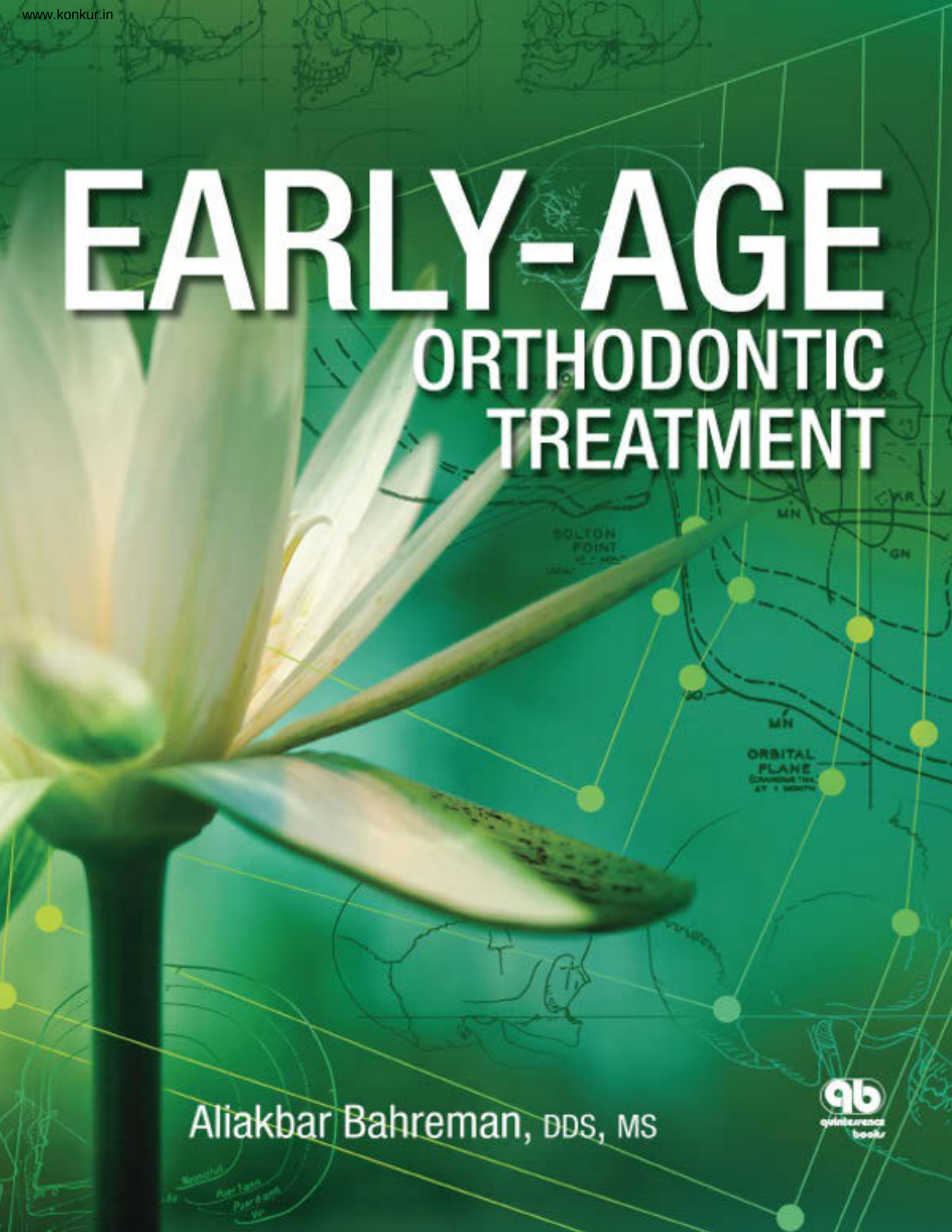
EARLY-AGE ORTHODONTIC TREATMENT



Aliakbar Bahreman, DDS, MS

quintessence books

Early-Age Orthodontic Treatment

EARLY-AGE ORTHODONTIC TREATMENT

Aliakbar Bahreman, DDS, MS

Clinical Professor
Orthodontic and Pediatric Dentistry Programs
Eastman Institute for Oral Health
University of Rochester
Rochester, New York

Quintessence Publishing Co, Inc

Chicago, Berlin, Tokyo, London, Paris, Milan, Barcelona, Beijing, Istanbul, Moscow, New Delhi, Prague, São Paulo, Seoul, Singapore, and Warsaw

Library of Congress Cataloging-in-Publication Data

Bahreman, Aliakbar.
Early-age orthodontic treatment / Aliakbar Bahreman.
p. ; cm.
Includes bibliographical references.
ISBN 978-0-86715-566-2
I. Title.
[DNLM: 1. Malocclusion--therapy. 2. Adolescent. 3. Child. 4. Orthodontics, Corrective--methods. WU 440]

617.6'45--dc23

2013001198

Quintessence Publishing Co, Inc
4350 Chandler Drive
Hanover Park, IL 60133
www.quintpub.com

5 4 3 2 1

Editor: Leah Huffman
Design: Ted Pereda
Production: Angelina Sanchez

Printed in China

Contents

Foreword

This book is a compendium of significant and pertinent information related to early-age orthodontic treatment, a subject that seems to have evolved into one of considerable controversy, with as many orthodontists expressing a negative reaction as a positive reaction to its benefits. Dr Bahreman is a believer in early-age orthodontic treatment, and he expresses some cogent arguments founded in years of experience in practice and teaching to back up his beliefs. In developing his treatise, Dr Bahreman outlines the development of the occlusion and/or malocclusion from the embryonic stages, when the foundation of the jaws and thereby the position of the dentition is first established.

Early-age orthodontics is not about the time it takes to orthodontically treat a problem; it is a story of growth, of variation in anatomy, and of muscle function and influences, a realization that it is the jaws that contain the teeth and that where the jaws go, the teeth will have to go, and both undergo varying influences as well as grow in varying directions. Early-age orthodontics necessitates recognition of this process and aims to alter and redirect it whenever feasible and possible. Dr Bahreman has undertaken a monumental effort in directing efforts along this path. An extensive exploration of the literature is an added bonus, as the mechanical approaches are based on this literature. In fact, the extensive review of the literature and its application to diagnosis and varying forms of therapy are worth a veritable fortune.

You may or may not agree with the basic premises, but you will have access to important information that will widen your scope of vision and thereby widen your treatment horizons. To my mind, an ounce of prevention, if possible, is worth a pound of cure. The reality of prevention can exist at the earliest stages of development.

J. Daniel Subtelny, DDS, MS, DDSc(Hon)

Professor Emeritus

Interim Chair and Director of Orthodontic Program

Eastman Institute for Oral Health

University of Rochester

Rochester, New York

Preface

After obtaining a master's degree in orthodontics in 1967, I began my career at a newly founded dental school in Tehran. My responsibilities included teaching and administrative duties at the university and maintenance of a very busy private practice. In addition, I established both the orthodontic and pediatric dentistry departments at the university.

Many patients were being referred to the orthodontic department, and there were no qualified faculty members to help me provide care. To rectify the situation, I designed an advanced level, comprehensive curriculum in orthodontics for undergraduate students, including classroom instruction, laboratory research, and clinical demonstrations. Once the students completed the course, they could work in the clinic, thus temporarily solving the issue of the heavy patient load in the orthodontic clinic. With additional staff now available, I could select patients, mostly children in the primary or mixed dentition, for some interceptive treatment.

Despite my difficulties in performing all of the aforementioned duties, this situation had a fortunate outcome. It helped me to understand and discover the advantages of early-age orthodontic treatment, which was not common in those years. During my more than 40 years of practice and teaching, especially in early orthodontic treatment, I have accumulated a considerable amount of educational data for teaching purposes. I would like to share this experience and information with readers.

The public's growing awareness of and desire for dental services, especially at an early age, have encouraged our profession to treat children earlier. Despite the recommendation by the American Association of Orthodontists that orthodontic screening begin by the time a child is 7 years old, many orthodontists still do not treat children prior to the complete eruption of the permanent teeth. I believe that this inconsistency is due to the educational background of orthodontists as well as a lack of familiarity with recent technical advancements and the various treatment options that are available for young patients.

The therapeutic devices available for this endeavor are not complex, but deciding which ones to use and when to employ them are important steps. As we make these decisions, we should also remember not to treat the symptom but rather to treat the cause. My goal is to present the basic information necessary to understand the problems, to differentiate among various conditions, and to review different treatment options. Case reports are examined to facilitate clinical application of the theory in a rational way.

To understand the morphogenesis of nonskeletal and skeletal occlusal problems, to detect problems early, and to intervene properly, we must look at all areas of occlusal development, including prenatal, neonatal, and postnatal changes of the dentoskeletal system, and explore all genetic and environmental factors that can affect occlusion at different stages of development. In other words, we must have a profound understanding of the fundamental basis and morphogenesis of each problem and then apply this knowledge to clinical practice. Thus, the goals of this book are:

- To provide a comprehensive overview of all areas of dental development, from tooth formation to permanent occlusion, to refresh the reader's memory of the fundamentals necessary for diagnosis and treatment planning.
- To emphasize all the important points of the developmental stages that must be recognized during examination of the patient to facilitate differential diagnosis. Each tooth can become anomalous in a number of ways and to different degrees. Occlusion and maxillomandibular relationships can vary in the sagittal, transverse, and vertical directions.
- To discuss the application of basic knowledge to practice by presenting several cases with different problems and different treatment options.
- To demonstrate the benefits of early-age orthodontic treatment, achieved by intervention in developing malocclusion and guidance of eruption.

Materials are presented in three parts: In Part I, "Clinical and Biologic Principles of Early-Age Orthodontic Treatment," three chapters introduce and explain the concept of early-age treatment, describe its necessity and advantages, and discuss the controversies surrounding this topic; discuss the basic foundation of occlusal development, empowering the practitioner to detect anomalies and intervene as necessary; and illustrate the procedures, tools, and techniques available for diagnosis, emphasizing differential diagnosis and treatment planning for early-age treatment.

Part II, "Early-Age Orthodontic Treatment of Nonskeletal Problems," consists of

seven chapters describing the non-skeletal problems that might develop during the primary and mixed dentitions. The chapters explain the ontogeny, diagnosis, and early detection of, and intervention for, these problems. Topics include space management, crowding, abnormal oral habits, abnormal frenum attachment, hypodontia, supernumerary teeth, and abnormal eruption problems.

Part III, "Early-Age Orthodontic Treatment of Dentoskeletal Problems," consists of three chapters on early intervention for the dentoskeletal problems that might arise during the primary and mixed dentitions in the three dimensions: sagittal problems (anterior crossbite and Class II and Class III malocclusions); transverse problems (posterior crossbites); and vertical problems (open bites and deep bites).

This book will provide the reader with a firm foundation of the basic science and case examples with various treatment options. It is my hope that the information provided will promote a better understanding of abnormalities and their causes and enable readers to recognize the clues for early detection and intervention.

Acknowledgments

First and foremost, I would like to gratefully acknowledge the valuable opportunity that was afforded me as a student in Dr Daniel Subtelny's orthodontic program. Between 1964 and 1967, I completed both my orthodontic specialty and master degree programs with Dr Subtelny as my mentor. As chairman and program director, researcher, and mentor, Dr Subtelny has dedicated over 57 years of his life to teaching, personally influencing the lives of over 350 students from around the world, myself included. In 1999, after over 32 years of teaching, practicing, and administrating in Tehran, I was fortunate enough to return to the Eastman Institute for Oral Health to work alongside Dr Subtelny as a faculty member in the Orthodontic and Pediatric Dentistry Programs.

In addition to Dr Subtelny, there are several individuals to whom I would like to express my deep gratitude for their help and encouragement in preparation of this book: the late Dr Estepan Alexanian, head of the Department of Histology at the Shahid Beheshti University Dental School in Tehran, whose dedication as an educator and preparation of superb histologic slides is remarkable and who allowed me to use his slides in my publication; Mr Aryan Salimi for scanning some of the slides and radiographs in this book; and Ms Elizabeth Kettle, Program Chair of the Dental Section of the Medical Library Association, head of Eastman's library, for her sincere help in editing this publication.

Finally, I wish to acknowledge the constant support of my family: Malahat, Nasreen, Saeid, Alireza, Tannaz, and Peymann Motevalei. Especially high gratitude goes to my wife, Malahat, for her tolerance, support, and encouragements. I also want to thank my son Alireza for his technical help and guidance in computer skills and my granddaughter Tannaz Motevalei for drawing some of the illustrations.

This publication is the product of 17 years spent organizing materials derived from my 45 years of practice and teaching as well as reviewing hundreds of articles and books. I herewith dedicate this book to the teachers, practitioners, residents, and students who are dedicated to treating malocclusion earlier in children, before it becomes more complicated and costly.

Introduction

Occlusal development is a long process starting around the sixth week of intrauterine life and concluding around the age of 20 years. This long developmental process is a sequence of events that occur in an orderly and timely fashion under the control of genetic and environmental factors. Dental occlusion is an integral part of craniofacial structure and coordination of skeletal growth changes. Occlusal development is essential for establishing a normal and harmonious arrangement of the occlusal system.

As we learn about craniofacial growth changes, the potential influences of function on the developing dentition, and the relationships of basal jawbones and head structure, we acquire a better understanding of when and how to intervene in the treatment guidance for each patient. It is more effective to intervene during the primary or mixed dentition period to reduce or, in some instances, avoid the need for multibanded mechanotherapy at a later age.

Untreated malocclusions can result in a variety of problems, including susceptibility to dental caries, periodontal disease, bone loss, temporomandibular disorders, and undesirable craniofacial growth changes. Moreover, the child's appearance may be harmed, which can be a social handicap. The benefits of improving a child's appearance at an early age should not be undervalued. The goals of many clinicians who provide early treatment are not only to reduce the time and complexity of comprehensive fixed appliance therapy but also to eliminate or reduce the damage to the dentition and supporting structures that can result from tooth irregularity at a later age. In short, early intervention of skeletal and dental malocclusions during the primary and mixed dentition stages can enable the greatest possible control over growth changes and occlusal development, improving the function, esthetics, and psychologic well-being of children.

For many decades, orthodontists have debated about the best age for children to start orthodontic treatment. While we agree on the results of high-quality orthodontic treatment, we often differ in our opinions as to how and when to treat the patient.

Some practitioners contend that starting treatment in the primary dentition is the most effective means of orthodontic care. Others prefer to begin the treatment in the mixed dentition. There is also controversy about whether the early, middle, or late mixed dentition is preferable.

Despite the fact that the American Association of Orthodontists recommends that orthodontic screening be started by the age of 7 years, many orthodontists do not treat children prior to the eruption of permanent teeth, and some postpone the treatment until the full permanent dentition has erupted, at approximately 12 years. The controversy surrounding early versus late treatment is often confusing to the dental community; therefore, clinicians must decide on a case-by-case basis when to provide orthodontic treatment. Indeed, there are occasions when delaying treatment until a later age may be advisable.

The long-term benefits of early treatment are also controversial. The majority of debates seem to revolve around early or late treatment of Class II malocclusions. There is less controversy regarding many other services that can be performed for the benefit of young patients during the primary or mixed dentition, such as treatment of anterior and posterior crossbite, habit control, elimination of crowding, space management, and management of eruption problems.

