, motor, and cognitive domains. Currently, there are no biomarkers available to diagnose CE; therefore, the diagnosis of this neurodegenerative disease is made at postmortem examination. CE is considered a tauopathy characterized by "the deposition of hyperphosphorylated tau (p-tau) protein as neurofibrillary tangles NFTs, thorned astrocytes (TA), and neurites in a unique pattern in the brain" [17]. Other diseases such as frontotemporal dementia, Alzheimer disease, progressive supranuclear palsy, and aging may have pathological findings of tau deposition in perivascular areas of the brain [5]. According to the 2016 Berlin Consensus Statement on concussion, potential of CE must be considered as this condition is associated with tauopathy, but the incidence in athletic populations is unknown [10]. At the present time, the literature is inconsistent, and further research is needed to better understand the incidence, prevalence, and diagnostic criteria to describe CE.

## 9.6 Role of Dentistry

The role of the dentist providing sideline care is to be aware that the forces transmitted through orofacial trauma may induce concussion. Having an emergency action plan and an established concussion policy prior to the season can assist those on the sidelines to know the roles and responsibilities [18]. Preseason planning should consist of drills and planning for coverage at the home venue and away. It is important to assess the availability of emergency medical responders and the locations of the nearest trauma centers. If an athlete is unconscious, a cervical spine injury must be suspected when approaching the injured player on the field. Identification of concussion can be a daunting task for any provider of sideline coverage. Many athletes may not reveal symptoms of concussion because they do not want to be removed from play or let their coach and teammates down. Visible signs of concussion may not be present, so coaches and parents may question why an athlete is unable to return to play. Regardless of the setting, education regarding concussions should be available to the athletes, coaches, parents, and school officials. Reporting symptoms of concussion to the medical staff is the responsibility of athletes, parents, and coaches. Once an athlete is suspected of having a concussion, a physician should be involved to assist in the return-to-play decision.

## 9.7 When to Refer

Based on the Berlin Consensus Statement, "persistent symptoms" following SRC describe cases in which recovery is prolonged, for example, longer than 10–14 days in adults and greater than 4 weeks in children. Patients should receive individualized treatments based on medical, physical, and psychosocial factors. In patients reporting persistent post-concussive symptoms, there is support for symptom-limited exercise programs, physical therapy in patients with cervical spine and vestibular dysfunction, and cognitive behavioral therapy to address mood and behavioral changes. In cases that are on a prolonged path to resolution, a multidisciplinary team should be assembled.

## 9.8 Concussion Prevention

Complete concussion prevention is impossible in contact sport. Incidental head impacts are often difficult to prevent, and individuals have different impact thresholds. As reported by Rowson, high school and college football players can experience 1000 impacts in one season [19]. Given certain individuals are more prone to injury, prevention should be attempted by teaching proper techniques, wearing safer equipment, and following rule changes.

### 9.8.1 Role of Mouthguards

In the late nineteenth century, mouthguards were developed to prevent lip lacerations for boxers [19]. Mouthguards are currently used to prevent dental injuries. Advocates for mouthguard usage reducing the incidence of SRC cite the following: limiting neck muscle activation, increasing space between the head of the condyle and mandibular fossa, and absorbing impact forces at the mandible and jaw [20]. There was no evidence in a reduction in SRC while wearing a mouthguard in football, rugby, ice hockey, and basketball in a review by Benson. At this time, there are limited prospective studies demonstrating that a specific brand or design of mouthguard can reduce the risk of SRC.

**Mouthguards and Concussion Protection**

FTC Warns Retailers About Mouthguard Concussion Claims

*Commission law is clear that making an objective claim for a product without a reasonable basis is deceptive, and that competent and reliable scientific evidence is generally needed to substantiate health and safety claims. Moreover, retailers, as well as a product's manufacturer, can be liable for violating the FTC Act by disseminating false or unsubstantiated claims.*

Athletes, however, should be encouraged to use mouth protection in collision and contact sports to protect the teeth and jaws from injury.

### 9.8.2 Role of Helmets and Headgear and Impact Sensors

The football helmet is used to protect the player from direct blows to the head and prevent skull fracture [28]. Current helmet designs are heavier and larger with the intent to absorb and dissipate impact forces [20]. The National Operating Committee on Standards in Athletic Equipment (NOCSAE) has developed guidelines that football helmets must meet in order to be used in high school football competition. Older helmets that have been reconditioned should only be used if they meet the NOCSAE impact standards. In the study by McGuine et al., SRC rates were similar whether a new or old helmet was used, and no specific helmet brand offered superior protection against SRC. Older helmets performed just as well as the newer models, therefore, challenging manufacturing recommendations of regular helmet replacement and annual reconditioning.

Head-impact-exposure patterns have been reported in several studies for collision sports, such as American football, Australian football, and ice hockey. Studies have reported frequency, head impact location, head kinematics, and injury outcomes by using helmet-based measurement devices. The use of these devices does not reflect the degree of concussion injury and cannot predict clinical outcomes. Therefore, "the use of helmet-based or other sensor systems to clinically diagnose or assess SRC cannot be supported at this time" [1]. Concussion is also likely dependent on other variables such as genetics, impact location, preexisting conditions, age, and contact phenomena [19]. Sensors can be useful to alert staff of severe impacts and the need for athlete evaluation based on this and other signs and symptoms. Individual impact thresholds are variable, and some athletes will have higher tolerances to head impact than others [33].

### 9.8.3 Role of Strength Programs

There are very few concussion prevention tools that are inexpensive. Neck strengthening is inexpensive, easy to adopt, readily available, and within the athlete's control. According to Tierney, females have less head mass and in response to an applied force have greater head acceleration when compared to males [31]. This may be related to less neck strength and girth. Risk factors for concussion include poor neck strength based on past studies. It has been suggested that athletes with decreased neck strength engage in a neck strengthening program to reduce their risk of concussion [21]. Further research is required to investigate that taking these preventive measures correlates with risk reduction and less concussions in female athletes.

### 9.8.4 Education

All 50 states and the District of Columbia have concussion legislation, but only 43 of the 51 require mandatory concussion education. In certain states, school districts have provided educational materials to student-athletes, coaches, and parents explaining concussion and risks that may occur if athletes continue to play their sport after injury. However, there are no current penalties for noncompliance. Some underlying assumptions for mandatory education were that high school athletes would report concussion symptoms, seek medical care, and have better overall outcomes. Studies have demonstrated mixed results in that increased concussion knowledge has not lead more athletes to report concussion symptoms and seek medical attention [22]. Concussion legislation provides a mode of delivery of mandatory concussion education materials, but continued efforts are required to increase knowledge and change reporting behavior [25, 27]. Ethical values such as fair play and respect should be promoted to reduce the incidence of SRC. Web-based resources and educational videos endorsed by sporting bodies, government, and private companies are available to reach a broader audience.

## References


1. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport- the 5th international conference on concussion in sport held in Berlin, October 2016. Br J Sports Med. 2017;51:838–47.
2. Meeuwisse WH, Schneider KJ, Dvorak J, et al. The Berlin 2016 process: a summary of methodology for the 5th International Consensus Conference on Concussion in Sport. Br J Sports Med. 2017;51:873–6.
3. Barkhoudarian G, Hovda DA, Giza CC. The molecular pathophysiology of concussive brain injury—an update. Phys Med Rehabil Clin N Am. 2016;27(2):373–93.
4. Harmon KG, Drezner JA, Gammons M. American Medical Society for Sports Medicine position statement: concussion in sport. Br J Sports Med. 2013;47:15–26.
5. Hobbs JG, Young JS, Bailes JE. Sports-related concussions: diagnosis, complications, and current management strategies. Neurosurg Focus. 2016;40(4):1–14.
6. McCrea M, Barr WB, Guskiewicz KM, et al. Standard regression-based methods for measuring recovery after sport-related concussion. J Int Neuropsychol Soc. 2005;11(1):58–69.
7. Davis GA, Anderson V, Babl FE, et al. What is the difference in concussion management in children as compared with adults? A systematic review. Br J Sports Med. 2017a;51:949–57.
8. Davis GA, Purcell L, Schneider KJ, et al. The child sport concussion assessment tool 5th edition (child SCAT5): background and rationale. Br J Sports Med. 2017b;51:859–61.
9. Echemendia RJ, Meeuvisse W, McCrory P, et al. The sport concussion assessment tool 5th edition (SCAT5): background and rationale. Br J Sports Med. 2017;51:870–1.
10. McCrory P, Collie A, Anderson V, et al. Can we manage sport related concussion in children the same as in adults? Br J Sports Med. 2004;38:516–9.
11. Rizzo JR, Hudson TE, Dai W, et al. Rapid number naming in chronic concussion: eye movements in the King-Devick test. Ann Clin Transl Neurol. 2016;3(10):801–11.
12. Patricios J, Fuller GW, Ellenbogen R, et al. What are the critical elements of sideline screening that can be used to establish the diagnosis of concussion? A systematic review. Br J Sports Med. 2017;51:888–94.
13. Putukian M. Clinical evaluation of the concussed athlete: a view from the sideline. J Athl Train. 2017;52(3):236–44.
14. Master CL, Mayer AR, Grady MF. Minds matter: concussion care for children. Curr Sports Med Rep. 2016;15(4):230–2.
15. Hebert O, Schlueter K, Hornsby M, et al. The diagnostic credibility of second impact syndrome: a systematic literature review. J Sci Med Sport. 2016;19:789–94.
16. McCrory P. Does second impact syndrome exist? Clin J Sport Med. 2001;11:144–9.
17. Huber BR, Alosco ML, Stein TD, et al. Potential long-term consequences of concussive and subconcussive injury. Phys Med Rehabil Clin N Am. 2016;27:503–11.
18. Guskiewicz KM, Broglio SP. Acute sports-related traumatic brain injury and repetitive concussion. Handb Clin Neurol. 2015;127:157–72.
19. Rowson S, Bland M, Eamon T, et al. Biomechanical perspectives on concussion in sport. Sports Med Arthrosc Rev. 2016;24(3):100–7.
20. McGuine TA, Hetzel S, McCrea M, Brooks MA. Protective equipment and player characteristics associate with the incidence of sport-related concussion in high school football players: a multifactorial prospective study. Am J Sports Med. 2014;42(10):2470–8.
21. Collins CL, Fletcher EN, Fields SK, et al. Neck strength: a protective factor reducing risk for concussion in high school sports. J Prim Prev. 2014;35:309–19.
22. Carroll-Alfano M. Mandated high school concussion education and collegiate athletes' understanding of concussion. J Athl Train. 2017;52(7):689–97.
23. Giza CC, Prins ML, Hovda DA. It's not all fun and games: sports, concussions, and neuroscience. Neuron. 2017;94:1051–5.
24. Hyden JH, Petty B. Sideline management of concussion. Phys Med Rehabil Clin N Am. 2016;27(2):395–409.
25. Kontos AP, Deitrick JM, Collins MW, et al. Review of vestibular and oculomotor screening and concussion rehabilitation. J Athl Train. 2017;52(3):256–61.
26. Matuszak JM, McVige J, McPherson J, et al. A practical concussion physical examination toolbox: evidence-based physical examination for concussion. Sports Health. 2016;8(3):260–9.
27. Provance AJ, Engelman GH, Terhune EB, et al. Management of sport-related concussion in the pediatric and adolescent population. Orthopedics. 2016;39(1):24–30.
28. Scorza KA, Raleigh MF, O'Connor FG. Current concepts in concussion: evaluation and management. Am Fam Physician. 2012;85(2):123–32.
29. Shrier I. Neuropsychological testing and concussions: a reasoned approach. Clin J Sport Med. 2012;22(3):211–3.
30. Stovitz SD, Weseman JD, Hooks MC, et al. What definition is used to describe second impact syndrome in sports? A systematic and critical review. Curr Sports Med Rep. 2017;16:50–5.
31. Tierney RT, Sitler MR, Swanik CB, et al. Gender differences in head-neck segment dynamic stabilization during head acceleration. Med Sci Sports Exerc. 2008;37(2):272–9.
32. Ventura RE, Balcer LJ, Galetta SL, et al. Ocular motor assessment in concussion: current status and future directions. J Neurol Sci. 2016;361:79–86.
33. Steven PB, Brock S, Jacob JS, Sunghoon S, Xingdong F, Xuming HE, Jerrad Z. Biomechanical properties of concussions in high school football. Medicine & Science in Sports & Exercise 42(11):2064–71.

# Dental Erosion in Sports

*Christopher Rivers and Michael Longlet*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_10

## 10.1 Introduction

Dental erosion is defined as the pathologic loss of dental hard tissues due to the chemical influence of acid, without bacterial involvement [1]. The acids responsible for the erosive wear of the dentition are not native to the oral environment; rather they can be introduced either extrinsically or intrinsically. Extrinsic erosion is most commonly dietary in origin. Acidic soft drinks, juices, and the increasingly popular sports and energy drinks are regular contributors. Erosion of an intrinsic nature is a result of contact between tooth structure and internal acids, most notably gastric acid. This contact is typically a result of gastroesophageal reflux, recurrent vomiting, or regurgitation. The frequency with which the acidic substances come into contact with tooth structure directly correlates with the degree of severity of the erosion [2]. That being said, erosion typically has a multifactorial etiology with several of the aforementioned sources at play.

**Causes of Sports-Related Dental Erosion**

*Extrinsic causes*

- Sports drinks: Hydration
- Energy drinks: Energy for competitive edge

*Intrinsic causes*: Gastric acid

- Self-induced vomiting (wrestling)
- Bulimia nervosa (body conscious sports)
- GERD (gastroesophageal reflux disease)

*Other sports-related sources*

- Improperly chlorinated swimming pools



Due to the sources of dental erosion, athletes are at risk. The consumption of sports drinks to aide in rehydration and replacement of electrolytes is a common practice. Some athletes are consuming energy drinks for the benefit of caffeine; however, the International Olympic Committee bans this substance as it has been found to increase endurance [3]. When compared to water, there are few benefits to using sports drinks for rehydration by athletes that are not competing at an elite level. In addition, there are little to no benefits of consuming energy drinks prior to competition.

Dental erosion can also affect athletes from an intrinsic route. Athletes competing in sports that value leanness and weight dependency are at risk of developing an eating disorder. A study by Byrne and McLean found that a higher percentage of eating disorders are found in athletes than in nonathletes and that athletes competing in body conscious sports showed a higher prevalence of eating disorder symptoms [4]. Eating disorders that involve purging, such as bulimia nervosa, allow for gastric contents to come in contact with the dentition. Depending on the frequency and duration of which one has been self-inducing vomiting, varying degrees of erosion can be observed.

As athletes are at risk of dental erosion, it is important for dentists to recognize the clinical presentation. The pattern of erosive wear on the dentition can be indicative of what is causing the erosion. The athlete may not even be aware that this process is happening, and the dentist could be the first to notice. The diagnosis of dental erosion could be the first step in changing a behavior causing irreversible damage to the athlete's dentition or even their systemic health.

## 10.2 Hydration Science

The creation of sports drinks began in 1965, at the University of Florida. A football coach had noticed his players losing weight during games but also found that the players were not urinating much. The athletes were sweating so much in the humid swamp air that they were not retaining enough fluid to urinate. It was thought that the amount of sodium and potassium lost in the players sweat was upsetting the chemical balance of their bodies. Researchers developed a beverage that contained not only water but salt and sugar. As the first batch was completely unpalatable, lemon juice was added to enhance the taste. Due to the improvement in performance that was observed as the Florida Gators football team consumed this drink throughout practice and games, it was named Gatorade. The formula has not changed much over the years, and Gatorade remains the most researched sports drink [5].

Despite the benefits observed by a college football team, is a sports drink necessary to maintain hydration and electrolyte balance during physical activity? It is well known that maintaining hydration during exercise facilitates the duration in which a person can exert more effort. However, if sweat loss is minor, dehydration may not be an issue. Greater sweat loss occurs in warmer and more humid conditions but also when the duration or intensity of the activity increases. Maintaining hydration during exercise is largely dependent on sweat loss, thus weight loss that happens during exercise is merely water lost from sweat.

The daily average fluid intake for male and female adults is 3.7 L and 2.7 L, respectively, which is equivalent to 16 cups for men and 11 cups for women [6]. These daily recommendations are for sedentary adults, and fluid requirements rise significantly when someone is engaged in strenuous physical activity. Data from a fitness club showed that 46% of people that arrive for a workout are not properly hydrated [7]. It has been suggested that consuming 14–20 ounces of fluid an hour or two before exercise can increase the likelihood of entering the workout hydrated. It is also important to note that hydration should be maintained throughout the workout to avoid suffering the consequences of dehydration, which include reduced blood volume, increased heart rate, and trouble losing body heat.

Monitoring hydration status during a workout can be accomplished by observing body weight, thirst status, and urine color [8]. Body weight should be maintained or only slightly reduced after exercise. As previously stated, weight loss during a workout is merely water lost as sweat. A body weight reduction of 2% or more can impair performance and lead to the negative physiologic effects associated with dehydration [7]. Thirst is also a measure of hydration status; however, it is less reliable. The hypothalamus is responsible for monitoring changes in the blood, and the effects of dehydration are detected as the salt content of blood increases and the blood volume decreases. Thirst is stimulated in this way and is reduced when the blood levels return to normal. For the sedentary person, thirst is a great indicator of fluid balance. However, by the time thirst is detected during exercise, a person is already 1–2% dehydrated, which leads to worsening of dehydration if thirst is the only factor at play [9]. Another way to gauge hydration is by observing urine color. Proper hydration is demonstrated by urine that is pale yellow, similar to lemonade. Urine that is dark yellow signifies dehydration and the need to drink more. Conversely, urine that is clear like water may be a sign of overhydration, and backing off on fluid intake might be called for.

The ideal fluid used to rehydrate during exercise depends on the level of intensity of the activity, as well as the amount of sweat lost during the workout. Water is very effective at replacing lost sweat if enough is consumed. As will be discussed next, water does not cause erosion of the dentition unlike acidic sports drinks. Despite its negative effects on the oral environment, properly formulated sports beverages can be beneficial for heavy sweat producers. The carbohydrates and electrolytes in sports drinks not only help maintain hydration but also can sustain the capacity to continue exercise. The carbs in sports drinks can help provide energy to the muscles that are working during exercise. However, since the body can only oxidize 60–90 g of carbohydrate per hour, it makes sense to choose a beverage that does not have too many carbs [10]. Soft drinks, fruit juices, and beer all contain a high amount of carbohydrates, which will lead to a reduction of gastric emptying and fluid absorption and ultimately a stomachache and nausea if consumed during strenuous activity. The sodium in sports drinks can also help replace what is lost in sweat. Sodium can help the body retain more fluid instead of losing it to sweat.

Despite the perceived benefits of sports drinks, research has shown that for individuals not involved in intense or prolonged physical activity, there is little benefit of sports drinks over water. The study showed that both water and sports drinks were successful in maintaining hydration and both resulted in equal levels of performance [11]. The only difference was that voluntary consumption of the sports drink was higher than that of water. The taste of the sports drink is typically perceived as more palatable and desirable than water, which can lead to a greater degree of hydration if the sports drink is being consumed willingly more often. However, for the recreational athlete or everyday exerciser, water is just as efficient at maintaining hydration as sports drinks, which have no additional benefit when it comes to replacement of sweat loss (◘ Fig. 10.1).

## 10.3 Extrinsic Erosion: Sports and Energy Drinks

As previously stated, sports drinks were originally created for and marketed to sports teams as a replacement for carbohydrates and electrolytes that were lost during competition. These athletes were engaging in intense activity lasting longer than 1 h. As time has passed since the creation of sports drinks and they have become more popular among consumers, the beverages are now being marketed to and consumed by those that are not involved in endurance sports or strenuous exercise. Whereas the consumption of carbohydrates during prolonged exercise can lead to an increase in endurance capacity, the average consumer of sports drinks does not necessarily derive a benefit to hydration or performance that water wouldn't fulfill. Consequently, there are negative effects on the dentition that can manifest as dental erosion or caries due to the acidic nature and sugar content of the drinks (◘ Fig. 10.2).

Dental erosion is the irreversible loss of tooth structure that can occur as a result of frequent consumption of acidic sports drinks, soft drinks, or energy drinks. The exposure of tooth structure to the drinks leads to demineralization of enamel and dentin surfaces. The optimal pH of saliva is between 6.5 and 7.5, whereas the critical pH that results in the demineralization of tooth structure is 5.5 [12]. An influencing factor is the pH of what is being consumed. As the pH level at the surface of the tooth decreases as a result of acidic exposure, calcium and phosphate ions are released from the tooth, resulting in demineralization. Popular sports and energy drinks such as Gatorade (pH = 2.95), Powerade (pH = 2.78), and Red Bull (pH = 3.32) all have a low pH that results in a high level of acidity at the tooth surface [13].

**pH of Popular Drinks**

| Condition/drink | pH |
|---|---|
| – Optimal oral pH | 6.5–7.5 |
| – Demineralization critical pH | 5.5 |
| – Water | 7.0 |
| – Gatorade | 2.95 |
| – Powerade | 2.78 |
| – Red Bull | 3.32 |
| – Cola | 2.16 |

Despite a similarly acidic pH, it has been shown that the demineralization of enamel from sports and energy drinks is 3–11 times greater than that of cola, which has a pH of 2.16.


**Fig. 10.1** Ubiquitous nature of sports drinks: Marketing. Sports drinks are big business (8 billion dollar per year) and are found in grocery stores, drug stores, and convenience stores, and anywhere soft drinks are found. Aggressive marketing grows market share at the expense of teeth. **a** Above shows a major pharmacy's stock of sports and energy drinks. The same store had end caps and other sites for sports drinks as well. **b** Shows marketing for popular Gatorade sports drink at a Minnesota convenience store. (Photo credit: Mark Roettger DDS)

10

### Erosion Potency of Sports Drinks [12]

All tested drinks were compared to black tea and erosion ratio figured

| | |
|---|---|
| 1. Coke | 7.94 × more erosive than tea |
| 2. AMP energy drink | 40.03 × more erosive than tea |
| 3. Gatorade | 57.29 × more erosive than tea |
| 4. KMX energy drink | 84.80 × more erosive than tea |
| 5. Powerade | 48.51 × more erosive than tea |
| 6. Red Bull energy drink | 61.14 × more erosive than tea |

Black tea 0.35 mg/cm$^2$—standard

This difference is likely due to the large amounts of citric acid in sports and energy drinks as compared to the presence of phosphoric acid that is found in cola drinks. Citric acid has a greater buffering capacity and is able to bind with calcium that is released from the tooth structure during demineralization. The chelation of calcium leads to a reduction in the positive effects of calcium to reduce acidity, which ultimately would lead to remineralization. The perpetuation of a low pH after the acid exposure results in a greater potential for erosive damage to tooth structure. That being said, research has shown that there appears to be less of a correlation between the pH of a beverage and the amount of enamel demineralization. One study found that beverages with a greater composition of acid that could chelate calcium determined the severity of enamel demineralization more so than the pH of the beverage [12]. This is consistent with sports and energy drinks exhibiting a greater propensity for enamel demineralization than cola, which is just as acidic.

Naturally, the body has a biologic defense in the form of saliva that can help combat the erosive nature of acidic sports drinks. Two important protective features of saliva are the flow rate and the buffering capacity. The presence of saliva leads to the dilution of erosive beverages when they are present in the oral environment. As previously discussed, prolonged exercise leads to a significant loss of fluids through perspiration, ultimately leading to dehydration. As dehydration progresses, salivary flow rate decreases, and the resulting xerostomia leads to a slower rate of clearance of the erosive beverage that is less diluted. Saliva also has the ability to neutralize and buffer acidic substances. The calcium and phosphate in saliva can help combat the drop in pH and encourage remineralization at


**Fig. 10.2** **a** Clinical erosion from sports drinks. **b** Clinical erosion from sports drink use. **a** Clinical photos of a 24-year-old female athlete with severe erosion and secondary dental caries resulting from multiple years of drinking 3–5 sports drinks per day. Treatment required multiple extractions, endodontic treatments, and restorative procedures on nearly every tooth. **b** Is the full-mouth radiographs of the same patient. Both show the incredible damage partially caused by abuse of sports drinks. (Photo credit: Mark Roettger DDS)

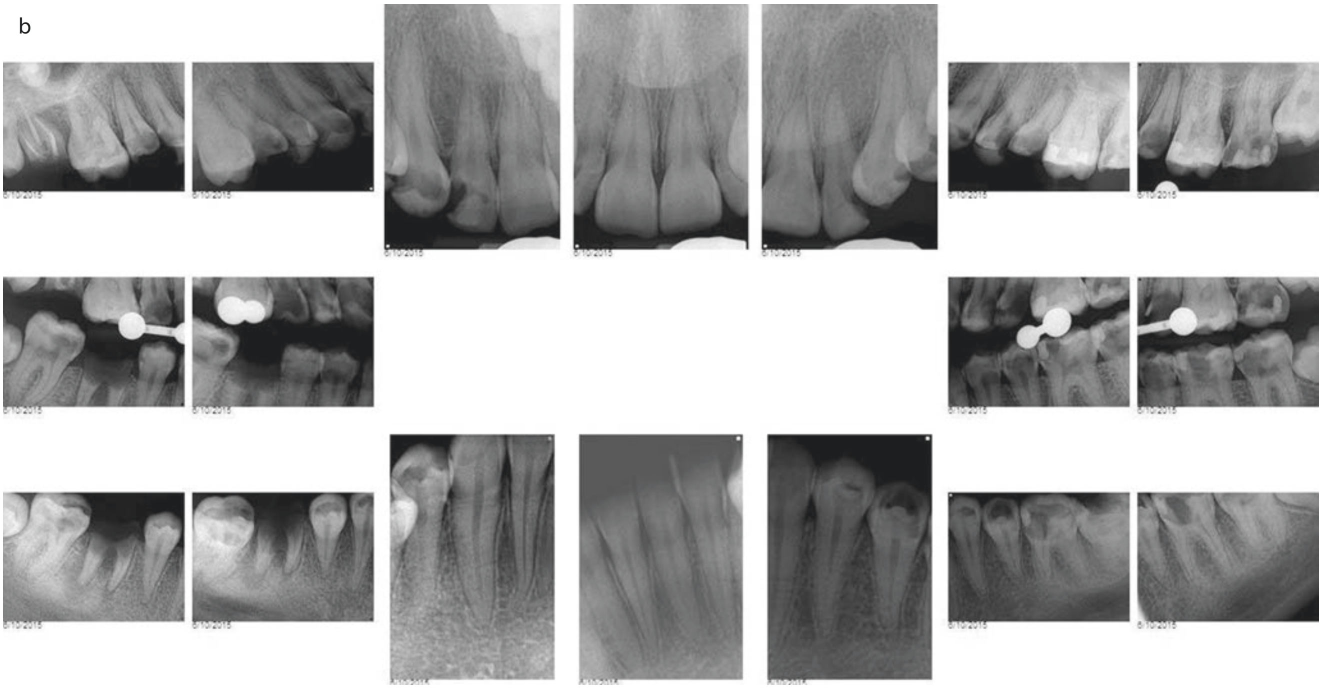




**Fig. 10.2** (continued)

the tooth surface. This was demonstrated by a study that measured the salivary pH, flow rate, and buffering capacity of saliva in children. It was discovered that children with erosive lesions had a low salivary buffering capacity compared with those without lesions [14]. As a result, it has been proposed that adding calcium and phosphate to sports drinks and soft drinks may reduce the erosive nature of the beverages.

In an attempt to combat the negative aspects of sports drinks on the dentition, studies have examined the effect of attempting to lower the pH of the beverage as well as adding calcium and phosphate. It was found that this could reduce the amount of erosion that is seen on enamel surfaces. One such study involved a prototype carbohydrate and electrolyte drink that was compared to a commercially available sports drink, as well as water. The prototype drink had a less acidic pH and a greater content of calcium (pH of 3.81 and 355 mg/L of calcium) than the sports drink that was commercially available (pH of 3.16 and 2.8 mg/L of calcium). The researchers found that the amount of enamel lost from subjects that consumed the prototype sports drink was the same as the group that consumed water. In contrast, the amount of erosion observed in the group that consumed the commercially available sports drink was almost 30 times greater than the other 2 groups [15]. The challenge with adding calcium and phosphate to sports drinks to reduce the erosive effect lies in the taste. It has been determined that people prefer the taste of beverages with a lower pH and attribute a repulsive taste to those drinks with an increased pH and calcium content [16]. The palatability problems have made a more favorable formulation to the dentition more challenging.

As it is impossible to reconstitute the ingredients of all sports drinks, consumers must develop good habits when it comes to drinking these beverages. Dentists have an important role, as they may be the first to notice the erosive changes to the dentition that may be unknown to the patient. Early diagnosis and behavior modification are essential. First and foremost, sports drinks should not be substituted for water, as a majority of people consuming these beverages will derive no greater benefit to hydration that water will not provide. If sports drinks are to be consumed, the method of consumption can be modified to reduce the erosive effects on the dentition, specifically the amount of time the drink spends in contact with the teeth. Slowly sipping the beverage, or swishing a drink in the mouth, leads to the dentition being exposed to a lower pH and acidic conditions for a longer period of time. To avoid this, consumers can attempt to rapidly swallow or gulp a sports drink to minimize the amount of time the liquid is in contact with the dentition.

Finally, in addition to sports drinks, some athletes are consuming energy drinks prior to competition, mainly for the effect of caffeine and increased alertness. Like sports drinks, energy drinks also have an erosive effect on the dentition; however, these drinks carry no benefits in regard to hydration or the replenishment of carbohydrates and electrolytes. The dangers of energy drinks lie in the ingredients. Energy drinks contain caffeine along with other ingredients and herbal supplements, such as taurine, guarana, ginseng, and B vitamins that all claim to provide an increase in energy. Despite being banned by the International Olympic Committee, consuming caffeine before exercise has been found to be safe; however when combined with the other ingredients in energy drinks, there is not enough evidence to determine safety [3].

**Possible Ingredients of Energy Drinks**

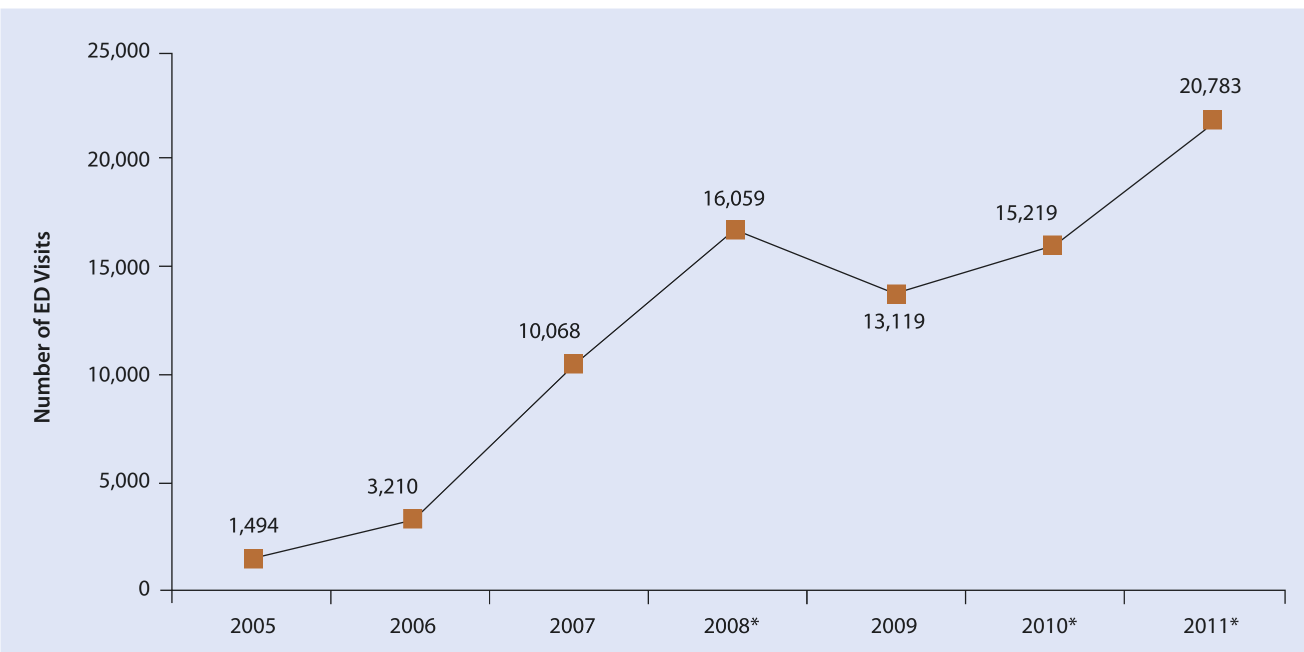
- *Caffeine* (high dose): cardiovascular effects
- *Taurine*: amino acid—anxiolytic
- *Guarana*: (more caffeine) cardiovascular effects
- *Ginseng*: cardiovascular effects
- *Yohimbe*: cardiovascular effects
- *β-phenylethylamine*: weight loss

Many ingredients of energy drinks have effects on the cardiovascular system including heart rate and blood pressure. Their use is not encouraged for sports performance.

High levels of caffeine have been associated with an increase in blood pressure. Supplemental ingredients yohimbe and β-phenylethylamine are also known to increase blood pressure; however, their chemical structure is similar to amphetamines, and their interaction with caffeine is not well known. One report described a patient that suffered from a hemorrhagic stroke after consuming an energy drink prior to doing yard work. In addition, the patient had chronic, untreated hypertension that was likely exacerbated due to energy drink consumption [17]. Unless the label is read, the serving size of most energy drinks can be misinterpreted. The 8 ounce can that was consumed by the patient with the stroke states there are two servings per can, although most people likely consume the entire can in one sitting. The FDA recommends a maximum allowance of 400 mg of caffeine per day for normal healthy adults, which is about the equivalent of 4–5 cups of coffee [18]. Energy drinks can contain almost 300 mg of caffeine per can. Ultimately, there are more negative effects than benefits when it comes to consuming energy drinks during athletic activities, and their use should be avoided (◘ Fig. 10.3) [19].