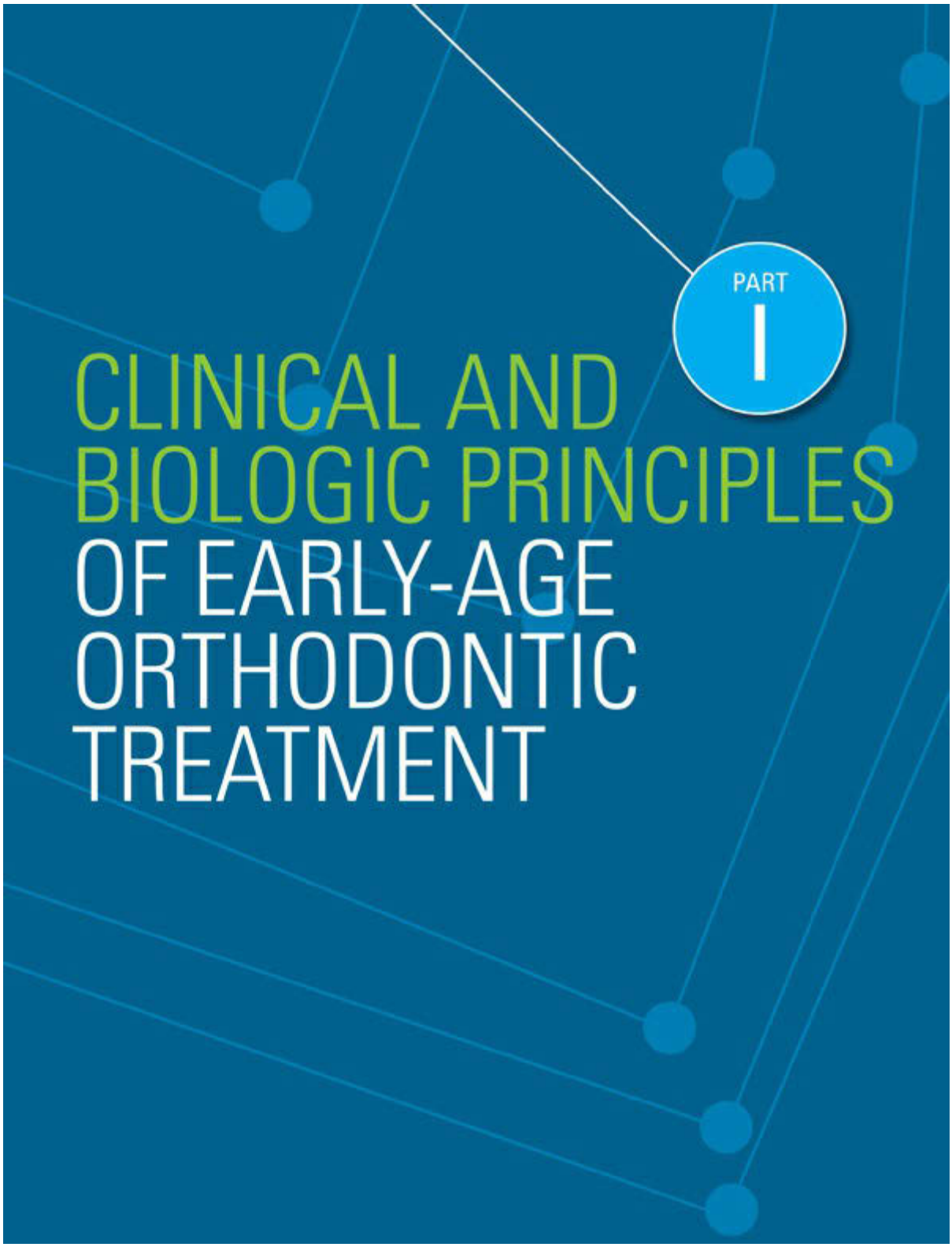
Practitioners who are in favor of early treatment of Class II problems contend that early intervention is the best choice for growth modification when the problem is skeletal and especially when it results from mandibular retrusion. On the other hand, opponents believe that there is no difference in the final result and that a single-phase treatment approach is preferable because of the advantages that accompany the reduced treatment time.

Unfortunately, some practitioners, without a profound evaluation of the indications for early treatment, conclude that late treatment is always preferable. However, broad conclusions drawn from narrowly focused research can be misleading. One cannot conclude that no birds can fly by considering the flight characteristics of the ostrich.

To evaluate and demonstrate the benefits of early treatment, I aim to discuss and clarify available treatments and services and discuss cases with different problems and different treatment options. An understanding of all aspects of early treatment requires a thorough knowledge of the basics of embryology, physiology, and growth and development. This includes development of the dentition, tooth formation, eruption, exfoliation, and all transitional changes. Therefore, my other goal is to integrate the basic science and the clinical, in order to refresh the reader's memory on important points about the bases of nonskeletal and skeletal problems that can

arise during the transitional stages of occlusion.

Each patient who enters our practice represents a new chapter and a new lesson that we can learn from. A thorough knowledge of the basis for early-age orthodontic treatment, an understanding of the proper treatment techniques, and a willingness to consider their appropriateness for each individual patient will allow us to intervene in ways that will provide the maximum benefit for a young and growing child.



Rationale for Early-Age Orthodontic Treatment

In the past, orthodontic treatment has been focused mainly on juvenile and adult treatment. Treatment options for patients in these age groups often are limited by complex dental and orthodontic problems and the lack of sufficient future craniofacial growth.

During the later part of the 18th century, orthodontic treatment of Class II malocclusion was limited primarily to retraction of the maxillary anterior teeth to decrease excessive overjet. In 1880, Norman Kingsley[1] published a description of techniques for addressing protrusion. He was among the first to use extraoral force to retract the maxillary anterior teeth after extraction of the maxillary first premolars; the extraoral force was applied with headgear. Later, Case[2] continued to refine these methods.

Angle's classification[3] of malocclusion, published in the 1890s, provided a simple definition of normal occlusion and was an important step in the development of orthodontic treatment. Angle opposed the extraction of teeth and favored the preservation of the full dentition. His position against tooth extraction led him to depend on extraoral force for the expansion of crowded dental arches and retraction of the anterior segment. Later he discontinued the use of extraoral force and advocated the use of intraoral elastics to treat sagittal jaw discrepancies.

Because of Angle's dominating belief that treatment with Class II elastics was just as effective as extraoral force, the use of headgear was abandoned by the 1920s. Then, in 1936, Oppenheim[4] reintroduced the concept of extraoral anchorage, employing extraoral traction to treat maxillary protrusion. Accepting the position of the mandible in Class II malocclusions, Oppenheim attempted to move the maxillary

dentition distally by employing a combination of occipital anchorage and an E-arch, allowing the mandible to continue its growth. This resulted in an improved relationship with the opposing jaw. In 1947, Silas Kloehn[5] reintroduced extraoral force, in the form of cervical headgear, for the treatment of skeletal Class II relationships.

In 1944, another student of Angle's, Charles Tweed,[6] was discouraged by the prevalence of relapse in many of his patients treated without extraction, so he decided to oppose the conventional wisdom of nonextraction.

In the early part of the 20th century, there was optimism about the influence of orthopedic force on skeletal growth. An almost universal belief was that orthodontic forces, if applied to the growing face, could alter the morphologic outcome.

In the United States, headgear was the principal appliance used for facial orthopedic treatment, whereas in Europe the functional appliance was predominantly used.

In 1941, Alan Brodie,[7] one of Angle's students, concluded that the growing face could not be significantly altered from its genetically predetermined form and that the only option for the orthodontist in cases of skeletal malocclusion would be dental camouflage, or the movement of teeth within their jaws. This idea led to tooth extraction.

In 1931, Broadbent[8] introduced the standardized lateral cephalometric radiograph as a tool for longitudinal evaluation of growth and developmental changes. This radiograph was significant because of its ability to demonstrate treatment results. Kloehn[5] used this technique to evaluate dentoskeletal changes after the application of extraoral forces and found that these forces could produce positive skeletal changes as well as dentoalveolar changes in the correction of skeletal Class II problems.

In the late 19th and early 20th centuries, interceptive orthopedic procedures were not common. Intraoral and extraoral devices such as face masks, functional appliances, and mini-implants were not as well known and accessible as they are today. At that time, the treatment focus was on alignment of irregularities after the complete development of malocclusions in older juveniles and adults. However, early orthodontic treatment is now more generally accepted as a means of gaining the greatest possible control over form and function and producing superior dentoskeletal changes over time.

The more that practitioners learn about growth and its potential, the influence of function on the developing dentition, the relationship of basal jawbones and head structure, and the influence of neuromuscular activity and functional balance, the

more they can apply these processes in clinical practice. With a thorough understanding of the biologic facts and the availability of new intraoral and extraoral devices and techniques, clinicians can acquire a better understanding of when and how to apply early intervention for prevention of the problems and control future adverse effect on the patient's growth pattern.

What Is Early-Age Orthodontic Treatment?

The term *early-age orthodontic treatment* encompasses all interventions and treatments that can be performed during the primary or mixed dentition, with the purpose of eliminating or minimizing dentoalveolar and skeletal disharmonies that can interfere with the normal growth and development of occlusion, function, esthetics, and the psychologic well-being of children. In other words, the main objective of this type of intervention is to prepare an environment that will enhance occlusal development.

General objectives

The following are the major goals of treatment started during the primary or mixed dentition:

- Enhancement of the normal dental and skeletal development
- Elimination or control of any environmental factors that might disturb normal occlusal development
- Preparation of the optimal environment for normal occlusal development
- Correction or guidance of a developing malocclusion to normal occlusion
- Elimination of the need for or reduction in the duration of second-phase treatment
- Maximization of growth potential for growth modification treatment

General strategy

The words *prevention* and *interception* have been used in many types of early treatment but are sometimes misleading. Neither of these terms can possibly include all areas of early-age orthodontic treatment. To eliminate these misunderstandings, the terms *preventive*, *interceptive*, and *corrective* must be explained:

- *Preventive orthodontic treatment* includes treatments that prevent the development of a malocclusion before it happens, such as space maintenance or habit control.
- *Interceptive orthodontic treatment* includes all types of treatment that can be used during the development of a malocclusion to guide the developing abnormality toward normality and to prevent further damage to the occlusion; these methods include space regaining, palatal expansion, correction of posterior and anterior dental crossbites, maintenance of leeway space in cases of moderate crowding, and management of severe arch size–tooth size discrepancy.
- *Corrective orthodontic treatment* is performed after a malocclusion is completely developed.

Early-age orthodontic treatment can include preventive, interceptive, or corrective treatment as well as combinations of all three types applied during the primary or mixed dentition period, before the complete development of a malocclusion. This timetable is necessary to take advantage of growth potential at the right time for skeletal problems by initiating treatment such as headgear or face mask therapy.

In 1975, Popovich and Thompson[9] evaluated preventive and interceptive orthodontic treatment in patients between 3 and 18 years of age. They concluded that few malocclusions can be prevented but that about 25% can be intercepted.

Why Is Early Orthodontic Intervention Recommended?

Despite progress in the understanding of growth and development and the physiology, morphology, and ontogeny of dentoskeletal anomalies, as well as the emergence of sophisticated diagnostic techniques, many practitioners do not know how to manage the problems they diagnose or when the right time is to refer their patients to a specialist for intervention.

One-phase versus two-phase orthodontic treatment

There are two schools of thought in regard to orthodontic treatment strategy. One philosophy advocates beginning treatment when the second molars and all premolars have erupted. These clinicians believe that it is easier to design the treatment plan and begin treatment when major growth has been completed. This approach avoids

the need to compensate for variations that might occur during or after treatment as a result of unexpected and unpredictable variations in growth patterns. Usually this type of procedure requires comprehensive appliance therapy. This can be time-consuming and pose serious risks to the health of the teeth and surrounding tissue structures. Some conditions are more prone to relapse and require long-term retention.

The other school of thought advocates early treatment because the development of the dental occlusion is a long process, beginning at 6 weeks of intrauterine life and ending 20 or more years later. The most important stage of the dental occlusion is the transitional dentition; hence, most dentoskeletal anomalies start and develop during the primary or mixed dentition. Therefore, proponents believe that early detection of the problem and proper intervention can guide the abnormality toward normality and, under some conditions, can prevent or at least reduce the severity of the problems.

The evidence regarding early versus late orthodontic treatment, a subject that has been debated for years, will be discussed later in the chapter. However, the fact that important occlusal developments and dentoskeletal changes happen during transitional dentition and most dentoskeletal anomalies develop during the primary and mixed dentition prompts the following question: Why should unfavorable dental, skeletal, or soft tissue relationships be allowed to remain for a number of years if they can be corrected completely or partially at an earlier age with a minimum of appliance therapy and treatment effort? Delayed treatment fails to take advantage of growth potential that could eliminate or modify deviations in skeletal growth and the functional matrix associated with mouth breathing, atypical swallowing, and other abnormal behavior. It also fails to take advantage of the opportunity to guide dentoalveolar development. Furthermore, early intervention can also prevent damage to teeth and surrounding structures that may result from a misarticulated and traumatized dentoskeletal relationship.

The author recommends a single- or two-phase treatment approach that begins around the time of the middle or late mixed dentition. There are also conditions that require early intervention during the primary or mixed dentition. All of these procedures will be discussed in detail in part 3 of this book and will include detailed descriptions of cases.

Mechanisms affecting occlusal development

To clarify the rationale for early-age treatment, it is necessary to describe some of

the important mechanisms that can influence occlusal development, such as the long process of occlusal development, genetics and environmental factors, form and function, and locked occlusions.

Long process of occlusal development

Postnatal development of dentofacial structures spans 18 to 25 years. This long process allows practitioners to observe and monitor these changes at different stages of the dentition. Understanding and memorizing all the events that take place during development of the occlusion (see chapter 2) gives clinicians the opportunity to detect and intercept incipient abnormalities at their early stage of development.

During this long process, craniofacial growth is also interacting with the development of the dentition. Different parts of the skull grow at different rates and mature at different ages. Carlson[10] stated that 80% of craniofacial growth is completed by the age of 6 to 8 years, while only 50% of midfacial and mandibular growth is completed by the age of 8 to 10 years. Thus, a considerable amount of midfacial and mandibular growth potential remains during the transitional dentition. Monitoring these important growth changes and their interactions with the transitional dentition will also provide an opportunity to detect and intervene in developing malocclusions.

Genetics and environmental factors

The process of occlusal development and its fundamental principles are under the influence of two basic mechanisms: genetic processes and environmental processes, which can act individually or interact with each other.

The relative contributions of genes and environment as an etiologic factor of malocclusions have been a matter of controversy in the past century. The previous emphasis on genetics was based on the results of early twin and family studies. These studies revealed that the effects of common environment were not separated from the gene-environment interaction. However, Christian[11] conducted an acceptable segregation of genetic from environmental effects.

Research also indicates that genetic mechanisms have more influence on morphogenesis of the craniofacial structure during embryonic life, while environmental factors influence developing occlusion, especially during the early postnatal period. Harris and Johnson,[12] distinguishing between skeletal variables and occlusal, tooth-based variables, evaluated serial assessments of 30 untreated

siblings from the ages of 4 years (full primary dentition) to 20 years (full permanent dentition). They concluded that while the skeletal variables had high heritability, nearly all of the occlusal variability was acquired.

Therefore, many incipient malocclusions that are under the influence of environmental factors during the primary or mixed dentition could be distinguished and prevented. Early recognition and interception of these factors can eliminate or reduce the severity of future problems. Examples of these common factors include:

- Early primary tooth loss
- Delayed exfoliation or overretained primary teeth
- Arch size–tooth size discrepancy
- Abnormalities of tooth number (hyperdontia and hypodontia)
- Eruption problems (ectopia, transposition, impaction, and ankylosis)
- Deleterious oral habits
- Craniofacial dysfunctions, including mouth breathing, abnormal swallowing patterns, and all aspects of cranial posture

Form and function

The orofacial structure is anatomically and functionally one of the most complex regions of the human body. The interplay between form and function is another important mechanism that affects orofacial structure during development of the occlusion. This mechanism is managed by the surrounding environment, which is called the *functional matrix*.

The effects of muscle dysfunction during the primary or mixed dentition are not self-corrected and become worse if continued in older age. Examples of this phenomenon are:

- The interplay between perioral musculature and the tongue and the interaction between tongue size and tongue volume and craniofacial skeletal growth
- Respiration and the capsular matrix influencing the nasomaxillary complex
- Activity of the muscles maintaining the head position, which also can affect craniofacial growth and occlusion

Locked occlusions

Some occlusal interferences, such as posterior or anterior crossbites, can have an adverse effect on the normal rate and direction of jaw growth. Unless treated during early stages of development, these types of irregularities, also called *locked occlusions*, can cause skeletal jaw deformity.

Examples of these malocclusions include the following:

- Collapsed maxillary arch, occurring during the primary or mixed dentition, which can prevent normal sagittal and transverse growth and displacement of the maxillary arch (Fig 1-1)
- Class II division 1 impinging bite, which can cause mandibular collapse within the maxilla, affecting normal anterior growth of the mandible (Fig 1-2)
- Unilateral crossbite with a functional shift, which is a common problem in primary and early mixed dentition and, if not treated at an early age, can affect mandibular growth, causing jaw asymmetry (Fig 1-3)

Early detection and intervention of occlusal interference by mechanical unlocking of occlusion will allow normal function and therefore normal development of the occlusal system.

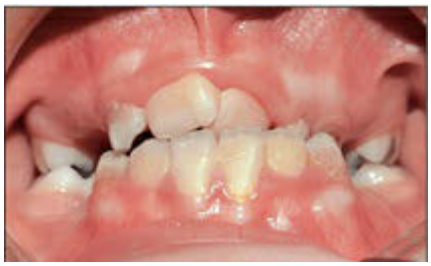


Fig 1-1 Maxillary arch collapse.

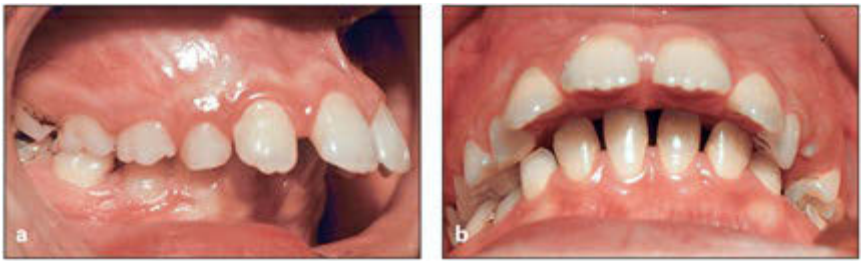


Fig 1-2 *(a and b)* Impinging deep bite and locked mandibular arch.

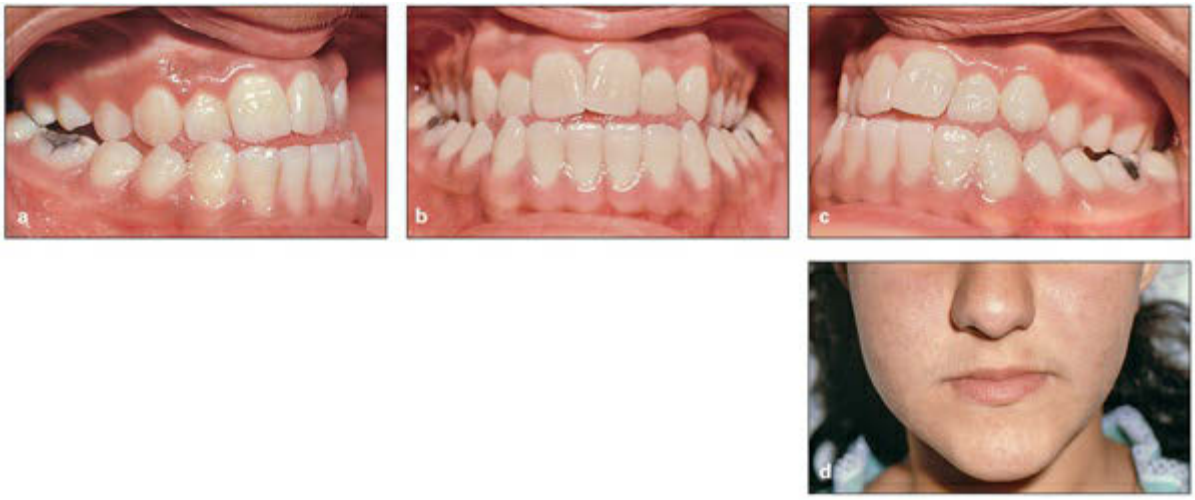


Fig 1-3 *(a to d)* Asymmetric mandibular growth.

Current interest in early-age treatment

Over the past two or three decades, interest in early orthodontic treatment has increased. Parents now seek treatment for their children at an earlier age. This interest has been stimulated by the following developments:

- Parents who underwent orthodontic treatment themselves are more likely to be aware of their children's orthodontic concerns.
- Members of the medical and dental community, including specialists and general practitioners, are more aware of current research and the influence of function on the developing dentition as well as the development of sophisticated appliances for treatment.

Scientific evidence has shown that orthodontic treatment is more than just the treatment of dental misalignment. It is also necessary to understand the cause and the mechanism of effects that are influenced by genetic and environmental factors. For example, functional activity of the surrounding tissues, such as neuromuscular functional balance, habits, and upper airway obstruction, can affect the proper development of occlusion. Therefore, early detection of the problem through systematic diagnostic procedures can ensure that proper interceptive and corrective measures are instituted at the proper time; in this way, many facial and dental problems can be minimized or averted.

Rationale

When considering the reasons why early treatment is recommended, the following important points should be considered:

- Almost all of the occlusal variability that happens during early stages of occlusal development is acquired rather than inherited.
- Most incipient irregularities are preventable and, if neglected, can result in more complicated deformities.
- A considerable amount of midfacial and mandibular growth occurs during the transitional dentition. This must be considered an advantage of early treatment and growth modification.
- It is easier for patients to adapt to a new environment and normal function at early ages, and therefore the outcome has better stability.
- The orofacial skeletons of young children have high plasticity, and thus occlusal guidance is easier to achieve.

When Is the Best Time to Start Early Intervention?

There is no simple answer to the question of timing, because there is no one "best time" to start early treatment. The answer depends on the nature of the patient's problems. Each patient must be evaluated individually, and the treatment plan must be designed according to each individual's problem. The challenge is to select the proper type of intervention at the proper time for each individual case.

As a general rule, clinicians should follow these guidelines when determining the best time to intervene:

- Orthodontic screening must be started by the age of 4 or 5 years.
- Treatment can begin after the permanent first molars and all incisors have erupted.

Occasionally there are situations where even earlier intervention is needed, even at the primary or early mixed dentition stage. Some examples are locked occlusions, posterior and anterior crossbites (especially accompanied with mandibular shift that disturbs normal growth and development of occlusion), or functional problems such as mouth breathing, abnormal tongue position and function, and other deleterious oral habits.

After a thorough examination and analysis of diagnostic findings, clinicians should ask the following questions:

- Is the problem damaging involved teeth, periodontium, temporomandibular joints, or other tissues?
- Is there a possibility for the problem to worsen?
- Is there a possibility of adverse effect on the growth of the other jaw?
- Is this situation psychologically problematic for the patient or others?

If the answer to any of these questions is "yes," then it is time to start early intervention.

How Are Early Intervention Procedures Applied?

In order to apply the proper treatment at the proper time, it is necessary to look first at the principles and strategy of early orthodontic intervention (for more detail, see chapter 11).

Goals of early orthodontic treatment

The strategy and major techniques of early orthodontic intervention include the following important goals:

- Elimination of primary etiologic factors, if possible
- Correction of obvious problems
- Interception of developing problems
- Prevention of worsening of obvious problems
- Preparation of an environment for normal occlusal development and function
- Guidance of growth in a more favorable direction by unlocking occlusal interferences, which can have an adverse effect on occlusion
- Management of arch size–tooth size discrepancy
- Reduction in susceptibility to trauma and incisor fractures (increased overjet)
- Correction of skeletal dysplasia at an early stage of development

Phases of early orthodontic treatment

The controversy surrounding early versus late orthodontic treatment is often

confusing to the dental community. The literature regarding early orthodontic treatment is often misleading. Some practitioners limit their practice only to the permanent dentition, and some believe that early treatment always needs a second phase of treatment, which is not true.

Early orthodontic treatment protocols can be performed in two separate phases. Depending on the type of problem, the age of the patient, and the stage of dentition, this procedure can be accomplished in one phase and sometimes in two phases. Occasionally, a three-phase treatment plan might be implemented if some correction of the primary dentition is needed, such as elimination of posterior cross-bite, and serial extractions in the mixed dentition must be followed by a final phase of treatment for the permanent dentition.

Some patients benefit from a single phase of treatment, which usually starts around the late mixed dentition and ends during the permanent dentition. Other patients might be managed better if treatment is initiated during the early or middle mixed dentition, followed by a phase of observation or retention, and then completed during the permanent dentition with the second phase of treatment. The challenge is to select the proper type of intervention at the proper time for each individual patient.

During each phase, the main objectives of intervention and treatment include reducing adverse growth, preventing dental and skeletal disharmonies, improving the esthetics of the smile, enhancing the patient's self-image, and improving the occlusion. First-phase early orthodontic treatments offer many advantages to both the doctor and the patient. These therapies should be part of every orthodontist's armamentarium.

One-phase early orthodontic treatment

To clarify the different types of early intervention used in orthodontic practice and clearly distinguish between the meanings of these terms, it is necessary to explain them first:

- *One-phase early treatment* consists of a type of interceptive or corrective treatment that is performed during the primary or mixed dentition stage to eliminate the cause and to correct the present abnormality.
- *One-phase treatment* is a single phase of comprehensive treatment for correction of an abnormality, whether in the primary, mixed, or permanent dentition.

For example, one-phase early treatment of Class II malocclusion is a single phase of comprehensive treatment usually started around the end of mixed dentition, just before the growth spurt and ending after the completion of canine eruption. This usually takes about 2 to 3 years.

For more than 40 years, the author has used this comprehensive, one-phase early treatment to correct severe Class II malocclusion (dental or dentoskeletal). Treatment begins around the late mixed dentition and finishes with completion of the permanent dentition. This method does not require a second phase, which is usually expected in early treatment procedures.

The major goal of late mixed dentition treatment is growth modification to take advantage of growth potential during the growth spurt. This type of treatment involves orthopedic management such as extraoral traction, functional therapy, and rapid maxillary expansion.

Some abnormalities may require only one phase of treatment for the correction of the present malocclusion, even at the early or middle mixed dentition stage; for example, regaining space, space maintenance, guidance of eruption, and correction of posterior or anterior dental crossbite may not need second-phase treatment.

Two-phase early orthodontic treatment

This type of early treatment involves an initial intervention during the primary, early, or middle mixed dentition stage, followed by an interim or resting stage, which is a transitional period. During this period, the patient is under observation or may be wearing some type of simple retainer (full- or part-time). After the transition to complete permanent dentition, the second phase of comprehensive fixed appliance therapy is performed for final adjustment.

Two-phase treatment is advocated to address skeletal, dental, and neuromuscular problems such as abnormal habits, hyperactive musculature, crowding, dental cross-bites, hypodontia, supernumerary teeth, and problems of tooth eruption in order to eliminate or reduce the severity of their effects and to facilitate the second phase of treatment. The treatment will be completed by a second phase of comprehensive fixed appliance therapy. The severity of the problem will have been considerably diminished during phase 1 treatment, and a short phase of treatment will be required to align the permanent occlusion.

An example of two-phase treatment is serial extraction, where preparation of anchorage, extractions, and guidance of canine eruption are performed in phase 1. After canine eruption and completion of the permanent dentition, the second phase

begins, with full bonding of appliances for uprighting of teeth, correction of rotation, and minor space closure to finalize the treatment.

Another example of two-phase treatment is early intervention and control of abnormal habits, which might be started during the primary or mixed dentition and followed by an interim phase of supervision during the transitional dentition, until eruption of the permanent dentition for phase 2 treatment.

Many other abnormalities, such as hypodontia and hyperdontia, anterior and posterior crossbite, eruption problems (impaction and transposition ankylosis), abnormal oral habits, and abnormal frenum attachment, require early intervention as a phase 1 treatment during the primary or mixed dentition and a second phase during the permanent dentition.

Occasionally, there are cases that have been planned for two-phase treatment but, after completion of phase 1 and enhancement of the conditions to achieve a normal environment, ultimately do not require a second phase of treatment.

Monitoring the dentition

Depending on the type of malocclusion, the chronologic and skeletal ages of the patient, and the stage of dentition, there are various situations that justify early detection and intervention. The importance of managing the developing dentition and occlusion and its effect on the well-being of infants, children, and adolescents is clear. This management includes the recognition, diagnosis, and appropriate treatment of dentofacial abnormalities. Comprehensive clinical examinations, appropriate pretreatment records, differential diagnosis, sequential treatment planning, and progress records are necessary to manage any condition affecting the developing dentition.

Systematic monitoring of the developing dentition at regular clinical examinations and periodic radiographic evaluation, from the beginning of the mixed dentition to complete eruption of the permanent dentition, can expose many developmental problems and facilitate early intervention. For more than 40 years, the author has consistently advised all undergraduate and postgraduate students that taking serial panoramic radiographs of patients at ages 6, 8, and 10 years is a necessary step in evaluating and monitoring the transitional dentition. Comparison of these serial radiographs taken at the early stages of occlusal development can guide treatment and aid early recognition of many ongoing problems throughout the developing dentition. Early detection also allows clinicians to inform the patient's parents and

plan and recommend the appropriate intervention.

For more detail, see the longitudinal panoramic radiograph monitoring technique in chapter 3.

What Is Controversial About Early Treatment?

Untreated malocclusions can result in a variety of problems, such as susceptibility to dental caries, periodontal disease, bone loss, and temporomandibular joint problems. The most significant detrimental effect can be on the appearance of the patient; studies by Shaw et al[13,14] have confirmed that severe malocclusion is likely to be a social handicap. Facial esthetics have also been found to be a significant determinant of self- and social perceptions and attributes. Tung and Kiyak[15] and Kilpeläinen et al[16] concluded that perceptions of facial esthetics influence psychologic development from early childhood to adulthood. The goal of many clinicians who provide early treatment is not only to reduce the time and complexity of fixed appliance therapy but also to eliminate or reduce the damage to occlusion that can be produced if treatment is postponed.

The majority of debates surrounding the long-term benefits of early orthodontic treatment center around early or late treatment of Class II malocclusion. Some claim that early intervention in Class II situations is the best choice when the problem is skeletal and especially if the problem is the result of mandibular retrusion. Others believe that there is no difference in the final result and a single-phase treatment approach is preferable because of the advantages that accompany the reduced treatment time. The questions related to early treatment have led to the need for critical analyses of the effectiveness of such an approach.

Clinical evidence

The relative merits of the early and late approaches to orthodontic treatment have been debated for years. Randomized clinical trials conducted by Ghafari et al,[17] Keeling et al,[18] and Tulloch et al[19] were specifically designed to address important issues in Class II treatment. All three studies concluded that, for children with moderate to severe Class II problems, treatment seems to be as effective in late childhood as it is at an earlier age. On the basis of these three ongoing clinical trials, these authors concluded that both the single- and two-phase approaches are effective

in the correction of Class II malocclusion and that this correction is the result of both skeletal and dental changes. In a review of the literature, Kluemper et al[20] similarly asserted that both the single- and two-phase approaches are effective in correcting Class II malocclusion. Moreover, depending on the type of appliances and therapeutic techniques used, the treatments seem to exert more influence on skeletal changes in one jaw than in the other.