## 10.4 Intrinsic Erosion: Gastric Acid

Athletes are not only at risk of dental erosion from what they consume but from internal sources as well, primarily in the form of gastric acid. Exposure of the dentition to gastric acid is a result of vomiting, regurgitation, or gastroesophageal reflux disease. As with extrinsic sources, the degree of erosion observed is dependent on the amount of time and frequency with which tooth structure is exposed to the acidic environment. Observable intrinsic erosion is typically a result of behavioral habits such as self-induced vomiting. Self-induced vomiting has been associated with eating disorders, and depending on the sport, certain athletes may be at risk. It has been found that eating disorders are more common in athletes than in the general population, more common in females than males, and with a greater predilection for athletes in body conscious sports that favor a lean body habitus [20]. Athletes in sports like gymnastics, dancing,




◘ **Fig. 10.3** ED visits related to energy drinks. Energy Drink-Related Emergency Department (ED) Visits, by Year: 2005 to 2011. *Compared with the number of visits in 2007, the difference was statistically significant at the 0.05 level. The number of visits in years prior to 2007 were not used in statistical tests because of low numbers; the number of visits in 2004 was not shown because of low statistical precision. (Source: 2011 SAMHSA Drug Abuse Warning Network (DAWN)) figure skating, and wrestling are often pressured to reduce weight to enhance their performance. The quest for leanness by athletes and the pressure to perform better can have potential to develop into an eating disorder.

Anorexia nervosa affects approximately 1% of women between the ages of 12 and 25, and bulimia nervosa is even more common, affecting between 1 and 5% of the same demographic. In addition, 40% of patients with anorexia also have bulimia [21]. Although some studies suggest a genetic component to the disorders, cultural factors such as involvement in athletics play a role, especially those that encourage a lean body appearance. For example, ballet dancers have a prevalence of anorexia that is ten times greater than the general population [20]. As anorexia and bulimia can both affect athletes in body conscious sports, bulimia is of greater concern to the dentition, as it can lead to debilitating erosion. Anorexia is the restriction of food intake and is diagnosed as weighing 85% or less than ideal weight. Despite being underweight, patients have a fear of becoming fat and often have an altered perception of themselves as it relates to body weight. Bulimia involves self-induced vomiting and a history of binge eating without major weight gain.



As stated, dental erosion is more common in individuals with eating disorders that practice self-induced vomiting since the pH of gastric acid is around 1–1.5. Dental erosion affects 20% of patients with anorexia and more than 90% of patients with bulimia [22]. The most common presentation of erosive wear in people with this disorder is observed on the palatal surfaces of the upper anterior teeth due to the contact of these areas with stomach contents during vomiting. However, as erosion of the dentition is dependent on the amount of time the tooth surfaces are exposed to the acidic environment, the habitual vomiting is typically a chronic issue of 1–2 years prior to noticeable changes on the dentition [23]. One study showed that patients who practiced self-induced vomiting for over 10 years demonstrated more erosions and a greater severity of the erosive lesions, including exposed dentin [2]. However, the same study also showed that almost one third of the individuals who had been inducing vomiting regularly for many years had no evidence of erosive lesions. This shows that the degree of erosion is likely multifactorial when it comes to eating disorders. In addition to the frequency of acid exposures, salivary flow also seems to play a role. Studies have shown that patients with bulimia also have a significantly lower unstimulated salivary flow rate. In addition, many patients seeking treatment for an eating disorder are also prescribed antidepressants or other drugs that are known to have xerostomia as a side effect [24].

The oral hygiene habits of patients who self-induce vomiting can also influence the degree of dental erosion. It has been observed that patients with bulimia who regularly brush their teeth immediately after vomiting have more severe erosions observed on tooth structure than those who only rinse with water [22]. The abrasive nature of brushing is damaging to enamel that is in a state of demineralization. It was observed that even brushing without toothpaste leads to the loss of enamel. It has been recommended that patients should wait at least an hour after vomiting before brushing their teeth.

Many patients that are affected by eating disorders tend to keep their food related issues to themselves. It may be more apparent when a patient has anorexia nervosa due to their emaciated figure; however, a patient with bulimia nervosa may be more difficult to detect since they tend to maintain their body weight. For this reason, erosive changes to the dentition from self-induced vomiting may be the only observable changes. Specific changes that may be observed in the dentition are not only limited to enamel erosion but can also include dentinal hypersensitivity to temperature, raised restorations above the eroded tooth structure, decreased salivary flow, and enlarged parotid glands. The restricted diet in patients with eating disorders results in a lack of intake of vitamins and nutrients. A lack of vitamin B, specifically B1, B6, and B12, has been associated with a decrease in epithelial cell turnover and can manifest as mucosal atrophy. A deficiency in vitamin C, also known as scurvy, can result in defective collagen synthesis. Faulty collagen can affect the periodontium and can lead to edematous gingiva, gingival bleeding, mobility of teeth, and gingival ulcerations [25].

As these changes involve the oral cavity, the dentist may be the first healthcare professional to notice, and a proper referral could lead to the diagnosis of an eating disorder. The dentist should be prepared to confront the patient and inform them of not only the risk to the dentition but also the serious health risks that could occur as a result. For example, patients with anorexia are at risk of sudden cardiac death from tachyarrhythmias [20]. Athletes in sports that value body leanness are at an increased risk, and the dentist should be mindful of patients involved in these activities. Oral manifestations can impair oral function, lead to oral discomfort, as well as lead to a decreased quality of life.

## 10.5 Other Sources of Erosion

Another unique source of erosion to the dentition of athletes that deserves mention is that which is observed after swimming in an improperly chlorinated pool. Poorly chlorinated swimming pools with an acidic pH can have been reported to result in dental erosion. Although technically a form of extrinsic erosion, the exposure of the dentition to chlorine is not as overtly intentional when compared to consuming a sports or energy drink. Nonetheless, erosive effects are still observed, but with a slightly different presentation.

Chlorine is the most common agent used to sustain the pH balance of swimming pools. The pH of a properly chlorinated pool is between 7.2 and 7.8, which happens to be the range in which the physiologic pH of 7.4 falls [26]. Improper chlorination of a swimming pool can result in acidic conditions. A low pH may not necessarily be evident to swimmers, aside from some eye irritation to those not wearing goggles [27]. A few times spent in an acidic pool may not result in symptoms to the swimmer. However when many long hours are spent each day in the pool, such as those experienced by competitive swimmers, the acidic pH can lead to the erosion of dental enamel.

One such case was described in which a patient reported to his dentist with the chief complaint of extremely sensitive teeth and dark staining. The patient was examined by the dentist who noticed the labial surfaces of his anterior teeth appeared as if they were being prepared for porcelain veneers. After a thorough investigation, it was discovered that the patient had been swimming for 90 min each day in his home pool; however, the pool was not being maintained by a professional and was found to be improperly chlorinated, resulting in an acidic environment. The patient's enamel had undergone irreversible erosion of his dentition from his time spent in the pool [28]. Similar reports exist and mutual symptoms include temperature sensitivity, anterior teeth resembling porcelain veneer preparations, the manifestation of diastemas that were not previously present, as well as a rough texture to the affected teeth.

## 10.6 Treatment

Treatment of dental erosion first involves finding the source. Without a proper identification and elimination of what is causing the erosion, treatment will be futile. Much like athletes in contact sports that wait for their professional careers to be over before pursuing a permanent solution for their damaged dentition, athletes with erosion must also delay treatment until they are able to manage to remove the source of erosion from exposure to the dentition.

For athletes suffering from eating disorders, treatment first involves making sure the patient is medically stable and receiving the appropriate treatment for their systemic condition. As previously stated, medical complications can include cardiac arrhythmias leading to death, which is clearly more serious than dental erosion. For short-term relief of symptoms, direct composite restorations can be placed to help relieve dentinal sensitivity and aide in prevention of further erosion. A long-term fixed prosthodontics treatment plan should not be pursued until the patient has recovered from the eating disorder by undergoing behavior modification techniques to help with weight gain.

Patients can be treated for their eating disorder in either an inpatient or outpatient setting. The role of the dentist during this time is to focus on oral hygiene improvement and sensitivity control. Patients can be prescribed desensitizing toothpaste or fluoride during this phase. The dentist could also provide the patient with customized trays and 1.1% fluoride gel that can be worn for 5 min daily [21]. When the patient is ready for a more permanent treatment, full coverage restorations are typically required, but this is based on the severity of the erosion. Definitive treatment could include bonded labial veneers in combination with onlays or full-coverage crowns. For teeth that have become eroded to the point where little to no clinical crown is left, endodontic treatment with a post, core, and crown may be necessary. Patients should be informed that if they relapse, the restorations might fail with more exposure of the supporting tooth structure to vomiting.

Athletes that are experiencing erosion from an extrinsic source, such as frequent sports drink consumption or swimming in an improperly chlorinated pool, have similar options with treatment. One thing remains the same and that is identifying the source of the erosion. A thorough history of daily habits can aide in the discovery of the cause. As these patients do not necessarily require medical intervention prior to definitive treatment, once the source is revealed and the habit addressed, the patient can proceed straight to a more definitive restorative approach. Despite the differences in the sources of erosion, treatment goals remain the same. Goals include eliminating dental sensitivity, replacing the tooth structure that has been lost to erosion, correcting esthetics, and reestablishing proper occlusion [27].

## 10.7 Conclusion

Despite the multiple sources of dental erosion, it is clear to see that athletes are at risk. Whether it is from extrinsic or intrinsic sources, the pathologic loss of tooth structure can result from an acid attack on the dentition. It is important for dentists to recognize the clinical presentation, which can lead to detection of the source of erosion. The athlete may not even be aware of the source. Early detection can lead to the preservation of tooth structure. Regardless of the source, it is apparent that behavior modification is key in eliminating the progression of enamel demineralization. Treatment may save the dentition from further destruction but prevention is best. The sports team dentist needs to be proactive with the players on his or her team regarding hydration and the use of sports or energy drinks, educating them on the dangers associated with their use and abuse. Through recognition and elimination, treatment can commence to eliminate discomfort and restore quality of life.

## References


1. Imfeld T. Dental erosion. Definition, classification and links. Eur J Oral Sci. 1996;104(2):151–5.
2. Uhlen MM, Tveit AB, Stenhagen KR, Mulic A. Self-induced vomiting and dental erosion–a clinical study. BMC Oral Health. 2014;14(1):92.
3. Heneman K, Zidenberg-Cherr S. Some facts about energy drinks. Nutrition and health info-sheet for health professionals. 2011.
4. Byrne S, McLean N. Eating disorders in athletes: a review of the literature. J Sci Med Sport. 2001;4(2):145–59.
5. Kays J, Phillips-Han A. Gatorade: the idea that launched an industry, vol. 8(1). Explore: Research at the University of Florida; 2003.
6. Institute of Medicine (US). Panel on Dietary Reference Intakes for Electrolytes, & Water. DRI, dietary reference intakes for water, potassium, sodium, chloride, and sulfate. Washington: National Academy Press; 2005.
7. Stover B, Murray B. Drink up!: the science of hydration. ACSMs Health Fit J. 2007;11(2):7–12.
8. Cheuvront SN, Sawka MN. SSE# 97: hydration assessment of athletes. Sports Sci Exch. 2005;18(2):1–12.
9. Hubbard RW, Szlyk PC, Armstrong LE. Influence of thirst and fluid palatability on fluid ingestion during exercise. In: Fluid homeostasis during exercise. Carmel: Benchmark Press; 1990. p. 39–95.
10. Maughan RJ, Murray R, editors. Sports drinks: basic science and practical aspects. Boca Raton: CRC Press; 2000.
11. Johnson HL, Nelson RA, Consolazio CF. Effects of electrolyte and nutrient solutions on performance and metabolic balance. Med Sci Sports Exerc. 1988;20(1):26–33.
12. Von Fraunhofer JA, Rogers MM. Effects of sports drinks and other beverages on dental enamel. Gen Dent. 2005;53(1):28–31.
13. Noble WH, Donovan TE, Geissberger M. Sports drinks and dental erosion. J Calif Dent Assoc. 2011;39(4):233–8.
14. Sanchez GA, Fernandez De Preliasco MV. Salivary pH changes during soft drinks consumption in children. Int J Paediatr Dent. 2003;13(4):251–7.
15. Venables MC, Shaw LINDA, Jeukendrup AE, Roedig-Penman A, Finke M, Newcombe RG, Parry J, Smith AJ. Erosive effect of a new sports drink on dental enamel during exercise. Med Sci Sports Exerc. 2005;37(1):39–44.
16. Cochrane NJ, Yuan Y, Walker GD, Shen P, Chang CH, Reynolds C, Reynolds EC. Erosive potential of sports beverages. Aust Dent J. 2012;57(3):359–64.
17. Venkatraman A, Khawaja A, Shapshak AH. Hemorrhagic stroke after consumption of an energy drink. Am J Emerg Med. 2017;35:522.
18. US Food and Drug Administration. FDA to investigate added caffeine. Consumer Updates. 2013.
19. Mattson ME. Update on emergency department visits involving energy drinks: a continuing public health concern. The CBHSQ report: January 10, 2013. Rockville, MD: Center for Behavioral Health Statistics and Quality, Substance Abuse and Mental Health Services Administration; 2013.
20. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. Clin J Sport Med. 2004;14(1):25–32.
21. Little JW. Eating disorders: dental implications. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;93(2):138–43.
22. Lifante-Oliva C, López-Jornet P, Camacho-Alonso F, Esteve-Salinas J. Study of oral changes in patients with eating disorders. Int J Dent Hyg. 2008;6(2):119–22.
23. Scheutzel P. Etiology of dental erosion–intrinsic factors. Eur J Oral Sci. 1996;104(2):178–90.
24. Dynesen AW, Bardow A, Petersson B, Nielsen LR, Nauntofte B. Salivary changes and dental erosion in bulimia nervosa. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;106(5):696–707.
25. Lo Russo L, Campisi G, Di Fede O, Di Liberto C, Panzarella V, Lo Muzio L. Oral manifestations of eating disorders: a critical review. Oral Dis. 2008;14(6):479–84.
26. Kane S, Keeton R. Tooth damage linked to public pool by Center for Disease Control. Fla Dent J. 1983;54(4):12–3.
27. Dawes C, Boroditsky CL. Rapid and severe tooth erosion from swimming in an improperly chlorinated pool: case report. J Can Dent Assoc. 2008;74(4):359–61.
28. Jahangiri L, Pigliacelli S, Kerr AR. Severe and rapid erosion of dental enamel from swimming: a clinical report. J Prosthet Dent. 2011;106(4):219–23.

Check for updates

# Tobacco Use in Sports

*Mark Roettger and Ryan Pacheco*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_11

## 11.1 Introduction

Tobacco use in sport is widely practiced and has a long history and has developed its own culture in athletics. Sports such as baseball have a long and open history of tobacco use while other sports the use of tobacco is more insidious and hidden. It is not uncommon to see a major league baseball player or manager with a large plug of tobacco in his mouth and even the telltale brown spittle that runs down the chin and onto a jersey. This in contrast to a male or female hockey player who places spit tobacco plug in his or her mouth between periods of a game thus hidden from family and fans. There is a stigma among many female athletes on spit tobacco use because of the need to spit or the brown staining of teeth and lips leading to the vaginal use of tobacco in some female athletes. Athletes use tobacco during sports because they perceive a quick boost of epinephrine which increases heart rate and blood pressure and creates rapid shallow breathing. This offers the illusion of performance enhancement. After the initial epinephrine burst wears off, the athlete may feel tired or lethargic requiring another dose. The performance enhancement phenomenon for spit tobacco use has been shown to be nonexistent in multiple studies [1].



**Athletes and Spit Tobacco**

- Athletes are led to believe smokeless is harmless.
- Myth: spit tobacco can enhance athletic performance.
- Not just baseball anymore: hockey, golf, rodeo, and football.
- Use by females is increasing: oral and axillary.
- NSTEP estimates one in ten high school boys use spit tobacco.
- Unknown link to incidence of oral cancer.

Nicotine use carries physical and psychological addiction issues with its use, and every effort to prevent its use and abuse is required. Tobacco is big business throughout the world, and tobacco companies have been less than helpful in efforts to educate the public to the dangers of tobacco use; in fact they have been engaged in creating doubt as to the dangers of tobacco use in the past [2]. This is extremely important considering the dangers associated with tobacco use. Tobacco companies are always in need of new consumers, and there have been marketing strategies aimed at kids. These strategies involve emulation of older athletes. Case in point is a bubble gum product called "Big League Chew." This bubble gum comes packaged the same way leafy tobacco chew often used by professional baseball players is packaged (Fig. 11.1). Copying the tobacco habits of major league baseball players is not a healthy practice for children, and health-care providers must be a voice against such insidious and devious marketing practices.


**Fig. 11.1** Tobacco and bubble gum. Shows similarities between kids bubble gum and tobacco products; similar in packaging and in the way the product looks inside the package; note how the gum is shredded like the leafy tobacco product

Tobacco in all forms kills an estimated 440,000 Americans each year. Tobacco has also been associated with cancers of the mouth, pharynx, larynx, esophagus, stomach, pancreas, cervix, kidney, bladder, and acute myeloid leukemia. It has also been well documented that smoking substantially increases the risk of heart disease, including stroke, heart attack, vascular disease, and aneurysm. Globally the consequences are horrific. By the year 2020, it is estimated that 8.4 million people will die annually as a result of tobacco-related diseases [3, 4]. This is too important public health issue to ignore, and we need to arm all health professionals with information to combat the tobacco industry.

Tobacco use also has significant oral health sequelae as well. Due to ingredients in spit tobacco, there is a firm link to increased periodontal disease and some potential links to dental caries and oral mucous membrane disease from its use. Smoking is also strongly associated with severity of periodontal disease as well [4].

The team dentist or dental consultant to any sports league or team should know the basics of tobacco carcinogenesis and oral disease associated with tobacco use. Our profession needs to be a source of truth about the dangers of tobacco use in sports. This chapter will give the reader a basic understanding of these important issues. We will explore the history of tobacco in the world and in sports, followed by the physiologic and addictive properties of nicotine and the carcinogenic potential of tobacco.

## 11.2 Tobacco History and Relation to Sports

We begin this section by looking at a brief history of tobacco and its economic impact. Then we will review the types of tobacco products used and how they are used in the context of sport. Properties of tobacco and the ingredients that affect users will be reviewed next. We then will explore the newest delivery system for tobacco in the e-cigarette. Finally we will review the use of tobacco by young athletes. We will look in detail of the special case of tobacco use in baseball in the next section.

### 11.2.1 Tobacco History, Types, and Economics

Tobacco is a leafy plant that grows in warm climates. After harvest the leaves are dried and prepared for use in its multiple forms. The dried leaves can be smoked in a pipe or rolled in a cigar or in a cigarette, they can be chewed as in spit tobacco, or they can be sniffed through the nose as snuff. Nicotine is one of the many chemicals that are found in tobacco, and its presence makes tobacco an addictive drug, even though it is not regulated as such.

Tobacco has a long history of use in the Americas that began long before the Europeans came to the New World. The Mayans depicted tobacco use in their art from times before European exploration. Native Americans smoked tobacco in pipes for religious ceremonies and medical purposes. Today tobacco is used with great frequency throughout the world. The same qualities that encouraged Native Americans to use tobacco as a medicine and as a religious aid are the same qualities that encourage its use today. Today we recognize these qualities as physiologic and pharmacologic, and we also recognize the dangers of tobacco use today that were unknown to earlier users of tobacco.

Tobacco was the first cash crop grown in the New World after European settlement in the 1600s. Tobacco helped to finance the American Revolution against England, and George Washington was a tobacco grower [5]. By the 1800s many people were using small amounts of tobacco either smoking or chewing. Invention of cigarette making automation in 1881 allowed mass production of cigarettes, and smoking took off as an American pastime [5]. World War I and World War II solidified tobacco's grip on the world. Soldiers were given free cigarettes, and they consumed large quantities of a very addictive drug, this before the dangers of tobacco use were known and world tobacco addiction was set in motion [5]. Tobacco use would now be guaranteed for generations to come. Tobacco would be used by athletes since it was a way of life around the world and still the dangers of tobacco use were unknown or hidden. Smoking though was not convenient for athletes engaged in competition, and chewing tobacco became the vehicle of choice to deliver the tobacco drugs to athletes.

The financial health of tobacco companies are good even if the health of their customers is not.

Selling an addictive drug to the masses without regulation or medical intervention is a recipe for wild profit. Tobacco companies in the 1950s and 1960s made large profits, and they wisely diversified their businesses. Tobacco companies show profits in the billions of dollars each year, and they pay incredible amounts of taxes to governments that are strapped for cash. We expect to see tobacco companies to be around for years to come even though the numbers of people smoking continues to go down.

From here on the major focus will be given to the use of spit tobacco since this is the form most often used by athletes.

### 11.2.2 Properties and Ingredients of Spit Tobacco

Spit tobacco is the preferred form of tobacco used by athletes during competition. It requires no use of hands to deliver the drug to the athletes and requires no flame to burn leaves to create smoke to inhale. Contact of the tobacco product with a mucous membrane is how the chemicals are delivered to the user. Spit tobacco comes in a few forms. There are plugs or blocks of tobacco where a plug is bitten or cut off, leafy tobacco for chew and snuff which is moisturized finely ground tobacco. In the Far East, betel quids with tobacco and areca nut products are popular.

**Oral Use Tobacco Products: General**

1. *Chewing tobacco*: leafy rough cut ex. Red Man
2. *Dipping tobacco*: fine ground moist ex. Copenhagen
3. *Snus*: similar to dip; Scandinavia
4. *Snuff*: fine ground tobacco that is inhaled
5. *Betel quid with tobacco*: betel leaf and tobacco leaf
6. *Gutka*: betel leaf areca nut and tobacco

There are wide varieties in the ingredients of the betel leaf and areca nut product used mainly in Asia. Generally areca nut products are the most carcinogenic.

The type of tobacco used in a particular product will determine the chemical content of the product. The composition of the tobacco will depend on genetic makeup, growth environmental conditions, and all steps of processing and handling [6]. There have been over 3000 possible chemical components identified that may exist in different tobaccos [7].

**Possible Ingredients of Spit Tobacco**

Spit tobacco possible ingredients:

- Polonium 210
- *N*-Nitrosamines
- Formaldehyde
- Nicotine
- Cadmium
- Cyanide
- Arsenic
- Benzene
- Lead

There are at least 28 carcinogens identified in spit tobacco products; most are nonvolatile alkaloid-derived, tobacco-specific *N*-nitrosamines; and these compounds are normal ingredients of these tobacco products [8]. Another group of carcinogens in spit tobacco are polycyclic aromatic hydrocarbons (PAH). These chemicals originate primarily from polluted air and possibly from the fire curing of some tobaccos. Additionally, formaldehyde another known carcinogen is found in most spit tobacco. Formaldehyde is formed from natural occurring alkyl aldehydes during heating in the tobacco processing steps. Radioactive polonium-210 is also found in most tobacco and comes from the soil where the tobacco grows [6].

Nicotine, a highly addictive drug, is found in all forms of spit tobacco in high concentrations. Nicotine, like most drugs of abuse, stimulates the release of dopamine in the brain. Dopamine is the primary neurotransmitter involved in pleasure and reward [9]. Release of pleasure and reward transmitter dopamine makes this drug very addictive.

### Neurochemical and Related Effects of Nicotine

| | | |
|---|---|---|
| N I C O T I N E | Increase dopamine | Increase pleasure, reward |
| | Increase norepinephrine | Arousal, appetite suppression |
| | Increase acetylcholine | Arousal, cognitive enhancement |
| | Increase glutamate | Learning, memory enhancement |
| | Increase serotonin | Mood modulation, appetite suppression |
| | Increase β-endorphin | Reduction of anxiety and tension |
| | Increase GABA | Reduction of anxiety and tension |

Still there is no regulation of this drug from normal regulatory agencies. The FDA controls issues like sales of tobacco to minor children, forces warning labels on packaging, and controls some of the marketing of tobacco products. Not only is this drug very addictive, but it also exhibits many potent cardiovascular effects.

### Nicotine Pharmacodynamics

Central nervous system
- Pleasure
- Arousal, enhanced vigilance
- Improved task performance
- Anxiety relief

Cardiovascular system
- Increased heart rate
- Increased cardiac output
- Increased blood pressure
- Coronary vasoconstriction
- Cutaneous vasoconstriction

Other
- Appetite suppression
- Increased metabolic rate
- Skeletal muscle relaxation

These cardiovascular effects are likely the effects that give athletes the false sense of improved athletic performance after tobacco use.

### Myth: Spit Tobacco Can Enhance Athletic Performance

*Nicotine*
- Increases heart rate
- Increases blood pressure
- Causes arrhythmias
- Causes vasoconstriction which can slow reaction time and cause dizziness
- Decrease pituitary secretion of vasopressin which can adversely affect memory and learning

There may be very transient increases in performance followed by performance reduction. Addiction, carcinogenesis and vasoactive properties make tobacco a very dangerous product.

The stimulant effects of nicotine give athletes some sense of improved performance, some are mental and some are physical. The pharmacology of nicotine leads some researchers to pose the question of whether the use of nicotine constitutes legal doping in sport. Nicotine has been found in some studies to enhance information processing and attentional processes in human subjects [10]. Multiple studies have been done to test the hypothesis of improved physical performance from use of spit tobacco. These studies have shown no statistically significant increase in strength, power, or athletic skills while using tobacco products [1, 11].

## 11.2.3 E-Cigarettes

Electronic cigarettes are one of the newest tobacco products being used by people today. E-cigarettes work by using a battery to heat and atomize a nicotine-filled liquid into a vapor for the user to inhale or "vape." E-cigarettes are diverse in that they are sold in many different forms, from small disposable cigarette-style types to larger reusable devices. Within this new market, submarkets have also developed for modifying reusable devices. These include larger reservoirs for the liquid, different batteries and atomizers, and coils to heat dry substances, such as marijuana.

E-cigarettes gained popularity as nicotine delivery alternatives to smoking. E-cigarettes are marketed as aids for cigarette cessation and as tolerable in more social settings where cigarette use is not permitted. Cigarettes have developed a social stigma, whereas the same stigma does not yet apply to their electronic counterparts. One survey of young adults, average age 30 who reported using e-cigarettes, was aimed at assessing perceptions on e-cigarette usage. This study found that users perceived e-cigarettes to be an aid to decrease or stop smoking and that they are more socially acceptable than smoking a cigarette. Other so-called benefits include the abundance of flavors and the perception that they are healthier than cigarettes. When prompted as to the side effects of use, most common responses were coughing, throat irritation, and lightheadedness. This survey also found that many users were not only unsure of the long-term health risks but they also unsure of the ingredients in the liquid itself [12].

The liquid used for e-cigarettes is available in over 7700 flavors and contains "propylene or polyethylene glycol, glycerin, additives and nicotine" [13]. Once heated, the composition of the aerosol/vapor has been found to include "nicotine, ultrafine particles, tobacco specific nitrosamines (TSNAs) and other toxic compounds such as acetaldehyde, acrolein, and toluene, although at significantly lower levels than in cigarettes" [13]. These ultrafine particles, in higher concentrations, have been associated with cardiovascular and respiratory disease. No direct relationship between e-cigarette use and oral health consequences has been reported, but given that the liquid does contain high levels of nicotine, the possibility still exists.

E-cigarettes do pose a dental trauma risk as we have begun to see. According to the US Fire Administration, between 2009 and 2014, there were 25 reported incidents involving explosions and fires as a result of e-cigarette use [14]. Some of the most common injuries reported after an e-cigarette explosion include "intraoral burns, luxation injuries, and chipped and fractured teeth" [14]. These may seem insignificant, but serious accidents have occurred. These include formation of an antra-oral communication, spinal fracture, and respiratory tract burns. Eighty percent of these adverse outcomes transpired during use [14]. Furthermore, if an explosion does occur while in use, it is important to evaluate the patient for trauma to the head and neck region, possible concussion, and maintenance of a patent airway [15].

### 11.2.4 Prevalence of Tobacco Use by Athletes: High School, College, and Professional

Overall use of tobacco products continues to decrease over time according to the CDC. Combustible tobacco product use is declining in high school students overall, and high school athletes are less likely to be smokers than nonathletes to maintain physical fitness and optimize athletic performance. Athletes in high school who use tobacco generally use spit tobacco as it can be used discreetly during practice and games unlike combustible tobacco products which are discouraged by sports organizations. Some trends emerge as we examine spit tobacco use by high school age individuals. Spit tobacco use among high school males is about 14% of total. These users frequently use spit tobacco while playing or watching sports and the greater their involvement in sports, the greater their likelihood of spit tobacco use [16]. Peer pressure is a large part of this spit tobacco use in high school. Among a group of 995 adolescent football players, it was found that 30% of these athletes had tried spit tobacco and that of the players that had tried spit tobacco 60% cited friends' use as the major reason that they tried tobacco [16]. College years see increases in all types of tobacco use, and students experiment with substances previously not legally available to them [17]. The use of tobacco products by college athletes also is increasing. Much of this spit tobacco use in college is sport specific with baseball having the highest prevalence. Spit tobacco use in baseball is well documented (see ► Sect. 11.3). Spit tobacco use prevalence among college baseball athletes has been seen in the 45–55% range [18, 19]. Of the baseball athletes who use spit tobacco in college, nearly half of these players began their tobacco use during high school years [19]. From the data reviewed, it is found that playing sports in all levels increases the prevalence of spit tobacco use. As health-care professionals, we have a duty to offer cessation programs to assist athletes to break the habit of tobacco use. Interventions with cessation programs are effective and can double the likelihood that an athlete will stop using spit tobacco [20].

## 11.3 History: Baseball and Tobacco

Baseball, more than any other sport, has been associated with spit tobacco use. This is a short narrative of how tobacco made its way into the culture of baseball. As discovered earlier in the chapter, tobacco has a long and strong economic and use history in the USA from the 1600s on. Baseball rules were being formalized in the mid-nineteenth century, long before the carcinogenic properties of tobacco were discovered. By the late nineteenth century, the average American was consuming more than 3 pounds of tobacco annually [5]; tobacco had been firmly inserted into the culture of baseball. Spit tobacco was the choice of baseball players for obvious reasons; they could use tobacco on the field without using hands to hold a cigarette. Additionally, the baseball fields were not as well-groomed as they are today, and baseball games were very dirty and dusty. Spit tobacco helped the players with increased saliva flow, and an added benefit was that tobacco-laced saliva helped them lube their gloves to keep the leather more supple, and this aided in fielding. The early twentieth century found baseball the most popular sport in the USA, and every professional baseball team had a tobacco sponsor [21]. Bull Durham is one of the oldest brands of spit tobacco in the USA and was a frequent advertiser on outfield fences in many baseball parks in the Southern USA. It is thought that the term "bullpen," the place where relief pitchers warm up, was coined because of all the Bull Durham signs on outfield fences. The famous "spitball" used by many pitchers was aided by use of tobacco-induced saliva.

It is easy to see how spit tobacco made its way into the culture of baseball from the very beginning. It wasn't until the Surgeon General's report in 1964 that linked cigarette smoking with cancer did we know officially that tobacco contained carcinogenic chemicals [22]. It naturally followed that spit tobacco also contained many of the same chemicals and also could be carcinogenic as well [8, 23]. By the mid- to late twentieth century, tobacco was a dangerous part of baseball culture. Perhaps the most famous baseball player of all time Babe Ruth died of head and neck cancer. He was diagnosed in the mid-1940s with nasopharyngeal cancer. Nasopharyngeal cancer today is known to have two major causative agents: alcohol and tobacco. Babe Ruth was known to have been a zealous user of both of these products.

Over the years it has become well-known that tobacco is one of the most significant threats to human health that we know. There have been a number of attempts made to limit the use of spit tobacco in baseball. In 1993 minor league players, coaches, and staff were prohibited from spit tobacco use during games. The thinking behind this was that if minor league player's tobacco use declined the major league players, tobacco use would naturally decline, since most major league players spend time in the minor leagues. A new MLB labor deal in 2011 prevents the use of spit tobacco in professional baseball during pre- and post-game interviews. This agreement does not ban the use of tobacco during games as long as the tobacco can or pouch is out of sight. Finally in 2016, a new collective bargaining agreement prohibits all new major league baseball players from using spit tobacco.



**Baseball Rules Aimed at Spit Tobacco Use**

- 1993: Minor league players, coaches, and staff prohibited from spit tobacco use during games.
- 2011: MLB labor deal prevents use of spit tobacco during pre- and post-game interviews and requires that spit tobacco cans or pouches are out of sight during games.
- 2016: Collective bargaining agreement prohibits all new major league baseball players from using spit tobacco.

Baseball has made strides to limit the use of spit tobacco at all levels and help purge this habit from the culture of a great game.

## 11.4 Health Effects of Spit Tobacco Use

Spit tobacco contains nicotine like all tobaccos as well as many of the carcinogens found in smoking tobacco products. There is a difference in the delivery of these chemicals into the user's body. The chemicals are taken in by the oral mucous membranes and are not burned and taken into the lungs. This may alter some of the effect of spit tobacco products, but they are far from harmless. All tobacco products are highly addictive.

Smoking is well-known to cause multiple morbidities and mortalities from lung cancer to head and neck cancers to cardiovascular disease in those who use these products. The question to sports dentistry is if any of that risk transfers to those who exclusively use spit tobacco. Spit tobacco has been implicated in the past in increased mortality from cancers of the oral cavity, cancers of the digestive tract, and cardiovascular disease [24]. A number of studies have been done to look into these general health issues as affected by spit tobacco use [24–26]. These articles find that it is difficult, due to low use, to generate the statistical power necessary to make definitive statements of association between spit tobacco use and many of the above diseases. There appears to be weak or no link of spit tobacco use with digestive system cancers [25]. We will review the issue of spit tobacco and head and neck cancers in a later section. One area that seems to be significantly affected by spit tobacco use is in mortality due to cardiovascular disease (CVD) [26]. They estimated from their data that use of spit tobacco carried a 24% increased risk for mortality due to CVD which is very similar to the risk from secondhand smoke.

## 11.5 Oral Effects of Spit Tobacco Use

There are effects on oral health from the use of spit tobacco products. It is important for dentists and other health practitioners to understand how spit tobacco use affects the oral cavity. How this use may affect dental hard tissues and affect dental caries, how it affects the supporting structures of the teeth and its effect on periodontal disease, and finally how spit tobacco use affects the mucous membranes and its effects on malignancy in the head and neck region. The most common oral conditions found with the use of spit tobacco are gingival recession, mucous membrane hyperkeratosis, and dental staining (Fig. 11.2).

**Fig. 11.2** Dental effects of ST. Shows a number of the oral effects of ST use. Recession of the gingival tissue, attrition of the hard tissue, and leukoplakia. (Photo credit Mark Roettger DDS)

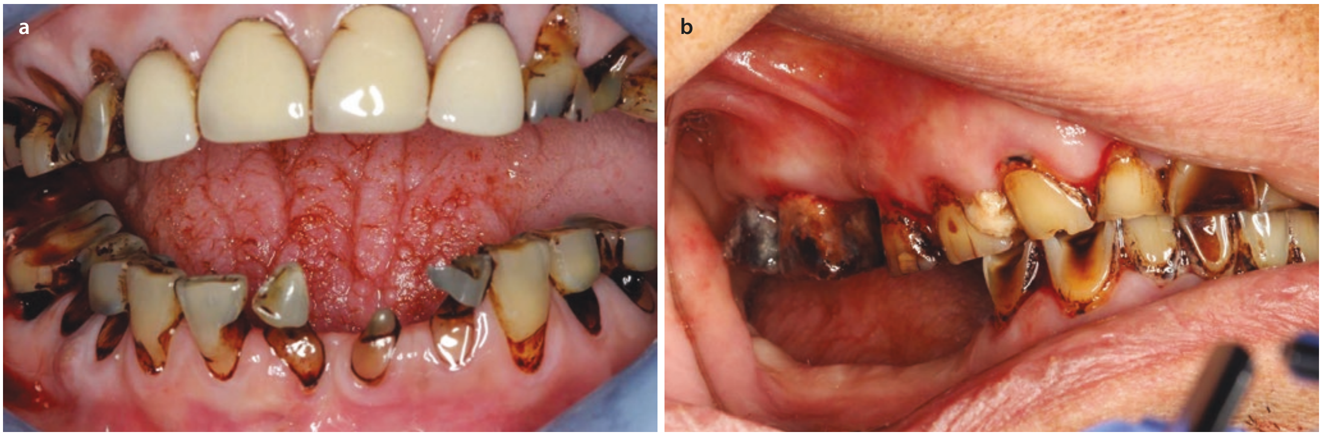



Fig. 11.3 Dental effects betel quid. **a** shows the dental effects of heavy use of betel quid with staining and erosion of tooth structure; note how the dental restoration crowns #7–10 have been spared. **b** shows a second patient that used similar products again with erosion and periodontal destruction staining of teeth and tissues

### 11.5.1 Dental Caries

There has been no strong link found between spit tobacco use and dental caries [4, 27]. It is known that long-term use of spit tobacco can hasten gingival recession and expose more root surface. There are varying amounts of sugar in the spit tobacco products, so there could be some caries increase as the use becomes more long term [28]. As the tobacco companies attempt to increase users of their products, there may well be more sugar added to make the flavor more appealing to a larger customer base to include new younger users (Fig. 11.3).

### 11.5.2 Periodontal Disease

Many studies have confirmed a link between smoking and periodontal disease [29]. There is mounting evidence for the mechanism of periodontal destruction from tobacco use [30]. Nicotine has a profound negative affect on the periodontal tissues. It affects the immune system and the response of the periodontal tissues to treatment modalities. Numerous studies have looked at the effects of tobacco use on periodontal health and report odds ratios for increased periodontal destruction on the order of 2–6 [31]. This data suggests a strong link between tobacco use and periodontal destruction.

A comprehensive review of the literature was done in the late 1990s that looked at the modifying effects of tobacco use on the immune and inflammatory response in humans [32]. This review verified the fact that tobacco users were 2.5–6 times more likely to have periodontal destruction than non-tobacco users. This review looked at the deleterious effects of tobacco on the host response to periodontal pathogens. Reviewed were the effects of tobacco on macrophages, neutrophils, natural killer (NK) cells, B lymphocytes, and T lymphocytes. All of these effects would make the host more susceptible to periodontal destruction. Most of this work has been done on smoking. Use of spit tobacco would be expected to have similar effects perhaps on a more localized area.

Periodontal disease progression is a multifactorial process that combines environmental-, host response-, genetic-, and host-related issues. This is a complex process, but it is clear that tobacco use hastens periodontal destruction.

### 11.5.3 Oral and Oropharyngeal Cancer

There is no doubt that tobacco contains multiple carcinogens and that smoking tobacco causes multiple forms of cancer from oral cancer to lung cancers. There is not a strong link between chewing tobacco and the development of oral and oropharyngeal cancers [33]. It is difficult to effectively study the links between spit tobacco use and head and neck cancers because of low prevalence of use, inconsistent definitions of exposures, and difficulties in controlling for concurrent use of combustible tobacco products [34]. Because the link between spit tobacco and the development of oral cancer is not as strong as it is with smoking tobacco, carcinogenic potential from the use of spit tobacco is associated with long-term use (Fig. 11.4). The use of spit tobacco among young people is growing. It has been estimated that in the USA, over 12 million people use spit tobacco, over 3 million of which are less than 21 years of age. Additionally, in the Southeastern USA where women use spit tobacco at higher frequency than in other parts of the country, there is a higher than expected rate of oral cancer in women along with higher mortality [4]. With spit tobacco use starting early, especially in young athletes, and dangers grow with long-term use, we as a professional and as members of the sports medicine team need to council young athletes that using chewing tobacco products holds potential future harm which may include cancer.

There are also stark differences in the carcinogenic potential in different spit tobacco products. Products used in Asia made from the areca nut appear to be more dangerous than tobacco products in other parts of the world. It has been estimated that around 10,000 annual deaths can be attributed to oral tobacco use in India [25]. American spit tobacco and Swedish snus appear to have decreased potential for causing oral cancers [33].

After long-term use of any oral tobacco product, there is a potential for the development of an oral neoplasm. The earliest change in the oral mucosa noted from the use of spit tobacco is leukoplakia or “white patch” (◘ Fig. 11.5). At this stage tissue damage is easily reversible, and tobacco use cessation can completely reverse any damage. Oral leukoplakia is a premalignant lesion in that there is a risk of malignant transformation. Continued tobacco use increases the risk for future development of squamous cell carcinoma [4]. Simply stopping the use of spit tobacco is sufficient to reverse the tissue damage from tobacco use. In cases where cessation does not reverse, the lesion chemoprevention strategies are being developed and studied. Chemoprevention agents are being developed to help high-risk individuals with precancerous




◘ **Fig. 11.4** ST-induced oral cancer. Shows a case of ST-induced oral cancer from a 20+-year ST habit; **a** shows the lesion clinically as it has come through the skin, the marker which shows the extent of the intraoral lesion. **b** shows the orthopantomogram of the lesion in the mandibular anterior region. **c** shows preparation for resection in the OR, again the skin marker shows the internal margins of the tumor. **d** shows the start of the resection as the surgeons resect to achieve “clean” margins. (Photo credit Bevan Yueh MD). **e** shows the resection continuing with mandibular saw cuts made. **f** Shows the resection about to be completed. It can be appreciated that these resections can be very extensive and disfiguring. Reconstruction can be done after resection. (Photo credit Bevan Yueh MD)


Fig. 11.4 (continued)

Fig. 11.5 Leukoplakia. Shows the type of leukoplakia that we may see in a young athlete using spit tobacco. Discontinued use of the tobacco products will usually result in complete resolution of the lesion. (Photo credit Mark Roettger DDS)

lesions to decrease the chance of malignant transformation. There are numerous agents that are being studied for use as chemoprevention agents. Some like the retinoids have been studied for many years, and some newer molecular-targeted agents are beginning to be tested.

**Chemoprevention Agents: Oral Cancer**

- Retinoids
  - Vitamin A
  - Ferentinide
- Beta-carotene
- Alpha-tocopherol
- Selenium
- NSAIDs
  - Cox-2 inhibitors
  - ASA
- Curcumin
- Green tea extract
- Polyphenols
- Blackberries
- Pioglitazone
- Molecularly targeted agents
  - EGFR-TKIs
  - EGFR-farnesyl
  - EGFR-EKB-569
  - Transferase inhibitors
- P53 gene
  - ONYX-015
  - Ad5CMV

The normal histologic progression of oral cancer is from normal squamous epithelium to epithelial dysplasia (moderate or severe) to frank invasive squamous cell carcinoma (Fig. 11.6). Clinically these lesions may all look similar, and it is impossible to diagnose by clinical examination alone. Suspicious lesions should undergo diagnostic procedures. Biopsy is the definitive diagnostic procedure for suspicious oral lesions. There are two types of biopsy techniques used in the oral cavity: *excisional biopsy* where the entire lesion is removed and *incisional biopsy* where a small but representative portion of the lesion is removed. For any lesion that has suspicion of malignancy, incisional biopsy should be done. Leaving a portion of the lesion intact will assist the surgeon in orientation if the results of the biopsy indicate malignancy. Wide local excision is the most common treatment for most oral tumors. This requires having clean (tumor free) margins around the entire lesion. If clean margins are not possible, or if there is local lymph node involvement or distant metastasis, radiation and chemotherapy may be used in the cancer management plan. Surgical resections of tumors on the oral cavity are many times extensive and disfiguring (Figs. 11.4 and 11.7). Five-year survival rates for oral and oropharyngeal malignancies still hover in the 50–60% range as we still too often get late diagnosis for these tumors. Prevention by tobacco counseling screening and surveillance is important in these malignancies.

## 11.6 Conclusion

The dangers of tobacco use are becoming more known with smoking being the worst and spit tobacco not as dangerous but still not harmless. When tobacco took hold in the sport of baseball, there was no knowledge of the dangers of tobacco use. Today there are efforts being made to discourage and stop the use of tobacco on the practice and game venues in most sports. Major league baseball as discussed above is a prime example. The future is likely to see a decrease in the use of all types of tobacco in sport. Health care providers who work with athletes and athletic teams must all be ready to counsel against the use of all tobacco products. These

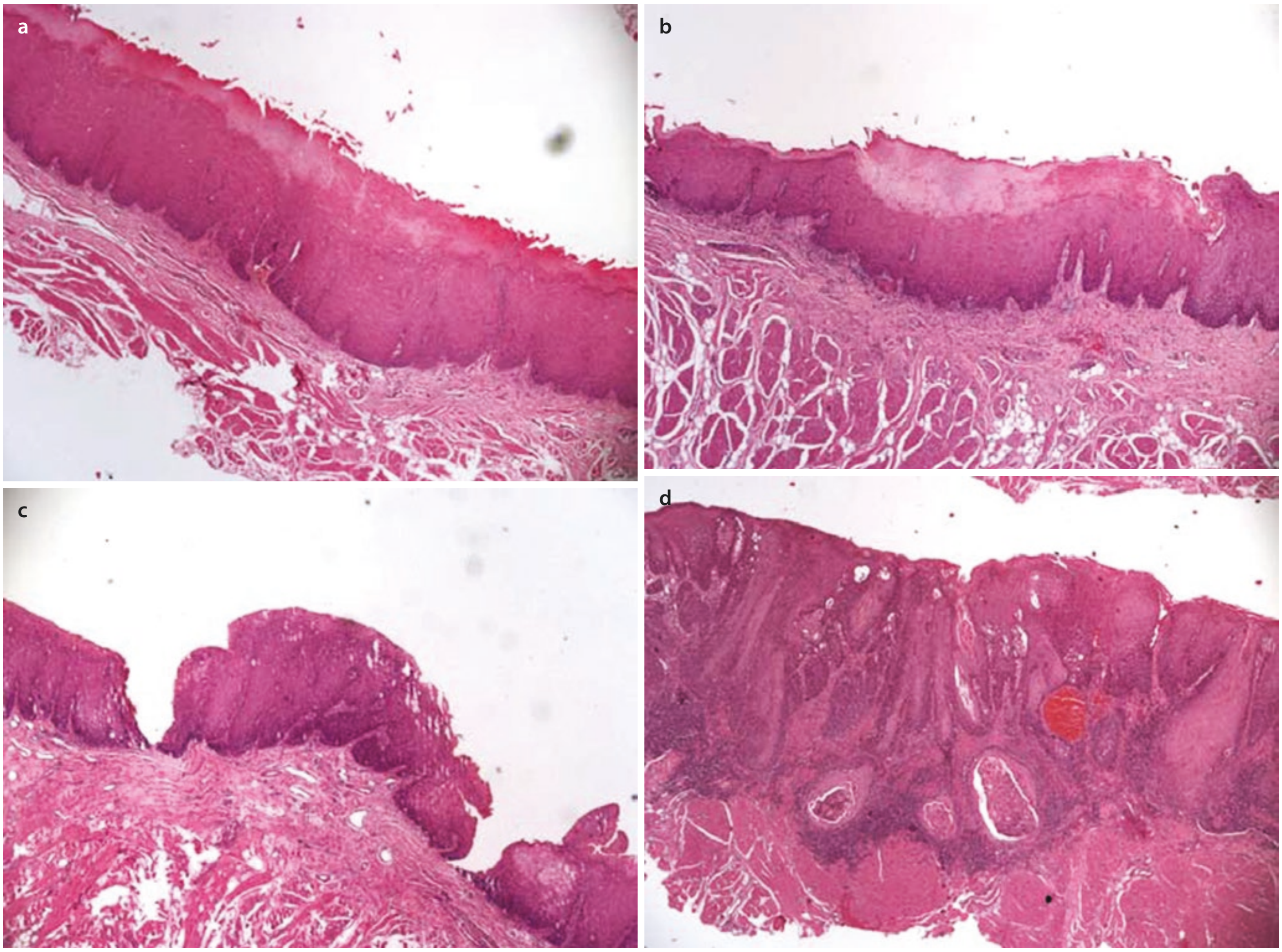



**Fig. 11.6** Histologic progression following a patient as their disease process from **a** mild dysplasia in June 2009 to **b** mild dysplasia with hyperkeratosis in May 2010 to **c** moderate to severe dysplasia in July 2010 and finally in **d** invasive squamous cell carcinoma in November of 2010. (Photo credit Mike Rohrer DDS MS)

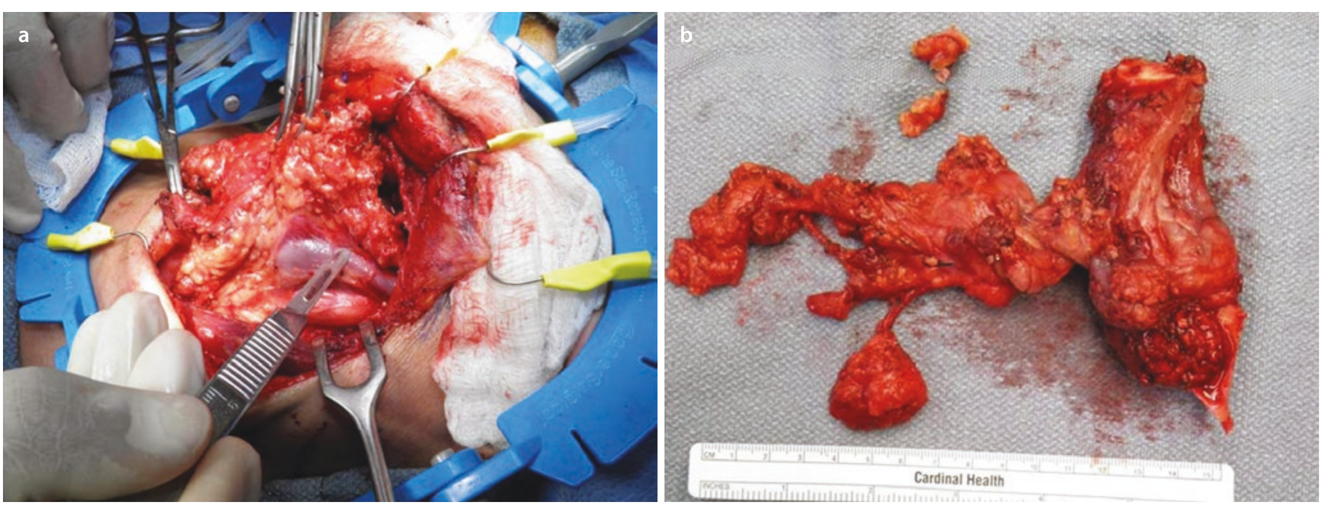



**Fig. 11.7** Neck dissection. Shows neck dissection surgery with the goal of eliminating involved lymph nodes. **a** shows the surgical procedure and **b** shows the nodes and tissues dissected from the neck which will be sent to pathology for histologic diagnosis. (Photo credit Priya Kamdar DDS)

providers should be able to cite peer reviewed literature to counter the performance enhancement claims of athletes who use tobacco products. They also must be able to talk with athletes on the addictive power of nicotine present in all forms of tobacco and the difficulties in cessation once the habit is begun. The future of sport is brighter as tobacco becomes a thing of the past.

## References

1. Robertson PB, Walsh MM, Greene JC. Oral effects of smokeless tobacco use by professional baseball players. Adv Dent Res. 1997;11:307–12.
2. Oreskes N, Conway E. Merchants of doubt. New York: Bloomsbury Press; 2010.
3. Abuse, National Institute on Drug. Tobacco nicotine. Bethesda: NIH; 2012. https://www.drugabuse.gov/publications/research-reports/tobacco/what-are-medical-consequences-tobacco-use.
4. Silverman S. Oral cancer. 5th ed. Hamilton: BC Decker; 2003.
5. Jacobs M. From the First to the last ash: the history, economics and hazards of tobacco. Cambridge: The Cambridge Tobacco Education Program; 1997.
6. IRC Working Group on Evaluation of Carcinogenic Risk to Humans, IRC Working Group. IARC monographs on the evaluation of carcinogenic risks to humans. Lyon: WHO; 2007.
7. Hoffman D, Hoffman I. The less harmful cigarette: a controversial issue. A tribute to Ernst L. Wynder. Chem Res Toxicol. 2001;14:767–90.
8. Brunemann KD, Cox JE, Hoffman D. Analysis of tobacco-specific N-nitrosamines in indoor air. Carcinogenesis. 1992;13:2415–8.
9. Domino E. Tobacco smoking and nicotine neuropsychopharmacology: some future research directions. Neuropsychopharmacology. 1998;18(6):456–68.
10. Wesnes K, Warburton DM. Smoking, nicotine and human performance. Pharmacol Ther. 1983;21:189–208.
11. Escher SA, Tucker AM, Lundin TM, Grabiner MD. Smokeless tobacco, reaction time and strength in athletes. Med Sci Sports Exerc. 1998;30:1548–51.
12. Coleman BN, Johnson SE, Tessman GK, Tworek C, Alexander J, Dickinson DM, Rath J, Green KM. It's not smoke. It's not tar. It's not 4000 chemicals. Case closed: exploring attitudes, beliefs, and perceived social norms of e-cigarette use among adult users. Drug Alcohol Depend. 2015;159:80–5.
13. Couch ET, Chaffee BW, Gansky SA, Walsh MM. The changing tobacco landscape. J Am Dent Assoc. 2016;147(7):561–9.
14. Harrison R, Hicklin D Jr. Electronic cigarette explosions involving the oral cavity. J Am Dent Assoc. 2016;147(11):891–6.
15. Brooks JK, Kleinman JW, Brooks JB, Reynolds MA. Electronic cigarette explosion associated with extensive intraoral injuries. Dent Traumatol. 2017;33(2):149–52.
16. Creath CJ, Bradley DH, Shelto WO, Wright JT, Feinstein RA, Wisniewski JF. The prevalence of smokeless tobacco use among adolescent male athletes. JADA. 1988;116:43–8.
17. Rigotti NA, Lee JE, Weschler H. US college students' use of tobacco products. JAMA. 2000;284(6):699–705.
18. Levenson-Gingiss P, Morrow JR, Dratt LM. Patterns of smokeless tobacco use among university athletes. J Am Coll Health. 1989;38:87–90.
19. Walsh M, Hilton JF, Ernster VL, Masouredis CM, Chesney M, Grady DG. Prevalence patterns and correlates of smokeless tobacco use in a college athlete population. J Addict Behav. 1994;19:411–27.
20. Walsh MM, Hilton JF, Ellison JA, Gee L, Chesney MA, Tomar SL, Ernster VL. Spit (smokeless) tobacco intervention for high school athletes: results after 1 year. Addict Behav. 2003;28:1095–113.
21. Blum A. Tobacco in sport: an endless addiction? BMJ. 2005;14(1):1–2. http://tobaccocontrol.bmj.com/content/14/1/1.full#xref-ref-2-1
22. American Cancer Society. Cancer facts and figures 2001. Atlanta: American Cancer Society; 2001.
23. IARC. Smokeless tobacco and some tobacco specific N-Nitrosamines. Lyon: WHO; 2007.
24. Accortt NA, Waterbor JW, Beall C, Howard G. Chronic disease mortality in a cohort of smokeless tobacco users. Am J Epidemiol. 2002;156:730–7.
25. Critchey JA, Unal B. Health effects associated with smokeless tobacco: a systematic review. Thorax. 2003;58:435–43.
26. Timberlake DS, Nikitin D, Johnson NJ, Altekruse SF. A longitudinal study of smokeless tobacco use and mortality in the United States. Int J Cancer. 2017;141:264–70.
27. Savitz DA, Meyer RE, Tanzer JM, Mirvish SS, Lewin F. Public health implications of smokeless tobacco use as a harm reduction strategy. Am J Public Health. 2006;96(11):1934–9.
28. Going RE, Hsu SC, Pollack RL, Haugh LD. Sugar and fluoride content of various forms of tobacco. J Am Dent Assoc. 1980;100:27–33.
29. Gonzalez YM, de Nardin A, Grossi SC, et al. Serum cotinine levels, smoking and periodontal attachment loss. J Dent Res. 1996;75:796–802.
30. Robertson PB, Walsh M, Greene J, Ernster V, Grady D, Hauck W. Periodontal effects associated with the use of smokeless tobacco. J Periodontol. 1990;61:438–43.
31. Kinane DF, Chesnutt IG. Smoking and periodontal disease. Crit Rev Oral Biol Med. 2000;11(3):356–65.
32. Barbour SE, Nakashima K, Zhang JB, Tangada S, et al. Tobacco and smoking: environmental factors that modify the host response (immune system) and have an impact on periodontal health. Crit Rev Oral Biol Med. 1997;l8(4):437–60.
33. Rodu B, Jansson C. Smokeless tobacco and oral cancer: a review of the risks and determinants. Crit Rev Oral Biol Med. 2004;15(5):252–63.
34. Wyss AB, Hashibe M, et al. Smokeless tobacco use and the risk of head and neck cancer: pooled analysis of US studies in the INHANCE consortium. Am J Epidemiol. 2016;10:703–16.

Check for updates

# Athletic Performance: Drugs and Ergogenic Aids

*Leslie Rye*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_12

## 12.1 Introduction

Appropriate therapeutic drug use goal for the athlete begins by knowing the athlete

**Goals for the Oral Healthcare Provider On Sports Medicine Team**

Primary Goal: Keep athletes ready to play, infection-free, and pain-free; avoid banned drugs contributing to drug addiction.

- Know your athlete and understand the drugs used to keep athletes infection and pain-free and the drug's influence on athlete's performance.
- Be knowledgeable about resource information for both therapeutically indicated and banned drugs.
- Understand performance-enhancing drugs (PEDs), banned dru gs, doping, and drug addiction.

and the athlete's profile.

**Athlete's Profile (Training/Competing)**

- Youth/adolescent/weekend warrior
- College
- Compromised/medically complex
- Special Olympics/Paralympics
- Adult recreational
- Professional
- Elite
  - Olympic level
  - World Tournaments

12

A thorough diagnosis and treatment of existing oral and dental conditions should be completed prior to training and competitions. Preseason oral exam contributes to reduced infections and pain, and need for therapeutic agents during competitive seasons. Early diagnosis of conditions allows timely treatment that may reduce problems during competition and training. The oral healthcare provider must know not only the therapeutic drug treatment for oral disease conditions of athletes, but they must also be familiar with the effects of these drugs on the athlete's ability to perform and possible contributions to addiction. Recommended treatment should keep the athlete infection- and pain-free.

**Athlete Ready to Play**

- Pain-free.
- Infection-free.
- Hydrated.
- Alert.
- No clotting/bleeding issues.
- *Keep within the rules.*

Athlete medical and dental treatment should not impair hydration or hemostasis. The drugs chosen for care should be used with awareness of addiction profiles and both the medical and drug history of the athlete. In addition, the provider should provide treatment that will not reduce alertness or interfere with sleep patterns during time of competition. All this should be done within the rules of the athlete's sport. Many sports have rules about drugs from their respective governing bodies as described further in the chapter.

As this chapter is reviewed, it is important to note that any recommended treatment or lists of banned substances can frequently change and should be checked often for updated protocols and information. This chapter describes acute care for the reasonable healthy adult competitor and recommendations of some, but not all indicated oral or topical therapeutic drugs, for acute conditions. Medically, cognitively, or physically compromised athletes require modification of therapeutic recommendations and close coordination with the team or personal physicians.

**Major Chapter Objectives**

Drugs

- Use: acute oral conditions
- ****** Special considerations for athletes
  - Misuse
  - Abuse
  - Addiction
  - PEDs (performance-enhancing drugs/substances/doping)
  - Drugs/doping
  - Useful references

## 12.2 Therapeutic Drug Uses to Control Oral Infection and Pain in the Competitive Athlete

Etiologies of acute conditions requiring dental care include infections, pain, autoimmune conditions, and trauma. Indicated anti-infectives, analgesics, and anti-inflammatory medications are used to treat these conditions. Many common oral conditions are listed in box "Acute Oral Conditions."

**Acute Oral Conditions**

- Infections
- Lacerations/fractures
- Bruxism/other facial pain
  - Ability to eat
  - Sensitive teeth
  - Headaches
  - Jaw and facial pain

Traumatic fractures of the teeth and jaws and lacerations of the oral area may require immediate emergency treatment followed with more definitive or long-term care and various drug therapies. Bruxism may be considered a long-term condition, but exacerbation of acute conditions such as pain while chewing, sensitive teeth, jaw and facial pain, and headache may require immediate drug therapy. Categories of medications for acute oral facial conditions include anti-infective agents, analgesics, anesthetics, muscle relaxants, and anti-inflammatory drugs.

**Medications for Acute Oral/Facial Conditions**

*Common Medications Used by Dentist to Treat*
- Infection
  - Anti-infective agents
  - Antimicrobials
  - Antivirals
  - Antifungals
- Pain
  - Analgesics and local anesthetics
  - Muscle relaxants (CNS depressants)
- Inflammatory lesions/ulceration
  - Anti-inflammatory or steroidal agents

Prescribers need to have knowledge about both the benefits and negative effects on athletes of indicated medications [1]. The long-term therapy of oral facial conditions is beyond the scope of this chapter.

### 12.2.1 Guidelines for the Anti-infective Agents for Common Bacterial, Fungal, and Viral Oral Pathology

Most commonly *oral infections* are acute conditions.

**Examples of Common Oral Infections**
- Pulpitis
- Pericoronitis
- Periodontal abscess
- Soft tissue lesions
  - Bacterial
  - Viral
    - Acute herpetic lesions
  - Fungal infections
    - Candidiasis
- Aphthous and traumatic ulcers (potential secondary infection)
- Traumatic lesion infections (lacerations/fractures)

The oral and topical anti-infective agents include antiviral, antifungal, and antibacterial.

#### 12.2.1.1 Management of Common Acute Head and Neck Bacterial Infections

Prescribing guidelines include selecting the simplest agent for effectively treating the infection. Common antimicrobials for these oral infections are listed in box “Antimicrobials—Rx: Systemic.”

**Antimicrobials**

Rx: Systemic
- Pen VK
- Amoxicillin
- Augmentin
- Clindamycin
- Erythromycin—xx
- Azithromycin
- Cephalosporins

Note that erythromycin is no longer an antimicrobial of choice due to its high incidence of GI distress. Use loading doses for antibacterial oral agents. This is generally double the standard dose for the initial dose.

**Antimicrobials**

Prescribing suggestions:
1. Loading dose (oral): double standard dose stat.
2. Stay simple.
3. Be alert to allergies.
4. Be alert to superinfections (i.e., fungal)
5. Be alert to resistance.
6. Be alert to side effects.

Review indications and contraindications before prescribing anti-infectives.

**Contraindications and Considerations**
- Hypersensitivity
- Side effects
  - GI: diarrhea/nausea
  - Yeast infection
  - Photosensitivity
  - Atypical effects
    - Tendon injury → ciprofloxin, levofloxacin
    - Cardiac arrhythmias → azithromycin, clarithromycin
    - Fatigue

Be alert to various allergic or adverse responses. Watch for superinfections, such as fungal infections, especially candidiasis. Monitor for poor responses, which may indicate inappropriate drug, or bacterial resistance. Be alert to side effects. Encourage athlete to report any of the above concerns immediately.

Hypersensitivity or prior allergic reactions should be reviewed in the medical history and reevaluated at time of treatment. Side effects of specific agents need to be reviewed in light of effect on athletic performance. Examples of side effects or adverse effects that might influence use or timing of the drug are diarrhea/nausea, drug-induced yeast infections, photosensitivity, and atypical effects.

Other contraindications include less common and atypical side effects from certain antimicrobials. Examples of cardiac arrhythmias may arise from use of azithromycin and clarithromycin. Atypical effects may include additional tendon pain or tendon damage as with fluoroquinolones (Levaquin®). Because of this, fluoroquinolones may be contraindicated for use in athletes. Dental care providers should also monitor for less common possible drug-induced fatigue or tiredness from some of these medications, such as amoxicillin, azithromycin, and ciprofloxacin. Further studies suggest fatigue may be related to illness or longer-term use of these drugs. Future studies may clarify any correlations between fatigue and certain antibacterial agents [2] (▶ https://www.nlm.nih.gov/medlineplus/druginfo/meds/a685001.html, ▶ https://www.nlm.nih.gov/medlineplus/druginfo/meds/a697037.html, ▶ https://www.nlm.nih.gov/medlineplus/druginfo/meds/a688016.html).

Additional bacterial skin infections of importance and frequency in athletes include *Streptococcus pyogenes* impetigo and methicillin-resistant *Staphylococcus aureus* (MRSA). These highly contagious infections are especially of concern in contact sports, with sporting equipment and contact exposure in locker rooms. Treating dentists must recognize these conditions especially on the face, and they should have knowledge of the use of appropriate agents. Small isolated impetigo lesions on the face may be treated with mupirocin (Bactroban®) topical ointment. If MRSA is suspected or more extensive lesions are noted, contact both the team physician and treating physician and athletic trainer.



### 12.2.1.2 Management of Common Acute Head and Neck Viral Infections

*Common oral viral lesions* may be treated both topically and systemically. Most common viral lesions are herpetiform lesions, which are most often caused by the herpes simplex virus (HSV) type I and type II. The athlete may have a history of frequent lesions found on the lips (herpes labialis), secondary to sun exposure or certain foods or drinks. To have the most effective reduction in pain or duration of lesions in patients with recurrent herpes lesions, they should be advised to recognize and start treatment at the first prodromal signs or prior to known etiology. Systemic treatment includes valacyclovir (Valtrex®). Topical treatment of small localized lesions includes use of penciclovir (Denavir®) [3–5].

**Herpetic/Viral Rx**

| Rx: systemic (Oral) | Rx: topical (oral) |
|---|---|
| – Valacyclovir (Valtrex®) 500 mg tablets | – Penciclovir (Denavir®) 1% cream |
| – Disp: 4 tablets | – Disp: 2 g tube |
| – Sig: take two tablets bid (12 h apart) | – Sig: apply a thin coat to affected area q2h for 4 days. Start at first sign of symptoms |


Fig. 12.1 Special concern with inhalers

### 12.2.1.3 Management of Common Acute Head and Neck Fungal Infections

*Fungal Candida* infections may arise secondary to antimicrobial use, inhaler use (Fig. 12.1), persistent dry mouth, or immune suppression conditions. Topical antifungals for localized oral lesions are the first line of defense, since systemic antifungals may have negative effects on liver function. Topical clotrimazole (Myclelex®) troches are effective, when used according to directions so as to maintain good contact with the effected oral tissue.

Another more common mixed fungal infection is seen in angular cheilitis. It may be seen more frequently in cold weather or climates. Topical treatment includes application of combination of clotrimazole 1% and betamethasone (Lotrisone®) cream. Combination topical nystatin and triamcinolone acetonide (Mycolog II®) cream to affected areas.

Other fungal lesions to observe in athletes include *tinea faciale* (ringworm). Again contact sports such as wrestling are a source of contagious spread of this fungal infection. Topical application of antifungal creams to small facial lesions is effective. Monitor for other lesions. Contact athletic trainer and treating and team physician with this information regarding contagious skin lesions.

### 12.2.1.4 Management of Common Autoimmune Mucosal Lesions

*Autoimmune lesions* that are painful or contribute to dehydration can be treated in acute phase with topical steroids. Simple and multiple aphthous ulcers can be painful and interfere with eating and drinking. Examples of topical ointment or gels include triamcinolone (Kenalog®) and 0.05% fluocinonide gel. For multiple intraoral lesions, dexamethasone (Decadron®) 0.5 mg/ml elixir may be indicated for topical effects. It should be used topically by rinsing, expectorating, and not swallowing to avoid systemic steroid effects.

These are considered weak corticosteroid agents and are not listed on the World Anti-Doping Agency (WADA) Prohibited List.

## 12.2.2 Management and Guidelines for Oral Analgesics (Adults)

Analgesics for oral pain can be selected based on severity of pain and expected duration of pain. Most oral/dental analgesic treatments are for short-term acute conditions.

The athlete's status for training, performance, or recovery should also be considered in devising pain management strategies. For all analgesics routinely use the lowest effective dose for the least amount of time to obtain pain control and reduce adverse and side effects. Always review with patient other analgesics he or she may be already taking in order to avoid overdosing or adding to addictive potential. Consult with lead team physician, if needed, to check for duplicate prescriptions in the case of opioid-containing analgesics.

**Pain Analgesics**

- *NSAIDS*
  - Anti-inflammatory
  - Platelet effects
- *Acetaminophen (APAP)*
  - Caution—dosage limitations
- *Narcotics/opioids*
  - Caution: head trauma and abuse potential
  - Sedation
- Combinations of the above

### 12.2.2.1 Analgesics for Mild to Moderate Acute Pain Management

Nonaddicting acetaminophen (APAP) and nonsteroidal anti-inflammatory drugs (NSAIDS) are first choices for mild to moderate acute pain control. NSAIDS have an additional anti-inflammatory effect.