All of the above studies advocate that both one- and two-phase approaches are effective in Class II correction, but critical evaluation of these studies reveals that the following questions must be clarified:

- Was the type of Class II malocclusion (dental, skeletal, or combination) determined for all subjects?
- What criteria were used for selecting the subjects (the amount of overjet or point A–nasion–point B angle cannot show the whole detail)?
- Was the skeletal discrepancy caused by the mandible, the maxilla, or a combination?
- Was the variability in skeletal growth patterns considered?
- Was skeletal age (growth spurt and growth maturation) considered in each individual case?
- Were variations in patient compliance considered in the analysis?
- Was early treatment managed properly?
- Were the interim phase and follow-up managed properly?

These are conditions that can influence the results of any investigation comparing the results of early and late treatments. Many experienced teachers and clinicians, including Ricketts,[21–23] Subtelny,[24] Gugino and Dus,[25] Bench et al,[26] Graber,[27] McNamara and Brudon,[28] and Dugoni[29] have compared the two approaches and concluded that there are many disadvantages to waiting for the second molars to erupt. In a survey of 159 diplomates of the American Board of Orthodontics,[30] participants were asked what they perceived to be the benefits of early treatment. The most common responses were:

- Greater ability to modify skeletal growth
- Improved patient self-esteem and parental satisfaction
- Better and more stable results

- Less extensive therapy required later
- Reduced potential for iatrogenic tooth damage such as trauma, root resorption, and decalcification

A review of all the literature related to the controversy about early versus late orthodontic treatment reveals that the major differences center around early Class II treatment. There does not seem to be as much controversy about the benefits of other services that can be performed for young patients during the primary or mixed dentition, such as anterior and posterior crossbite correction, habit control, and crowding and space management. Unfortunately, many practitioners, without attempting a thorough evaluation of the many services that can benefit young patients, extend the controversy surrounding Class II treatment to all kinds of early treatment and thus prefer late treatment.

First-phase, or early, orthodontic treatment offers many advantages to both the doctor and the patient, and these therapies should be a part of every orthodontist's armamentarium.

Misconceptions about early treatment

The following are the most controversial claims expressed by opponents of the early-age treatment approach, together with some counterpoints:

Claim 1. Most patients who undergo early treatment require a second phase of treatment.

Counterpoint 1. Some patients do need a second phase of treatment but not most patients. It is also true that, when needed, the second phase of treatment is easier and shorter and produces better skeletal results. Most of the correction has already been accomplished when a second phase of treatment is needed.

Claim 2. Two-phase treatment lengthens the treatment time.

Counterpoint 2. Some two-phase treatments do require a longer period of time because of the patient's age and the need to wait for complete eruption of the permanent dentition. However, the length of treatment must be measured by chair time, not calendar time. Most intervals between visits are longer in early treatment than they are during comprehensive treatment, and each visit needs considerably less chair time. For example, changing an archwire requires more time than does adjusting headgear or a removable appliance.

Claim 3. Improper early treatment can be harmful to the patient's growth pattern.

The reasoning goes that, if growth can be directed advantageously in early treatment, it can also be misdirected.

Counterpoint 3. Current knowledge of growth changes and growth patterns can be very helpful in treatment planning. It is true that in rare cases, growth prediction is more complex and unpredictable, but the length of treatment and continuous observation of a growing patient can guide modifications of the treatment plan, if needed. As the saying goes, it does no good to drive faster if you are on the wrong road. You have to have a map and correct driving directions before starting.

Claim 4. Early diagnosis and treatment planning are sometimes more tentative. When growth has terminated, the future of a malocclusion is more visible and the diagnosis is more certain.

Counterpoint 4. It is true that early diagnosis and treatment planning are more tentative, but periodic reassessment of cephalometric radiographs and study casts (as an absolute necessity) aids in this process. Furthermore, it is easier to monitor treatment progress and the developing occlusion, which is a slow process.

What Are the Benefits of the Early Treatment Approach?

The types of treatments and services that can be provided to young children during the developmental stages of the dentition and skeletal growth are tremendous. Many dental and skeletal anomalies can be prevented or intercepted during the primary or mixed dentition. Some might be treated in one phase and others in two phases, but proper intervention can definitely reduce the duration and complications of second-phase treatment. The following services can be provided to young patients if treatment is initiated at the proper time:

- Space management (see chapter 4)
- Management of incisor crowding (see chapter 5)
- Management of deleterious oral habits (see chapter 6)
- Orthodontic management of missing teeth (see chapter 7)
- Orthodontic management of supernumerary teeth (see chapter 8)
- Diagnosis and management of abnormal frenum attachments (see chapter 9)
- Early detection and treatment of eruption problems (see chapter 10)
- Management of sagittal (see chapter 11), transverse (see chapter 12), and vertical (see chapter 13) dentoskeletal problems in the primary and early mixed dentition

stages

Benefits to patients

- Improvement of facial appearance and self-esteem. Facial esthetics has been found to be a significant determinant of self- and social perceptions. Early intervention in and improvement of a patient's appearance are very important, especially in preadolescents.
- Easier resolution or interception of developing malocclusion. Any incipient orthodontic problem detected during the transitional period is easier to correct than it would be after complete formation of the malocclusion. The resulting longer periods between appointments and shorter chair time of each visit is desirable for patients and their parents.
- Minimization of severe malocclusions. Early detection and interception will minimize the severity of the problem.
- Correction of functional problems. Some tooth malalignments, such as prematurities and incorrect dental inclinations (anterior and posterior crossbites), can result in mandibular shift, functional discomfort, and structural defects and have an adverse effect on normal growth patterns. Early correction can eliminate the patient's discomfort and prevent many complicated problems that can happen later.
- Prevention of damage to teeth and dentoskeletal structures. Many irregularities can cause structural damage. Some irregularities, such as severe dental protrusion and overjet, increase the risk of fracture of the maxillary incisors because of trauma. Early correction of these irregularities would prevent such problems.
- Reduction in the need for extraction. Much of the crowding that develops during the mixed dentition can be corrected by space regaining, expansion, or growth modification. These corrections might not be possible after completion of the growth spurt and eruption of the permanent dentition.
- Greater patient compliance. Prior to their teens, most children are enthusiastic about getting braces and are more comfortable wearing appliances.
- More stable results. A major goal of early intervention is to provide a normal environment to enhance the normal development of occlusion. Teeth in young patients are more adaptive to the changes of orthodontic tooth movement, and the results are more stable.

- Less traumatic, and therefore less painful, treatment procedures. Most early treatments do not require massive tooth movement. Hence, less force is applied, there is less pain, and children are more comfortable. Younger children seem to have a lower resistance to bone and tooth movement and tend to complain less.
- Prevention of psychologic problems, which may occur in some children. The anterior teeth play an important role in appearances. Irregularities can cause psychologic problems for young children and elicit teasing from classmates. Early correction of defects will give children more confidence and lessen the hardship.
- Lower treatment costs. The total fee for early orthodontic treatment is less than that of comprehensive treatment of the permanent dentition because therapy is less extensive, there is less chair time per visit, and appointment intervals are longer. Also, if a second phase of treatment is needed, it tends to be easier and for a shorter period of time.

Benefits to practitioners

- Availability of more treatment options. Because of the patient's age, the possibility of guidance of eruption, and the growth potential, more options are available in treatment planning, especially because the abnormalities are still in developmental stages.
- Better patient compliance. A patient's compliance in orthodontic treatment is one of the most important factors in treatment success. Cooperation is much greater among children aged 7 to 10 years than among older patients. If an adolescent or adult insists on having the appliances removed before the proper results are obtained, the orthodontist might be forced to compromise treatment goals.
- Better use of growth potential. One of the most important benefits of early treatment is utilization of the growth potential to correct skeletal abnormalities and the potential to modify growth, which is impossible to achieve after termination of growth. Proper orthopedic treatment during jaw growth can result in significant reduction of dentoskeletal deformities in three dimensions. These include early Class III correction involving overgrowth of the mandible or underdevelopment of the maxilla, correction of the constricted maxilla for better mandibular growth, or stimulation of mandibular growth in the retrognathic mandible.
- Reduction in the need for extraction. As mentioned earlier, the availability of more options for regaining lost space or creating new space in crowding situations has the potential to reduce the need for tooth extraction.

- Easier control of habits. Control of serious deleterious oral habits is easier to obtain in younger children. Also, future structural damage to the dentition and alveolarskeletal structure can be prevented. If abnormal habits continue, there is more potential for damage, and management of the problems will be more complicated.
- Better management of problematic growth patterns. Long-term observation facilitates control of abnormal, complex problems in the growth pattern, for example, by guiding adjustments to the treatment plan when necessary.
- Less need for mass tooth movement and complex therapy. En masse tooth movements, such as torquing movements, dental compensation, and other complicated mechanics, usually are not necessary in early treatment. Early treatment also reduces the need for second-phase treatment.
- Shorter treatment time in the second phase. Proper intervention at the proper time reduces the duration of the second phase and significantly reduces the severity of the problems treated in phase 2.
- More stable results. Relapse after the completion of treatment is one of the most disturbing events to confront an orthodontist. This failure happens more often after treatment initiated in the permanent dentition than it does after early-age treatment. Early-age treatments are usually performed during occlusal development, providing a normal environment for the dentition. Most tooth movements in early treatment are the result of natural eruption processes caused by guidance techniques. Therefore, the dentition has more potential to adapt to the changes, making posttreatment relapse less likely.

How Can the Profession Encourage Early-Age Treatment?

Traditionally, early orthodontic treatment has not been taught effectively in many orthodontic specialty programs, for the following reasons:

- Some instructors' lack of awareness, interest, or experience in early-age treatment techniques.
- Management of early orthodontic treatment in some cases may last several years; treatment of these patients cannot fit into a 2-year curriculum. However, it is still possible to continue these cases with a new resident and through an organized system of patient transfer. Furthermore, all residents would be exposed to different phases of treatment for these types of problems.

• Some insurance companies do not provide coverage for early-age treatment.

For the benefit of patients and community care and to prevent the high expense of comprehensive treatment, the author suggests that the American Association of Orthodontists place more emphasis on the inclusion of the early treatment approach in all orthodontic specialty programs, including a mandate that it be a required part of the curriculum. It would also be beneficial to provide an introductory course on this topic to both undergraduate dental students and pediatric dental residents.

Summary

- Early orthodontic treatment includes all types of preventive, interceptive, or corrective treatments applied during the primary or mixed dentition, before the complete development of occlusion.
- Most dentoskeletal malocclusions initiate and develop during the transitional dentition.
- A considerable amount of midfacial and mandibular growth occurs during the transitional dentition.
- Studies indicate that genetic mechanisms have more influence during embryonic life, while environmental factors influence the developing occlusion, especially during the early postnatal period.
- Many incipient malocclusions, which are under the influence of environmental factors during the primary or mixed dentition, are detectable and preventable.
- Examples of these factors include early primary tooth loss, delayed exfoliation, overretained primary teeth, arch size–tooth size discrepancy, hyperdontia and hypodontia, eruption problems (ectopia, transposition, impaction, and ankylosis), deleterious oral habits, all craniofacial dysfunctions including mouth breathing, abnormal swallowing patterns, and all aspects of cranial posture.
- Why should an unfavorable dental, skeletal, or soft tissue relationship be allowed to remain for a number of years if it can be corrected completely or partially at an earlier age, with a minimum of appliance therapy and treatment effort?
- Early orthodontic protocols can be applied during occlusal development and facial growth period into separate phases. One-phase treatment is an

interceptive treatment that can be performed during the primary or mixed dentition to correct existing abnormalities, to eliminate or reduce severity of problems, and to facilitate the second phase of treatment. Two-phase treatment consists of an initial intervention followed by an interim or arresting phase; after eruption of the complete permanent dentition, the second or final phase of comprehensive therapy is performed.

- The main objectives of early-age treatment are to prepare an environment for normal occlusal development, to eliminate or control any environmental factor disturbing normal occlusal development, to correct or to guide a developing malocclusion to normal occlusion to take advantage of growth potential to modify skeletal growth, and to eliminate the need for or reduce the duration of second-phase of treatment.
- Early treatment has various benefits for patients and practitioners, such as better patient compliance, better final esthetic results due to growth modification, more stable results, less damage to teeth and supporting structures, the availability of more treatment options, a better chance to prevent extraction, and better use of growth potential.

References

1. Kingsley NW. Treatise on Oral Deformities as a Branch of Mechanical Surgery. New York: Appleton & Lange, 1880.
2. Case C. Dental Orthopedia and Cleft Palate. New York: Les L. Bruder, 1921.
3. Angle EH. Treatment of Malocclusion of the Teeth, ed 7. Philadelphia: SS White, 1907.
4. Oppenheim A. A possibility for physiologic orthodontic movement. Dent Rec (London) 1945;65:278–280.
5. Kloehn S. Guiding alveolar growth and eruption of the teeth to reduce treatment time and produce a more balanced denture and face. Angle Orthod 1947;17:10–33.
6. Tweed CH. Indication for the extraction of teeth in orthodontic procedure. Am J Orthod Oral Surg 1944–1945;42:22–45.
7. Brodie AG. On the growth pattern of the human head from the third month to eighth year of life. Am J Anat 1941;68:209–262.
8. Broadbent BH. A new x-ray technique and its application to orthodontia. Angle Orthod 1981;51:93–114.
9. Popovich F, Thompson GW. Evaluation of preventive orthodontic treatment between three and eighteen years of age. In: Cook JR (ed). Transactions of the Third International Orthodontic Congress. St Louis: Mosby, 1975:260–281.
10. Carlson DS. Biological rationale for early treatment of dentofacial deformities. Am J Orthod Dentofacial Orthop 2002;121:554–558.
11. Christian JC. Testing twin means and estimating genetic variance: Basic methodology for the analysis of quantitative twin data. Acta Genet Med Gemellol (Roma) 1979;28:35–40.
12. Harris EF, Johnson MG. Heritability of craniometric and occlusal variables: A longitudinal sib analysis. Am J Orthod Dentofacial Orthop 1991;99:258–268.

13. Shaw WC, Rees G, Dawe M, Charles CR. The influence of dentofacial appearance on the social attractiveness of young adults. Am J Orthod 1985;87:21–26.
14. Shaw WC. The influence of children's dentofacial appearance on their social attractiveness as judged by peers and lay adults. Am J Orthod 1981;79:399–415.
15. Tung AW, Kiyak HK. Psychological influence on timing of orthodontic treatment. Am J Orthod Dentofacial Orthop 1998;113:29–39.
16. Kilpeläinen PV, Phillips C, Tulloch JF. Anterior tooth position and motivation for early treatment. Angle Orthod 1993;63:171–174.
17. Ghafari J, Shofer FS, Jacobsson-Hunt U, Markowitz DL, Laster LL. Headgear versus function regulator in the early treatment of Class II, division 1 malocclusion: A randomized clinical trial. Am J Orthod Dentofacial Orthop 1998;113:51–61.
18. Keeling SD, Wheeler TT, King GJ, et al. Anteroposterior skeletal and dental changes after early Class II treatment with Bionators and headgear. Am J Orthod Dentofacial Orthop 1998;113:40–50.
19. Tulloch JF, Phillips C, Proffit WR. Benefit of early Class II treatment: Progress report of a two-phase randomized clinical trial. Am J Orthod Dentofacial Orthop 1998;113:62–72.
20. Kluemper T, Beeman C, Hicks P. Early orthodontic treatment: What are the imperatives? J Am Dent Assoc 2000;131:613–620.
21. Ricketts RM. Early treatment. 1. J Clin Orthod 1979;13:23–38.
22. Ricketts RM. Early treatment. 2. J Clin Orthod 1979;13:115–127.
23. Ricketts RM. Early treatment. 3. J Clin Orthod 1979;13:181– 199.
24. Subtelny JD. Early Orthodontic Treatment. Chicago: Quintessence, 2000.
25. Gugino CF, Dus I. Unlocking orthodontic malocclusions: An interplay between form and function. Semin Orthod 1998;4:246–255.
26. Bench RW, Gugino CF, Hilgers J. Bioprogressive therapy. 11. J Clin Orthod 1978;12:505–521.
27. Graber TM. Extraoral force: Facts and fallacies. Am J Orthod 1955;41:490–505.
28. McNamara JA, Brudon W. Orthodontic and Orthopedic Treatment in the Mixed Dentition. Ann Arbor, MI: Needham Press, 1995.
29. Dugoni SA. Comprehensive mixed dentition treatment. Am J Orthod Dentofacial Orthop 1998;113:75–84.
30. Bishara SE, Justus R, Graber TM. Proceedings of the workshop discussions on early treatment. Am J Orthod Dentofacial Orthop 1998;113:5–6.