*Acetaminophen* maximum dosage recommended by the Food and Drug Administration (FDA) for adults is 4000 mg/24 h.

**Maximum Acetaminophen Dose**

- Adult dose: 325–650 MG Q4–6H
- Maximum daily dose: 4000 mg/24 h
- The liver can only metabolize limited amounts before toxic metabolite builds up.
- Boxed warning:
  - FDA drug safety communication: prescription of acetaminophen products to be limited to 325 mg per dosage unit
  - Boxed warning will highlight potential for severe liver failure
  - FDA Safety Announcement 1-13-2011

Johnson & Johnson the producer of acetaminophen (Tylenol®) suggests not to exceed ten tablets of 325 mg (3250 mg) or six tablets of 500 mg (3000 mg) in a 24 h period. This dosage limit is due to the concern that individuals may be taking other drugs simultaneously that also contain acetaminophen. Combination drugs containing acetaminophen are limited to 300-325 mg per tablet. Athletes should be advised to avoid taking multiple acetaminophen-containing products together in order to avoid daily overdosing. Simultaneous alcohol consumption should also be discouraged, because of detrimental effects on the liver. Inform the athlete that severe, even life-threatening liver injury will occur when they exceed consuming above therapeutic doses. As with all medications, they should report any adverse effect during use of acetaminophen. The effect of acetaminophen on bleeding when consumed at proper dosage in otherwise healthy individual is minimal. If additional drugs are also needed to treat a condition, athletes should let all healthcare providers know if they are taking acetaminophen.

### 12.2.2.2 NSAIDS (Nonsteroidal Anti-inflammatory Drugs)

*Aspirin (ASA)* acts as an analgesic and an anti-inflammatory.

**Aspirin**

- NSAID
  - Analgesic
  - Antipyretic
  - Anti-inflammatory
- Antiplatelet effect: nonreversible platelet binding
  - Prolonged bleeding after injury
- Variable recommendations for delay prior to oral surgery
- Non-addicting
- Adult dosage: 325–650 MG Q4H

Dosage recommendations for adults: take 1 or 2 325 mg tablets every 4 h or 3 tablets every 6 h, but do not exceed 12 tablets in 24 h. Aspirin is also a nonreversible platelet-binding agent, which may contribute to prolonged bleeding after an injury. Bleeding or platelet effect gradually reverses itself as new platelets form over 10 days. This can be very significant in athletes since impaired clotting may lead to more bleeding in musculoskeletal injuries that are often seen in sports, especially hemarthrosis and deep tissue bruises. Recent studies recommend various time durations for delay of oral surgery procedures following ASA consumption [6, 7].

Contraindications of aspirin usage include but are not limited to pregnancy, breastfeeding, allergy history, gastric ulcer, asthma and nasal polyps, drug interactions, concomitant blood thinners, defects in blood clotting system, active peptic ulcers, and compromised renal conditions. An important side effect presents as gastrointestinal distress. There are many drug interactions with ASA, and the list should be checked before using ASA. Interactions with aspirin can be of major significance in individuals taking other medication that interfere with clotting mechanisms such as clopidogrel (Plavix®) and warfarin (Coumadin®). Alcohol should be avoided while taking ASA to avoid increased chance of gastric bleeding. Additionally, other NSAIDS and steroids may also interact negatively with ASA in some individuals. Always check for other aspirin-containing medications that the athlete may already be using. The prescriber should also be aware of the athlete who may be taking low dose aspirin to reduce probability of cardiovascular disease. This is often under reported by the patient [8].

#### 12.2.2.3 Non-ASA NSAIDS

Non-ASA NSAIDS include but are not limited to over-the-counter (OTC) ibuprofen (Advil®), naproxen sodium (Aleve®), and prescription etodolac (Ultradol®) which act as analgesic and anti-inflammatory agents. Usage of these NSAIDs contribute to anticlotting effects similar to ASA; however this category of NSAIDS has a reversible effect on platelets and therefore a reversible effect on bleeding when the drug is discontinued.

**Non-ASA NSAIDS**

- Reversible effect on platelet binding
  - This is very different from ASA.
- Antipyretic
- Analgesic
- Non-addicting



Dosage information is seen in box "NSAID Analgesics."

Dosage guidelines suggest using the lowest possible dose of ibuprofen for the least possible time to accomplish adequate pain control and minimize adverse and side effects. Continue to monitor the patient, and modify dose and use for shortest duration as appropriate for pain control.

Contraindications to NSAIDS include evaluating athlete's history for allergies, asthma with nasal polyps, pregnancy, breastfeeding, concomitant blood thinners, defects in blood clotting system, active peptic ulcers, and compromised renal conditions.

**When Are NSAIDS Not So Great**

Drug interactions

- Especially lithium
- SSRI (class of antidepressants)

NSAIDs cannot be used (are contraindicated) in the following cases:

- Allergy to ASA or any NSAID
- Some asthmatics—especially with nasal polyps
- During pregnancy and during breast feeding
- Concurrent with other anticoagulants
- Suffering from a defect of the blood clotting system

Duration of use is important in anticipating peptic ulcers or "delayed healing." There are increasing numbers of reviews correlating some delay in healing of soft tissue and perhaps bone secondary to the reduction in the inflammatory process of these drugs. It still needs to be clarified whether dosage, duration, or genetics are factors in these possible adverse effects [9–11].

There is a black box warning initiated in 2005 by the Food and Drug Administration.

**NSAID Black Box Warning**

- NSAIDs black box warning for both prescription and OTC products in the USA. FDA has requested that sponsors of all nonsteroidal anti-inflammatory drugs (NSAIDs) make labelling changes to their products. The FDA has recommended label changes for both the prescription and over-the-counter (OTC) NSAIDs and a medication guide for the entire class of prescription products. All sponsors of marketed prescription NSAIDs, including Celebrex (celecoxib), a cyclooxygenase-2 (COX-2) selective NSAID, have been asked to revise the labelling (package insert) for their products to include a boxed warning, highlighting the potential for increased risk of cardiovascular (CV) events and the well-described, serious, potentially life-threatening gastrointestinal (GI) bleeding associated with their use. The agency based its advice on a review of the regulatory histories and databases on the various NSAIDs. *Reference:Drug Information Page. United States Food and Drug Administration, 16 June 2005 (http://www.fda.gov).*

Other adverse effects include but are not limited to renal, cardiovascular, hepatic, and respiratory function.

Use of *acetaminophen-NSAID combination* agents continues to be evaluated. Publications support their combined use as another method of acute pain control [12–15]. Potential success of this combination would contribute to reduced prescriptions for opioid and opioid combination drugs.

**Case Study**

**NSAID Analgesics Examples (Mild to Moderate Pain)**

| | | |
|---|---|---|
| Ibuprofen (OTC) | Motrin/Advil (200 mg) | 400–600 mg Q4H |
| Naprosyn (OTC) | Aleve® (220 mg) | 1 unit Q6–8H |
| Etodolac (RX) | Lodine® (200–400 mg) | 1 UNIT Q6–8H *MAX 1000 mg/day |

#### 12.2.2.4 Analgesics for Moderate to Severe Acute Pain Management

Opioids and opioid combination drugs are indicated for moderate to severe acute pain control. Opioid-containing analgesics are considered banned substances by some sporting organizations, and their use is limited in these cases.

**WADA Prohibited In-competition Opioids**

| |
|---|
| Fentanyl and its derivatives |
| Hydromorphone |
| Methadone |
| Morphine |
| Oxycodone |
| Oxymorphone |
| Pentazocine |
| Pethidine |

Side effects are dose-dependent. Therefore lower doses express fewer or less severe side effects. The most common side effects and adverse effects are dizziness, sedation, nausea, vomiting, and constipation. Other important adverse and side effects one should monitor include respiratory depression, tolerance, and addiction.

Addictive potential is of great concern for all athletes and their healthcare providers. Opioid chemical addiction is related to multiple factors. The varied opioid receptor sites in the brain and nervous system include sites for analgesia (kappa and delta), sedation (kappa), and euphoria along with respiratory depression and reduced GI motility (mu). These receptors and others contribute to several other effects. With prolonged use of these opioid drugs, tolerance develops, and higher dosages are required to obtain the same effects of pain relief and euphoria, which also contributes to abuse and addiction.

The team dentist who prescribes opioid medications for dental or oral pain in athletes must be aware that these patients may be taking opioids for other concurrent injuries and should prescribe accordingly to minimize contributing to overdose, abuse, and addiction. Misuse of opioids is correlated with several different individual profiles. Pain, concussions, and concurrent alcohol use correlated with misuse in an NFL player study [16]. Misuse of opioids by adolescent athletes is higher in those with a current history of substance abuse [17].

In the USA, any opioid-containing agents fall into Schedule II DEA classification, because of their abuse potential. This requires written prescriptions without refills. Emergency prescriptions must follow state prescribing regulations. Some sports medicine teams request that all opioid prescriptions go through one provider for their athletic team. This provides for better monitoring of multiple opioid prescriptions and their contributions to misuse and overdosage.

#### 12.2.2.5 Opioid Combination Drugs

Also for moderate to severe pain, acetaminophen or NSAID combination with opioids has the same issues as each of these agents creates independently. The combination agents allow for lower doses of opioids and therefore less adverse opioid effects for equivalent pain control.

**Case Study**

**Opioid Analgesics Examples (Moderate to Severe Pain)**

| | | |
|---|---|---|
| APAP (300mg)* + codeine (30 mg) | Tylenol 3 | 1 tab Q4H *MAX APAP 4000MG |
| Hydrocodone (5 mg) + APAP (300 mg) | Vicodin® | 1–2 tab Q4–6H; do not exceed 8 tablets per day |
| Hydrocodone (7.5 mg) + APAP (300 mg) | Vicodin ES® | 1 tab Q4–6H; do not exceed 5 tablets per day |
| Hydrocodone (2.5 mg) + ibuprofen (200 mg) | Vicoprofen ® | 1 tab Q4–6H; do not exceed 5 tablets per day; use less than 10 days |

The same concerns about addiction exist for the opioid combination drugs as the individual opioid analgesics. Same guidelines for selecting analgesic dosage apply for opioid-containing drugs and recommend to routinely use the lowest effective dose for the least amount of time to obtain pain control and reduce adverse and side effects

#### 12.2.2.6 Concomitant Use of Other Addictive Drugs

Athletes, especially those with frequent travel schedules through time zones such as younger and professional athletes, may be using hypnotic drugs to combat disrupted sleep patterns. Zolpidem (Ambien®) and eszopiclone (Lunesta®) belong to a class of hypnotic drugs to treat insomnia. They are not currently banned by the World Anti-Doping Agency (WADA). These drugs also have an addictive profile. Dentists must be aware that the athlete may be taking these drugs, which add to the addictive and depressive potential, when they are used alone or in combination with opioid-containing drugs.

### 12.2.3 Local Anesthetics for Analgesia

Local anesthetics are used for pain control during dental procedures and for short-term pain control, when definitive care may be delayed.

**Case Study**

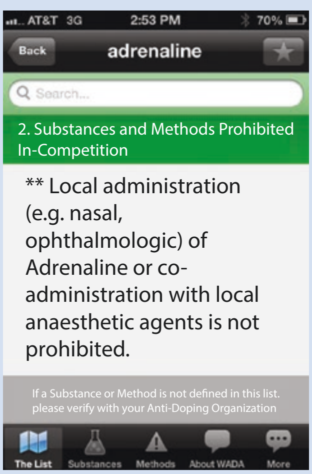
**Local Anesthetics (Duration of Pulpal Anesthesia)**

| Local anesthetics | Duration of pulpal anesthesia (infiltration) (approximate values) |
|---|---|
| 2% lidocaine | 5 min |
| 2% lidocaine plus 1:100,000 epinephrine | 60 min |
| 3% mepivacaine | 30 min |
| 4% articaine plus 1:100,000 epinephrine | 60 min |
| 4% articaine 1:200,000 epinephrine | 60 min |
| 0.5% bupivacaine Plus 1:200,000 epinephrine | Up to 7 h |

Reference: ► http://www.dentalanesthesia.com/pdfs/LA_ADA_PainPosterFront.pdf

**Case Study**

**Local Anesthetics *according to WADA(2015)**
Lidocaine w/ and w/o epinephrine*
Carbocaine
Articaine w/ and w/o epinephrine*
Duration of action
§*WADA Permitted—2015




The commonly used local anesthetics, including those combined with epinephrine, are not banned during elite athletic competitions governed by the World Anti-Doping Agency (WADA: website ► www.wada-ama.org/) policies. (WADA LIST-2015)

## 12.3 Drug Sources Available to the Athlete

Within the multidisciplinary sports medicine team, several prescribers may be prescribing drugs to the athlete. The physician, dentist, athletic trainer, physical therapist, and psychologist may be a therapeutic drug provider. In addition to the sports medicine team, the athlete may be receiving drugs from family, friends, team members, and street drug vendors, overseas, and by way of the Internet and through social media contacts.

**Other Drug Sources Used and Abused by Athletes**

- Rx by dentist
- Rx by physician
- Dispensing by team athletic trainers
- Over the counter (OTC)
- Herbals/supplements
- Performance-enhancing substances
- Illegal drugs
- *Online ordering
- Unknown contaminants (in supplements)

Use of excess drugs from multiple prescriptions is a common source of additional drugs. Sports medicine healthcare providers who manage athlete's pain must closely monitor the amount of addictive drug prescribed for the athletes and communicate with other members of the medical team in order to use the minimum amount of drug to effectively manage the pain and to minimize "leftover drugs" that may be shared with team members.

Addictive, performance-enhancing, and banned drugs may also be obtained through herbals, supplements, illegal drugs, and unknown contaminants. Drug interactions, banned substances, and side effects can also contribute to overdose and addiction.

**Additional Drugs in the Mix**

- Drug interactions
- Banned substances
  - Some may be illegal.
  - Some illegal substances not banned.
- Side effects
  - Performance impairment
  - Medical compromise
- Multiple drug sources
  - Overdose
  - Addiction

*Drug addiction* and abuse is a major concern in today's sports arena. The sports medicine team's contributions to long-term drug problems come from lack of oversight, multiple sources of addictive drugs, and pain/depression profiles developed because of chronic pain from injuries and concussions. This includes abuse of alcohol and illegal drugs. Specific psychiatric and psychological aspects of substance abuse and addiction are beyond the scope of this text but play a major role in their development. Members of the sports medicine team are encouraged to consult with those healthcare providers experienced and trained in the treatment of addiction and substance abuse. Proper drug prescribing by the team or sports dentist can contribute to helping reduce this severe abuse pattern in some athletes [18, 19].

## 12.4 Performance-Enhancing Drugs/Substances and Doping

### 12.4.1 Doping

Doping is defined by the use and misuse of substances banned by sports governing bodies and anti-doping organizations or methods that may enhance athletic performance. Major organizations governing doping regulations are the World Anti-Doping Agency (WADA), US Anti-Doping Agency (USADA), and National Collegiate Athletic Association (NCAA). Doping includes pharmacologic doping with performance-enhancing drugs and substances (PEDs).

**Performance-Enhancing Drugs/Doping**

- *Innocent-banned substances*
  - In OTC preparations
  - In herbal preparations
    - As contaminant
    - As metabolic breakdown product

In addition, doping includes blood transfusions and artificial oxygen carriers such as modified hemoglobins, which increase oxygen levels.

### 12.4.2 Performance-Enhancing Drugs/Substances (Referred to in This Chapter as PEDs)

The rationale for use of performance-enhancing drugs (PEDs) varies among athletes.

**Performance-Enhancing Drugs/Doping**

Athlete's rules to follow:

1. Avoid prohibited/banned drugs and substances/doping.
2. Avoid drug masking.
3. Use only permitted dosages.
4. Verify that all substances are known and permitted for use.

Several surveys of athletes in many age ranges and skill levels indicate the drive to win and succeed in sport and image are among the many psychological factors influencing the use of PEDs. The lure of awards and money drive some. Image, social acceptance, and influence of peers may drive others to use PEDs. Concurrent substance abuse is also a consideration. For athletes, doping's lure is to improve skill strength, endurance, and recovery specific to their sport [20–23]. Correct data on use of PEDs is difficult to obtain, since most studies evaluate one drug, one sport, and one age group or gender and make good evaluations across the board difficult [23].

These specific desires drive the athlete's search for enhancement of energy bursts, endurance, strength, muscle mass, and improved recovery by using performance-enhancing drugs. Some PEDs are consumed to influence and effect weight control, focus, attention, and recovery time. Additionally, some of these agents increase pain masking and general competitiveness. Use of PEDs is found in a wide range of age and skill levels including but not limited to adolescent athletes and elite competitors. Efforts to test and survey for the use of PEDs continue throughout the sports world [24, 25].

Examples of PEDs cross a wide spectrum of drug categories.

**Categories of Performance-Enhancing Drugs (PEDs)**

| Examples | Examples |
|---|---|
| AAS—anabolic androgenic steroids | Non-AAS ergogenic agents |
| | – Human growth hormone |
| | – DHEA |
| Herbals (ephedra) | Stimulants |
| Supplements (creatine) | Insulin |
| Laxatives | Beta blockers |
| Caffeine | Diuretics |
| Nicotine | Illegal drugs |
| NSAIDs | Erythropoietin-stimulating agents |
| Alcohol | Combinations of the above |

Each of these categories' effects may add to one or more aspects of performance enhancement as listed in box "Performance-Enhancing Drugs/Doping." Many substances are believed by athletes to increase performance levels or mask use of performance-enhancing substances. Although measurements of the actual benefits are not well-supported by quality research, the probable mechanisms of many PEDs are described below.

One of the most common drug categories that test positive is the group of *anabolic adrenergic steroid (AAS) agents* [26, 27].

They include testosterone, testosterone analogs, and designer steroid drugs. Testosterone analogs are drugs that mimic the chemistry and physiologic function of testosterone. Testosterone is a hormone that is naturally found in the body and along with several of the testosterone analogs is used for specific medical care. Androgenic refers to male sex characteristics, and anabolic refers to muscle-building ability. These AAS agents are taken to enhance muscle mass, image, and performance. These agents are not approved for performance enhancement in sports. In addition to use as PEDs, strong psychosocial factors contribute to the initiation of androgenic steroid use [28].

These androgenic anabolic steroids (AAS) are chemicals that attach to specific steroid muscle receptor sites triggering a cascade of protein synthesis, which contributes to increased muscle mass. Besides increasing muscle mass, anabolic steroids may help athletes recover from a hard workout more quickly by reducing the muscle damage that occurs during the session. This enables athletes to work out harder and more frequently without overtraining.

Some athletes use testosterone to boost their performance. Synthetic modifications of testosterone known as testosterone analogs or designer steroids are the adrenergic anabolic agents that are more frequently used by athletes. Designer steroid agents are illegal and created to (1) duplicate the physiologic effects of androgenic anabolic steroids and (2) to avoid detection by current doping drug tests. Several different forms may be taken together in what is called a "stacking" protocol. They may also be taken in a "cycling" protocol where the steroid agents are taken for time periods around performance, stopped, and then started again. Pyramiding combines the cycling and stacking [29].

Antiestrogen drugs such as *Tamoxifen®* can be used to mask synthetic testosterone from binding to receptors. High doses of synthetic testosterone (testosterone agonists) can increase estrogen production in the body. *Tamoxifen®* can block the estrogen receptors and then can hide or mask the effects of the extra testosterone-like drugs.

Stimulants are agents that simulate natural endogenous adrenaline (epinephrine) and norepinephrine. The physiologic effects are increased alertness, reaction time, concentration, sense of increased energy, and a decreased appetite. Any of these effects may be considered contributions to specific performance or fitness for sport training and competition. These are banned on the WADA list. Other types of stimulants include *caffeine* and *nicotine* and *some herbals* and supplements such as *ephedrine.*

**INNOCENT BANNED SUBSTANCES**
**in**
**DRUGS/DOPING**

- In OTC preparations
- In herbal preparations
  - As contaminant
  - As metabolic breakdown product
  - Some foods

**Fig. 12.2** Banned substances that may be overlooked

*Human growth hormone* (HGH) has a gonadotrophic effect resulting in increased muscle mass and muscle repair. *Insulin* also leads to increased muscle mass along with increased glycogen as muscle fuel, which may influence endurance [26].

*Beta-blocking agents* block the adrenergic beta receptors in the cardiovascular and respiratory system. This may result in a calming effect and slower heart rate by blocking the normal effects of adrenaline (epinephrine) on these receptors.

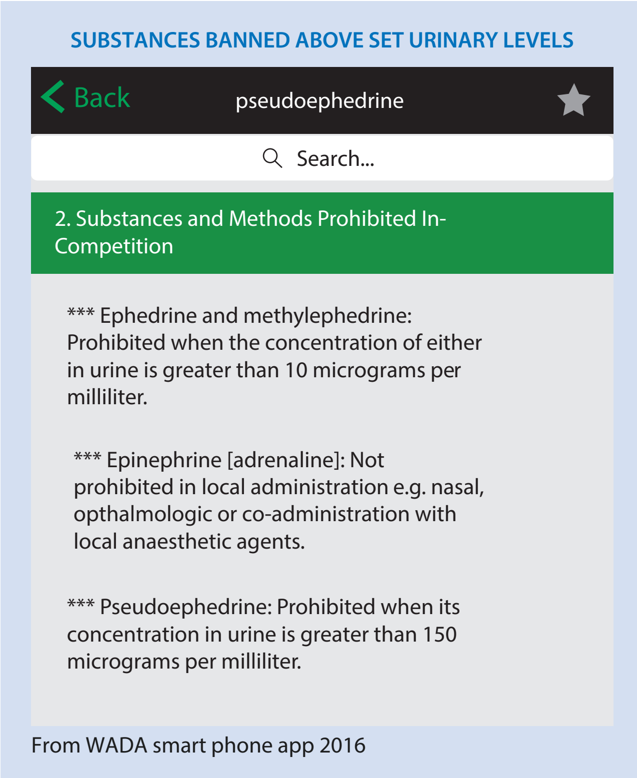
NSAIDS are analgesics, which can delay perception of pain or soreness. This would allow continuation of activity by the athlete when injured.

*Laxatives* increase bowel movements and may be used for weight loss. *Diuretics* result in increased urine excretion. This may also contribute to weight loss. It is also a way to dilute and eliminate banned drugs and mask them from detection in the urine.

*Creatine* is made in the body and is also available in some foods and as a supplement. Creatine contributes to increased contraction power by increasing the body's production of adenosine triphosphate (ATP), which is an energy source for muscle cells. It is thought to contribute to power by quick and short bursts of energy.

Erythropoietin-stimulating agents (EPA) such as erythropoietin (EPO) increase the production of red blood cells. Increased levels of red blood cells allow for more oxygen binding and increased oxygen available for muscle tissue. This additional oxygenation may contribute to more endurance and faster recovery [28].

*Inadvertent doping or innocent-banned performance-enhancing drugs* can be found in OTC preparations and in herbal preparations and some foods as a contaminant or a metabolic breakdown product (Fig. 12.2). Inadvertent doping occurs when an athlete uses a therapeutic drug to treat an illness or consumes food, drink, or OTC preparations without realizing that it also contains a banned substance.



From WADA smart phone app 2016

**Fig. 12.3** Example of phone app infrormation to assist providers who treat elite athletes

Examples might include cold or sinus medications and untested dietary supplements. An example is consumption of pseudoephedrine in cold and allergy preps. Even though the athlete may not be taking the drug to enhance performance, when they are actively participating in competitive sport, they are at risk of testing positive for a prohibited substance or for banned levels of a substance.

*Drug masking* is taking a drug or drugs to hide the existence of a banned drug from urine or blood testing. Diuretics are an example. Diuretics may be used to flush a banned or illegal substance from urine prior to time of testing.

*Drug urinary level limitations for accepted drugs*: For athletes falling under WADA or NCAA regulations, some accepted drugs may be banned when blood or urine levels exceed certain limitations. An example is pseudoephedrine, which is not on the WADA (2015) banned list. However it is not acceptable above 150 mg/ml in the urine (Fig. 12.3). In the case of caffeine, it is not banned by WADA (2015) but is a monitored drug in 2018. For NCAA regulations, caffeine is not allowed in urinary levels above 15 mcg/ml.

*Gene doping* is the use of genetically modified cells or nucleic acids or their analogs to enhance performance (WADA definition). As gene therapies develop, the possibility of using these procedures for performance enhancement will contribute to sophisticated doping [30].

### 12.4.3 National and International Organizations Governing Doping in Sports

A sports governing body has many functions including the following:

1. Upholding the rules of the sport and punishing those who break the rules (fining and banning teams and clubs and athletes)
2. Promoting the sport to attract new players and spectators
3. Organizing competitions (such as the World Cup, championships, college championships, and running the national teams)
4. Fight against doping and corruption in sport (▶ http://www.teachpe.com/gcse_society/governing_bodies.php)

Many governing bodies fall under the International Olympic Committee (IOC), and in the USA, many college sports fall under the National Collegiate Athletic Association (NCAA).

Sports governing bodies coordinate with anti-doping organizations and drug regulations to reduce doping and the use of PED substances in sports by athletes: both athletes and prescribers have rules to follow to maintain doping-free sport competition. Both athletes and prescriber must be familiar with banned substances for a sport, avoid drug masking and inadvertent doping, and stay within permitted drug dosages. Drug testing for PEDs is more commonly done with urine and blood level testing. Because of harmful effects of some legal drugs, some suggest monitoring these levels also [1].

Prescribers should be alert to misuse and PEDs in addition to considering preventive measures for and education of athletes. Testing for specific substances has become more advanced as doping has become more sophisticated [31]. Student PED drug surveys expose incomplete conclusions and legal issues [32].

### 12.4.4 Anti-doping Organizations

The *World Anti-Doping Agency (WADA)* was established in 1999 (Fig. 12.4).

WADA -- World Anti-Doping Agency
WORLD ANTI-DOPING AGENCY
play true

It is an international and independent agency whose mission is to support drug-free sport. The organization's goal is to bring consistency to anti-doping policies and regulations

WADA -- World Anti-Doping Agency

WORLD ANTI-DOPING AGENCY
play true

<u>The Prohibited List</u> Provides 3 areas of information

1-Substances and Methods Prohibited at all times
2-Substances and Methods Prohibited in-Competition
3-Substances Prohibited in particular sports

**Fig. 12.4** Levels of substance prohibition



across the world through governing sport bodies. Of importance to the prescriber for elite athletes is the annual *Prohibited List*. This allows the prescriber to search for drugs that are banned in particular sports, in competition, or out of competition. As part of the sports medicine team, a dentist should be aware if the athlete and his or her sport are covered by a governing body or affiliation to *WADA*. As stated and described on the WADA*)* website (▶ www.wada-ama.org.org), "The World Anti-Doping Code is the document that brings consistency to anti-doping rules, regulations and policies worldwide." Drugs, substances, and methods of performance enhancement are described in several categories by WADA.

According to the WADA website (▶ www.wada-ama.org), a substance can be placed on the Prohibited List "According to the Code, if a substance or method is found to meet two of three following criteria: enhances performance, poses a threat to athlete health, violates the spirit of sport. The Prohibited List includes the following: those substances and methods that are banned or prohibited from use both in and out of competition, prohibited in competition only, and prohibited in particular sports. The list is updated annually. Some drugs are banned only when their urine or blood levels exceed an indicated concentration. Monitored substances: some substances are being monitored for potential performance enhancement abuse, and they may be banned at a later date. Monitored drugs are currently not prohibited, but the list*)* must be checked annually for possible movement of these monitored substances to the Prohibited List.

Therapeutic Use Exemption substances (TUEs): These are drugs that may be banned; however they are approved for use through therapeutic use exemption applications. These are specified drugs used by athletes to treat specific diagnosed illnesses. WADA works with the standards of International Standard for Therapeutic Use Exemptions (ISTUE) to ensure uniformity between sports and countries in the process of granting TUEs. TUE Physician Guideline documents are created by the TUE committee working with WADA and international standards for TUE.

The Prohibited Methods List is divided into three categories: manipulation of blood and blood components, chemical and physical manipulation, and gene doping.

If a substance is not listed, athletes and healthcare providers should check with the sports governing body to verify allowed use of the drug in sport.

*The US Anti-Doping Agency* (*USADA* website: ▶ www.usada.org) is a signatory to the World Anti-Doping Code. This organization manages the anti-doping program for all US Olympic Committee-recognized governing bodies, events, and athletes in and out of competition.

**Case Study**

**USADA**

The US Anti-Doping Agency (USADA) is the national anti-doping organization (NADO) in the USA for:

- Olympic sport
- Paralympic sport
- Pan-American sport
- Parapan American sport

The organization is charged with managing the anti-doping program, including in-competition and out-of-competition testing, result management processes, drug reference resources, and athlete education for all US Olympic Committee (USOC)-recognized sport national governing bodies, their athletes, and events.

The *National College Athletic Association* (*NCAA* website: ▶ www.ncaa.org) publishes the US guidelines for proper drug use in college sports.

The guidelines are published on their website (▶ http://www.ncaa.org/2015-16-ncaa-banned-drugs). The website posts a very important statement to all athletes: *Note to Student-Athletes: It is your responsibility to check with the appropriate or designated athletics staff before using any substance. There is NO complete list of banned substances. Do not rely on this list to rule out any label ingredient. It is your responsibility to check with the appropriate or designated athletics staff before using any substance.*

It is also the prescriber's responsibility to be familiar with these rules and prescribing guidelines [33].

Many other resources for appropriate therapeutic drug use and information for drug-free sport are available from governing sports bodies, Internet, and specific apps for smartphones and tablets. At this time, the WADA Prohibited List is available as an app for smartphones and tablets. It is updated in January of each year. Position papers and

**Anti-doping Specific Resources Available to Health Professionals**

| USADA Resources | Description | Weblink |
|---|---|---|
| GlobalDRO | Check the WADA status of medications by ingredient or brand name. | http://www.globaldro.com/us-en/ |
| Wallet Card | List of medicines that are not prohibited by WADA in sport. | http://www.usada.org/substances/tue/ |
| WADA Prohibited List | List of substances and methods that are prohibited in sport. | http://www.usada.org/substances/prohibited-list/ |
| Supplement 411 | Information on Dictary Supplements including a High- Risk List. | http://www.supplement411.org/supplement411/ |
| Drug Reference Line | Speak to an export to understand the Prohibited List, TUEs, and Dictary Supplements. | http://www.usada.org/substances/drug-reference-phone-line/ |
| Ask the Scientist | Ask USADA's scientists and experts questions related to anti-doping science. | http://www.usada.org/science/ask-the-scientists/ |
| Surgery Checklist | Information on anti-doping considerations in preparation for surgery. | http://www.usada.org/substances/tue/ |
| HealthPro Advantage | Online anti-doping educational tutorial, specific to health professionals. | http://www.usada.org/resources/healthpro/ |
| Athlete Express | Information on the basics of drug-testing. | http://www.usada.org/athletes/ |
| Play Clean Tip Center | Anonymously report doping in sport. | http://www.usada.org/athletes/playclean/ |

126|112:2|March/April 2015|Missouri Medicine

**Fig. 12.5** Anti-doping specific resources that are available to the health professional (adapted from REF figureTandon S, Bowers LD, Fedoruk MN Treating the elite athlete: anti-doping information for the health professional. Mo Med. 2015 Mar-Apr;112(2):122–8)

consensus reports written by various athletic-related organizations also provide guidelines for therapeutic agent use in sport. The National Athletic Trainers' Association has published valuable resources for athletic trainers and those on the sports medicine team [34].

Other important resources are listed in Fig. 12.5.

## 12.5 Summary

In summary, the team dentist must assess and diagnose the athlete. They should (1) be aware of the indicated therapeutic drugs to treat acute oral conditions, (2) not violate rules of governing bodies, or (3) compromise the athlete's ability to compete. Therapeutic drugs should help the athlete remain pain-free and infection-free. These drugs should not interfere with hydration, alertness, or clotting. In addition, the team dentist must be aware of addictive drugs by responsible prescribing (Fig. 12.6) and realize that other prescribers and sources may provide the athlete with addictive drugs. The team/sports dentist can use alternative medications when possible and minimize dosage and amount prescribed as appropriate for the individual athlete's needs.

**SUMMARY**

- ASSESS YOUR ATHLETE
- DIAGNOSIS
- KNOW THE DRUGS AND RULES TO TREAT
- ENJOY THE GAME

**Fig. 12.6** Know your Athletes patients

## References

1. Alaranta A, Alaranta H, et al. Use of prescription drugs in athletes. Sports Med. 2008;38:449–63.
2. Burstein R, Hourvitz A, Epstein Y, Dvir Z, Moran D, Altar J, Shemer J, Shalev A, Galun E. The relationship between short-term antibiotic treatments and fatigue in healthy individuals. Eur J Appl Physiol Occup Physiol. 1993;66(4):372–5.
3. Schmid-Wendtner MH, Korting HC. Penciclovir cream—improved topical treatment for herpes simplex infections. Skin Pharmacol Physiol. 2004;17(5):214–8.

4. Tubridy E, Kelsberg G, et al. Which drugs are most effective for recurrent herpes labialis? J Fam Pract. 2014;63(2):104–5.
5. Opstelten W, Knuistingh A, et al. Treatment and prevention of herpes labialis. Can Fam Physician. 2008;54:1683–7.
6. Lee J, Kim JK, et al. Recovery time of platelet function after aspirin withdrawal. Curr Ther Res Clin Exp. 2014;76:26–31.
7. Cahill RA, McGreal GT, et al. Duration of increased bleeding tendency after cessation of aspirin therapy. J Am Coll Surg. 2005;200(4):564–73.
8. Cea Soriano L, Soriano-Gabarró M, García Rodríguez LA. Validation of low-dose aspirin prescription data in The Health Improvement Network: how much misclassification due to over-the-counter use? Pharmacoepidemiol Drug Saf. 2015. https://doi.org/10.1002/pds.3926. [Epub ahead of print].
9. Anderson K, Hamm RL. Review article: Factors that impair wound healing. J Am Coll Clin Wound Spec. 2014;4(4):84–91.
10. Bailey S, Patrick O'Connor J. NSAID therapy effects on healing of bone, tendon, and the enthesis. J Appl Physiol. 2013;115(6):892–9. https://doi.org/10.1152/japplphysiol.00053.2013. Epub 2013 Jul 18.
11. Patrignani P, Tacconelli S, Bruno A, Sostres C, Lanas A. Managing the adverse effects of nonsteroidal anti- inflammatory drugs. Expert Rev Clin Pharmacol. 2011;4(5):605–21.
12. Moore PA, Hersh EV. Combining ibuprofen and acetaminophen for acute pain management after third-molar extractions: translating clinical research to dental practice. J Am Dent Assoc. 2013;144(8):898–908.
13. Menhinick KA, Gutmann JL, et al. The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-controlled study. Int Endod J. 2004;37(8):531–41.
14. Mehlisch DR, Aspley S, et al. Comparison of the analgesic efficacy of concurrent ibuprofen and paracetamol with ibuprofen or paracetamol alone in the management of moderate to severe acute postoperative dental pain in adolescents and adults: a randomized, double-blind, placebo-controlled, parallel- group, single-dose, two-center, modified factorial study. Clin Ther. 2010;32(5):882–95.
15. Mehlisch DR, Aspley S, et al. A single-tablet fixed-dose combination of racemic ibuprofen/paracetamol in the management of moderate to severe postoperative dental pain in adult and adolescent patients: a multicenter, two-stage, randomized, double- blind, parallel-group, placebo-controlled, factorial study. Clin Ther. 2010;32(6):1033–49.
16. Cottler LB, Ben Abdallah A, et al. Injury, pain, and prescription opioid use among former National Football League (NFL) players. Drug Alcohol Depend. 2011;116(1-3):188–94.
17. McCabe SE, West BT, et al. Motives for medical misuse of prescription opioids among adolescents. J Pain. 2013;14(10):1208–16. https://doi.org/10.1016/j.jpain.2013.05.004. Epub 2013 Aug 15.
18. Pennsylvania guidelines on the use of opioids in dental practice. July 2015. proper reference format.
19. Denisco RC, Kenna GA, et al. Prevention of prescription opioid abuse: the role of the dentist. J Am Dent Assoc. 2011;142(7):800–10.
20. Diehl K, Thiel A, et al. Substance use among elite adolescent athletes: findings from the GOAL Study. Scand J Med Sci Sports. 2014;24:250–8.
21. Petróczi A, Aidman E. Psychological drivers in doping: the life-cycle model of performance enhancement. Subst Abuse Treat Prev Policy. 2008;3:7.
22. Terry-McElrath YM, O'Malley P, et al. Middle and high school drug testing and student illicit drug use: a national study 1998-2011. J Adolesc Health. 2013;52(6):707–15.
23. Castillo EM, Dawn Comstock R. Performance enhancing drugs prevalence of use of performance-enhancing substances among United States adolescents. Pediatr Clin. 2007;54(4):663–75.
24. Thevis M, Kuuranne T, et al. Annual banned-substance review: analytical approaches in human sports drug testing. Drug Test Anal. 2016;8(1):7–29.
25. Momaya A, Fawal M, et al. Performance-enhancing substances in sports: a review of the literature. Sports Med. 2015;45(4):517–31.
26. Handelsman DJ. Performance enhancing hormone doping in sport. In: De Groot LJ, Beck-Peccoz P, Chrousos G, Dungan K, Grossman A, Hershman JM, Koch C, McLachlan R, New M, Rebar R, Singer F, Vinik A, Weickert MO, editors. 2015. Source: Endotext. South Dartmouth: MDText.com; 2000.
27. Parkinson AB, Evans NA. Anabolic androgenic steroids: a survey of 500 users. Med Sci Sports Exerc. 2006;38(4):644–51.
28. Sagoe D, Andreassen CS, et al. The aetiology and trajectory of anabolic-androgenic steroid use initiation: a systematic review and synthesis of qualitative research. Subst Abuse Treat Prev Policy. 2014;9:27.
29. DrugFacts: anabolic steroids. Rev July 2012. National Institute on Drug Abuse; National Institutes of Health; U.S. Department of Health and Human Services. http://www.steroidabuse.gov/.
30. Sci American. 2004;291(1).
31. Reardon CL, Creado S. Drug abuse in athletes. Subst Abuse Rehabil. 2014;(5):95–105.
32. Tandon S, Bowers LD, et al. Treating the elite athlete: anti-doping information for the health professional. Mo Med. 2015;112(2):122–8.
33. Kersey RD, Elliot DL. National Athletic Trainers' Association position statement: anabolic-androgenic steroids. J Athl Train. 2012;47(5):567–88.
34. Zinder SM, Basler RS. National Athletic Trainers' Association position statement: skin diseases. J Athl Train. 2010;45(4):411–28.

### Books

Glick M, editor. Burket's oral medicine, 12th ed. Raleigh: Peoples Medical Publishing House; 2015.
Malamed S. Handbook of local anesthesia. 6th ed. St. Louis: Elsevier Mosby; 2011.

12

# Oral Appliances and Athletic Performance

*Dena P. Garner*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_13

## 13.1 Introduction

This chapter will include a comprehensive literature review and history of oral appliances used to improve physical performance. This will be helpful in obvious ways in the sports arena but may also have additional medical implications. Discussion of the literature will be complete and thorough. Then theories of possible mechanisms will be discussed, and the chapter concludes with a discussion of potential future research areas to be explored.

### 13.1.1 History of Crude Oral Appliances

The history of oral appliances to improve breathing may be dated back to Pierre Robin who developed a device to improve breathing in those individuals diagnosed with Pierre Robin syndrome [1]. This syndrome has characteristics of a cleft palate, glossoptosis, and a retrusive mandible [2]. In the early 1900s, Robin developed two devices first treat glossoptosis and then later a device that was purported to change the position of the mandible [1]. Yet even before oral appliances emerged in the early 1900s, it has been cited that both soldiers in battle and women during childbirth were given leather straps and sticks to bite on during the pain of surgery or child delivery to alleviate and endure physical stress [3]. Although the history of oral appliances has been long, there is limited understanding of the physiological impact of such devices on the individual during *physical* stress. Thus, the purpose of this chapter is to point to potential physiological mechanisms occurring during the stress of exercise while using a mouthguard/mouthpiece.



### 13.1.2 Use of Oral Appliances for Protection

Recent research on oral appliances, in the form of mouthguards, have been used in a variety of sports to prevent oral-facial injury (see ▶ Chap. 7) [4]. In a review of dental trauma literature, Glendor noted that participation in sports resulted in the greatest cause of dental injury [5]. A review by Newsome cited that early research estimated that players in contact sports such as American football and rugby had a one in ten chance of receiving a dental injury during a year of play, with a one in two chance in one's lifetime of playing such a sport [6] as cited in [7]. Injuries without mouthguard protection range from crown fractures via high-velocity objects, root fractures, mandibular fractures, and tooth fractures to luxations from low-velocity trauma [8]. Of these injuries, the American Dental Association (ADA) cites that close to 80% of these oral injuries occur with the maxillary incisors [9]. Thus, due to the correlation between dental injuries and sport participation, the ADA recommends that athletes use a mouthguard during contact sports [9]. In addition, other governing bodies such as the National Federation of High Schools and the National Collegiate Athletic Association mandate mouthguard use for athletes in a variety of contact sports such as football, field hockey, ice hockey, and lacrosse in order to minimize dental trauma during sport participation [10, 11].

There is substantial evidence that mouthguard use reduces dental injury for individuals during contact sports/activities [12, 13]. Early research in the field of mouthguard use and prevention of injury cited a significant reduction in injury as it related to mouth protections for high school football players [14]. In a more recent review of mouthguard use and injuries, Knapik and colleagues cited 69 quantitative studies on mouthguard use and prevention of injury [15]. Although there were difficulties in analyzing the data from the studies due to the methodology used, Knapik and colleagues concluded that there is a significant reduction in overall risk of orofacial injury with mouthguard use. They cited a 1.6–1.9 times higher risk of injury without mouthguard protection [15]. De la Cruz and colleagues supported this finding in their research with military individuals, specifically finding an overall risk of orofacial injury being 1.7 times greater during a period without mandated mouthguard use for all training events versus during periods when mouthguards were required for all training events [12].

Although the use of mouthguards in protecting the athlete cannot be refuted, compliance by the athlete is an issue. While the use of mouthguards during contact sports is of utmost importance to the dental health of the athlete, adherence to the use of the mouthguard should continually be monitored based on studies citing a range between 16 and 46% of athletes who do not wear the appliance [16–18]. Hawn and colleagues examined the enforcement and use of mouthguards in a men's collegiate ice hockey season. Of the 127 NCAA-affiliated institutions, it was cited that while a 93% of athletic trainers believe that mouthguards reduce dental injury, only 63% of the athletes actually wear the appliance. Interestingly, the study found that athletes in Division I were less likely to wear a mouthguard than to not wear a mouthguard ($N = 462$ reported wearing mouthguard, while $N = 481$ reported that they did not wear mouthguard) as compared to athletes in Division II and III [18]. Similar outcomes were found with players in the Rugby World Cup, with an average of 16% of the players from Ireland, Scotland, Wales, and Australia citing that they do not wear a mouthguard, while 100% of the players from all countries believe a mouthguard decreases injuries [17]. Berry and colleagues noted an overall negative attitude toward mouthguards in collegiate ice hockey players due to the bulkiness, uncomfortableness, and decreased ability to talk and breathe [19]. Thus, the question that continues to plague dental professionals and others associated with contact sports is how to encourage the athlete to wear a mouthguard during play.

## 13.2 Literature Review on Oral Appliances and Performance

### 13.2.1 Early Research on the Effect of Mouthguard Use on Performance

To potentially answer this question and to encourage athletes to wear a mouthguard, dental professionals in the late 1970s and early 1980s began to practice a new type of dentistry called "sports dentistry" [20]. Sports dentistry involved fitting athletes with mouthguards to correct malocclusions and temporomandibular joint (TMJ), while touting an improvement performance along with protection of the teeth, being named as physiologic dentistry by Fonder [21] and Moore [20]. However the idea of properly fitting mouthguards to improve TMJ issues was not without its critics. Smith, a physical anthropologist, cited that there was no theory to support a complex interaction between the TMJ and other parts of the body [20]. Yet, subjective data associated with use of a mandibular orthopedic repositioning appliance (MORA) stated athletes improved performance in sports such as football, luge, and running. Garabee cited improvements in endurance, leg strength, and resiliency in long-distance runners [22]. Kaufman, in utilizing the MORA with Olympic luge athletes, found that the device prevented headaches and resulted in greater endurance in their training runs [23]. Yet as intimated earlier, much of the purported benefits of mouthguard use were subjective in nature; thus, the credibility of these findings was seen as controversial [24].

To aid to the understanding of these subjective claims, dentists and researchers sought to quantify any increases or improvements in strength and performance with the use of a MORA device [23, 25–27]. Smith cited significant increases in strength in the isometric deltoid press in NFL football players ($N = 25$) when wearing a wax bite versus during a teeth together condition while completing an isolated muscle movement [25]. In a later study, Smith supported these findings citing a 66% significant improvement in strength using a custom vinyl mouthguard in professional football players [26]. In addition, Grunwaldt found in 41 members of the Green Bay Packers an 8–11% improvement in Cybex muscle testing in using corrective mouthguards [20]. However, comparing a MORA device, with no mouthguard, and a mouthguard condition, Yates et al. did not cite any significant differences in the isometric dead lift in college football players using whole-body movement [28]. In addition, in testing isolated muscle groups, Welch and colleagues found no differences in strength when measuring strength outcomes; specifically there were no differences in the maximal grip strength and knee extension and flexion [29]. However, problems of small sample sizes (the Welch study sample was small, $N = 9$), lack of control subjects, potential influence of the placebo effect, and the types of athletes (female volleyball players, NFL football players) studied makes it difficult to compare results. In addition, although Smith used an isolated relatively smaller muscle group (deltoids) and Yates studied a whole-body movement, critics could argue that differences may lie in the resistance training techniques and the impact of a mouthguard/mouthpiece on these movements. Forgione and colleagues suggest mouthguards with diverse designs could result in varied strength outcomes based on the construction of the mouthguard which is difficult to compare between studies [30, 31].

The design of the mouthguards in early research studying the effect of mouthguards on athletic performance primarily utilized what was labeled as a MORA device. Jackush cites the MORA device (also known as the Gelb appliance) derived from Dr. Harold Gelb [32] (Fig. 13.1). The MORA device was intended to cover the occlusal surfaces of the posterior teeth of the mandible with an appropriate vertical thickness. In addition, splints which cover the occlusal surfaces were often made of acrylic resin but may be lesser durometer if used in a mouthguard for the maxilla [32]. Yet, the issue with describing these appliances was the technique employed by the researcher to determine proper vertical dimensions which typically involved subjective methodology. For example, Welch and colleagues set the ideal vertical dimension based on the subject's manual resistance of the deltoid muscle. This technique is similar in other studies utilizing the MORA, in which the researcher(s) manually applied opposing force and measured the associated vertical dimension of occlusion during the greatest force production by the subject [25, 29]. The subject is then said to be in a state of optimal mandibular position that could then positively affect other parts of the body [27, 32]. Thus, in many of the studies in the late 1980s, vertical displacement and its varying degrees of displacement was often used as a gauge to measure if differences in performance occurred with the MORA device.

**Fig. 13.1** MORA appliance. MORA (mandibular orthopedic repositioning appliance) appliance with hard acrylic covering the posterior teeth designed to reposition the mandible in the anterior direction

Consequently, in order to assess differing impacts of vertical dimensions and subsequent effects on performance, researchers employed various methods to measure impact on performance. Greenberg and colleagues conducted a study by creating a placebo appliance (no vertical dimension) versus a MORA/Gelb device, testing strength differences in a university basketball team ($N$ = 14). The authors were not clear on how much vertical dimension the appliance provided but did cite that the placebo appliance did not provide any resin on the occlusal services, resulting in no change in vertical dimension for this appliance. In testing shoulder abduction and adduction on the dynamometer, there were no differences between the placebo appliance, the no appliance, and the MORA device [33, 34]. However, the sample size was small, no vertical dimensions were reported, and the authors noted that these athletes were not involved in any strengthening program at the time of testing. Therefore, in order to address some of these issues, Yates and colleagues set the vertical dimensions to a specific 2–3 mm for 14 college football player subjects studying the effect of the MORA on isokinetic and isokinetic upright rower, isometric deadlift, or isometric two-arm pull. They too found no significant positive or negative outcomes in muscular strength with this position at 2–3 mm vertical dimension [28]. However, more recently, Lee et al. [35] utilized a MORA device which was designed using a precise protocol (see paper for all aspects of the protocol) that provided 3 mm vertical dimension at the centric occlusion and adjusted such that all teeth would come in contact evenly with the MORA device [35]. Their findings revealed significant EMG outcomes with isometric improvements in the following muscle groups: sternocleidomastoid muscles, cervical and lumbar erector spinae, upper trapezius, biceps, triceps, rectus abdominis, and internal and external oblique [35].



It is evident in many of the earlier studies that a few methodological issues were apparent as it relates to the vertical dimensions, sample size, types of athletes and sports studied, and design of the mouthguards. However, despite these issues, each study added valuable knowledge to the understanding of the effect of MORA and/or mouthguard use during exercise performance. Yet the question still remained on how the MORA and/or mouthguard use during exercise performance elicited performance outcomes, if any. Were any positive performance outcomes due to the placebo effect, or was there a link to whole-body physiology with the use of such a device as described by Stenger and Kaufman [23, 27]? Thus, due to the complexities of the issues, the difficulties involved for practicing dental professionals in conducting research, and the lack of interest by researchers in the field of exercise physiology, investigation of this issue remained stagnant for several years.

### 13.2.2 Recent Research on the Effect of Mouthguard Use on Performance

However, in the early 2000s, research interest in the use of custom-fit mouthpieces regained momentum, and this is partly due to the subjective feedback provided by athletes wearing mouthpieces designed by Shock Doctor, Bite Tech, and Makkar Athletics, mouthguard companies that marketed the effectiveness of mouthguard use during exercise performance (Fig. 13.2). For example, Shock Doctor's website stated that "Performance mouthguards, mouthpieces and mouthwear that advertise increased strength and performance are typically called MORA (Mandibular Oral Repositioning Appliance) mouthguards. MORA technology provides optimum positioning and 'bracing' of the lower jaw, neck and shoulders so that the muscles work more

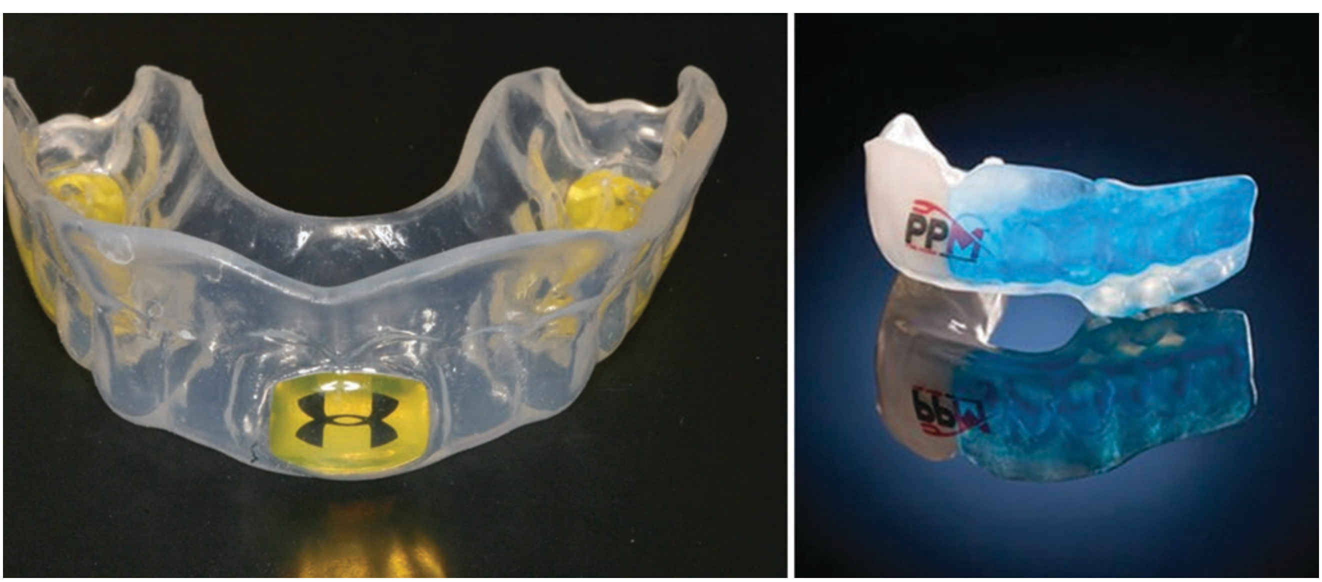



**Fig. 13.2** Performance-enhancing mouthguards. Two styles of performance mouthguards Under Armour MG that relies on wedges to move the mandible in an anterior inferior direction. The pure power MG utilizes neuromuscular dentistry to locate the desired bite to build into the appliance

efficiently, thus conserving energy for the muscles controlling the arms and legs, which may increase strength" [36]. However, such claims are based on subjective data by athletes. Therefore, with the increased use of mouthguards as a performance-enhancing device, researchers have taken up the mantle left by their predecessors to determine if there are effects on performance and then to link these effects to more objective measures. Mouthguards tested from the 2000s to present can be classified into three categories: stock mouthguards, boil and bite mouthguards, and custom-made mouthguards [9]. Yet, these vary greatly between study to study, including suppositions of forward mandibular placement, optimal temporomandibular placement, and lower mouthpieces versus upper mouthguards. Thus, in critically examining if any effects are physiological or placebo, a review of the studies is needed with a description of the mouthguard(s) used, the testing procedures, and the differences in performance as it relates to anaerobic and aerobic activity with use of the mouthguard. With all this information, plausible theories will be suggested to explain potential physiological effects in order to encourage future research to clarify the mouthguard effect.

### 13.2.3 Mouthguard Effect on Anaerobic Performance

There have been a number of current studies published which cite improvements with mouthguard use during anaerobic performance which includes varying mouthguards and methodology [37–41]. Ebben and colleagues using a vinyl mouthpiece found significant improvements during knee extensions, with an 11% increased average torque and 10% increase in peak torque with subjects clenching on a mouthpiece versus no-mouthpiece condition [40]. Dunn-Lewis and colleagues also cited improvements with a Pure Balance mouthguard made of an EVA material which incorporates pronounced bite pads on both sides of the maxillary mouthguard. Among their findings, they cited a significant increase in bench throw power and force, increased rate of power production in the vertical jump for the Pure Balance mouthguard versus no mouthguard and an over-the-counter mouthguard. Yet no differences were seen in the Pure Balance mouthguard between conditions as it related to 10 m sprint time, sit and reach distance, a visual reaction test, and balance [38]. Utilizing a TMJ repositioning mouthguard, Arnet and colleagues gauged the effect on physical performance parameters in collegiate and professional athletes using neuromuscular dentistry (a method in which TENS surface electromyography is applied to the jaw to facilitate muscular relaxation resulting in a neuromuscular optimal bite position, with a mouthguard fabricated based on this position). They found that when subjects wore a TMJ repositioning device, there was a 3% improvement in vertical jump and average mean power for the Wingate anaerobic test versus a standard custom-fit mouthpiece designed to protect the teeth [37]. These findings as it relates to the Wingate protocol were later substantiated using a maxillary mouthguard (CleverBite®, CleverBite SL, Terrassa, Spain) fabricated using digital scans of the maxillary and mandibular dental arches as well as a recording of the interocclusal relation of the rest position of the mandible. They found a 4% improvement in peak power and a 1% improvement with mean power during the Wingate anaerobic test in the mouthguard condition, these being the same findings (4% and 1%, respectively) by Arnet and colleagues using the same protocol [37, 41]. In addition, researchers cited an 8% improvement in lactate measures with the mouthguard use and significant improvements in all variables associated with anaerobic testing with mouthguard use compared to no-mouthguard condition [41]. Durante-Pereira and colleagues also cited significant improvements with a maxillary custom mouthguard (described as pressure laminated with gum shield consisting of ethylene vinyl acetate) in testing countermovement jumps (CMJ) in 10 rugby players. Yet they found no differences in a 15 second rebound jump nor in the spirometer data with each of these tests. Yet, the improvement in the CMJ test should be viewed with caution due to the small sample size [39]. Using the same CMJ test, Busca and colleagues utilized a larger sample size ($N$ = 28) and measured vertical CMJ and found significant improvements in mean power and height in the mouthguard condition versus a clenching no-mouthguard condition and a no-clenching no-mouthguard condition. The mouthguard used in this study was the CleverBite (as described earlier in this review) which relies on digital scans of the maxilla and mandible with a resultant maxillary mouthguard of 1.4 mm EVA overlaid with 4 mm polyethylenterephthalat-1. In addition, they cited significant improvements during the hand-grip test and the back-row isometric force test (force development and peak force) in the mouthguard condition versus the other two conditions (clenching no mouthguard and no clenching no mouthguard) [42].

While there are many studies which have cited improvements in performance with mouthguard use during exercise, there are numerous others which have cited no difference in anaerobic performance with mouthguard use. A study by Allen and colleagues examined recreationally trained individuals and found no differences in countermovement vertical jump (CMJ) using a force plate and Vertec device, one-repetition bench press, and measurements of peak force or rate of force development. However, the caveat of this study was that the authors admitted they had not informed the subjects to clench or not to clench but to try to perform each exercise as "normally as possible" [43]. Thus, the difference between those articles finding improvements with mouthpiece use and those which did not cite differences may be due to the clenching effect. Goelm and Arent also found no differences with a maxillary mouthpiece in assessing measures of vertical jump and power output, balance, flexibility, range of motion, strength (though trended to significance with $p$ = 0.06), and agility. They cited that this lack of evidence in any of these parameters may be due to the fact that they used a basic custom mouthpiece that may not have provided optimal jaw-repositioning versus a custom dental appliance used in a previous study done in their lab which found differences using a jaw-repositioning mouthpiece [37, 44]. Drum and colleagues also noted no differences in anaerobic, aerobic, reaction time, and flexibility measures between a custom-fit maxillary mouthguard, boil and bite mouthguard, and a no-mouthguard condition. Their study utilized a collegiate football team, highly conditioned athletes, yet sample size ($N$ = 10) was small and would be an impetus for more studies with a larger sample size [45].

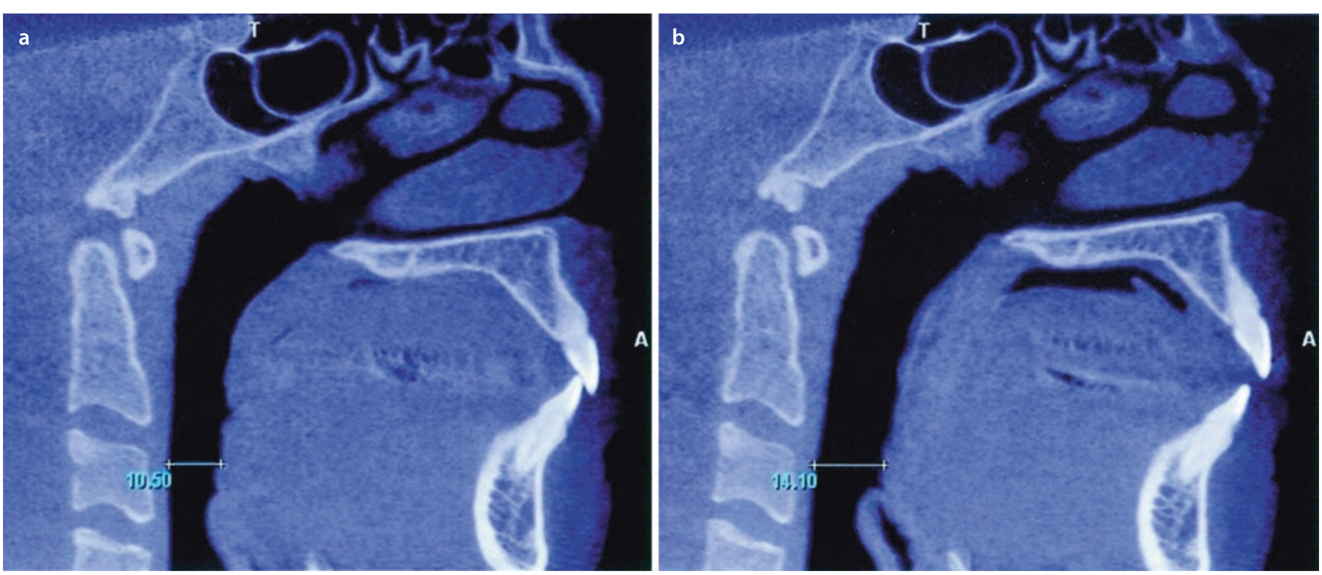
### 13.2.4 Oxygen Uptake and Ventilation

While much of the research has focused on anaerobic performance outcomes with mouthguard use, other research has focused on objective measures assessing differences in oxygen uptake, heart rate, and ventilation and mouthguard use [46–55]. In a study examining impact of exercise between no-mouthguard condition, commercially available maxillary mouthguard, and custom maxillary mouthguard in 19 trained males, Bourdin and colleagues cited no differences in respiratory parameters as well as no differences in visual reaction time and explosive power at rest and during exercise [47]. Yet interestingly, as it relates to respiratory parameters, the commercially available mouthguard showed differences in respiratory rate during stages of incremental exercise on the cycle ergometer. The use of this mouthguard resulted in a 9% decrease in respiratory rate during stage 1 of the incremental protocol using the commercially available mouthguard as compared to the no-mouthguard condition. These appear important in light of later research by Garner and colleagues which found significantly lowered respiratory rates with various mouthpieces utilized in their studies [47–50]. Gebauer and colleagues also found no differences in respiratory function (ventilation, oxygen uptake, and heart rate) during a graded exercise test (two 5 min stages at 6.2 and 7.5 mph) between no mouthguard, normal-palate maxillary mouthguard, and an open-palate maxillary mouthguard, with $N$ = 27 [53]. However, they cited a 3.9% change between no-mouthguard condition and palate-free condition and a 2.1% change between no mouthguard and normal-palate mouthguard in comparing maximum oxygen uptake (ml/kg/min) during each stage [53]. During an incremental exercise protocol in which the workload was increased each minute by 30 W, von Arx and colleagues showed that subjects experienced no difference between no mouthguard and custom mouthguard as it relates to peak oxygen uptake, breathing frequency, and peak minute ventilation. However, von Arx and colleagues did note a 5% improvement in workload scores with the mouthguard versus no mouthguard [55]. In another graded exercise protocol in which subjects were asked to cycle for 5 min during four stages which increased by 50 W for each stage, Bailey and colleagues cited a significant difference in ventilation in the vented moldable maxillary mouthguard versus the no mouthguard and standard boil and bite maxillary mouthguard. Specifically, the vented mouthguard ventilation was 9% lowered at maximum workload as compared to the no-mouthguard condition and was 6% lower at 200 W in vented versus no mouthguard. In addition, they cited a significant reduction in blood lactate levels with the vented mouthguard as compared to the no mouthguard and standard boil and bite mouthguard at both the 200 W and maximum workloads [46]. Finally, while most studies cited involve males, Rapisura and colleagues using an all-female population ($N$ = 11) cited no significant differences in heart rate, oxygen consumption, and minute ventilation between women with a self-adapted mandibular mouthguard, boil and bite maxillary mouthguard, and no-mouthguard condition during 2 min incremental exercise on the cycle ergometer [54]. Yet issues with this study were small sample size and potential differences in the use of the maxillary versus mandibular mouthguard as well as inability to compare respiratory parameters due to the duration of incremental exercise chosen for this study (2 min stages). While these studies have utilized various sample sizes and protocols, what appears to be apparent is that a trend or a significant difference occurs with respiratory parameters with mouthguard use during higher-intensity exercise. Yet why would such changes in respiratory parameters during exercise be important to individuals during exercise?

An earlier study by Francis and Brasher helps shed light on possible mechanisms and impact on exercise performance with mouthguard use [56]. In this study, they had subjects perform 20 min of continuous exercise with varying intensities with the following conditions: no mouthguard (No), unfitted upper mouthguard (MG1), unfitted bimaxillary mouthguard (MG2), and a bimaxillary guard with a breathing hole (MG3). In comparing all conditions for the subjects with conditions randomly assigned, they found that during heavy-intensity exercise, subjects had significantly lower ventilation with the mouthguard conditions as compared to the no-mouthguard condition, with expired volume of gas being higher in the mouthguard condition. They then concluded that the use of the mouthguard may actually result in an improved breathing pattern that would enhance alveolar ventilation. They cited that this could be due to a type of pursed-lip breathing that would enable subjects to take in less air with a given amount of oxygen thereby affecting ventilation and expired gas [56]. In their protocol they examined effects during both light and maximum exercise on a cycle ergometer, with only the maximum exercise demonstrating differences. Similar to this protocol, Garner and colleagues utilized mandibular mouthpieces against a no-mouthpiece condition in assessing effects on respiratory parameters [48–50]. In one study, they found significant decreases in respiratory rate with both the boil and bite and the custom mouthpiece, specifically a 3% reduction in respiratory rate during the first 5 min of moderate-intensity activity [49, 50]. Then in comparing the boil and bite to the custom, the boil and bite had lowered respiratory rate versus the custom, with a 9% reduction in the respiratory rate with the mandibular boil and bite mouthpiece [48–50]. As noted earlier, comparing these outcomes is difficult to relate to other studies as none have provided steady-state exercise parameters. However, the study by Bourdin and colleagues was the most similar with an incremental exercise protocol of 4 min. Bourdin and colleagues found a 2% difference in ventilation (L/min) with the custom upper mouthpiece versus the no-mouthguard condition during the first 4 min of the exercise, with Garner finding 1.4% difference in ventilation between their custom lower mouthpiece and the no-mouthpiece condition during the first 5 minutes of steady-state exercise [49, 50]. In addition, Bourdin and colleagues found a 5% difference in breaths per minute between the boil and bite mouthguard and no-mouthguard condition, while Garner found a 5% decrease in respiratory rate (similar to breaths per minute) between custom mouthpiece and no mouthpiece and a 9.7% decrease in respiratory rate between a boil and bite and the no-mouthpiece condition during 10 min of steady-state exercise [47–50]. Although the protocols are different, cycling versus running and steady state versus graded, they are the most similar and enable a greater understanding the effect of various mouthguards/mouthpieces versus a no-mouthguard/no-mouthpiece condition. A potential explanation on the differences may be due to the amount of material with boil and bite mouthguards/mouthpieces versus the custom devices as it relates to the tongue placement (to be discussed in a later section). Thus in these studies, the objective measures of respiratory function demonstrated trending or significant improvements in ventilation and or breathing rate with mouthguard use during maximal exercise. This is critical as it shows that breathing rate and ventilation is slower while taking in the required oxygen needed for exercise with mouthguard use, potentially resulting in improved alveolar ventilation and reduced workload as seen in the study by Bailey and von Arx studies [46, 55, 56].

### 13.2.5 Lactate and Cortisol

While understanding the effect of mouthguard use during exercise on respiratory physiology provides more objective measures of identifying the mouthguard effect, Garner and colleagues and Dudgeon and colleagues have sought to add to the body of objective measures by assessing the effect of mouthpiece use during exercise on lactate and cortisol [49–51, 57–59] . Thus based on the differences cited in respiratory physiology and the potential mechanisms to explain the mouthguard effect, Garner and McDivitt measured the width and diameter of the oropharynx with and without a mouthpiece using cone beam computed tomography (CBCT) scans. They cited a 9% increase in both diameter and width for subjects using a mouthpiece but found no difference in lactate levels during an exercise protocol, yet the sample was small ($N$ = 10) [51] (◘ Fig. 13.3). Thus, based on the changes in the airway parameters, they conducted a study with a larger population ($N = 24$) and found that lactate levels were significantly improved after 30 min of running at moderate-intensity exercise, noting a 23% change of lowered lactate levels with mouthpiece use versus a no-mouthpiece condition [59]. In addition to differences in lactate during running, researchers also studied effects of mouthpiece use during exercise on cortisol levels, citing a trend toward lowered cortisol levels with mouthpiece after 30 min of running [58]. Yet, the exercise intensity may not have been substantial enough to elicit significant changes in cortisol. Thus, to test this theory, researchers utilized a more intense protocol of 1 hour with resistance exercise. In this protocol a Division I football team completed a routine resistance training session while cortisol was measured before, during, and after the session. With mandibular mouthpieces being randomly assigned, they found a 51% reduction in cortisol levels 10




◘ **Fig. 13.3** CBCT scans airway. CBCT scans of subject airways with and without oral appliance. Appliance used was Under Armour mouthpiece (lower arch) seen in ◘ Fig. 13.4. **a** shows the no appliance airway and **b** shows the airway while wearing the appliance. There was consistent significant improvement in airway volume while wearing the appliances minutes post exercise ($N$ = 28). This is significant for a few reasons. Firstly, cortisol with mouthpiece use had not been measured in humans, though a similar measure had been assessed in rats under stress while biting on a stick [60, 61]. Secondly, research in resistance exercise without a mouthpiece shows that cortisol levels increase significantly from baseline to post-intensive resistance exercise, and this was the case in the no-mouthpiece condition, thus corresponding with the literature [49, 50, 62, 63]. Thirdly, it is well understood that elevated cortisol affects protein synthesis and immunity [64, 65], thus these findings link mouthpiece use with a potential recovery aspect of exercise. Supporting these findings by Garner et al. [49, 50] was a follow-up study by Dudgeon and colleagues in which they had subjects complete a highly intensive protocol of ten sets of six repetitions of back squats at 80% of the individual's one-repetition maximum with and without a mandibular mouthpiece. They cited significant reductions in cortisol and lactate, finding a 39% reduction in cortisol and 22% reduction in lactate 30 min post exercise with mouthpiece use [57]. In conclusion, the studies by Garner and colleagues and Dudgeon and colleagues cite credible results and potential mechanisms that would support the mouthguard effect and be difficult to link to a placebo effect.