Development of the Dentition and Dental Occlusion

In any difficult situation, the first step toward remediation is to recognize the problem. The first step in recognizing the problem is to know how the problem has developed. Thus, the best "cure" is early recognition and proper intervention before the problem arises. A review of the prenatal stages of tooth development, especially those occurring during the embryonic period, helps to clarify the normal relationships of adult body structures and the causes of congenital anomalies.

It is not within the scope of this chapter to discuss all aspects of embryology and odontogenesis. There are many reference texts on oral histology, embryology, and developmental anatomy. There are also many contemporary research reports about the molecular level of tooth development. However, this chapter will provide a brief review of the different stages of dentition during prenatal life.

The embryonic period is one of the most critical developmental events, including weeks 3 to 8 of intrauterine life. During this stage, all major external and internal structures are established. Most developmental processes are controlled by precise coordination and interaction of genetic and environmental factors. This control mechanism guides differentiation and synchronizes tissue interactions, migration of cells, and controlled proliferation.

Most facial structure is ultimately derived from migration of neural crest cells, which occurs during the embryonic period. Any interference with this migration can cause different craniofacial anomalies. For example, teratogenic agents, such as viruses and some drugs, can produce this interference and trigger the incidence of congenital anomalies.

Development of the dentition is also a sequence of events that occurs in an orderly and timely fashion; it begins during this stage (week 6 of intrauterine life), continues

as a very long process, and ends after the age of 20 years. There are many normal transitional changes during this course of development that must be separated from the abnormal.

Development of the dentition is an integral part of craniofacial structure. Skeletal growth of craniofacial bony structures also interacts with the development of the dentition, which leads to normal interdigitation between the maxillary and mandibular dental arches. Any disruption of these complicated processes may affect the ultimate occlusal status. Appropriate actions are needed to restore the normal processes of occlusal development. Depending on the age of the patient and the stage of dentition, these treatment procedures may be preventive, interceptive, or corrective.

An understanding of the complex processes of the growth and development of the face and dentition can play an important role in recognition of dentofacial disharmony and help in diagnosis and treatment planning. The growth and changes that take place during the development of the dentition are also a basis for clinical application of early-age orthodontic treatment within the complex processes of occlusal development. An understanding of the structural components can help clinicians to recognize developing problems at their initial stages and to determine the proper time of intervention.

Therefore, a thorough grasp of all stages of development of this system is a must for any dental practitioner treating children who are in the developmental stages of occlusion.

The formation, eruption, and exfoliation of teeth; their changes from a deep position within the jawbones into the oral cavity; and the concomitant growth of bones comprise one of the most fascinating processes of biology. This chapter discusses different stages of tooth formation and eruption and developmental stages of occlusion at three different stages:

1. Prenatal stage
2. Neonatal stage
3. Postnatal stage

Prenatal Development of the Dentition

The embryonic period, weeks 3 to 8 of intrauterine life, is the most critical stage of

developmental events. Development of the occlusion initiates during week 6 of intrauterine life and ends after 20 years of age. Neural crest cells begin migration during the embryonic period, forming most of the facial structure. Any interference with this migration can cause different craniofacial anomalies. By the end of this period, all of the main organ systems have begun to develop.

During the first 2 months of intrauterine life, the ectomesenchyme cells, from which the orofacial structures develop, undergo a complex and coordinated series of steps of proliferation and differentiation. At approximately 7 weeks of intrauterine life, a bandlike ectodermal thickening called the *dental lamina* develops in maxillary and mandibular processes. Odontogenesis (tooth development) begins with budding of the dental lamina and continues into the next week of life.

Massler and Schour[1] have divided the life history of the primary dentition, from initiation of tooth formation until birth and exfoliation, into six major stages: *(1)* growth, *(2)* calcification, *(3)* eruption, *(4)* attrition, *(5)* resorption, and *(6)* exfoliation.

The growth stage, or tooth formation, consists of the following five subdivisions:

1. Initiation
2. Proliferation
3. Histodifferentiation
4. Morphodifferentiation
5. Calcification

Initiation stage

At this stage, the primitive oral cavity, or *stomodeum*, is lined by epithelium that consists of two to three cell layers. These cells cover the embryonic connective tissue originating from the neural crest. This is known as *ectomesenchyme*.

Epithelial thickening occurs after 37 days and is the first sign of development of the dentition.[2]

At this stage, certain cells of the basal layer of the oral epithelium proliferate at a more rapid rate than adjacent cells and form a horseshoe-shaped thickened band of epithelium around the mouth, called the *primary epithelial band*, which corresponds in position to the future dental arch (Fig 2-1). The primary epithelial band quickly gives rise to two subdivisions, known as the *dental lamina* and the *vestibular*

lamina.

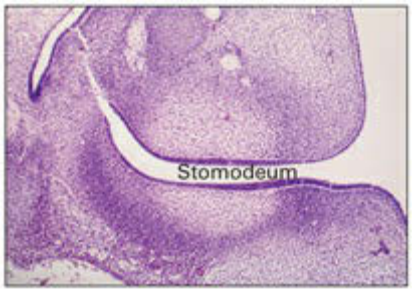


Fig 2-1 Epithelial thickening.

Dental lamina

The dental lamina develops by proliferation of the basal layer of the oral epithelium at a rate that is faster than that of adjacent cells and produces a thicker region extending along the entire free margin of the jaw (Figs 2-2 and 2-3).

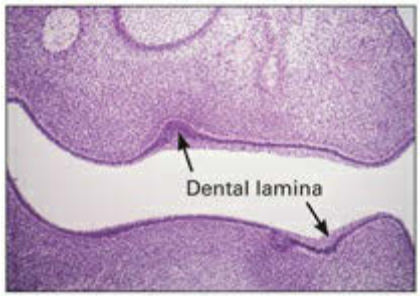


Fig 2-2 Upper and lower dental lamina.

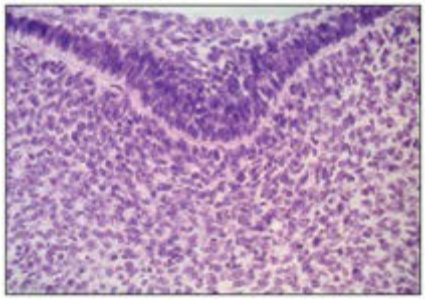


Fig 2-3 Higher magnification of the dental lamina.

Vestibular lamina

The vestibular lamina is another proliferation of the oral epithelium; it occurs outside of the dental lamina (buccally and labially). As a result of proliferation of the vestibular lamina into the ectomesenchyme, the vestibule develops and forms a separate plate between the cheeks, lips, and developing dental lamina. This separating plate rapidly degenerates and forms a sulcus, or vestibule, between the

cheek and tooth area (Fig 2-4).

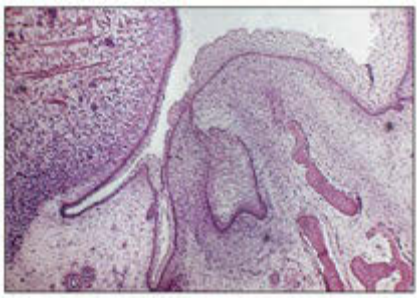


Fig 2-4 Vestibular lamina.

From this point, tooth development proceeds in three stages (bud, cap, and bell stages), and 10 round or ovoid swellings occur in each arch in the position to be occupied by the primary teeth.

Although these stages are discussed separately here, tooth formation is a continuous process, and a clear distinction between the transition stages is not possible.

Bud stage (proliferation stage)

Within the dental lamina, a localized proliferative activity leads to the formation of tooth buds as a series of 10 round or ovoid swellings of epithelial ingrowth into the ectomesenchyme, corresponding to the position of future primary teeth (Fig 2-5). According to Ten Cate,[3] at this time the mitotic index, the labeling index, and the growth of the epithelial cells are significantly lower than corresponding indices in the underlying ectomesenchyme, which suggests that part of the "ingrowth" is achieved by ectomesenchymal upgrowth.

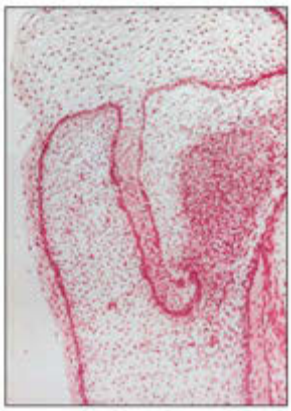


Fig 2-5 Proliferation stage (bud formation).

Within 6 to 8 weeks, 10 primary tooth buds develop from anterior to posterior.

These proliferating cells contain the entire growth potential of the teeth. The permanent tooth germs of the incisors, canines, and premolars develop as a result of new extension and further proliferation of the buds of their primary predecessors, within the dental lamina, attached to the dental organ of the primary tooth germ, which leads to the formation of a new cap on the lingual aspect of primary germs (Fig 2-6).

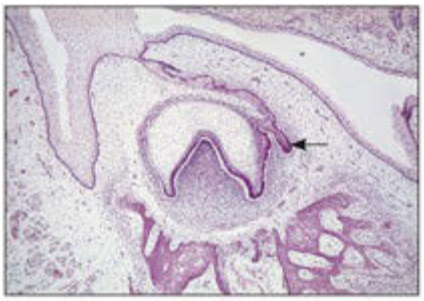


Fig 2-6 Permanent tooth bud formation.

Permanent molars follow a different process, when the jaw growth and bone remodeling increase the body length of the jaw. The dental lamina burrows posteriorly beneath the epithelium of oral mucosa into the ectomesenchymal tissue and forms the first, second, and third molar germs.

Enamel knot

The enamel knot is a localization of cells on an enamel organ that appear thickened in the center of the inner enamel epithelium during the late bud stage of development at the site of the primary tooth cusps (see Fig 2-9).

According to Ten Cate,[3] the function of this structure is not known, but it seems that it is involved in determining the initial position of the first cusp of the tooth during crown pattern formation.

Developing abnormalities at the bud stage

During the bud stage, abnormal proliferation, lack of initiation, or hyperactivity of the dental lamina can cause different abnormalities in tooth formation:

- Hypodontia: Congenital absence of a tooth can be the result of a lack of initiation or an arrest of the proliferation of dental lamina cells.
- Hyperdontia: Supernumerary teeth are the result of a hyperactivity of the dental lamina and continued bud formation of the enamel organ.

- Odontoma: During bud formation, and depending on the degree of differentiation from the enamel organ, a supernumerary tooth or a dysmorphic tooth tissue called an *odontoma* can be produced.
- Cyst formation: Excessive proliferation of cells can produce epithelial rests. When epithelial rest cells are stimulated and become active, they assume secretory functions, which can result in cyst formation.
- For additional detail on these phenomena, see chapters 7 and 8.

Cap stage (proliferation stage)

During this stage, proliferation of the cells continues. As a result of unequal growth and proliferation of different parts of the dental organ, a caplike configuration develops, and a shallow invagination appears on the deep surface of the bud. This consists of the ball of condensed ectomesenchymal cells, called the *dental papilla*, that later form the dentin and the pulp (Fig 2-7). Surrounding the enamel organ, the ectomesenchymal cells form the dental follicles (Fig 2-8). The peripheral cells of the cap later form the outer and inner enamel epithelium.

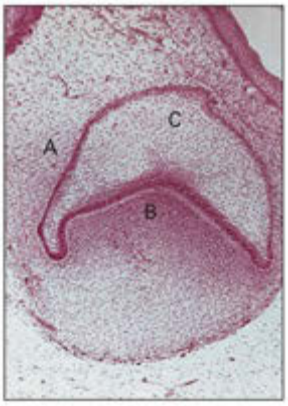


Fig 2-7 Early cap stage. A— dental follicle; B—dental papilla; C—enamel organ.

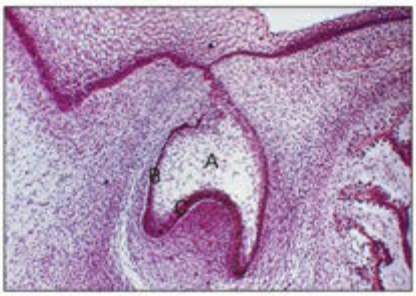


Fig 2-8 Early bell stage. A—enamel organ; B—outer enamel epithelium; C—inner enamel epithelium.

At this stage, each bud consists of three parts (see Fig 2-8): *(1)* the enamel organ, derived from ectoderm; *(2)* the dental follicle, derived from mesenchyme; and *(3)* the dental papilla, derived from mesenchyme.

Early bell stage (histodifferentiation stage)

At this stage, the epithelium continues to invaginate and deepen. The enamel organ changes from a caplike configuration to a bell-shaped form; dentinogenesis and amelogenesis both begin (Fig 2-9). Late in the cap stage, during the transition of the tooth germ from cap to bell, important developmental changes begin. This process is called *histodifferentiation*.

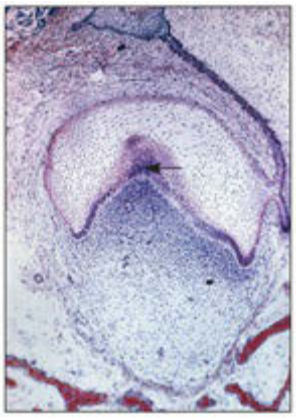


Fig 2-9 Enamel knot *(arrow)*.

The enamel organ undergoes histodifferentiation, and the cells in the center of the dental organ continue to synthesize and secrete glycosaminoglycans into the extracellular space between the epithelial cells. Glycosaminoglycans are hydrophilic and absorb water into the dental organ.

The increasing amount of fluid increases the volume of the extracellular compartment of the dental organ. This causes the cells to separate while they are connected with each other by desmosomal contacts. Next, the stretched cells become star shaped and are then known as the *stellate reticulum* (Fig 2-10).

Histodifferentiation is actually the end of the proliferative stage, as the cells lose their capacity to multiply.

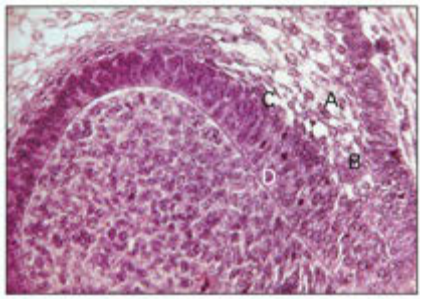


Fig 2-10 Histodifferentiation. A—stellate re-ticulum; B—outer enamel epithelium; C— stratum intermedium; D—ameloblasts.

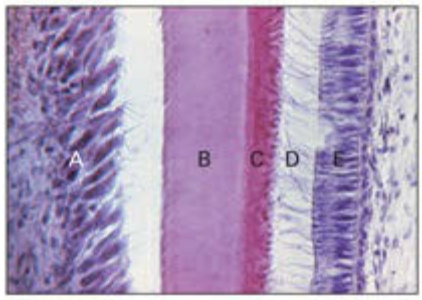


Fig 2-11 Histodifferentiation. A—ameloblast; B—enamel; C—dentin; D—cytoplasmic extension; E—odontoblast.