In conclusion, although these recent studies have cited improvements, of importance is understanding if there is an impact of the placebo effect with any of these studies, i.e., if a person is told he/she will or won't improve performance outcomes with an appliance, this is likely to affect results. It is unclear whether researchers informed subjects of a potential effect in studies that found improvements. In studies within the Garner laboratory, subjects were not told whether the mouthpiece would or would not affect their performance [59]. However, due to the popularity of such products, it still may be a factor in influencing subjects. Thus, research which is less subjective is needed to support or refute the positive physiological findings with mouthpiece use. Much of the evidence citing positive physiological performance effects leads this author to believe that there is a physiological mechanism resulting from mouthguard use during exercise. Thus, the next sections of this review will delve into these theories of the mouthguard effect which have been substantiated in other fields of research, thus providing a greater understanding and knowledge of how to better study this area of sport dentistry.



## 13.3 Literature Review of Theories to Support Performance Enhancement

### 13.3.1 Genioglossus and Tongue Position

It has been cited that the "the tongue is a small member and has dominion" (James 3:5, Aramaic Bible in Plain English). Although this statement was not in reference to the tongue being a physiologic marvel, it cannot be overstated the importance and involvement of the tongue within several physiologic functions. The complexity of the organ can be found in studying its involvement in respiration, swallowing, speech, and mastication [66–72]. The tongue muscle, specifically the genioglossus, is the main protruding muscle. The genioglossus is innervated by the hypoglossal (cranial nerve XII) which, along with the hypoglossus, causes a pressing down of the tongue base [66, 69]. The importance of the genioglossus is its role in increasing muscular tone during the inspiratory phase of breathing [73, 74] which in turn is important for dilating of the pharyngeal area. The importance of the genioglossus' role in dilating the airway has been extensively researched in the area of sleep apnea and will be discussed later in this chapter [75–79].

In addition to the genioglossus's role in dilating the airway, there has been a body of literature which states the tongue's role in temporomandibular (TMJ) disorders, serving as a way to mediate or reduce the severity of the disorder. Schmidt and colleagues cite the use of the tongue as a treatment option as minimizing muscle activity and thereby reducing pain in the orofacial area; specifically by placing the tongue in a position of "rest" will maximize relaxation and subsequently reduce muscle-related pain in the TMJ [80]. Optimal tongue placement providing a "rest" position suggests that it should be positioned on the floor of the mouth. Evidence of this optimal tongue "rest" position cites decreased EMG activity in the right masseter, suprahyoid, right temporalis, and left temporalis with the tongue on the floor of the mouth versus against the hard palate [80]. To a degree, others have supported this, finding decreased activity in the anterior temporalis and suprahyoid with the tongue on the floor of the mouth versus on the hard palate yet with an increase in masseter EMG activity with the tongue on the floor versus the hard palate [81]. Yet, before the research of how the genioglossus elicits effects in the body, there must be an understanding of mechanisms involved with innervating the tongue muscle and its reflex response.

Miller [69] states that the tongue, in order to operate optimally, receives complex somatosensory input via the central nervous system, resulting in both complex and simple reflex actions [69]. Initial animal and human research to more recent research supports this hypothesis [69, 82–87]. With the animal model, Lowe and Sessle cite the interaction between the jaw and tongue when they opened the cat jaw as little as 4 mm, resulting in genioglossus activity. This outcome led to their conclusion that the temporomandibular joint significantly affects the activity of the tongue due to reflexes originating in orofacial regions [88]. In earlier human research, Weber and Smith [87] stated a reflex exists between the jaw, tongue, and lip by demonstrating increased EMG activity of the masseter, orbicularis oris inferior, and the genioglossus with mechanical stimulation [87]. In the human model, Takata and colleagues found genioglossus and orbicularis oris EMG activity increased with jaw opening and ceased with jaw closing during gum chewing, suggesting the link between the tongue, lip, and jaw [89]. Hiyama's laboratory also found similar outcomes with EMG activity of the genioglossus, with EMG increasing during jaw opening. Yet, they also found increases during jaw closing. They state that this may be due to the hypoglossal nerve which innervates the tongue protruding muscle, thus resulting in EMG activity in both the opening and closing phases of the jaw. They hypothesize that this collaboration of activity between the jaw and tongue would not be explained by a sequential reflex response but possibly preset into the central nervous system within the lower brain stem [67]. Miller [69] explains these complex oral reflexes as it relates to the genioglossus, stating that either via the lingual nerve or mechanical stimulation of the tongue will result in a potential excitation or inhibition in various tongue muscles [69]. In addition, Miller cites that this interaction between the tongue and jaw plays a critical role in the function of the pharyngeal pathway during respiration; specifically that protrusion of the tongue muscle will function to open in the pharyngeal airway [69].

The importance of tongue muscle placement has been cited as playing a key role in the opening of the pharyngeal area, with sleep apneic studies citing a forward shift of the mandible and subsequent forward protrusion of the tongue using sleep apneic mouthpieces designed to promote enhanced breathing mechanics [75, 76, 90, 91]. These devices have been shown to increase the pharyngeal area, with Kyung and colleagues citing a 19% improvement in cross-sectional area of the retroglossal (defined as the back of the tongue to the wall of the pharynx) area of the pharynx using a 75% mandibular advancement mouthpiece [91]. Mann and colleagues cited increases in the diameter of the hypopharyngeal area with genioglossal stimulation, resulting in a mean 133% increase from baseline [68]. Earlier research cited that contracting the genioglossus results in pulling the base of the tongue down and forward, with later researcher citing that this occurs with the help of the protruder muscles, which will subsequently open the pharyngeal area [92, 93]. To clarify how this occurs, Saboisky et al. [71] cited a complexity of networks linking the hypoglossal motoneurons which innervate the genioglossus. They cite increased genioglossus discharge rates during both inspiration and expiration thereby leading to tongue protrusion [71]. In addition, research has shown that the number of hypoglossal motoneurons will also be affected by exercise, citing an increased number of these motoneurons activated with increased exercise intensity, resulting in increased EMG activity of the genioglossus [94].

### 13.3.2 Role of Clenching

In addition to the important role the genioglossus plays in dilating the airway as innervated by the hypoglossal motoneurons, research has also examined the effect that clenching has on the EMG activity of this muscle. Firstly, researchers have cited an increase in EMG activity in the genioglossus with mild to maximal clenching during non-exercise protocols [86, 95]. Valdés and colleagues cite a link between the masseter while clenching and its effect on the tongue, noting the interaction using 30 healthy subjects with no current or past pain in the TMJ, mouth, or tongue. In measuring the EMG activity of the masseter and temporalis during clenching and swallowing, they cited significantly lower EMG activity in the masseter during clenching with the tongue on the floor of the mouth versus on the hard palate, this being explained by the effect the tongue creates when placed on the floor of the mouth, against the mandibular, lingual side of the incisors, which consequently linked to the masticatory muscles [86]. Igarashi then demonstrated outcomes in the genioglossus during clenching, finding an increase in EMG activity of the genioglossus during clenching [96]. Finally, as it relates to force production, placement of the tongue has also been cited as an important factor. Saboisky et al. [97] found that optimal position for the tongue resulting in the greatest tongue force production is when it is retracted between 12 and 32 mm, with the mean maximal force being 28.3 N at 24 mm and the lowest forces (14.9 N) produced with tongue protrusion at 12 mm [97]. Not only did they find increased force production but also cited in a significant decrease in breathing rate with the tongue on the floor of the mouth (15.47 BPM) versus the tongue on the roof of the mouth (16.15 BPM, $p = 0.023$). These findings support observational studies in our laboratory with various mouthguard/mouthpieces utilized during exercise (versus at rest as in the Saboisky et al. study) and will be discussed later in this chapter.

Not only has clenching been cited to effect genioglossus activity and masticatory muscles, clenching has also been shown to affect cerebral activity in activation of the cortical areas in the brain, thereby affecting the hormone response [60, 61, 98–100]. As cited earlier, studies have cited decreases in cortisol levels with both clenching and chewing, with and without physical activity [49, 50, 100]. Yet what mechanism can explain the purported improvements in hormone levels with clenching? A rat model may explain the potential mechanisms that occur with a reduced stress response during clenching. Researchers have cited that restrained and stressed rats, when biting on a stick, had reductions in corticotrophin releasing factor and c-Fos in the hypothalamus which may be modulated by suppression of extracellular signal-regulated protein kinase 1/2 (pERK 1/2) in the paraventricular nucleus [60, 61, 101]. This link between the hypothalamus and the involvement in the jaw muscle via clenching may be explained by neuronal projections from the lateral hypothalamic connecting to the trigeminal motor nucleus in the rat model [102]. In addition, it was observed that the trigeminal motor nucleus is innervated by corticotropin-releasing factor-immunoreactive fibers within the amygdala, providing another explanation of effects on hormonal response during clenching [102].

Yet rat models cannot completely explain the stress response mechanisms involved in humans during chewing and clenching; thus, researchers use functional magnetic resonance imaging or positron-emission tomography to assess cortical activity and blood flow dynamics during clenching and chewing which have been cited to be a valid measures of assessing these tasks [98, 99, 103–106]. Momose et al. [105] demonstrated mastication increased cerebral blood flow in the sensorimotor cortex by approximately 26.5% during clenching [105]. Later studies cited significantly increased middle cerebral blood flow and significant activation of the sensorimotor cortex with clenching versus other tasks such as gum chewing and a hand motor task [98, 103]. Research also cites that activation within the dorsolateral prefrontal cortex (DLPFC, an area in the cerebral cortex) is most likely dependent on continuous teeth contact as occurs during clenching, and that intensity of the clenching most likely influences that magnitude of the cerebral activity within the sensorimotor cortex (area in cerebral cortex responsible for motor function) [99, 107]. Qin et al. [108] cited that the function of the DLPFC is likely affected by the HPA axis by decreasing levels of the catecholamines [108]. These findings are significant as it relates to mouthpiece use during exercise as they provide potential explanations for the cited decreases in cortisol and lactate with mouthpiece use during exercise [49–51, 58, 59]. Thus, enhanced cerebral blood flow may be a key piece in understanding these effects, with researchers citing improved cerebral blood flow rate when subjects are in a mandibular physiologic rest position [109]. Research by Otsuka and colleagues demonstrated how an experimentally induced retrusive mandibular position using a splint (defined as placing the mandible in a more backward position) resulted in an activation of the hypothalamus during clenching in two of eight subjects [110]. Though this evidence is not sufficient to make any definitive links between malocclusion and activation of the hypothalamus and subsequent stress response, it is another step in understanding a mechanism that could explain the cortisol response during exercise with a mouthpiece, a mouthpiece which has been cited as placing the mandible in a more forward mandibular position [48–50, 58]. In closing, more recent research aims to elucidate how increased cerebral blood flow could affect the hypothalamic response from stress with subsequent hormonal production such as cortisol. Miyake et al. [104] demonstrate that biting during stress and its effect on the hypothalamic response appears to be mediated by nitric oxide levels, specifically with biting resulting in decreased levels of nitric oxide versus not biting which leads to elevated nitric oxide levels [104]. They surmise that masticatory activity (biting down) during physiological stress results in an anti-stress response that may be facilitated by nitric oxide in the brain in which nitric oxide acts as an amplifier or feedback mechanism for neuronal activity during stress [104].

13

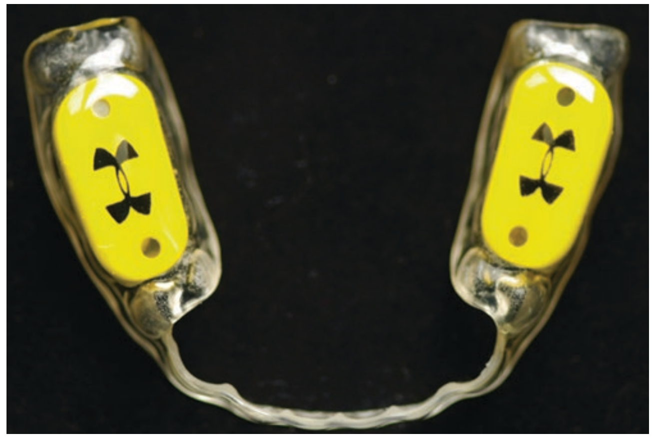
Although the interaction between the mandible and tongue has been clearly established in the literature, the relationship between such a reflex and effect on whole-body movement is sparsely cited. However, some research suggests that concurrent activation potentiation is the result of the jaw-repositioning and subsequent mandibular muscle contraction which thereby affect neuromuscular outcomes during exercise [42, 91]. Ebben first defined this phenomenon in his review of concurrent activation potentiation which he defined as an interaction between the Jendrassik maneuver, a type of remote voluntary contraction, in which individuals can increase the strength of reflexes by clenching their teeth and motor overflow, referring to communication between cortical areas through various parts of the brain [111]. In essence, Ebben cites literature suggesting that remote voluntary contractions acting through the H reflex will positively affect lower body musculature [111]. Examples of this phenomenon can be found in studies that show an increase in the soleus H reflex, which is described as a measure of the excitation of the spinal monosynaptic reflex in humans, during clenching, with a strong correlation in the increase of EMG activity of the masseter [112, 113]. The conclusion is that such a clenching response would be beneficial during stabilizing posture and improving fluidity of movement during muscular contraction, this being due to enhanced H reflex of the leg muscles with a concurrent reduction of the reciprocal Ia inhibition [113].

### 13.3.3 Genioglossus and Clenching and Involvement of the Mouthguard

Although there is substantial evidence of the importance of the genioglossus, as well as the effect of clenching, how does this relate to mouthguard use during exercise? Firstly, an appliance provides some type of stimulus to the tongue muscle as well as an increased opportunity for the individual to clench during exercise. Hidaka and colleagues found that with increased clenching, there was a resulting shift on the bite force such that there was a more balanced bite force (with balance bite force being defined as force placed on all occlusal contacts). They hypothesized that this outcome may be a mechanism which prevents damage to teeth and to the temporomandibular joint [114]. Thus, using a mouthguard may improve clenching capacity thereby resulting in changes of cerebral blood flow and hypothalamic response as noted earlier. Secondly, the design of the mouthguard is important to understand in light of its potential effect on the tongue muscle, i.e., a mandibular mouthguard versus a maxillary mouthguard differs in its impact on the tongue and may thereby affect outcomes associated with the genioglossus.

Accordingly, reviewing what is known will enable researchers to clarify the precise mechanism(s) that may be occurring with mouthguard/mouthpiece use during exercise. With research as it relates to the genioglossus and clenching effect, there is compelling evidence that a few aspects of mouthguard/mouthpiece use should be more carefully studied. Firstly, understanding the types of oral appliances used in previous and current studies should be evaluated for potential effect on vertical displacement, then understanding how the angle of the bite created by the mouthpieces affects vertical displacement as it relates to the TMJ should be reviewed. Murakami and colleagues who used magnetic resonance imaging (MRI) with and without a mouthguard made of a EVA material (Erkosoft Erkodent, Pfalzgrafenweiler, Germany) studied 26 healthy subjects wearing two different EVA mouthguards; MG1 created a 3 mm vertical displacement, while MG2 created a 6 mm vertical displacement [115]. It was cited in both mouthguard conditions that the condyle moved backward and upward in all subjects during clenching yet to a lesser extent than the non-clenching condition. During the non-clenching, MG1 condition, the condyle moved 1.15 mm downward and 2.10 mm forward, with MG2 moving 2.10 mm downward and 2.33 forward, a statistical difference between MG1 and MG2 [115]. Thus vertical displacement alone seems to create varying degrees of condylar distance from the mandibular fossa. A case study assessing the positioning of the temporomandibular joint between the boil and bite UA mouthguard, the custom mouthguard, and no-mouthpiece condition showed that the boil and bite provided greater distance between the condyle and the mandibular fossa, the custom provided lesser distance, and the no-mouthpiece condition resulted in the least distance created between the condyle and fossa, which may be due to the type of vertical displacement created by the two mouthpieces and no-mouthpiece conditions (unpublished data).

In addition to vertical displacement affecting the TMJ, the degrees of vertical displacement created by the different mouthpieces may also have an effect on the genioglossus. Appliances used in the 1970s and 1980s were defined as the MORA devices (◘ Fig. 13.1). These devices are narrowly described in the literature but appear to all have a hard resin which results in increased vertical displacement and subsequent effect on the tongue. In viewing a model of a mouthguard in an article by Stenger, the subject has a substantial vertical displacement, with the tongue visibly elevated to the roof of the mouth and the tongue tip touching the interior teeth. However, it is unclear from the photo the degree of mandibular or vertical displacement [27]. Welch and colleagues state that "the appliances (MORA) were designed to maintain this programmed centric relation/occlusion at the previously determined increased vertical dimension" [29]. Thus, in most cases, this vertical dimension, though vaguely described, involves manually manipulating the patient to find increased muscle force production and citing the position of the mandible along with the increased vertical displacement that coincides with the optimal force production and thereby temporomandibular repositioning [32]. However, it was doubtful that tongue placement was considered in any of these studies. In addition to the potential effect vertical displacement can create on the tongue, some of the appliances studied cited increased mandibular forward placement when utilized in the mouth of the wearer, yet this was never confirmed with CBCT scans which were not available during the time of these studies [26, 29, 33, 116]. As stated earlier in this review, it is well researched within sleep apneic research that forward mandibular placement devices are utilized to open the airway and improve breathing for this population [75–77]. Consequently, due to findings in the sleep apneic literature, Garner and McDivitt studied a mandibular forward placement mouthpiece (as advertised by the company, Bite Tech, Inc.) (◘ Fig. 13.4) Utilizing CBCT scans they studied the effects of mouthguard use on exercise performance and found a significant 9% increase in width and an increase in diameter of the oropharynx with mouthpiece use versus a no-mouthpiece condition [51] (◘ Fig. 13.3). They surmised



◘ **Fig. 13.4** Appliance used in airway study. Under Armour performance mouthpiece used in the CT studies of airway. Lower appliance used when protection is not required

that the enhanced airway openings as seen in the CBCT scans with mouthpiece use signified forward mandibular placement and thereby explained improvements in lactate [51]. In addition they suggested a link between the mandibular placement of this mouthpiece and effect on the genioglossus, citing increased activation of the genioglossus in a case study referred to in a published study [48]. Thus, understanding the degree of mandibular advancement and vertical displacement and subsequent effect on the genioglossus in future mouthguard/mouthpiece studies should further clarify our understanding of the mouthpiece effect.

Finally the design of the mouthpiece and how it affects tongue placement may be of importance understanding outcome differences within these studies. Francis and Brasher [56] cited that in comparing their three mouthguards, that the one which resulted in the most significant improved ventilation was a bimaxillary mouthguard with a small breathing hole. Bailey and colleagues also noted significantly lowered lactate levels and ventilation at 200 W and maximum workloads with a vented mouthguard versus no-mouthguard condition and a traditional boil and bite maxillary mouthguard [46]. While Francis and Basher cited that the improvements in ventilation in their study may be due to a type of pursed-lip breathing, Bailey and colleagues stated that plausible differences in ventilation could be due to the construction of the mouthguard [46, 56]. Garner and colleagues also hypothesized that a type of pursed-lip breathing could be occurring with their lower mouthpieces as a potential explanation in the decreased respiratory rate noted in the lower custom and boil and bite mouthpieces [48–50]. As mentioned earlier, this type of breathing leads to improved ventilation in COPD patients both at rest and during exercise [117, 118], and this being may be linked to displacement of the tongue. However, Garrod and colleagues stated that they were unable to explain the mechanisms for the improvements in respiratory parameters with pursed-lip breathing [117]. Garner hypothesized that the decreased vertical displacement created by the mouthpiece used in the study may have resulted in less space to allow air in and out of the mouth, causing subjects to contract the tongue, thereby opening the airway and in turn explaining the respiratory improvements in this study [48]. Nevertheless, studies to confirm a potential link between pursed-lip breathing and tongue placement as well as other mechanisms to explain respiratory improvements with pursed-lip breathing should be explored.

In addition to understanding the effects of the mouthguard design on tongue placement and activity, the degree of even occlusal contact and subsequent effect on clenching may be of importance in studying effect on exercise performance. Murakami and colleagues stated that the surface of the mouthguards was adjusted such that there was an even bite surface between the mouthguard and the opposing occlusal surface [115]. This is supported by Pae and colleagues who noted that in creating their mouthguard, "all teeth contacted equally at maximal intercuspal positions" a study in which they cited significant improvements in club head speed and driving distance in professional golfers [119]. Similar to these findings was a study by Lee and colleagues in which they utilized a MORA device but noted that the fit of the appliance required that all teeth have even contact [35]. Their findings revealed significant EMG measurements in isometric improvements with the following muscle groups: sternocleidomastoid muscles, cervical and lumbar erector spinae, upper trapezius, biceps, triceps, rectus abdominis, and internal and external oblique [35]. Furthermore, the studies by Garner and colleagues primarily used a mandibular device (Bite Tech mouthpiece, later branded as the Under Armour Mouthguard Performance Mouthwear) which incorporated bite pads that varied between the custom mouthpiece and the boil and bite mouthpiece. Both mouthpieces had a 2 mm posterior to 1 mm anterior wedge; however, the custom mouthpieces did not have a slope, while the boil and bite mouthpieces utilized a bite surface that was continuous and provided even contact with the occlusal surfaces [48]. That study cited that two of the mouthpieces tested had elevated pads which would have prevented even contact between all teeth and could have negatively affected the performance parameters [48]. Thus, more research should focus on the impact of vertical displacement with a continuous and noncontinuous bite surface between the mouthguard and the opposing occlusal surface and its impact clenching intensity.

In understanding both the degree of vertical displacement and occlusal contact, the types of materials used in the various mouthguards should also be considered within those earlier studies. Smith provided a photograph and description of the devices (described as a wax bite that "locks the jaws" into their ideal position) used in his study which found significant improvements in strength measures in professional football players with the wax bite [26, 120]. Yet he also cited that his MORA mouthguards actually transferred the bite pressures, placing more pressure on the molars versus the incisors [26]. This is important as evaluation of the mouthguard research reveals that differences may be in even contact between all teeth.

13

### Oral Conditions that May Improve Performance

- Jaw position
  - Vertical
  - Anteroposterior (anterior)
- Tongue position
  - Anterior/inferior
- Clenching: balanced and complete occlusion
  - Increased cerebral blood flow
  - Decreased cortisol

Jakush concedes that there were differences between the lower MORA devices and the maxillary MORA devices, with the maxillary mouthguard MORA devices using a softer material than the typically hard acrylic resin in the mandibular MORA devices [32]. In revisiting the photo by Stenger, the type of material typically used for these mandibular MORA devices (hard acrylic resin), it is apparent that the vertical displacement (greater jaw opening) is accentuated [27].

In more recent research, the materials used in the basic three types of mouthguards, custom mouthguard, boil and bite mouthguard(s), and placebo condition, vary among researchers and may affect vertical displacement and occlusal contact. For example, Francis and Basher used moldable over-the-counter upper and lower mouthguards [56]. The difference with these and the MORA appliances is the contact between the upper and lower teeth would be less rigid, due to the nature of the rubberized material in the moldable mouthguards. However, it is unclear, how much vertical dimension is created with these. Yet it may be surmised, due to the hinge component of the bimaxillary mouthguard, that this mouthguard would create an increase in vertical dimension. The mouthguards, as described by both Arent and colleagues and Pae and colleagues, consisted of an ethylene vinyl acetate (EVA) polymer [37, 119].

### MG Design Considerations for Performance Enhancement

- Mandibular or bimaxillary
  - Optimize tongue position
  - Pursed-lip breathing
- Balanced occlusal contacts
  - Enhances clenching ability
- Thickness
- Materials used
  - Hard vs soft

While the Under Armour custom-fit lower mouthpiece incorporates a polypropylene material versus the more common ethylene vinyl acetate used as a comparison mouthpiece in Garner study [48]. Gould and colleagues cited that the use of EVA is most common within mouthguards due to the fact that EVA is accessible, is easy to work with, and has the appropriate mechanical properties to meet the needs of the product [121]. Gould found that EVA materials in mouthguards ranged from 67.6 to 81.4 on the Shore A hardness factor at 37 °C [121]. Polyurethane bite pads have not been tested in the Under Armour mouthpieces for durometer values, but these plastics typically have lower durometers than the acrylics and EVA materials utilized in much of the mouthguard research [122]. Thus, due to the different outcomes for many of the studies as noted previously, the durometer of the material may be one area of future study to clarify its effect on outcomes during exercise performance. It should be noted that those outcomes in the Francis and Basher study were similar to those in the Garner studies, with these two groups of researchers using lower durometer materials which this author hypothesizes as creating less vertical dimension with a subsequent forward placement of the tongue.

### 13.3.4 Exercise Physiology and Practical Applications of the Science

Although there has been a great deal of research presented in this review to suggest the importance of tongue placement and the clenching effect, it is important to link the science to the application of exercise and use of the mouthpiece. Firstly, fatigue during exercise has many causes ranging from peripheral to central nervous system fatigue. Yet, one site of fatigue with high-intensity exercise relates to the production of lactic acid. Lactic acid is produced during higher-intensity exercise in which the glycolytic pathway is heavily called upon to produce energy at a high rate (see Fig. 13.5). An end product of this glycolytic pathway is the production of lactic acid. Lactic acid then dissociates into lactate and hydrogen ions. The increased production of hydrogen is one of the fatiguing aspects of higher-intensity exercise as hydrogens cause the blood to become more acidic.

**Why Lactate Matters to Athletes**

- *Pyruvate* is converted to *lactate* in the absence of *oxygen*.
- When exercise intensity increases, the body is less able to obtain the needed oxygen to exercise. Thus, it relies on the glycolytic pathway to produce energy. This pathway does not require oxygen but does produce a greater amount of lactate.
- The breakdown products:
  - Lactic acid → Lactate + $H^+$ ions
- Lactate production must equal lactate clearance; if not, the results are fatigue (OBLA = 4 mmol/L).
- *The $H^+$ ions are considered by many to be the cause of fatigue*:
  - It binds with troponin to inhibit muscle contraction.
  - It inhibits phosphofructokinase, important enzyme in glycolysis.

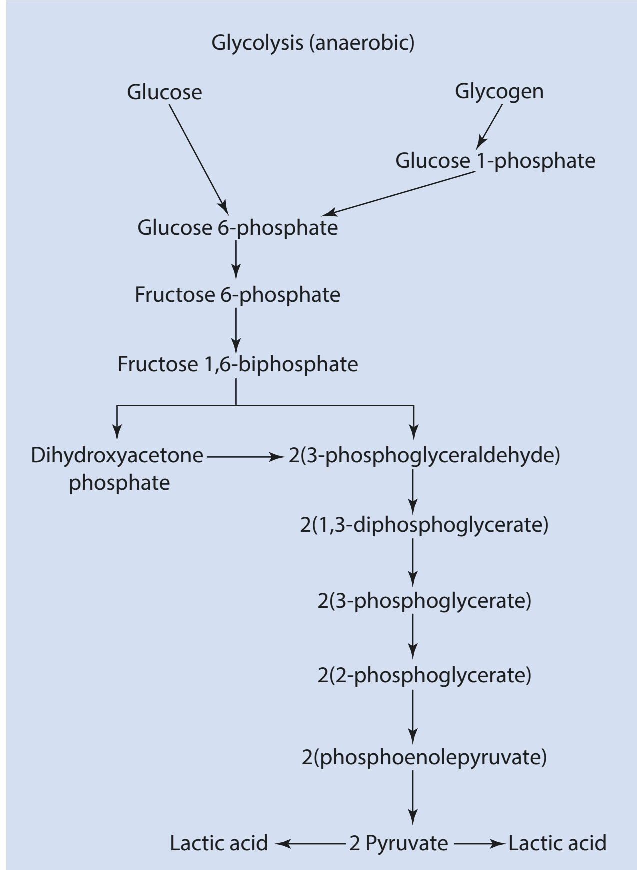



**Fig. 13.5** Glycolysis. Glycolysis is a catabolic pathway in the cytoplasm of cells where glucose is broken down into two molecules of pyruvate+ 2 ATP molecules and 2 NADH+ $H^+$. In times of depleted oxygen, glycolysis becomes the main pathway for energy production and pyruvate is converted to lactic acid

The acid-base balance is thus tightly regulated by mechanisms in the body in order to keep the pH in a tolerable range. One mechanism to regulate the acidity of the blood is the bicarbonate ion, which is formed when carbon dioxide combines with water forming carbonic acid that then quickly loses a hydrogen becoming bicarbonate. It has been cited that increased exhalation of carbon dioxide leads to decreased lactate acid which is precisely what was found in the studies by Garner and colleagues with the use of a mouthpiece during exercise. They cited that the improved breathing patterns created by the mouthpiece resulted in the increased carbon dioxide production noted in their study [49, 50]. In this study, they linked the improved carbon dioxide response to the decreased lactate cited in an earlier study [49–51]. Thus, the mouthpiece effect has been associated with changes in breathing patterns which in turn affect exercise outcomes such as lactate.

Another aspect of the mouthpiece effect is the decrease in cortisol during intense resistance exercise. During exercise, particularly during intense resistance exercise for approximately an hour, there is an increase in cortisol [62, 65, 123]. Cortisol is the product of the hypothalamic-pituitary axis (see Fig. 13.6),

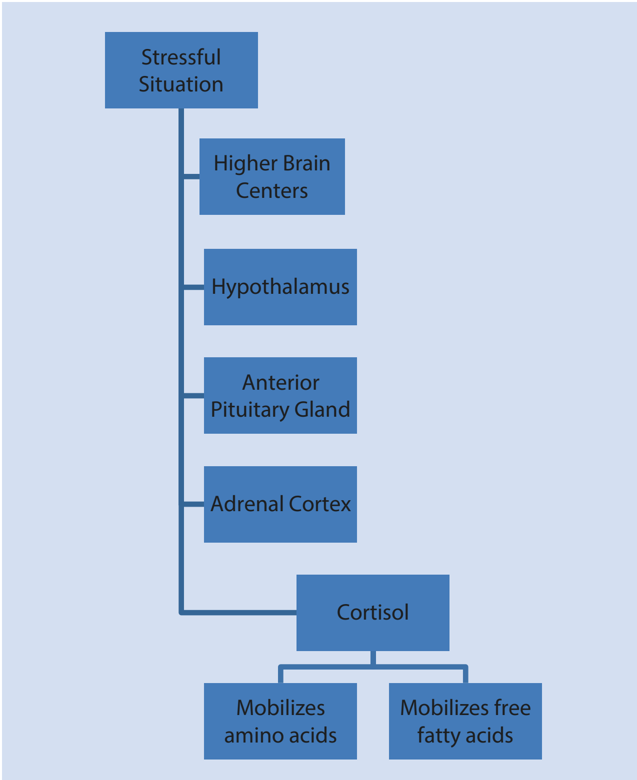



**Fig. 13.6** HPA axis. HPA axis schematic shows how stress stimulates the release of cortisol



with prolonged elevated levels being linked to a decreased protein repair and increased protein breakdown, negatively affecting the function and condition of the muscle [65].

**Effects on the Body from Excess Cortisol or Stress**

- Gluconeogenesis at the expense of muscle proteins
- Decreased immune function
- Decreased metabolism
- Depression
- Hypertension
- Chronic fatigue
- Sleep deprivation
- Migraines
- Tunnel vision

Thus, the findings by Garner and colleagues and Dudgeon and colleagues that the mouthpiece has a significant reduction in cortisol after intensive resistance exercise are critical to examine and seek to replicate [49, 50, 57]. Their findings support those found in the literature with the rat model as it relates to a reduction of stress parameters when biting on a stick and may be linked to increased cerebral blood flow when clenching [60, 61, 99, 100]. Thus interventions such as the use of a mouthpiece during exercise may affect both breathing mechanisms, thereby affecting fatigue pathways as well as potentially affecting the hormone response due to clenching during exercise.

## 13.4 Conclusions

Findings during this review reveal that there are a variety of acute outcomes with mouthguard/mouthpiece use during exercise. Yet, it is meaningful to understand that making comparisons between studies is difficult due to the complexities of the issues which include the type of mouthguard/mouthpiece used, exercise protocol, sufficient sample size, and techniques utilized to assess differences. Instead of making conclusive remarks about the use of a mouthguard, it is the goal of this review to raise appropriate questions to enable researchers to illuminate appropriate avenues to explore as it relates to mouthguard use during exercise. Finally, in noting the acute improvements cited in many of these studies, results denote seemingly minimal improvements, i.e., on average ranging from 3 to 10%. Thus dental professionals should examine if these improvements are meaningful for the client in fitting the individual with an appliance. However, it is the belief of the author that research in this area may be more promising in understanding the consistent use of mouthpiece/mouthguards during and post exercise in light of the research related to lactate and cortisol. Research should focus on impact on recovery and subsequent training sessions. If, as the research suggests, there are reductions in lactate and cortisol post-training sessions as indicated in the studies, then the impact on physiological recovery is remarkable. We know that elevated cortisol impairs muscle recovery and immune function, while elevated lactate impedes training by prolonging post-oxygen exercise consumption enabling the body to rid itself of elevated hydrogen ions associated with lactate [124]. Thus research should explore use of mouthguards/mouthpieces during and after exercise to determine training impact on the individual over a longer period of time. In addition, as practitioners, many of the common complaints of nonuse of the mouthguard are attributed to the negative effect athletes feel it has on their performance, as it relates to breathing patterns. The research has clearly cited that there appears to be no negative impact on performance, with some of the research citing an improvement in breathing parameters. Thus, as dental professionals, the research can be referred to as it relates to these common complaints made by the athlete and therefore encourage mouthguard use during play.

## 13.5 Future Research

The question remains, “Where do we go from here?” As intimated with this short review of the mouthguards, tongue placement, cortical communication via clenching, and resultant effect on hormones, the human body during rest and exercise involves a complexity of interactions which are not yet fully understood. As Chakfa and colleagues stated in establishing a link between increased isometric strength with application of increased vertical dimension using bite plates, "as data increase, the physiological mechanism underlying this phenomenon will become clear" [125]. Indeed the goal of this chapter was to summarize the research in the area of mouthguard/mouthpiece use with exercise and subsequently provide potential physiological mechanisms to explain these outcomes. With evidence in various disciplines, it is difficult to refute the mouthpiece effect during exercise. Therefore, researchers should be encouraged to investigate to what extent the mouthpiece plays during different types of sports, studying a variety of athletes, ranging from recreational to elite, male and female, and the types of mouthpieces utilized to create the effect. Specific areas to study include assessing the effect of clenching during exercise and its effect on cerebral blood flow while including an assessment of endocrine functions with and without a mouthpiece. In addition, using biological markers connecting to the response of clenching with mouthguard use appears to be a promising avenue, unlocking the mystery of the mouthguard effect which has eluded researchers over the years. Using biological markers helps minimize bias or the placebo effect that may accompany the use of a mouthguard during exercise performance. Thus, gaining clarity in this area of science would enable researchers to further elucidate the mouthguard effect during performance. As researchers take into account the interactions of these various factors, then the answers may become clearer for the practitioner to prescribe the correct mouthguard/mouthpiece which offers protection, as well as performance improvements.

## References


1. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. Sleep Breath. 2007;11(1):1–22.
2. Andrews SA, Sam M, Krishman R, Ramesh M, Kunjappan SM. Surgical management of a large cleft palate in a Pierre Robin sequence: a case report and review of literature. J Pharm Bioallied Sci. 2015;7(2):S718–20.
3. Roettger M. Performance enhancement and oral appliances. Compend Contin Educ Dent. 2009;30:4–8.
4. Hughston JC. Prevention of dental injuries in sports. Am J Sports Med. 1980;8:61–2.
5. Glendor U. Aetiology and risk factors related to traumatic dental injuries - a review of literature. Dent Traumatol. 2009;25:19–31.
6. Heintz W. Mouth protectors: a progress report. J Am Dent Assoc. 1968;77:632–6.
7. Newsome PR, Tran DC, Cooke MS. The role of the mouthguard in the prevention of sports-related dental injuries: a review. Int J Paediatr Dent. 2001;11:396–404.
8. Ranalli DN, Demas PN. Orofacial injuries from sport preventive measures for sports medicine. Sports Med. 2002;32:409–18.
9. ADA Council on Access, P. A. I. R. & ADA Council on Scientific Affairs. Using mouthguards to reduce the incidence and severity of sports-related oral injuries. J Am Dent Assoc. 2006;137:1712–20.
10. National Federation of State High Schools and Sports Medicine Advisory Committee. Position statement and recommendations for mouthguard use in sports. 2014. http://www.nfhs.org/sports-resource-content/position-statement-and-recommendations-for-mouthguard-use-in-sports/.
11. NCAA. In: Klossner D, editor. 2013-2014 NCAA sports medicine handbook. 24th ed. Indianapolis: NCAA; 2013. p. 113–4.
12. de la Cruz GG, Knapik JJ, Birk MG. Evaluation of mouthguards for the prevention of orofacial injuries during United States Army basic military training. Dent Traumatol. 2008;24:86–90.
13. Kerr I. Mouthguards for the prevention of injuries in contact sports. Sports Med. 1986;3:415–27.
14. Cohen A, Borish A. Mouth protector project for football players in Philadelphia high schools. J Am Dent Assoc. 1957;56:863–4.
15. Knapik JJ, Marshall SW, Lee RB, Darakjy SS, Jones SB, Mitchener TA, et al. Mouthguards in sport activities: history, physical properties and injury prevention effectiveness. Sports Med. 2007;37:117–44.
16. Boffano P, Boffano M, Gallesio C, Roccia F, Cignetti R, Piana R. Rugby athletes' awareness and compliance in the use of mouthguards in the North West of Italy. Dent Traumatol. 2012;28:210–3.
17. Chapman PJ, Nasser BP. Attitudes to mouthguards and prevalence of orofacial injuries in four teams competing at the second Rugby World Cup. Br J Sports Med. 1993;27:197–9.
18. Hawn KL, Visser MF, Sexton PJ. Enforcement of mouthguard use and athlete compliance in National Collegiate Athletic Association men's collegiate ice hockey competition. J Athl Train. 2002;37:204–8.
19. Berry DC, Miller MG, Leow W. Attitudes of central collegiate hockey association ice hockey players toward athletic mouthguard usage. J Public Health Dent. 2005;65:71–5.
20. Moore M. Corrective mouth guards: performance aids or expensive placebos? Phys Sportsmed. 1981;9:127–32.
21. Fonder AC. The profound effect of the 1973 Nobel Prize on dentistry. J Am Acad Func Prosthodontics. 1976;1:21–9.
22. Garabee WF Jr. Craniomandibular orthopedics and athletic performance in the long distance runner: a three year study. Basal Facts. 1981;4:77–81.
23. Kaufman RS. Case reports of TMJ repositioning to improve scoliosis and the performance of athletes. N Y State Dent J. 1980;42:206–9.
24. Gelb H, Mehta NR, Forgione AG. The relationship between jaw posture and muscular strength in sport dentistry: a reappraisal. Cranio. 1996;14:320–5.
25. Smith SD. Muscular strength correlated to jaw posture and the temporomandibular joint. N Y State Dent J. 1978;44:278–85.
26. Smith SD. Adjusting mouthguards kinesiologically in professional football players. N Y State Dent J. 1982;48:298–301.
27. Stenger JM. Physiologic dentistry with Notre Dame athletes. Basal Facts. 1977;2:8–18.
28. Yates JW, Koen TJ, Semenick DM, et al. Effect of a mandibular orthopedic repositioning appliance on muscular strength. J Am Dent Assoc. 1984;108:331–3.
29. Welch MJ, Edington DM, Ritter R. Muscular strength and temporomandibular joint repositioning. J Orthop Sports Phys Ther. 1986;7:236–9.
30. Forgione AG, Mehta NR, McQuade CF, Westcott WL. Strength and bite, part 2: testing isometric strength using MORA set to a functional criterion. Cranio. 1992;10:13–20.
31. Forgione AG, Mehta NR, Westcott WL. Strength and bite, part 1: an analytical review. Cranio. 1991;9:305–15.
32. Jakush J. Divergent views: can dental therapy enhance athletic performance? J Am Dent Assoc. 1982;104:292–8.
33. Greenberg MS, Cohen SG, Springer P, Kotwick JE, Vegso JJ. Mandibular position and upper body strength: a controlled clinical trial. J Am Dent Assoc. 1981;103:576–9.
34. Vesgo JJ, Kotwich JE, Cohen SG, Greenberg MS. The effect of an orthopaedic intraoral mandibular appliance on upper body strength. Med Sci Sports Exerc. 1981;13:115–6.
35. Lee SY, Hong MH, Park MC, Choi SM. Effect of the mandibular orthopedic repositioning appliance on trunk and upper limb muscle activation during maximum isometric contraction. J Phys Ther Sci. 2013;25:1387–9.
36. Shock Doctor, Inc. We give technology more bite. 2015. https://www.shockdoctor.com/technology/mouthguard. Retrieved 22 Sep 2015.

37. Arent SM, McKenna J, Golem DL. Effects of neuromuscular dentistry-designed mouthguard on muscular endurance and anaerobic power. J Comp Physiol. 2010;7:73–9.
38. Dunn-Lewis C, Luk HY, Comstock BA, et al. The effects of a customized over-the-counter mouth guard on neuromuscular force and power production in trained men and women. J Strength Cond Res. 2012;26:1085–93.
39. Durante-Pereira DMV, del Rey-Santamaria M, Javierre-Garces C, et al. Wearability and physiological effects of custom-fitted vs self-adapted mouthguards. Dent Traumatol. 2008;24:439–42.
40. Ebben WP, Leigh DH, Geiser CF. The effect of remote voluntary contractions on knee extensor torque. Med Sci Sports Exerc. 2008;40:1805–9.
41. Morales J, Busca B, Solana-Tramunt M, et al. Acute effects of jaw clenching using a customized mouthguard on anaerobic ability and ventilatory flows. Hum Mov Sci. 2015;44:270–6.
42. Busca B, Morales J, Solana-Tramunt M, Miro A, Garcia M. Effects of jaw clenching while wearing a customized bite-aligning mouthpiece on strength in healthy young men. J Strength Cond Res. 2015;30(4):1102–10. https://doi.org/10.1519/JSC.0000000000001192.
43. Allen CR, Dabbs NC, Zachary CS, et al. The acute effect of a commercial bite-aligning mouthpiece on strength and power in recreationally trained men. J Strength Cond Res. 2014;28:499–503.
44. Golem D, Arent SM. Effects of jaw-repositioning mouthguards on multiple aspects of physical performance. Rutgers: Rutgers University; 2012.
45. Drum SN, Swisher A, Buchanan CA, et al. Effects of a custom bite-aligning mouth guard on performance in college football players. J Strength Cond Res. 2015;30(5):1409–15. https://doi.org/10.1519/JSC.0000000000001235.
46. Bailey SP, Willauer TJ, Balilionis G, Wilson LE, Salley JT, Bailey EK, et al. Effects of an over-the-counter vented mouthguard on cardiorespiratory responses to exercise and physical agility. J Strength Cond Res. 2015;29:678–84.
47. Bourdin M, Brunet-Patru I, Hager PE, Allard Y, Hager JP, Lacour JR, et al. Influence of maxillary mouthguards on physiological parameters. Med Sci Sports Exerc. 2006;38:1500–4.
48. Garner DP. Effects of various mouthpieces on respiratory physiology during steady state exercise in college-aged subjects. Gen Dent. 2015;63:30–4.
49. Garner DP, Dudgeon WD, McDivitt E. The effects of mouthpiece use on cortisol levels during an intense bout of resistance exercise. J Strength Cond Res. 2011;25:2866–71.
50. Garner DP, Dudgeon WD, Scheett TP, et al. The effects of mouthpiece use on gas exchange parameters during steady-state exercise in college-aged men and women. J Am Dent Assoc. 2011;142:1041–7.
51. Garner DP, McDivitt E. Effects of mouthpiece use on airway openings and lactate levels in healthy college males. Compend Contin Educ Dent. 2009;30(2):9–13.
52. Garner DP, Miskimin J. Effects of mouthpiece use on auditory and visual reaction time in college males and females. Compend Contin Educ Dent. 2009;30(2):14–7.
53. Gebauer DP, Williamson RA, Wallman KE, Dawson BT. The effect of mouthguard design on respiratory function in athletes. Clin J Sport Med. 2011;21:95–100.
54. Rapisura KP, Coburn JW, Borwn LE, Kersey RD. Physiological variables and mouthguard use in women during exercise. J Strength Cond Res. 2010;24:1263–8.
55. von Arx T, Flury R, Tschan J, Buergin W, Geiser T. Exercise capacity in athletes with mouthguards. Int J Sports Med. 2008;29:435–8.
56. Francis KT, Brasher J. Physiological effects of wearing mouthguards. Br J Sports Med. 1991;25:227–31.
57. Dudgeon, WD, Buchanan, LA, Strickland, AE, Scheett, TP, and Garner, DP, Mouthpiece use during heavy resistance exercise affects serum cortisol and lactate. Cogent Med 2017;4:1403728.
58. Garner DP, McDivitt EJ. The effects of mouthpiece use on salivary cortisol and lactate levels during exercise. Med Sci Sports Exerc. 2009;41:S448.
59. Garner DP, McDivitt EJ. Effects of mouthpiece use on lactate and cortisol levels during and after 30 minutes of treadmill running. Open Access J Sci Technol. 2015;3:101148. https://doi.org/10.11131/2015/101148.
60. Hori N, Yuyama N, Tamura K. Biting suppresses stress-induced expression of corticotropin-releasing factor (CRF) in the rat hypothalamus. J Dent Res. 2004;83:124–8.
61. Sasaguri K, Kikuchi M, Hori N, et al. Suppression of stress immobilization-induced phosphorylation of ERK 1/2 by biting in the rat hypothalamic paraventricular nucleus. Neurosci Lett. 2005;383:160–4.
62. Ahtiainen JP, Pakarinen A, Kraemer WJ, Hakkinen K. Acute hormonal responses to heavy resistance exercise in strength athletes versus nonathletes. Can J Appl Physiol. 2004;29:527–43.
63. McGuigan MR, Egan AD, Foster C. Salivary cortisol responses and perceived exertion during high intensity and low intensity bouts of resistance exercise. J Sports Sci Med. 2004;3:8–15.
64. Chrousos GP. The role of stress and the hypothalamic-pituitary-adrenal axis in the pathogenesis of the metabolic syndrome: neuro-endocrine and target tissue-related causes. Int J Obes Relat Metab Disord. 2000;24:S50–5.
65. Kraemer WJ, Ratamess NA. Hormonal responses and adaptations to resistance exercise and training. Sports Med. 2005;35:339–61.
66. Fregosi RF, Ludlow CL. Activation of upper airway muscles during breathing and swallowing. J Appl Physiol. 2014;116:291–301.
67. Hiyama S, Iwamoto S, Ono T, Ishiwata Y, Kurodo T. Genioglossus muscle activity during rhythmic open-close jaw movements. J Oral Rehabil. 2000;27:664–70.
68. Mann EA, Burnett T, Cornell S, Ludlow CL. The effect of neuromuscular stimulation of the genioglossus on the hypopharyngeal airway. Laryngoscope. 2002;112:351–6.
69. Miller AJ. Oral and pharyngeal reflexes in the mammalian nervous system: their diverse range in complexity and the pivotal role of the tongue. Crit Rev Oral Biol Med. 2002;13:409–25.
70. Remmers JE. Wagging the tongue and guarding the airway: reflex control of the genioglossus. Am J Respir Crit Care Med. 2010;164:2013–4.
71. Saboisky JP, Butler JE, Fogel RB, Taylor JL, Trinder JA, White DP, et al. Tonic and phasic respiratory drives to human genioglossus motoneurons during breathing. J Neurophysiol. 2006;95:2213–21.
72. Saito H, Itoh I. Three-dimensional architecture of the intrinsic tongue muscles, particularly the longitudinal muscle, by the chemical-maceration method. Anat Sci Int. 2003;78:168–76.
73. Eastwood PR, Allison GT, Shepherd KL, Szollosi I, Hillman DR. Heterogeneous activity of the human genioglossus muscle assessed by multiple bipolar fine-wire electrodes. J Appl Physiol. 2003;94:1849–58.
74. Fogel RB, Malhotra A, Pillar G, Edwards J, Beauregard J, Shea SA, et al. Genioglossal activation in patients with obstructive sleep apnea versus control subjects. Am J Respir Crit Care Med. 2001;164:2025–30.
75. Gale DJ, Sawyer RH, Woodcock A, Stone P, Thompson R, O'Brien K. Do oral appliances enlarge the airway in patients with obstructive sleep apnoea? A prospective computerized tomographic study. Eur J Orthod. 2000;22:159–68.
76. Gao X, Otsuka R, Ono R, Honda E, Saski T, Kuroda T. Effect of titrated mandibular advancement and jaw opening in nonapneic men: a magnetic resonance imaging and cephalometric study. Am J Orthod Dentofac Orthop. 2004;125:191–9.
77. Lim J, Lasserson TJ, Fleetham J, Wright J. Oral appliances for obstructive sleep apnoea. Cochrane Database Syst Rev. 2006;4:CD004435.
78. Ryan CF, Love LL, Peat D, Fleetham JA, Lowe AA. Mandibular advancement oral appliance therapy for obstructive sleep apnoea: effect on awake calibre of the velopharynx. Thorax. 1999;54:972–7.
79. Zhao X, Liu Y, Gao Y. Three-dimensional upper-airway changes associated with various amounts of mandibular advancement in awake apnea patients. Am J Orthod Dentofac Orthop. 2008;133:661–8.
80. Schmidt JE, Carlson CR, Usery AR, Quevedo AS. Effects of tongue position on mandibular muscle activity and heart rate function. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;108:881–8.

81. Takahashi S, Kuribayashi G, Ono T, Ishiwata Y, Kurodo T. Modulation of masticatory muscle activity by tongue position. Angle Orthod. 2005;75:35–9.
82. Gerstner GE, Goldberg LJ. Genioglossus EMG activity during rhythmic jaw movements in anesthetized guinea pig. Brain Res. 1991;562:79–84.
83. Ishiwata Y, Hiyama S, Igarashi K, Ono T, Kurodo T. Human jaw-tongue reflex as revealed by intraoral surface recording. J Oral Rehabil. 1997;24:857–62.
84. Liu ZJ, Masuda Y, Inque T, Fuchihata H, Sumida A, Takada K, et al. Coordination of cortically induced rhythmic jaw and tongue movements in the rabbit. J Neurophysiol. 1993;69:569–84.
85. Liu ZJ, Shcherbatyy V, Kayalioglu M, Seifi A. Internal kinematics of the tongue in relation to muscle activity and jaw movement in the pig. J Oral Rehabil. 2009;36:660–74.
86. Valdes C, Astaburuaga F, Falace D, Ramirez V, Manns A. Effect of tongue position on masseter and temporalis electromyographic activity during swallowing and maximal voluntary clenching: a cross-sectional study. J Oral Rehabil. 2014;41:881–9.
87. Weber CM, Smith A. Reflex responses in human jaw, lip, and tongue muscles elicited by mechanical stimulation. J Speech Hear Res. 1987;30:70–9.
88. Lowe AA, Sessle BJ. Tongue activity during respiration, jaw opening, and swallowing in the cat. Can J Physiol Parmacol. 1973;51: 1009–11.
89. Takada K, Yashiro K, Sorihashi Y, Morimoto T, Sakuda M. Tongue, jaw, and lip muscle activity and jaw movement during experimental chewing efforts in man. J Dent Res. 1996;75:1598–606.
90. Johal A, Gill G, Ferman A, McLaughlin K. The effect of mandibular advancement appliances on awake upper airway and masticatory muscle activity in patients with obstructive sleep apnoea. Clin Physiol Funct Imaging. 2007;27:47–53.
91. Kyung SH, Park YC, Pae EK. Obstructive sleep apnea patients with the oral appliance experience pharyngeal size and shape changes in three dimensions. Angle Orthod. 2005;75:15–22.
92. Brouillette RT, Thach BT. A neuromuscular mechanism maintaining extrathoracic airway patency. J Appl Physiol. 1979;46:772–9.
93. Lowe AA. The neural regulation of tongue movements. Prog Neurobiol. 1980;15:295–344.
94. Walls CE, Laine CM, Kidder IJ, Bailey EF. Human hypoglossal motor unit activities in exercise. J Physiol. 2013;591:3579–90.
95. Roark AL, Glaros AG, O'Mahony AM. Effects of interocclusal appliances on EMG activity during parafunctional tooth contact. J Oral Rehabil. 2003;30:573–7.
96. Igarashi K. Neurophysiological mechanism of jaw-tongue reflex in man. J Stomatol Soc. 1996;63:108–21.
97. Saboisky JP, Luu BL, Butler JE, Gandevia SC. Effects of tongue position and lung volume on voluntary maximal tongue protrusion force in humans. Respir Physiol Neurobiol. 2015;206:61–6.
98. Hasegawa Y, Ono T, Hori K, et al. Influence of human jaw movement on cerebral blood flow. J Dent Res. 2007;86:64–8.
99. Shibusawa M, Takeda T, Nakajima K, et al. Functional near infrared spectroscopy study on primary motor and sensory cortex response to clenching. Neurosci Lett. 2009;449:98–102.
100. Tahara Y, Sakurai K, Ando T. Influence of chewing and clenching on salivary cortisol levels as an indicator of stress. J Prosthodont. 2007;16:129–35.
101. Kaneko M, Hori N, Yuyama N, et al. Biting suppresses Fos expression in various regions of the rat brain: further evidence that the masticatory organ functions to manage stress. Stomatologic. 2004;101:151–6.
102. Mascaro MB, Prosdocimi FC, Bittencourt JC, et al. Forebrain projections to brainstem nuclei involved in the control of mandibular movements in rats. Eur J Oral Sci. 2009;117:676–84.
103. Iida T, Kato M, Komiyama O, et al. Comparison of cerebral activity during teeth clenching and fist clenching: a functional magnetic resonance imaging study. Eur J Oral Sci. 2010;118:635–41.
104. Miyake S, Takahashi S, Yoshino F, Todoki K, Sasaguri K, Sato S, et al. Nitric oxide levels in rat hypothalamus are increased by restraint stress and decreased by biting. Redox Rep. 2008;13:31–9.
105. Momose E, Niskikawa J, Watanabe T, et al. Effect of mastication on regional cerebral blood flow in humans examined by positron-emission tomography with O-labelled water and magnetic resonance imaging. Arch Oral Biol. 1997;42:57–61.
106. Tamura T, Kanayama T, Yoshida S, Kawasaki T. Analysis of brain activity during clenching by fMRI. J Oral Rehabil. 2002;29: 467–72.
107. Iida T, Sakayanagi M, Svensson P, et al. Influence of periodontal afferent inputs for human cerebral blood oxygenation during jaw movements. Exp Brain Res. 2012;216:375–84.
108. Qin S, Hermans EJ, van Marle HJF, et al. Acute psychological stress reduces working memory-related activing in the dorsolateral prefrontal cortex. Biol Psychiatry. 2009;66:25–32.
109. Heit T, Derkson C, Bierkos J, Saqqur M. The effect of the physiological rest position of the mandible on cerebral blood flow and physical balance: an observational study. Cranio. 2015;33:195–205.
110. Otsuka T, Hayashi Y, Sasaguri K, Kawata T. Correlation of hypothalamic activation with malocclusion: an fMRI study. Biomed Res. 2015;26:203–6.
111. Ebben WP. A brief review of concurrent activation potentiation: theoretical and practical constructs. J Strength Cond Res. 2006;20:985–91.
112. Miyahara T, Hagiya N, Ohyama T, Nakamura Y. Modulation of human soleus H reflex in association with voluntary clenching of the teeth. J Neurophysiol. 1996;76:2033–41.
113. Takada Y, Miyahara T, Tanaka T, Ohyama T, Makamura Y. Modulation of H reflex of pretibial muscles and reciprocal Ia inhibition of soleus muscle during voluntary teeth clenching in humans. J Neurophysiol. 2000;83:2063–70.
114. Hidaka O, Iwasaki M, Saito M, Morimoto T. Influence of clenching intensity on bite force balance, occlusal contact area, and average bite pressure. J Dent Res. 1999;78:1336–44.
115. Murakami S, Maeda Y, Ghanem A, Uchiyama Y, Kreilborg S. Influence of mouthguard on temporomandibular joint. Scand J Med Sports. 2008;18:591–5.
116. Smith-Bindman R, Lipson J, Marcus R, Kim KP, Mahesh M, Gould R, et al. Radiation dose associated with common computed tomography examinations and the associated lifetime attributable risk of cancer. Arch Intern Med. 2009;169:2078–86.
117. Garrod R, Dallimore K, Cook J, Davies V, Quade K. An evaluation of the acute impact of pursed lips breathing on walking distance in nonspontaneous pursed lips breathing chronic obstructive pulmonary disease patients. Chron Respir Dis. 2005;2:67–72.
118. Ramos EMC, Vadnerlei LCM, Ramos D, Teizeira LM, Pitta F, Veloso M. Influence of pursed-lip breathing on heart rate variability and cardiorespiratory parameters in subjects with chronic obstructive pulmonary disease (COPD). Rev Bras Fisioter. 2009;13:288–93.
119. Pae A, Yoo R, Noh K, Pake J, Kwon K. The effects of mouthguards on the athletic ability of professional golfers. Dent Traumatol. 2013;29:47–51.
120. Smith SD. Atlas of temporomandibular orthopedics: dentistry in sports medicine. Philadelphia: Philadelphia College of Osteopathic Medicine Press; 1981.
121. Gould TE, Piland SG, Shin J, Hoyle CE, Nazarenko S. Characterization of mouthguard materials: physical and mechanical properties of commercialized products. Dent Mater. 2009;25: 771–80.
122. Plastics International. Hardness scale: durometer comparisons of materials. 2015. http://www.plasticsintl.com/polyhardness.htm.
123. Acevedo EO, Kraemer RR, Kamimori GH, Durand RJ, Johnson LG, Castracane VD. Stress hormones, effort sense, and perceptions of stress during incremental exercise: an exploratory investigation. J Strength Cond Res. 2007;21:283–8.
124. Kenney WL, Wilmore JH, Costill DL. Physiology of sport and exercise. 6th ed. Champaign: Human Kinetics; 2015.
125. Chakfa AM, Mehta NR, Forgione AG, Al-Badawi EA, Lobo SL, Zawawi KH. The effect of stepwise increases in vertical dimension of occlusion on isometric strength of cervical flexors and deltoid muscles in nonsymptomatic females. Cranio. 2002;20: 264–73.

# Team Dentist: Role of the Dentist in the Modern Sports Medicine Team

*Jeff Hoy, Mark Roettger, and Paul Nativi*




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1_14

## 14.1 Introduction: Team Dentist

The position of team dentist will vary from team to team and from sport to sport. Taking care of a high school baseball team will have different demands that taking care of a professional hockey team. Dentist's roles will vary significantly from team to team. Preseason exams may or may not be a part of their duties. On-field or venue participation may or may not be necessary. The dentist will need to be fluid to provide the needed care to the team as directed by the lead physician or certified athletic trainer (CAT). Sports medicine teams will look different at different levels of competition. The head physician and the head certified athletic trainer (CAT) are always leaders in these teams.

The commitment required to be a team dentist is significant. There will be additional call required, additional time spent away from family, or dental practice. For the right dentists, the rewards of working with a team will outweigh the time commitment of the position. Involvement with a local high school team can be a very good practice builder for a dentist. Communities take great pride in their high school athletic teams, and being a member of the sports medicine team creates a niche that encourages new patients to join a practice. Many dentists are competitive people and association with sports teams helps them to be a "part" of competition.

## 14.2 The Sports Medicine Team

The sports medicine team is an integrated team of health-care providers dedicated to the health and performance of athletes of all ages. Sports medicine teams will always have physicians and certified athletic trainers. We are working to get dentists involved in these teams at all levels as well. Currently there are many teams who don't have a dentist. Sports medicine teams may also have nutritionists, massage therapists, as well as sports psychologists. This chapter will focus mainly on the duties of physicians, certified athletic trainers, and dentists.

### 14.2.1 Physicians

Physicians will usually lead the sports medicine team. The most common types of physicians on most sports medicine teams are family practice physicians, many who have served a fellowship in sports medicine, and orthopedic surgeons. The team physician must make decisions on athlete's health and ability to participate safely in practices or games. Sports medicine physicians will manage infections, illness, and concussion injuries in team members. Orthopedic surgeons will diagnose, repair, and oversee rehabilitation of musculoskeletal injuries in athletes. Team physicians will usually be involved in preseason evaluations of each team member. This is more common the higher the level of competition. Team physicians or designee are available at all times during the season and beyond to care for athletes on the team.

### 14.2.2 Certified Athletic Trainers (CAT)

Certified athletic trainers are the frontline providers in sports medicine. They are intimately involved with the team members on a day-to-day basis. CATs need to be well versed in all aspects of sports medicine as they will interface with all other members of the team. They will have duties in prevention of illness and injury working directly with the athletes. They are in constant communication with the team physicians and coaches. Control of the training room is also a duty of the CAT. Trainers also have a large educational role with their athletes in the areas of nutrition, hydration, and environmental issues.

### 14.2.3 Dentists

The team dentist will direct all members of the dental team. Most team dentists are general dentists who have the experience to manage all aspects of oral care. Additional dental team members are most often oral and maxillofacial surgeons who will diagnose, repair, and oversee rehabilitation of jaw fractures. The team dentist may be responsible for all dental needs of the team. This can vary at different levels as high school athletes will likely have their own family dentist nearby. College and professional athletes are more likely to rely on the team dentist for all of their dental needs. The team dentist may be involved with preseason screening at the same time the team physicians are screening. This is a good time to look for issues that may limit participation during the season. Partially erupted third molars could flare up during the season. Looking for caries that have or are close to invasion of the pulp tissue could cause severe pain and some disability during the season. The time of preseason exam is also a pevrfect time to take impressions for mouthguards. Prevention of dentoalveolar trauma with mouthguards is a major goal of the team dentist. Management of dentoalveolar trauma (▸ Chap. 3) is also an important function of the team dentist (◘ Fig. 14.1). The position of team dentist is a significant time commitment and should not be entered with no thought to the time required to do this job.

## 14.3 Qualifications of a Team Dentist

The Academy for Sports Dentistry (ASD) has defined the qualifications necessary to be a team dentist. ASD has developed a course to qualify member dentists to be certified team dentists. The course covers topics of definitions of the sports medicine team members, preseason preparation, dentoalveolar trauma, performance-enhancing drugs, concussion and prevention of dental trauma, and the athletic mouthguard. These qualifications have been adopted by the Major League Soccer organization with other professional sports organizations certain to follow. The MLS requests the team dentists of all Major League Soccer teams be certified by the Academy for Sports Dentistry.

**Fig. 14.1** Trauma to the oral cavity from an elbow to the mouth in a soccer game. (Photo credit Mark Roettger DDS)

**Qualifications of a Sports Team Dentist**

- Licensed dentist.
- Member of the Academy for Sports Dentistry (ASD).
- Attend and complete the ASD's Team Dentist Course.
- Complete at least 15 sports dentistry-related CEUs every 3 years.
- Educate allied professionals, MDs, CATs, etc.
- Fabricate and deliver properly fitted mouthguards.
- Diagnose and treat all types of dentoalveolar trauma.
- Oral-facial first aid (contusions, lacerations, etc.) suture.
- Identify and treat temporomandibular joint injuries and dislocations.
- Identify medical complications of head injuries (concussion).
- Familiar with doping and drug issues.
- Establish a dental support team.
  - Oral maxillofacial surgeon
  - Dental lab support
- Cooperate with other members of the sports medicine team to insure the health and well-being of the athletes.

It was the vision of the Board of Directors of the Academy for Sports Dentistry to work toward being able to place qualified dentists with sports teams of all levels.

## 14.4 Education of the Sports Medicine Team: Dental Issues

The nineteenth century in the USA saw the separation of dental education from medical education. Education of dentists and physicians has remained segregated ever since the formation of the first dental school in the USA in Baltimore Maryland in 1840. Physicians receive very little training in anything dental. This requires the team dentist to take on training for team physicians and also for the certified athletic trainers.

Team dentists need to teach trainers and physicians which dentoalveolar injuries require immediate treatment and which injuries do not and can receive delayed treatment. Subluxation and crown fractures both simple and complex are usually fine with delayed treatment. Extrusive luxation, lateral luxation, intrusion, alveolar fractures gingival and oral lacerations, and avulsion injuries usually require immediate care. Most of the injuries that require immediate treatment are due to occlusal issues. Avulsion is one injury that if not treated immediately there is a rapid decrease in the survival chances of the tooth long term. For this reason physicians and trainers need to have first aid training for avulsion injuries. The team dentist may not be at a game and will likely not be at practice, so avulsion first aid is required at least of trainers. Avulsed teeth need to be reimplanted or placed in an appropriate storage medium preferably within 5 min of the injury (▶ Chap. 3). Effective storage media in order of effectiveness are cold skimmed milk, cold whole milk, fresh Hank's Balanced Salt Solution (HBSS), and Save-A-Tooth (HBSS). The team dentist then needs to be immediately involved in emergent care, splinting, and long-term management. The team dentist should have dental first aid protocols for all the dentoalveolar trauma types that require immediate dental care. This needs to be reviewed at regular intervals with the other members of the sports medicine team. Reference materials may also be prepared for these injuries to be used by the trainers. Laminated cards with easy access cookbook-type references and diagrams may be the best materials. The Academy for Sports Dentistry (ASD) has a laminated trauma card that is available to academy members. (Fig. 14.2)

Educating the rest of the sports medicine team on the benefits of wearing properly fitted mouthguards is another important educational duty of the team dentist (▶ Chap. 7). Prevention of athletic injuries is the duty of all members of the sports medicine team, and the athletic mouthguard is dentistry's main area of prevention of injury in sports.

## 14.5 Preseason Dental Screening

Athletes at every level devote countless hours of time and energy preparing themselves to compete. The further the athletes continue pursuing their sport, through college and even into a professional career, the more their sport places demands on their time and energy. This may include additional demands placed on them by coaches, team athletic trainers, personal trainers, agents, and the media, not to mention personal and family time demands. All too often, an athlete's oral health is over looked until there is a dental problem.