Stratum intermedium

Some epithelial cells between the internal dental epithelium and the stellate reticulum differentiate into a layer called the *stratum intermedium*. This layer is composed of two to three layers of flattened cells. The stratum intermedium gives rise to ameloblasts at a later stage, which produce enamel (Figs 2-10 and 2-11). Although the cells of this layer are histologically distinct from the cells of the internal dental epithelium, both layers should be considered as a single functional unit responsible for the formation of enamel.[4]

The outer surface of the enamel organ consists of simple cubical cells called the *external enamel epithelium* or *outer enamel epithelium*. Stellate reticulum cells are connected to each other and to the cells of the external dental epithelium and stratum intermedium by attachment plaques known as *desmosomes*.

Odontoblasts differentiate under an organizing influence stemming from the cells of the internal dental epithelium. During this stage, the cells of the dental papilla differentiate into odontoblasts, and the cells of the internal dental epithelium differentiate into ameloblasts (Figs 2-11 and 2-12; see also Fig 2-6).

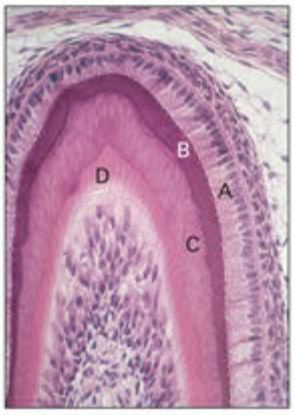


Fig 2-12 Morphodifferentiation. A—ameloblast; B—enamel; C— dentin; D—odontoblast.

Developing abnormalities at the histodifferentiation stage

Disturbances during the histodifferentiation stage of tooth formation cause abnormalities in the differentiation of the formative cells of tooth germs. This results in abnormal structures of the dentin or enamel:

- Amelogenesis imperfecta: Amelogenesis imperfecta is a clinical example of the failure of ameloblasts to differentiate properly to enamel at this stage.
- Dentinogenesis imperfecta: Dentinogenesis imperfecta is a clinical example of failure of the odontoblasts to differentiate properly to dentin during the histodifferentiation stage of tooth formation.

When the tooth germ is growing rapidly during the early bell stage, cell division occurs throughout the internal dental epithelium. As development continues, cell division ceases because the cells are beginning to differentiate and assume their eventual function of producing enamel.

Late bell stage (morphodifferentiation stage)

In this stage of tooth formation, the formative cells of ameloblasts and odontoblasts are arranged to outline the final form and size of the teeth. Until the completion of the bell stage, all cells of the internal dental epithelium are continually dividing and tooth germs are rapidly growing. At the end of this stage, the overall growth and final morphologic pattern of the tooth germ have been established.

The cessation of mitotic division determines the final shape and size of a tooth. This process occurs before matrix deposition.

When the inner enamel epithelium is arranged, the boundary between it and the odontoblast outlines the future dentinoenamel junction (see Fig 2-12).

Developing abnormalities at the morphodifferentiation stage

At the end of the morphodifferentiation stage, proliferation ends, and the morphology and size of the tooth germ reach their final form. Disturbances and aberrations occurring during the morphodifferentiation stage lead to teeth with abnormal forms and sizes. Some examples of these abnormalities are peg-shaped lateral incisors, mulberry molars, microdontia, and macrodontia.

Formation of the permanent dentition

The permanent teeth (successional teeth) also arise from the dental lamina, but not all permanent tooth formation is alike. Permanent incisors, canines, and premolars form as a result of extraproliferative activity from the dental organ of the primary teeth within the dental lamina. This extraproliferative activity leads to the formation of another epithelial cap located on the lingual aspect of the primary tooth germ that initiates the formation of the permanent tooth bud (see Fig 2-6).

The tooth germs of the first, second, and third permanent molars, which have no primary tooth predecessor, originate in a different way. When jaw growth and bone remodeling increase the body length of the jaw, the dental lamina burrows posteriorly beneath the epithelium of the oral mucosa. This, and the associated ectomesenchymal response, forms the tooth germs of the first, second, and third molars. This backward extension of dental lamina into the ramus area develops from the mandibular body, which has a more horizontal position at this age.

Occasionally, insufficient mandibular growth or insufficient resorption of the anterior border of the ramus during remodeling results in a space deficiency that can lead to third molar impaction in the bony ramus of the adult mandible.

Hard tissue formation

Dentin and enamel are two principle hard tissues of the tooth that form during the late bell stage. Dentin, as the bulk-forming tissue of the tooth, is a specialized hard connective tissue; dentin formation always precedes enamel formation and marks the onset of the crown stage of tooth development.

Overall growth of the tooth germ continues by cell division of all the cells of the

internal dental epithelium until the completion of the bell stage (Fig 2-13).

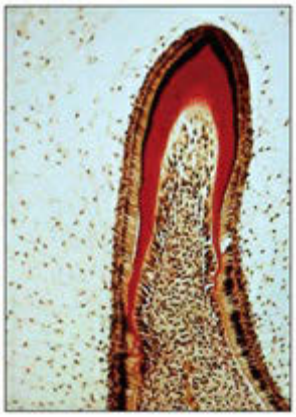


Fig 2-13 Early crown formation.

The first area of dentinogenesis appears at the future cusp tip, or the growth center, where mitotic cell division ceases. The internal dental epithelial cells differentiate and assume their role in producing enamel. The occurrence of second, third, and more zones of maturation within the internal dental epithelium lead to the final cuspal pattern of the tooth.

Role of the enamel knot in cusp formation. The enamel knot is a localized thickening of cells that appears in the center of the inner enamel epithelium of the enamel organ during the late bud stage of development. This corresponds to the site of the primary tooth cusps (see Fig 2-9). Vaahtokari et al[5] concluded that the enamel knot acts as a signaling center, responsible for directing cell proliferation and subsequent cuspal morphogenesis in the developing enamel organ. The enamel knot is seemingly necessary for morphogenesis of the tooth germ to progress from the bud to the cap stage. The ectomesenchyme eventually assumes the dominant role in determining the final pattern of the cusps and the shape of the teeth.

Dentinogenesis. Dentinogenesis, or dentin formation, in the crown stage is the first identifiable feature of tooth development, which starts at the tip of future cusps. At this time, mitotic activity ceases, and the ectomesenchymal cells increase rapidly in size and differentiate into odontoblasts. Odontoblasts are the dentin-forming cells.

Odontoblasts start collagen formation and elaborate the organic matrix of dentin by formation of collagen fibers and ground substance. Mineralization takes place following matrix deposition and involves the precipitation of inorganic calcium salts.

When organic matrix is deposited, the odontoblasts move toward the center of the

dental papilla, leaving behind a cytoplasmic extension called the *odontoblastic process*. The odontoblastic process is embedded in mineralized dentin tissues, and the tubular character of dentin is established. Dentin formation proceeds toward the inside of the dental papilla and odontoblastic process and results in the secretion of hydroxyapatite crystals and mineralization of the matrix (see Fig 2-11).

Amelogenesis. Although dentin must be present for enamel formation, within the enamel organ the preameloblasts differentiate before the odontoblasts and have an inductive influence on the odontoblasts, sending a message that causes them to differentiate and secrete dentin. Ameloblasts also require the signal of dentin formation to initiate their secretory activities. A message is sent from the newly differentiated odontoblasts to the inner enamel epithelium, causing further cell differentiation and activating secretory ameloblasts to form enamel tissue. This prerequisite is an example of the biologic concept termed *reciprocal induction*.

Enamel formation generally occurs in two stages: *(1)* the secretory stage and *(2)* the maturation stage. In the first stage of amelogenesis, the function of the ameloblasts is to secrete and release enamel proteins into the surrounding area and produce an organic matrix against the newly formed dentin surface. Ameloblasts immediately become mineralized by alkaline phosphatase enzymes and form the first layer of enamel tissue, causing a separation of the ameloblastic cells from the dentin and leaving enamel tissue behind (see Fig 2-6).

Initial mineralization of enamel matrix takes place almost simultaneously with organic matrix production; an unmineralized organic matrix cannot be found.

In the maturation stage, ameloblasts change their function from enamel matrix production to mineralization. Mineralization takes place by transferring substances used in the formation of enamel. In this stage, most of the materials transported by ameloblasts are proteins used to complete mineralization.

Mineralization of dental hard tissues (calcification stage)

The production of mineralized tissues involves two major processes, apposition and calcification.

Apposition

As explained earlier, at the morphodifferentiation stage, enamel and dentin tissues

are initially secreted by ameloblasts and odontoblasts as a nonvital extracellular secretion in the form of a tissue matrix. The tissue matrix is deposited layer by layer along the future dentinoenamel and dentinocemental junctions. The organic matrix for enamel tissue is not collagenous, but the organic matrix for dentin, cementum, and bone is collagenous. The organic matrix for enamel comprises mainly unique proteins (mostly amelogenin), reflecting the tissue's epithelial origin. The organic matrix in dentin forms a substantial part of the mineralized tissue.

The organic matrix of enamel is removed during the final stage of mineralization and leaves less than 1% of the weight of organic matrix. Matured enamel organic matrix is replaced with hydroxyapatite. Thus, enamel is physically different from dentin and cementum because it is formed from a noncollagenous matrix (mostly amelogenin) that is almost completely removed after mineralization.

As already mentioned, the formation of dentin always precedes enamel formation and marks the onset of the crown stage of tooth development. At this stage, the dental lamina is disintegrating, so the tooth germ now continues its development separated from the oral epithelium. The crown pattern of the tooth is established by folding of the internal dental epithelium. This folding reduces the amount of stellate reticulum over the future cusp tip. Dentin and enamel have begun to form at the crest of the folded internal dental epithelium. This stage is associated with the formation of the dental hard tissues, commencing at about the 18th week (Fig 2-14).

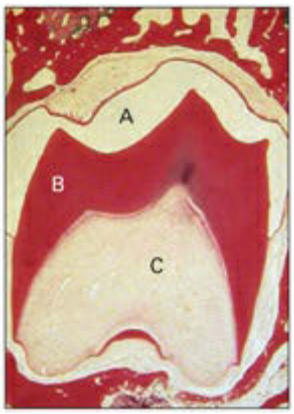


Fig 2-14 Crown stage. A shows the enamel space that has been lost during slide preparation. B—dentin; C—dental papilla.

Calcification

The second stage of dental hard tissue formation, like other hard tissues, is calcification or mineralization. This process takes place following the matrix deposition by precipitation of inorganic calcium salts (primarily in the form of calcium hydroxyapatite crystals) within the deposited matrix.

The mechanism involved in mineralization is not completely understood, especially in the first step, the formation of first crystals. According to Berkovitz et al,[4] any tissue could calcify after crystal formation because blood plasma is supersaturated with calcium and phosphate ions, meaning that mineralization is not limited by the supply of basic ions. An example of this phenomenon is the pathologic calcification that can occur in soft tissues (eg, muscle and tendon).

The structural unit of calcified tissues is the crystal or crystallite, which has a basic shape in all four calcified tissues (enamel, dentin, cementum, and bone). The sizes of the crystals within these tissues are all different.

Calcification of cartilage, which is controlled by the cells, is another mechanism that seems to be occurring in the initial formation of dentin and bone. These cells form small matrix vesicles that contain calcium and phosphate ions, alkaline phosphatase, and calcium-binding lipids. These vesicles separate from the cells, and conditions within the vesicles permit formation of hydroxyapatite crystals.

Precipitation of small niduses of crystals continues with precipitation of further niduses (calcospherites) spreading around the original nidus and increasing its size by the addition of concentric lamination. Finally, through approximation and fusion, these individual calcospherites transform into a homogenously mineralized layer of tissue matrix.

After the secretory phase of amelogenesis is completed and the ameloblasts enter the maturation stage, they perform a functional and structural task, removing proteins and water from the maturing enamel and allowing them to achieve complete mineralization. Once the enamel is completely mineralized, the ameloblasts shrink from columnar to cubical or flattened cells but remain a part of the reduced enamel epithelium that forms a more or less continuous lining over the completed enamel.

Figure 2-14 shows the crown stage of tooth development at a point when hard tissue formation is well advanced.

Developing abnormalities at the mineralization stage

Any systemic disturbance or local trauma that injures the ameloblastic cells during enamel formation can interrupt or arrest matrix apposition, resulting in enamel hypoplasia. These disturbances include tetracycline deposition, which causes yellow to brown hypoplastic enamel, and dental fluorosis, which results when an individual receives too much fluoride during tooth development.

Dentin hypoplasia is less common than enamel hypoplasia and occurs only after severe systemic disturbances. If the calcification process is disturbed, there is a lack

of fusion of the calcospherites. These deficiencies are not readily identified in enamel, but they are evident microscopically in dentin and are referred to as *interglobular dentin*.

Root formation

After enamel formation of the crown is complete and the crown is fully formed, root formation begins, and the inner and outer dental epithelium reaches and joins at the rim of the dental organ. This junctional zone is known as the *cervical loop* (Fig 2-15).

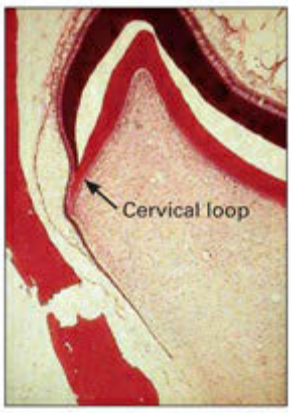


Fig 2-15 Root formation.

As epithelial cells of the external and internal dental epithelium continue proliferation, the cervical loop forms a double layer of cells known as *Hertwig's epithelial root sheath* (Fig 2-16).

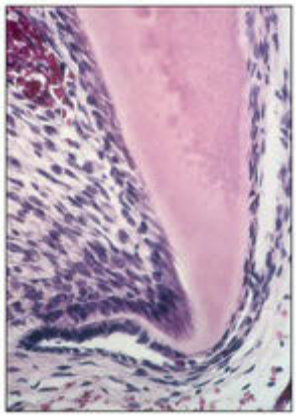


Fig 2-16 Hertwig's epithelial root sheath.

This epithelial sheath grows around the dental papilla, between the papilla and the dental follicle, and covers the entire dental follicle except the basal portion of

the papilla that encloses the primary apical foramen. Hertwig's epithelial root sheath is responsible for the shape and number of root formations.

The dental follicle lies external to the root sheath and forms cementum, periodontal ligament (PDL), and alveolar bone. Root development involves interactions between the dental follicles and the epithelial root sheath. The onset of root development coincides with the axial phase of tooth eruption.

As the inner epithelial cells of the root sheath progressively enclose more and more of the expanding dental papilla, they initiate the differentiation of odontoblasts from cells at the periphery of the dental papilla. These cells eventually form the dentin of the root. In this way, a single-rooted tooth is formed. Multirooted teeth are formed in the same way, and all teeth undergo the same process. It is still not clear, however, how various crown shapes develop.

Molecular level of tooth development

The main objective in discussion of the molecular level of tooth development is to give an overview of the more established theories on the molecular regulation of tooth development and to briefly introduce some recent advances.

The prechordal mesoderm, the craniofacial ectoderm, and the neural crest cells that develop on the dorsal side of the neural tube play a complex interaction in the formation of all craniofacial structures. The migration of neural crest cells begins cranially and gradually extends caudally, ultimately differentiating into a wide variety of adult structures.

Neural crest cells are derived from the ectoderm, but because of their importance in ectodermal-mesenchymal interaction, they are called the *fourth germ layer*. Neural crest cells express homeobox genes in the mandibular and maxillary arches for orofacial development. Homeobox genes are a large group of genes that code for transcription factors responsible for regulating the expression of downstream target genes. A homeobox is a DNA sequence found within genes that are involved in the regulation of patterns of morphogenesis in animals, fungi, and plants.

The different stages of tooth formation and morphologic changes, including bud, cap, and bell stages and terminal differentiation, have been known for several decades, but the molecular interactions involved in this process have been researched widely only in the past two to three decades. In this process, a series of inductive and reciprocal signals between the epithelium and mesenchyme determine the number, growth, morphology, and the ultimate differentiation of craniofacial and

tooth tissues and organs.