Dental literature contains information from surveys taken at championship events, including Olympic events that clearly demonstrate the need for basic dental care. Even

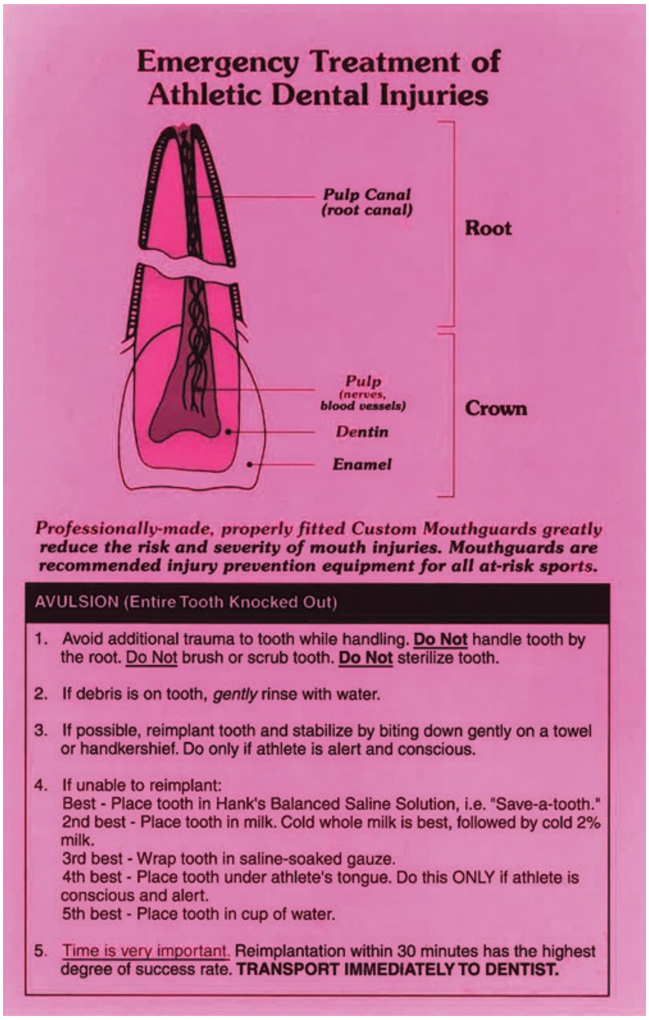



**Fig. 14.2** Trauma card from Academy for Sports Dentistry; available to members and printed in multiple languages. (Courtesy of ASD)



though these athletes are in pristine condition to compete in their sport, many athletes present with dental conditions that are below the level of what would be considered normal for the average population.

In addition to the demands of time placed on athletes, as mentioned above, there are other reasons an athlete may not see regular dental care. While many athletes may have limited financial assistance and possibly no dental insurance, a major reason athletes do not seek regular dental care is their lack of knowledge and awareness of the importance of oral health as it relates to their general health and ability to perform their sport. That is a major reason why it is very important for the team dentist to provide education toward the realization of the importance of good oral health for the athlete. This responsibility falls directly on the shoulder of the team dentist and if he or she fails in this respect, the athlete is underserved.

Most advanced levels of organized sports include some form of preseason exams and screenings and many sports require a preseason exam. The purpose of this exam is to assess the athlete's physical and mental condition and to establish baselines for the conditions of the athletes prior to their competing and to identify any conditions that might preclude or impair the athlete's performance. Valuable medical information is gained at these exams, and preexisting medical conditions may be uncovered at these examinations. Cardiovascular problems, hypertension, medication allergies, vitamin deficiencies, thyroid problems, and numerous other medical conditions may be detected by the team physician at the preseason examination, so the physician can optimize the health of the athlete. Even in high school, athlete participants are required to undergo a preseason physical exam. This is usually done by the athlete's primary care physician not by a team physician, but the rationale is the same, identify preexisting conditions and optimize the health of the athlete.

The ideal time for the dentist, and possibly the only time for the dentist and athlete to discuss dental health, is at a preseason dental screening. The preseason screening and exam is an ideal opportunity for the dentist to gather this valuable information and to meet the athletes and discuss the importance of oral health as well as the importance of wearing a mouthguard for all sports activities including conditioning, practice, and competitions. While most athletes are free from dental-related life-threatening conditions, other oral conditions may be discovered which, if not treated, may pose a significant health risk or possible loss of playing time during the upcoming season. Among these may include general unhealthy dental conditions; broken, missing, and decayed teeth; gingivitis and periodontal conditions; soft tissue anomalies; impacted or partially impacted third molars; pericoronitis; and pathologies of the head, neck, and oral cavity. Additionally, it would be an appropriate time to talk to the athletes, one on one, while performing the screening, regarding the pitfalls of sports drinks and the health hazards of smokeless tobacco. While most athletes consume sports drinks, far fewer engage in smokeless tobacco. Nevertheless, smokeless tobacco is used by a significant number of athletes in some sports. Oral conditions are noted and then may be treated to optimize the athlete's oral health as the season begins.

Of the five major professional sports leagues, NFL, NBA, NHL, MLB, and MLS, only the NHL requires preseason screening exams to include a dental screening as well. While dental preseason screenings are not required by the NBA, NFL, MLB, and MLS, specific teams within these leagues do include dental screenings at the preseason screening day. Even though it is not a required, preseason dental screening exams have become part of the overall preseason exam process for many teams due to the recognized importance of dental screenings. If you are the dentist on the sports medicine team in a league that does not require dental involvement in the prescreening exam process, it is very worthwhile and beneficial to start including dental involvement. Some of the components of a preseason dental evaluation can be seen in Box "Components of a Preseason Dental Examination".


Fig. 14.3 Shows preseason examination using a good quality headlight. **a** shows preseason exam outside a dental clinic setting using a good quality headlight. **b** shows the headlight used and **c** shows the exam in a coach's office using loupes for magnification as well as an adequate headlight. (Photo credit: Mark Roettger DDS)

### Components of a Preseason Dental Examination

- Medical history
- Dental history
- Complete X-rays
- Comprehensive dental exam/screening
- Soft tissue exam
- Mouthguard evaluation
  - Examine existing mouthguards
  - Impression for new mouthguards

Ideally, the preseason dental exams would occur at the dental office or facility where x-rays, mouthguard impressions, and complete exams can take place. However, as most sports team dentists will agree, this is rarely the case. Most preseason exams and screenings occur at locations other than a dental office or facility. Many preseason screenings are held at practice and game facilities including coach's office, training rooms, and even hotel rooms. This requires a very insightful level of preparation on the part of the dentist and dental staff performing the exams; every detail must be anticipated. As such, it is incumbent upon the dentist to prepare a sort of mobile dental office.

Providing a good visual clinical oral examination is only possible with adequate lighting which will rarely exist outside the dental office. The use of a good headlight (Fig. 14.3) is recommended to maximize intraoral vision during the examination. A list of necessary and suggested supplies for providing preseason dental screening examinations for up to 60 athletes is shown in Box "Preseason Exam List for 60 Exams and Mouthguards".

### Preseason Exam List for 60 Exams and Mouthguards

1. Doctor
2. Assistants
   1. Gloves
      - 2 boxes small
      - 2 boxes large
   2. Masks
   3. Gowns
   4. Loupes
   5. Head light
   6. Patient bibs
   7. Bib clips
   8. Clipboard
   9. Writing pens
   10. Labels for impressions
   11. Ziplock bags for impressions
   12. Paper towels
   13. Note pad

14. Rope wax
15. Alginate (3–4 bags)
16. Alginate cans for refill
17. Alginate measuring scoops
    - And water vial
18. Mixing bowls (3)
19. Mixing spatulas
20. PAM spray for metal trays
21. Impression trays (plastic)
    - 50 large
    - 10 medium
    - 5 small
22. Alcohol/torch and matches (to modify trays)
23. 1 can sani-cloth wipes
24. Empty box for impressions
25. Luggage wheels
26. Electrical extension cord
27. 60 mouth mirrors
28. 60 Explorers
29. 60 perio probes
30. Canned air
31. 5oz cups (40–50)

This list should be considered a starting point and can be modified as needed for each individual case. With the lack of radiographs, this cannot be considered a comprehensive examination, and if possible oral conditions are discovered during this screening examination, the athlete can be referred to your dental office for a more complete examination and treatment.



## 14.6 Team Mouthguard Programs

Providing mouthguards for an entire team can be challenging. Often this is done in a training room or another inconvenient place outside the comfort of a dental office. Infection control becomes more of a challenge and for large teams keeping the appropriate impressions, casts and finally mouthguards with the correct names can be an issue. Systems for infection control and the identification system for impressions, casts, and mouthguards must be thought out in advance, and the impression team, both clinical and lab, needs to be briefed prior to the event.

When mass mouthguard impression events are planned, there needs to be a significant team assembled to be sure that the process moves smoothly and that the patient identification follows the fabrication steps. It is prudent to have four teams for large mass impression clinics: first, the registration team who have the patient fill out an identification slip with either a name or identification number; second, the impression team who are the licensed practitioners who will take the impressions; third, the clinic support team who will assist with preparation of the alginate material measures and water measures, as well as cleanup of the bowls after mixing; and last, the lab team who will immediately pour the alginate impressions with either improved stone or die stone as well as stone cleanup (▣ Figs. 14.4 and 14.5).

The materials for impression-taking for mouthguards should be chosen by the team dentist. There is a sample list of supplies in Box "Preseason Exam List for 60 Exams and Mouthguards" that will serve as a starting point for each team. Most mouthguard programs utilize alginate impression material which must be poured immediately to avoid desiccation and distortion. Another variation that is sometimes used is to take impressions for mouthguards is to use a material such as vinyl-poly-siloxane (VPS). This impression material is more dimensionally stable and does not require immediate cast pouring. The impressions can then be brought to a lab for pouring in a more controlled environment. Digital impressions are fast-becoming accepted practice in dentistry and will likely become more utilized in mass mouthguard impression days. Each patient can be scanned by the digital camera and the impression data stored in the computer. The data is then used to fabricate a cast on which a mouthguard may be made (▣ Fig. 14.6). The digital option will become more popular because it is much cleaner and does not require shipment of large quantities of supplies to impression a team at a distant site.

Once the casts are made, the mouthguards are fabricated using either the vacuum-forming technique or using the heat-pressure lamination technique as discussed and described in ▶ Chap. 7. The mouthguards should then be delivered to the team members, and if occlusion has not yet been balanced on the appliance, it should be done at the fitting time. This is the time also to review proper care and maintenance of this appliance as well as instruction to properly wear the guard so that each appliance is properly fitted and properly worn. Team logos, player names, and jersey numbers can be added to custom mouthguards (▣ Fig. 14.7).

## 14.7 Delivery of Team Dental Care

Responsibility for routine dental care for team members increases as the level of competition increases. When covering high school teams, the team dentist will usually tend


**Fig. 14.4** **a** Team MG clinic youth sports incentives for volunteers. **b** Impression with alginate in this case the impression day was held in the dental clinic. (Photo credit Mark Roettger DDS). **c** Impressions bagged and identified ready to be poured in the lab photo credit. **d** Impressions poured and trimmed. Note the casts are identified in the lab; de-identified for the photo; (Photo credit Mark Roettger DDS). **e** MG fabrication in process with strap attachment (see ► Chap. 7). **f** MG ready to be bagged and delivered. (Photo credit Mark Roettger DDS). **g** MG bagged and ready to deliver to the players. (Photo Credit Mark Roettger DDS)

Fig. 14.4 (continued)

to routine dental care for only the athletes who are patients of record in their practice. The other team members will likely continue care at the dental practice where they have been previously seen. In college, some of the athletes are further from home and will rely more heavily on the team dentist for more routine care. This care will usually be set up and overseen by the training staff of the team. In the professional ranks, the team dentist is usually called on to care for the team members, coaching staff, and other team personnel. Often professional athletes are far from previous homes and trades can upset their care, so a reliance on the sports medicine team is heavier in the professional sports teams.

No matter what level the team dentist is working, there is one constant in the care of elite athletes, and that is time is sparse. In college, the demands are high on athletes and they still need to take classes and attend to all the team duties. The team dentist must be available on short notice and be flexible to balance the schedules between athletics, school, and dental care. In the professional sports, the classes are usually a thing of the past, but team meetings, practices, and often brutal travel schedules will complicate routine dental care.



Emergent dental care for patient athletes at any level requires the dentist to be fluid in his or her schedule. Keeping the athletes on the field of play is of utmost importance to any elite sports team. Making room in the dentist's schedule to see players or coaches with dental or oral emergencies will become a way of life for the college or professional sports team dentist.

In the college or professional levels, the sports team dentist, as a member of the sports medicine team, will be called upon and will have the opportunity to consult and to provide his or her expertise to other members of the sports medicine team, the athletes themselves, the athlete's agents and family, the general management and coaching staff, and other members of the sports organization. Additionally, the team dentist will be asked questions and even interviewed by the public relations staff as well as members of the media and the press. In short, the team dentist is regarded as the dental expert and the go-to person for all dental matters. Great care and preparation must be realized on the part of the team dentist when consulting and answering questions pertaining to dentistry. After all, you as the team dentist will be regarded as an expert and will be quoted in the printed and nonprinted media. First and foremost is the stringent protection of privacy of any patient. The term patient can have a very broad definition. Suffice it to say that once a person is touched or advised, dentally or medically, a doctor-patient relationship has been established.

These days, with the advent of HIPAA rules and regulations, extreme caution and prudence must be exercised on the part of the team dentist. Great care must be given to protect any information that was gathered in a doctor-patient relationship. In most, if not all, professional sports organizations, players sign their consent allowing team doctors to speak to other members of the organization and to appropriate league officials with regard to their injury and or health status. If there is ever a question regarding what information can be given to whom, it is wise to consult with the head physician or the head athletic trainer prior to discussing confidential information with anyone other than the patient themselves.

**Fig. 14.5** **a**, **b** Impression day at a D1 college training facility shows volunteers from the Sports Dentistry Club at the University of Minnesota preparing for football MG impressions (note the amount of supplies necessary to conduct this operation). **c** Players awaiting impressions in the training room using alginate impressions. Note the plastic covering the floor to avoid alginate messes. Panel **d** shows actual impression being taken and **e** shows the result. Panel **f** shows mix under vacuum. Panel **g** shows the pour using vibration to minimize air entrapment in the cast and **h** shows the finished pour; remember to always keep identification with the cast. When alginate is used for impressions, they must be poured immediately to avoid desiccation and distortion. Pouring must be planned for impression day when alginate is used. Panel **i** shows the cast after separation from impression, and **j** and **k** show the casts trimmed to make a vacuum MG; note the palate has been removed for vacuum MG fabrication; vacuum of pressure lamination may be used to fabricate (see ▶ Chap. 7). Panel **l** shows the finishing of the MG and **m** shows the finished product. When making MG for the entire team, vacuum guards are made; pressure laminate guards are made for many players after the initial push. (Photo credit Sports Dentistry Club University of Minnesota and Mark Roettger DDS)


a
b
c
d
MassMutual
800-886-1720

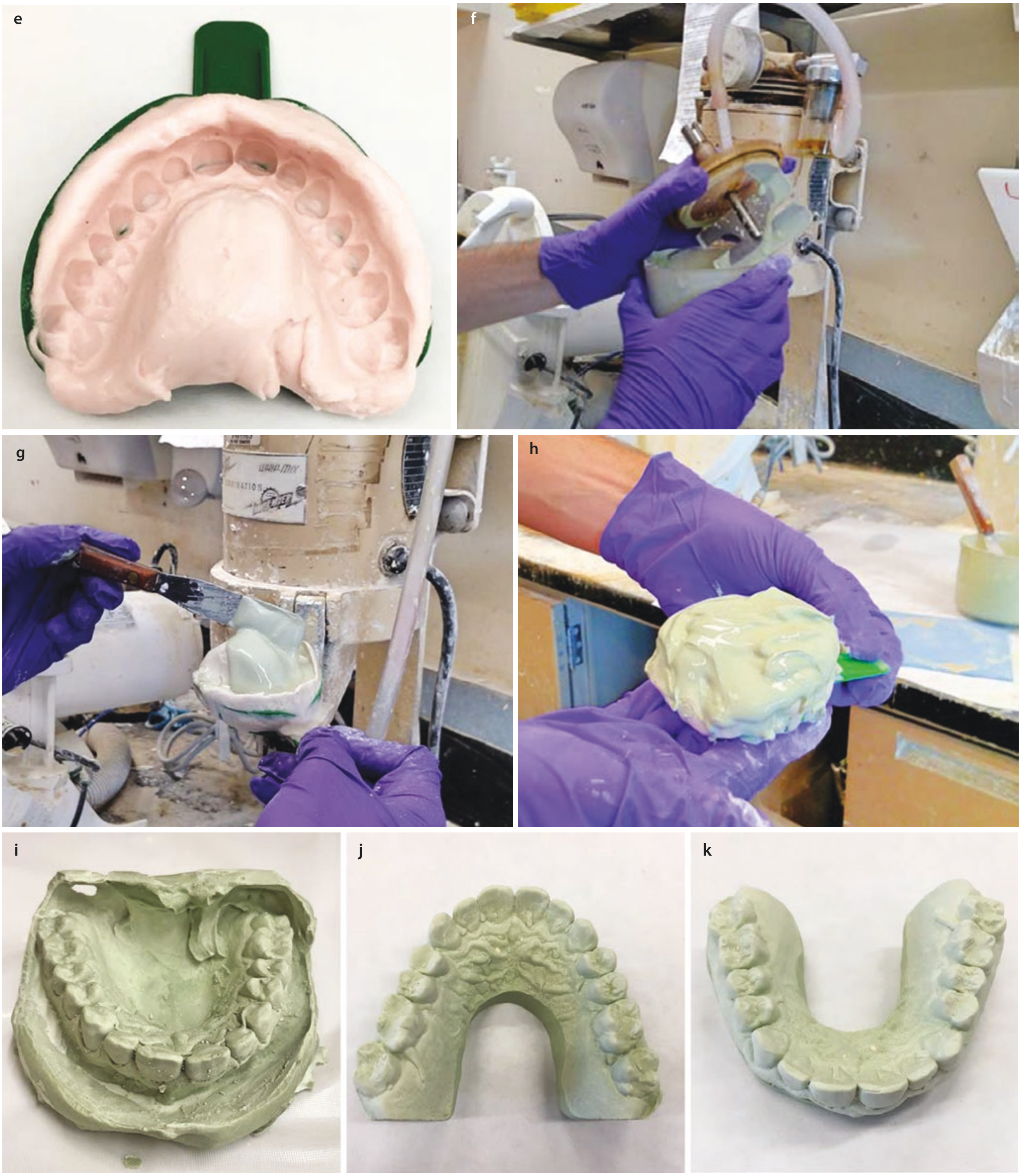



**Fig. 14.5** (continued)

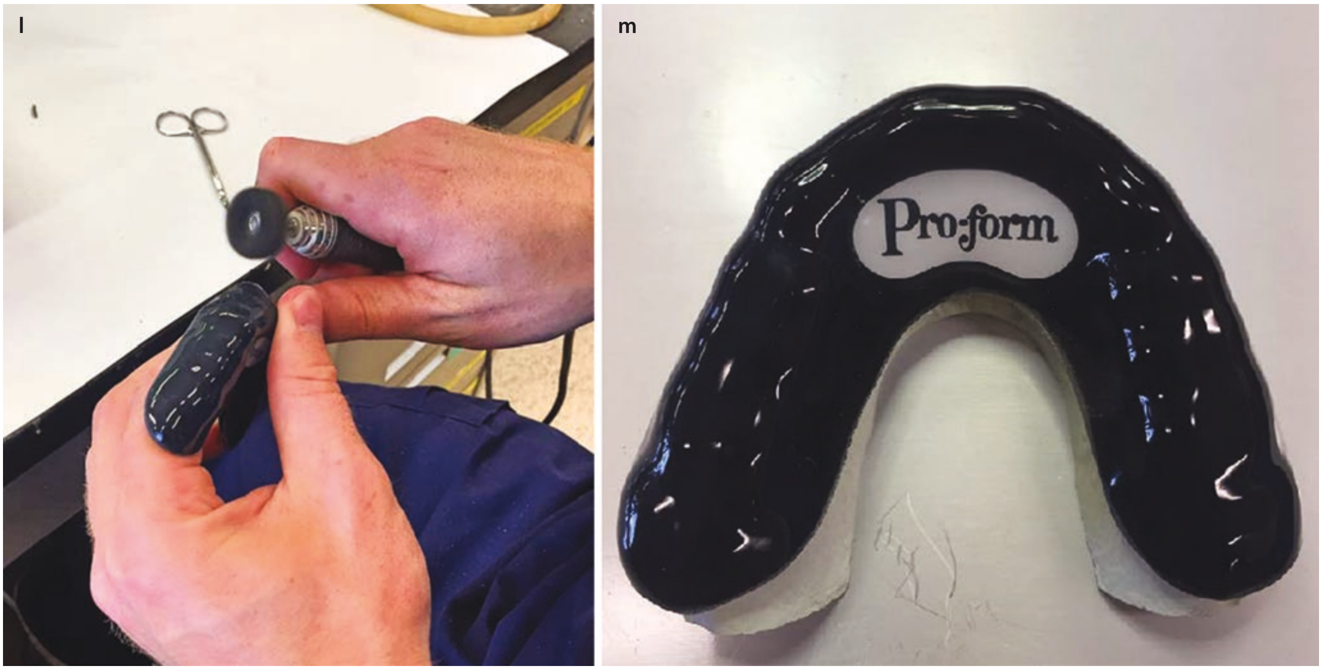



**Fig. 14.5** (continued)

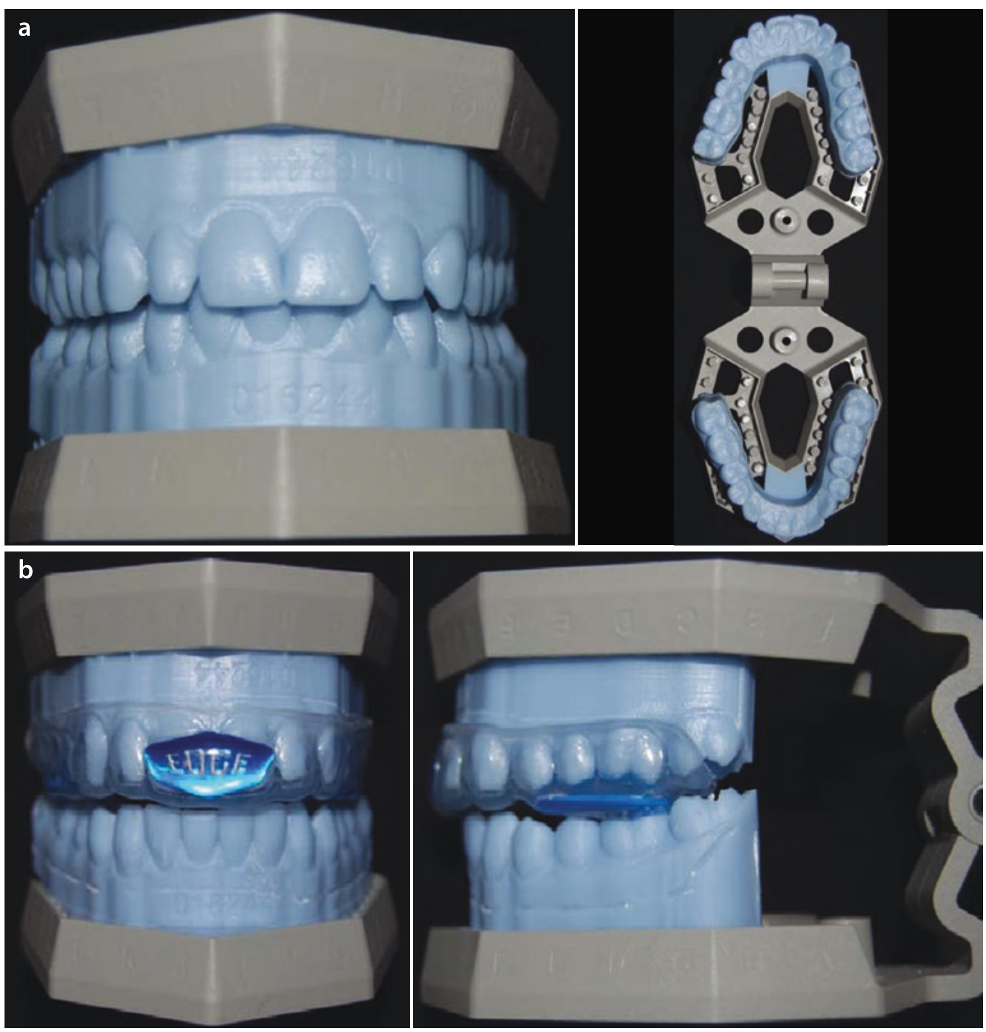



**Fig. 14.6** **a** After digital impressions, the casts are milled and ready to make the mouthguards. Digital impressions allow cleaner and easier methods of cast and MG fabrication for athletic teams when impressions are done outside of a dental office: (Photo credit Mark Roettger DDS). **b** Mouthguards have been fabricated on casts milled from digital impressions of an athlete, the guards are occlusally balanced prior to seating, and the occlusion is verified at delivery. (Photo credit Mark Roettger DDS)

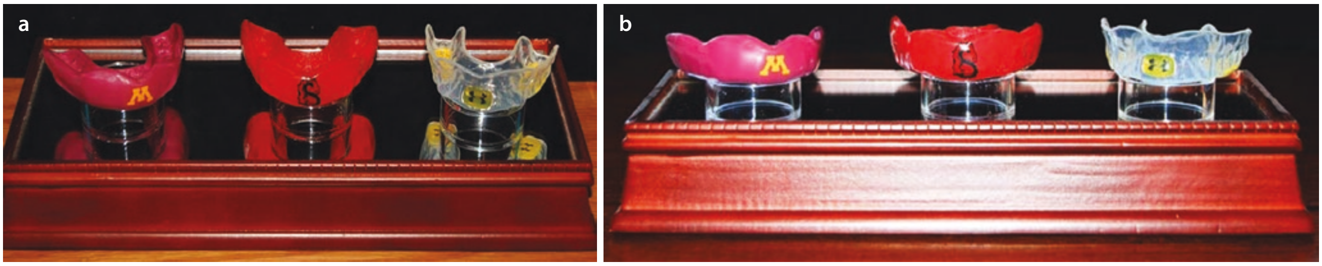



**Fig. 14.7** Finished mouthguards can have team logos if desired when they are custom made. (Photo credit Mark Roettger DDS)

## 14.8 Game Coverage for the Team Dentist

As a team dentist, you will likely be called upon to provide dental coverage for games. If so, this coverage will include all home games and may or may not include road or away games. Many colleges do not require game coverage where the dentist is required to be on site during the contest. They require only that there is emergent coverage via call to the team dentist or designee in the case that there is a dental injury.

At the professional level, the sports that require a dentist in attendance at all home games include NHL and the NFL. Typically, the home team dentist will provide coverage for the visiting team as well. This extends to the home physicians as well; the home team physicians provide coverage for the visiting team as well as the home team. In addition to providing medical and dental coverage for the athletes, the physicians and dentists may be called upon to attend to game officials and the coaching staff for both home and visiting teams. The topic of attending to fans and the general public sustaining an injury while attending sporting events is beyond the scope of this text and can be a complicated topic. Most arenas have personnel and facilities for fans in attendance, including EMTs, paramedics, and ambulance personnel.

For the teams in leagues that do not require the attendance of a dentist at games, many of the individual teams will require on-site dental coverage. For the teams not requiring their dentist to be in attendance at the games, the dentist will usually be on call for the needs of the team as required. The benefits and importance of having a dentist in attendance at all games appear overwhelmingly obvious especially in sports where orofacial injuries are most common.

For game dental coverage, it is essential to have the proper armamentarium to enable proper treatment of sports-related dental and oral injuries.



### Emergency Dental Trauma Kit: Suggestion for Contents

- Ace bandages
- Ammonia inhalers
- Anesthetics local topical long and short needles
- (Various types with and without epi)
- Ball burnisher
- Biohazard bags
- Alcohol or butane torch, matches
- Cotton rolls, cotton tip applicators, cotton pellets
- Endo files
- Formocresol
- Dental floss
- Dycal (calcium hydroxide paste system) or life, calcium hydroxide powder
- Explorer, perio probe, cotton pliers. Mouth mirror
- $2 \times 2$ gauze
- Latex gloves, masks
- Hemostats
- Hydrogen peroxide, betadine
- Curing light
- Mixing pads, spatula
- Plugger, plastic instrument, spoon excavator
- Pocket flashlight, headlight
- Hank's balanced saline solution (tooth storage medium)
- Scissors and crown and bridge scissors
- Dremel motor
- Burs, stones, polishing
- Syringe (aspirating)
- Tongue depressors
- ZOE, IRM, temporary cement
- Ice packs
- Soft wax (rope or utility wax)
- Suture material (silk, nylon, resorbable)
- Tissue glue
- Suturing instruments (needle driver, pickups, suture scissors)
- Light cured composite, flowable composite, self-curing composite
- Bonding resin
- Acid etch, etch/bond
- Canned air
- Cellophane strips, finishing strips
- Splint materials (flexible and rigid wire), wire cutters
- Ribbond
- RX pads
- 60-grit sandpaper


**Fig. 14.8** On-field emergency kit used for dentistry game coverage. The suggested contents for the kit are found in Box "Emergency Dental Trauma Kit: Suggestion for Contents". Each team dentist should design the contents of the kit to meet individual needs and to be sport specific. **a** Shows the kit being transported. **b** Shows small kit. **c** Shows the large on-field kit. **d** Shows a dental trauma splint kit. (Photo credit Jeffrey Hoy DDS)

It is suggested that each sports team dentist creates their own emergency kits as needed for the level and sport where they work (Fig. 14.8). When covering a sporting event, sports-related dental trauma may not be the only dental need the team dentist may be called upon to treat. Many other non-sports-related disorders can and will arise. The team dentist must be prepared to provide consultation and treatment for a variety of dental issues, including but not limited to dental pain, discomfort and swelling arising from untreated dental conditions; re-cementing crowns and bridges; lost or loose dental restorations; adjustment of dental appliances, including mouthguards; soft tissue lesions; and other non-trauma-related dental issues. It is imperative the team dentist be prepared and very knowledgeable in all aspects of dentistry, in most cases a general dentist.

## 14.9 Getting Started as a Team Dentist

The reasons a dentist might have for pursuing a position with a sports team as a team dentist are varied. Often the motivation arises from the dentists' enjoyment of sports in general or a specific sport. The dentist may have a relative, including a son or daughter, on a particular sports team. There is certainly an opportunity to be part of something special being part of the camaraderie of a sports team and forming relationships with staff and members of the sports team. Whatever the reason for getting involved as a team dentist, it is imperative to review the motives as well as the level of commitment required to do the job properly.

At any level of sports, amateur to professional, the dentist will not realize a financial gain as a sports team dentist. In fact, many times the dentists' time and efforts are donated. That is not to say there is no benefit for the sports team dentist. The goodwill and personal satisfaction from helping others and being part of a team will usually outweigh financial gain. There are also a possible practice building elements from community involvement. The dentist may also enjoy the diversion from the day-to-day regular practice routine. Additionally, from time to time, there may be fringe benefits of some type such as attendance at games, tournaments, or team celebrations or banquets. For the committed sports dentist, the experience will be fun and valuable at any level of sports.

As far as seeking a position as a sports team dentist, there is really no set or clear pathway. The individual dentist must, in a sense, blaze his or her own trail. There are, however, some general guidelines. After identifying the reasons a dentist has decided to seek a position as a team dentist, the dentist must make a very critical assessment of his or her skills not only in the area of sports dentistry but in all aspects of dentistry. Overseeing the delivery of dental care to a sports team, at any level, carries with it a lot of responsibility, and it is assumed that the dentist be aware of and be prepared to meet those responsibilities. The sports team dentist must be very knowledgeable in the proper diagnosis and treatment of dentoalveolar injuries, and this knowledge needs to be updated each year. The team dentist needs to take continuing education in this field regularly. There is significant preparation required to be a good team dentist. The qualifications and responsibilities of a sports team dentist have been thoroughly discussed earlier in this chapter, and the interested dentist needs to thoroughly research and follow the requirements to become a sports team dentist.



After preparation is underway to become a team dentist, it is time to begin the process to become connected to a team. At different levels of sports, from amateur to professional, the initial contact person is different.

### 14.9.1 Amateur Sports

At the amateur level, the initial contact person is usually someone the dentist knows on the team, a coach or friend. At the high school level, the contact person will usually be the school's athletic director or athletic trainer who will, in turn, usually discuss the request with the team's coaching staff. At the college level, the contact person is usually the same as at the high school level, usually the athletic trainer or the athletic director. If there is currently no team dentist in an amateur team, be prepared to explain the need for dental coverage and what services that a team dentist can provide and how that will be of benefit to the team and the school as well as the players. Be prepared for your presentation to the team or school.

### 14.9.2 Professional Sports Team Dentist

The path for seeking a team dentist position at the professional sports level is quite varied and obscure. Every team dentist for every type of sport at the professional level has attained his or her position quite differently. Discussion of the stories of how professional sports team dentists reveals many unique stories. There is no level of sports dentistry that requires more creative pathfinding than seeking the position of a professional sports team dentist. While there are thousands of amateur teams, high schools and colleges, there are only approximately 30 professional sports teams in each of the major professional sports, and these positions are highly coveted.

The acquisition of some team dentist positions is serendipitous, arising from good luck. Positions arise being a friend or the dentist for the team's owner, coach, general manager, or athletic trainer. Perhaps one knows an existing sports team dentist who would like assistance from a colleague or who may be ready to retire. If none of these situations are familiar, probably the best person to contact, at the professional sports level, would be the team's head athletic trainer. In the area of professional sports team dentist, it may


**Fig. 14.9** The Academy for Sports Dentistry (ASD) is an organization that offers a Team Dentist Course that can be used to improve credentials when approaching teams about a team dentist position. (Photo courtesy of the Academy for Sports Dentistry)

be who you know and not what you know that is most important. If you become known as a knowledgeable sports dentist in a community, it is possible that a call for an opportunity may come your way.

## 14.10 Conclusion

The position of sports team dentist at any level can be fun, rewarding, and professionally satisfying. It requires a significant investment of time and resources. There are constant demands for continuing education to stay abreast of the current diagnosis and management of not only traumatic injuries of the head and neck but in all aspects of dentistry. Often a team dentist must overcome skepticism from coaches and athletic directors. Certified athletic trainers are our greatest allies currently on the sports medicine team and they are powerful allies. The team dentist must always nurture the relationship between dentistry and medicine via the athletic trainer. If you have a desire to become a sports team dentist, be prepared for the need for additional education, hard work, and perseverance. Join organizations that will assist you in these efforts. The most helpful organization known to sports dentists in North America is the *Academy for Sports Dentistry* (◘ Fig. 14.9).

# Supplementary Information




M. Roettger (ed.), *Modern Sports Dentistry*, Textbooks in Contemporary Dentistry,
https://doi.org/10.1007/978-3-319-44416-1

# Index

## E



## F



## G



## H



## I

# J



# K



# L



# M



# N



# O



# P

## U



## V



## W



## Z