Development of the mammalian teeth as a model system has been intensively studied for epithelial-mesenchymal interactions during organogenesis, and progress has been made in identifying key molecules involved in this signaling. It is known that the morphogenesis and cell differentiation in developing teeth are governed by interactions between the oral epithelium and neural crest–derived ectomesenchyme. It has also become evident recently that the inductive interactions between epithelial and mesenchymal cells that operate in these processes are the same signaling molecules involved in the development of most vertebrate organs.

Coin et al[6] reported that bone morphogenetic protein 2 (BMP-2), combined with apatite, could induce both morphologic and functional differentiation of ameloblasts using the in vitro culture system. BMPs are known to be a group of growth factors and cytokines that have the ability to induce the formation of bone, tooth, and cartilage. BMPs are also available commercially as oral supplements.

Fibroblast growth factors (FGFs), a family of growth factors involved in embryonic development, have been implicated as regulators of mesenchymal gene expression and cell proliferation during tooth initiation, epithelial folding morphogenesis, and the establishment of tooth shape (specifically FGF-4, -8, and -9). Kettunen et al[7] analyzed the roles of FGF-3, -7, and -10 in developing mouse teeth. They found that FGF-10 stimulated cell proliferation in the dental epithelium and that FGF-3 may participate in signaling functions of the enamel knot. They concluded that the expression patterns and interactions of FGFs in dental epithelium and mesenchyme suggest their involvement in regulatory signaling pathways during tooth development.

Tompkins[8] stated that the FGF, BMP, hedgehog, and wingless protein families and their downstream transcription factors have been identified as key players in the epithelial-mesenchymal signaling loops that drive tooth development. He also added that recent results suggest that phenotypic proteins of both ameloblasts and odontoblasts, such as amelogenin and dentin matrix protein 2 (DMP-2), may act as the final instructive signals in cytodifferentiation. Amelogenin is a protein found in the developing tooth enamel, and it belongs to a family of extracellular matrix proteins that constitute 90% of enamel proteins.

DMP is an extracellular matrix protein that is critical for proper mineralization of bone and dentin. DMP is primarily present as a nuclear protein in an undifferentiated osteoblast that regulates the expression of osteoblast-specific genes; during osteoblast maturation, it becomes phosphorylated and is exported to the extracellular

matrix, where it coordinates mineralized matrix formation. Mutations in the *DMP* gene are known to cause autosomal-recessive hypophosphatemia, a disease that manifests as rickets and osteomalacia.

Advancements in molecular biology approaches, coupled with mapping of the complete human genome sequence, have shown a number of putative disease genes and loci to be associated with the hypodontia and oligodontia phenotypes. Missing maxillary lateral incisors along with the lack of *BMP4* expression were reported by Neubuser et a[19] to be the manifestation of odontogenic potential shifts from the epithelium to the dental mesenchyme.

Family studies by Burzynski and Escobar[10] have established that incisor and premolar hypodontia is inherited via an autosomal-dominant gene that demonstrates incomplete penetrance. Expression of the human *MSX1* and *MSX2* genes in relation to hypodontia, affecting one or a few teeth, has recently been investigated by many researchers, including Nieminen et al.[11]

Formation of tooth-supporting tissues

While roots are forming, the supporting tissues of the teeth, such as cementum, PDL, and alveolar bone, are also developing from the dental follicle in the late stages of tooth formation.

The dental follicle (sac) is a condensation of the ectomesenchymal cells that initially surrounds the enamel organ and the enclosed dental papilla. ElNesr and Avery[2] stated that the dental follicle has an important role in the initiation, formation, and maintenance of the tissues of the tooth root, PDL, cementum, and alveolar bone.

Cementum

Cementum formation, or *cementogenesis*, occurs late in tooth formation, just before the degeneration of the epithelial root sheath and root dentin deposition. It manifests as a thin, amorphous, structureless, and highly mineralized secretion on the surface of the root dentin. ElNesr and Avery[2] noted that this secretion is more evident in the apical region of the root and averages some 10 to 20 μm in thickness.

After root formation and proliferation of the epithelial root sheath, some fragmentation occurs. Ectomesenchymal cells of the dental follicle penetrate between the epithelial fenestrations and become closed to the newly formed dentin

of the root. The cells differentiate into cementum-forming cells, or *cementoblasts*. Cementoblasts secrete an organic matrix, consisting of fine collagen fibrils, ground substance, osteocalcin, and sialoprotein, that is mineralized later by minerals in the oral fluid (Fig 2-17).

Fig 2-17 Formation of tooth-supporting tissue.

Following mineralization, the cementoblasts move away from the cementum and collagen deposition continues to lengthen and thicken the bundle fibers. Finally, the surface fibers join the forming PDLs and become anchored with them.

During cementogenesis, two types of cementum form: *(1)* acellular cementum and *(2)* cellular cementum. Acellular cementum forms first, in the coronal two-thirds of the root. When the cementoblasts are trapped in lacunae in their own matrixlike bone cells, the cementum is called *cellular* or *secondary cementum*. This cementum is present only in the apical third of the root. At this time, the cementoblasts lose their secretory activity and become cementocytes. Cellular cementum develops after most of the tooth formation is complete and after contact is established with a tooth in the opposing arch.

According to Ten Cate,[3] cellular cementum is not found in teeth with one root; in premolars and molars, it is found only in the part of the root closest to the apex and in interradicular areas between multiple roots.

Periodontal ligament

The supporting tissues of the tooth develop during root formation from the dental follicle, a fibrocellular layer investing the dental papilla and enamel organ. The cells and the fiber bundles of the PDL differentiate from the primitive dental sac or dental follicle (Fig 2-18).

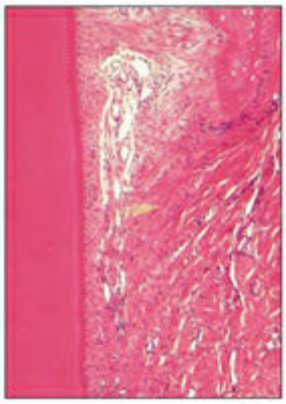


Fig 2-18 Principal collagen fiber bundles of the PDL.

When the root sheath fragments, ectomesenchymal cells of the dental follicle penetrate between the epithelial root sheath and the newly formed dentin of the root. These cells differentiate into cementum-forming cells (cementoblasts); according to Ten Cate,[3] some cells from Hertwig's epithelial root sheath may also transform into cementoblasts. These cells elaborate an organic matrix that becomes mineralized and in which collagen fiber bundles of the PDL become anchored.

The dental follicle also differentiates the cells and fiber bundles of the PDL. Recent evidence indicates that dental follicle cells also form the bone in which the ligament fiber bundles are embedded.[3]

Fibroblasts of the dental follicle also secrete collagen fibers, which interact with fibers on the surface of adjacent bone and cementum. This interaction between the alveolar socket and the tooth root is believed to help the process of tooth eruption.

The formation and arrangement of different PDL fibers are influenced by the way teeth occlude with the teeth in the opposing arch. The force and function of occlusion can lead to the formation of groups of fibers in different orientations, such as horizontal and oblique fibers. In other words, local environmental factors regulate the ability of precursor cells for cementoblasts and PDL fibroblasts within the dental follicle to function as cementoblasts that form root cementum or as fibroblasts of the PDL, respectively.

Some of these fibers incorporate as Sharpey's fibers into alveolar bone; others, called *cemental fibers*, embed in cementum. These two kinds of fibers are linked by a middle zone of fiber called the *intermediate plexus*.

The formation of the PDL varies between the primary and permanent teeth and also among various species.

Alveolar bone

During root and cementum formation, bone is also developing from the dental follicle in the adjacent area. During root formation, primary cementum covering new bone is also deposited, and this bone gradually reduces the space between the cementum and crypt wall and diminishes the PDL space.

According to Ten Cate,[3] evidence shows that the bone in which the ligament fiber bundles are embedded is also formed by osteoblastic cells that differentiate from the dental follicle. Similar to the primary cementum, collagen fibers are created on the surface nearest the tooth, and they remain there until they attach to PDLs and are mineralized. Like any other bone, alveolar bone is prone to remodeling throughout life, such as modification induced by osteoblastic and osteoclastic activity arising from the force applied during orthodontic treatment.

Dentogingival junction

The connection between the gingiva and tooth structure is called the *dentogingival junction*. Luke[12] explained that this junction and the PDL perform unique functions: The dentogingival junction makes a seal wherever a tooth punctures the integument, while the PDL cushions the tooth during mastication, sheltering the delicate cells that remodel the socket.

A special feature of both tissues is the rapid turnover of their major components. The dentogingival junction has three epithelial types: the gingival, sulcular, and junctional epithelia. These three types form a mass of epithelial cells, known as the *epithelial cuff*, between the tooth and the mouth.

Before eruption, tooth crowns are covered by a double layer of epithelial cells; the cells of the inner layer, which is in contact with the enamel, are the ameloblasts, which have completed their formative function and develop hemidesmosomes, secrete a basal lamina, and become firmly attached to the enamel surface. The outer layer forms from the remaining cells of the dental organ; these two layers of cells are called the *reduced dental epithelium*.

When tooth eruption begins, the connective tissue that supports both layers breaks down. The remnants of amelo-blasts provide hemidesmosomes, which are developed by the inner layer between the gingival epithelium. Ten Cate[3] indicated that much about gingival formation is not fully understood, but it is known that hemidesmosomes that form between the gingival epithelium and the tooth are responsible for the primary epithelial attachment.

The tissues of the dentogingival junction are dynamic rather than static. Even when they are pathologic, they can be reconstituted by repair, and they have a high

rate of turnover. The cells are capable of movement and of positional change. Its unique structural and functional adaptation enables the junctional epithelium to control the constant microbiologic challenges.

Role of form and function in occlusal development

The phenomenon of "structure related to function" plays a major role in dentofacial structures, including dental occlusion. The human face is anatomically and functionally one of the most complex regions of the human body. Several important functions reside within the face, including the senses of vision, hearing, smell, and taste. At the same time, the face provides proprioceptive input with respect to pressure, temperature, and stereognosis. The oronasal complex is also an important and complex region of the face, serving vital functions such as mastication, deglutition, speech, and breathing.

Dental occlusion is an important part of the oral cavity. To understand the complexity of the development of occlusion, which involves the interaction of orofacial growth and structure, tooth formation, and the influence of functional activity, a brief review of mandibular bone growth and development and their interrelationship are presented here. Examination of the initial stage of embryologic development and morphogenesis of the mandibular component can clearly explain the close relationship and interaction of this phenomenon.

Within the mandibular process of the first branchial arch, Meckel's cartilage forms; this later becomes atrophied and is replaced by intramembranous ossification of the mandible. Ossification starts at about the 6th week, laterally to Meckel's cartilage near the mental foramen between the inferior dental nerve and mental branch. Ossification spreads anteriorly and posteriorly to form the mandibular base and ramus. Condylar, coronoid, and symphyseal secondary cartilage is derived from mesenchyme. Tooth buds are located in a bone crypt and are surrounded by alveolar bone. Figure 2-19 shows a sagittal section of an embryo at this age, showing bone deposition and the relationship of bone, nerve, Meckel's cartilage, tooth germs, and the alveolar plate of the mandibular bone.

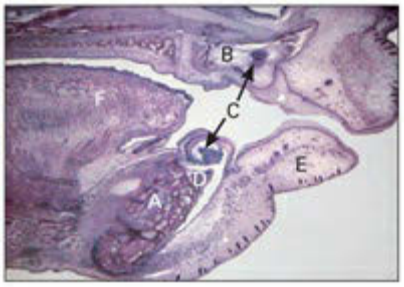


Fig 2-19 Jaw ontogenesis and tooth formation. A—mandible; B—maxilla; C—maxillary and mandibular tooth buds; D—alveoli; E— lower lip; F—tongue.

Maxillary bone is also derived from the first branchial arch. The maxilla, unlike the mandibular bone, has no precursor cartilage, and ossification appears from a single center on each side at the angle between the infraorbital nerve and its anterior superior dental branch.

Role of craniofacial growth in development of the dentition

During development of the dentition, craniofacial and jaw growth are also interacting and influencing the final result of occlusion, leading to normal interdigitation and normal intercuspation. Many difficult orthodontic problems or skeletal malocclusions are the result of maldevelopment, malrelationship, or malfunctional conditions of the skeletal structure. Simply, it can be said that wherever the jaw goes or grows, the teeth also must go. Thus, any disruption or disharmony between these structures can affect the rate, amount, or pattern of growth of each of these different parts. The end result, dental occlusion, can be profoundly affected. It can be concluded that, if these problems are recognized and treated early, it might be possible to minimize or even eliminate the consequences of undesirable craniofacial growth.

On the basis of development, growth, and function, mandibular bone has been divided into four parts: *(1)* the body or basal portion, *(2)* the alveolar portion, *(3)* the muscular portion, and *(4)* the condylar process. All of these parts show their own distinct growth characteristics (Fig 2-20).

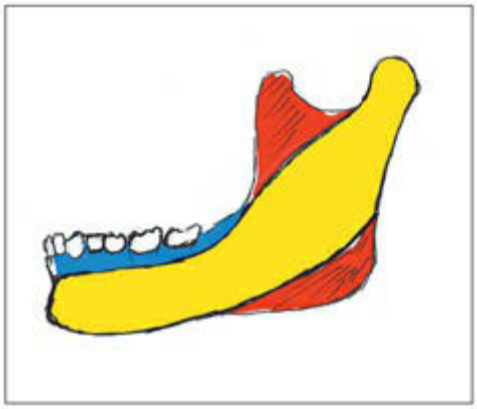


Fig 2-20 Different portions of the mandible: basal bone *(yellow)*, muscular part *(red)*, and alveolar part *(blue)*.

From this platform, the ideal dental occlusion, which is the accomplishment of perfect interdigitation between the maxillary and mandibular dental arches, is the result of a developmental process that consists of three main events: jaw growth, tooth formation, and tooth eruption. All of these processes are under the control of genetic, environmental, and functional factors that determine the rate of growth, the amount of growth, and the pattern of growth of the basal bone.

Mandibular bone is not only a major determinant in the masticatory function but also plays an important role in facial appearance and dental occlusion. Deviant growth of the mandible can result in various malocclusions, ranging from anterior crossbite to open bite to deep bite to excessive overjet. Asymmetric disturbances in mandibular growth lead to lateral malocclusion and asymmetry in skeletal morphology.

Growth of the alveolar process

The mandibular alveolar process functions as supporting tissue for the teeth, and there is a definite interplay between teeth and alveolar bone, as is clearly evident in the following situations:

- Congenital absence of the teeth accompanied by lack of alveolar bone development
- Extraction of teeth that results in resorption of the alveolar bone
- Tooth ankylosis that leads to a retardation of tooth eruption and slows the growth of the alveolar bone
- Orthodontic tooth movement and bone reaction against orthodontic forces

Vertical drift and mesial drift are important concepts and play a key role in maxillary and mandibular morphogenesis. Enlow[13] stated that the dentition drifts vertically and horizontally as the maxilla and mandible develop to keep pace with respect to anatomical position.

Enlow[13] also indicated that the periodontal connective tissue membrane (not ligament) provides the intramembranous bone remodeling that changes the location of the alveolar socket and moves the tooth itself. The developmental vertical movements of the teeth in normal growth are not the same as eruption; the process of drift moves the whole tooth and its socket as a unit.

Role of tooth migration

After clinical eruption of a tooth and achievement of occlusal contact, functional forces of occlusion acting horizontally on the teeth also affect the growth of the alveolar process. Studies by Björk and Skieller[14] and Björk[15] demonstrated that, depending on the growth pattern of the mandible, there is marked difference in the eruption direction of the teeth. For instance, clockwise rotation of the mandible changes the tooth eruption pathway to a more labial direction.

Muscle force and function

The human jaw muscles are essential to mastication, and they play an important part in craniofacial growth; their contraction is controlled by the nervous system.[16]

The coronoid process is an attachment site for the temporalis muscle. This process grows, or drifts, upward and backward by bone apposition at its posterior border and resorption along the anterior border. Many studies[13,17,18] have shown that the size and shape of the process are interrelated with the size of the temporalis muscle. They also have found underdevelopment or diminution of the coronoid process after removal of the temporalis muscle.

The angular portion of the mandible is also the site of attachment for two major masticatory muscles, namely, the masseter and medial pterygoid muscles. This angle grows mainly by periosteal apposition, while in the coronoid process secondary cartilage plays a role, both prenatally and during early postnatal life.

As Björk[15] illustrated, there is great variation between different individuals.

Growth of the mandibular condyle

The mandibular condyle is a major site of mandibular growth that contributes to both anteroposterior and vertical growth. The vital importance of mandibular growth is also clear in normal development of occlusion and growth of the face. The main growth direction of the condyle is upward, but Björk[15] has also shown that the direction and extent of condylar growth are variable.

Mandibular growth direction is one of the fundamental considerations of any type of orthodontic treatment, whether early-age or adult treatment. Three factors play an important role in the regulation of mandibular growth direction: *(1)* factors lying outside the temporomandibular joint, *(2)* factors inside the temporomandibular joint, and *(3)* intermediate factors. Some hypotheses in this regard have been proposed; for instance, the functional matrix concept proposed by Moss[18] suggests that the mandibular condyle is merely a passive and adaptive component.

Examples of factors lying outside the temporomandibular joint are the effect of head posture and mouth breathing on mandibular growth and the effect of muscles on the direction of mandibular growth. The effect of muscles is not as obvious as the other factors. However, as many investigators have indicated, there is some correlation between facial morphology and muscle strength or activity.[16,19,20] The effect of muscle fiber orientation on morphology of the bone has also been reported by Takada et al.[21] Congenital muscular dystrophy is an example of a pathologic condition of muscle force and function that influences the growth of the facial bone.

Solow and Kreiborg[22] and Houston[23] questioned the effect of soft tissue stretching on dentofacial morphology. They found a high correlation between craniofacial morphology and head posture. Extension of the head in relation to cervical column was found in connection with large anterior and small posterior facial height, small anteroposterior craniofacial dimension, clockwise mandibular rotation, facial retrognathism, and small nasopharyngeal space.

Houston[23] proposed an intermediate hypothesis, where factors both outside and inside the temporomandibular joint interact, resulting in a specific growth pattern.

However, there are examples of factors within the temporomandibular joint that influence the mandibular growth pattern; for example, juvenile rheumatoid arthritis and ankylosis of the mandibular condyle are conditions that cause posterior rotation of the mandible.[24–26] This indicates that in the presence of a shallow glenoid fossa, the mandible rotates downward (vertical pattern); in the presence of a deep fossa, an upward and forward growth pattern of the mandible is evident.

These mechanisms and their influence on the ontogeny and morphology of the mandibular bone and dental occlusion illustrate the complexity of the phenomena that

are acting in different areas of the dentoskeletal structure and, either directly or indirectly, affecting the development of occlusion.

Growth of the nasomaxillary complex

The nasomaxillary complex has multidirectional growth potential. There are several components of sutures in different planes of space with adjacent bony structues, especially near the cranial base. In addition, many neuromuscular activities and functions are channeled through the working environment of the oronasal cavity, referred to as the *oronasal functional matrix*.

Role of the tongue

Normal formation and development of the dental arches also require a normal balance between the perioral musculature and the tongue. The tongue itself can affect occlusion through its size, function, and position (see chapter 3).

Conclusion

Development of the occlusion is a long and complicated process requiring a normal interaction of several genetic and environmental factors affecting bone growth, muscle force, and the functional matrix environment. The early resolution of oral maldevelopmental or malfunctional problems is not only an essential part of early orthodontic treatment but also a vital component of treatment stability.

Neonatal Dentition

Gum pads

The maxillary gum pad is horseshoe shaped; the mandibular gum pad is U-shaped and somewhat flattened anteriorly.

Sheldon Friel[27] studied the form of gum pads at birth. He noted the presence of some divisions of the pad that formed sections corresponding to the underlying primary teeth. (The elevation related to the primary canine is well defined by grooves distal to the crypts of the primary canine in both arches. This is referred to as the *gingival groove*.) His observation indicates that the section related to the

primary second molar is not clearly defined at the time of birth and arises by about 5 months of age. Friel[27] also observed that the sagittal and transverse dimensions of the maxillary pad are larger than those of the mandibular pad, causing an overlap of the maxillary gum pad over the mandibular gum pad (Fig 2-21). The gum pads are not in contact when the jaws are at rest.

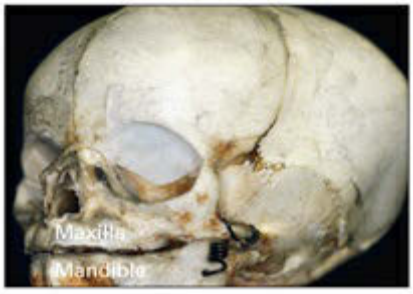


Fig 2-21 Newborn skull showing the jaws' basal bone as part of the gum pads.

In a careful study of a group of 709 infants ranging from 1 to 11 days, Sillman[28] indicated that the mandibular gum pad is distal to the maxillary gum pad in all cases, on average by 2.7 mm in the male and 2.5 mm in the female. He found that the mandibular gum pad progressively moves forward until the primary first molars erupt. The space between the anterior segments of the gum pads varies in form.

In another serial study of occlusion from birth to 3 years, Sillman[28] examined a group of 50 infants and found a limited anteroposterior movement of the mandible but no lateral movement. Material was drawn from casts of more than 50 children, followed continuously from birth to 11 years. As the child ages, the dimensions of the mandibular gum pad also change, and its distal position is reduced. The length and anterior width of the mandibular gum pad increase while the posterior width decreases.

The mandible "grows" forward at a greater rate than the maxilla to catch up with the maxilla in varying magnitude over time. Sillman[28] also found that the interrelationship of the gum pads at birth has no bearing on the future interrelationship of the jaws. There is no relationship between the anterior openings of the gum pads and future open bite tendency.

Leighton,[29] in a longitudinal study, examined the records of 109 children and 30 pairs of twins to determine the form of the maxillary gum pad at the age of 6 months and before the emergence of teeth. Heredity was found to make less contribution to the form of the gum pad at 6 months than at birth, mainly because the contribution made by underlying teeth had increased during the first 6 months of life.

Leighton[29] also concluded that the shape of the alveolar process may be modified

by physical forces, such as sucking habits, before tooth eruption. During the first year, the gum pads enlarge, and the arches widen to accommodate all the teeth.

There is also some adjustment in maxillomandibular relationships. When the jaws are at rest, the gums pads are not brought together in function, because at this stage the mouth is designed for suckling. The space between the gum pads is occupied by the tongue, which, at this age, is advanced in development and larger than the surrounding jaws.

Natal teeth

Natal teeth are defined as those types of teeth present in the mouth at birth. They are uncommon and most frequently occur in the anterior part of the mandible as a pair of incisors. They are usually small and poorly developed with little or no root formation. Only 10% of these teeth are supernumerary.

Natal teeth sometimes cause problems for the child and mother such as difficulty in suckling and severe ulceration of the child's tongue or the mother's breast, and they may present the risk of aspiration. If the natal teeth are loose and problematic, extraction is the recommended treatment. However, if the teeth are firm and cause no problems for the child or mother, conservative management and observation are indicated.

Chawla[30] stated that the management of natal, neonatal, and early infancy teeth should aim at preservation of esthetics and maintenance of space for eruption of the permanent successors. He reported on 50 children with natal, neonatal, or early infancy teeth. In 10% of the cases, the teeth caused trauma, and 94% presented with mobility and a danger of inhalation; in 97% of cases, extraction was carried out. He also indicated that the adjacent primary teeth tended to move into the extraction space.

Development of tooth germs

Tooth buds develop from the anterior teeth to the posterior. This process is synchronized with the growth of the tuberosity and anterior resorption and remodeling of the ramus, providing room for new tooth germs.

Each tooth develops sequentially, first from the crown and tip of canines, then gradually toward the root to the apex. Initially there is a great deal of space between teeth germs, but because of rapid interstitial growth of the tooth germs, which

continues until mineralization, tooth germs become crowded.

At the same time, rapid growth of the basal part of the jaws occurs in three dimensions. This will accommodate space for all of the tooth germs and crowding will disappear. This growth increment occurs in the mandible by sagittal growth of the condyle and remodeling of the ramus and transversely by sutural growth of the symphysis, between the two parts of the mandible. In the maxilla, the sagittal and transverse growth of the jaw is provided by rapid growth of the tuberosity and midpalatal suture.

The mandibular sutures fuse before incisor eruption (around 6 years of age), and the maxillary sutures fuse after puberty. Successional tooth germs first develop in the same crypt as their primary predecessors. Permanent tooth germs move also with their predecessors.

The first tooth buds to form are the mandibular anterior teeth, between 6 and 7 weeks in utero. All mandibular and maxillary primary buds are formed at 8 weeks in utero.

Tooth eruption

Eruption is an axial or occlusal physiologic movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane.

During tooth formation and interaction with maxillomandibular growth, tooth buds also undergo a complex movement to maintain their position in the growing jaw to compensate for masticatory function and growth.

Physiologic tooth movement, stages of eruption, theories of tooth eruption, and mechanisms of tooth eruption are discussed in detail in chapter 10.

Postnatal Development of the Dentition

The development of the dentition during postnatal life has been divided into six stages:

1. Birth to complete development of the primary dentition
2. First intertransitional period
3. First transitional period

4. Second intertransitional period
5. Second transitional period
6. Adult dentition

Intertransitional periods are actually more like dormant interludes during which the external appearance of the dentition remains unchanged.

Primary Dentition

The primary dentition starts with eruption of the first primary tooth (mandibular primary central incisor) and is considered complete around the age of 3 years, when the roots of the second molars are completed. This stage of dentition lasts until the age of 6 years, when the permanent first molars begin to erupt.

From ages 3 to 5 years, the dental arch is relatively stable and changes very slightly. This is the first intertransitional period. Preservation of the dental arch circumferences of the primary dentition is very important during the transitional dentition. From ages 5 to 6 years, the arch length begins to decrease because of the eruption of permanent first molars, which push the primary molars forward to reduce interdental spacing.

Both dental arches are semicircular. Spaces are present between all teeth, particularly in the anterior segment. Spacing in the primary dentition is very common, and the prevalence of spaced dentition varies between different ethnic groups. According to Baume,[31–34] there are distinct spaces between the maxillary lateral incisors and canines, the mandibular canines, and the first molars called *primate spaces* (mesial to the maxillary primary canine and distal to the mandibular canine) (Fig 2-22).

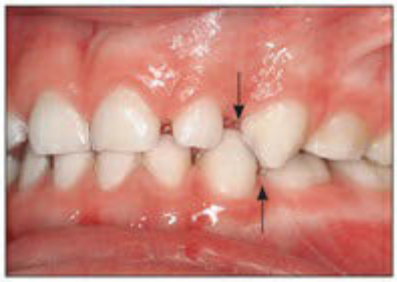


Fig 2-22 Maxillary and mandibular primate spaces *(arrows)* in the primary dentition.

The spaces found between incisors in the primary dentition are also called *secondary* or *developmental spaces*. The presence of these spaces is helpful for

aligning permanent teeth, and the absence of these spaces in the primary dentition indicates a lack of proportion between the jaw and tooth sizes.

In 1819, Delabarre was the first to describe interproximal spacing in the primary dentition of children between the ages of 4 and 6 years and suggested that this space is necessary for the permanent dentition.[31] Baume classified the primary dentition as follows:

- Class I: Primary dentition with spacing; spaces are present between all teeth, particularly in the anterior segment (see Fig 2-22)
- Class II: Primary dentition with no spacing

Baume[31–34] investigated physiologic changes in human dental arches in a clinical study of 60 children over a period of 8 years. The first part of this study examined developmental changes in the primary dentition. Annual impressions of 30 children aged between 3 and 4.5 years were made, and different measurements were taken. Regarding the developmental changes happening between 3 and 5.5 years of age, Baume[31–34] reported that:

- After completion of the dental arch, the sagittal and transverse dimensions are not altered.
- Two consistent morphologic arches are found: one continuously spaced, one continuously closed. No physiologic spacing occurs after eruption of the primary teeth, and an individual has either a spaced or a closed dentition.
- A primary dentition without spacing is followed by crowding in the permanent dentition 40% of the time. Primary spacing occurs in the maxilla in 70% of patients and in the mandible in 63%. No spaces exist in the maxillary dentition in 30% of patients and in the mandibular dentition in 37%.
- The maxillary intercanine distance is 1.7 mm greater in spaced dentitions than in closed dentitions. The mandibular intercanine distance is 1.5 mm greater in spaced dentitions.
- Spaced arches show two distinct spaces: mesial to maxillary primary canines and distal to mandibular primary canines. These spaces are interpreted as primate spaces.
- The terminal planes of the arches in occlusion remain constant.
- Alveolar vertical growth is observed concomitant with the development of successional tooth germs, and sagittal growth is observed concomitant with the development of accessional tooth germs.

Terminal plane

The relationship of the distal surfaces of the maxillary and mandibular primary second molars, termed the *terminal plane*, is an important part of the primary occlusion that must be checked by practitioners when examining a child after completion of the primary occlusion. The terminal plane is one of the most important factors that influences the future occlusion of the permanent dentition.

Baume[31–34] classified the terminal plane into three categories (Fig 2-23):

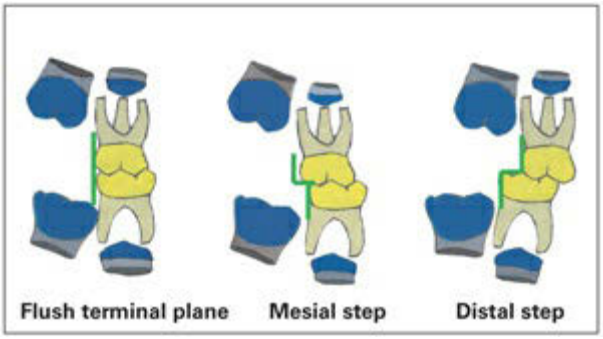


Fig 2-23 Baume's classification31–34 of the terminal plane.

1. Flush terminal plane (flush terminus): In centric occlusion, the anterior-posterior positions of the distal surfaces of opposing primary second molars are in the same vertical plane.
2. Mesial step terminus: In centric occlusion, the distal surface of the mandibular second primary molar is more mesial than the distal surface of the maxillary primary second molar terminus.
3. Distal step terminus: In centric occlusion, the mandibular primary second molar terminus is distal relative to the maxillary primary second molar terminus.

Frequency of different terminal planes

Physiologic interdental spacing is common in the primary dentition but varies among different ethnic groups. Studies have reported different values regarding the frequency of different types of terminal plane. Baume[31–34] reported that a flush terminal plane was present in 76%, a mesial step was present in 14%, and a distal step was present in 10% of the 60 children he studied. In contrast, statistical studies of the primary terminal plane status by Arya et al[35] showed a mesial step 49% of the time, a flush terminal plane 37% of the time, and a distal step 14% of the time. Bishara et al[36] reported a mesial step of . 1 mm in 42% of patients, a mesial step of

> 1 mm in 19% of patients, a flush terminal plane in 29% of patients, and a distal step in 10% of patients.

Importance of the primary dentition

Because of the presence of a secondary dentition (permanent dentition), the importance of primary dentition is sometimes neglected. However, careful maintenance of a healthy primary occlusion is necessary for the child because of the important roles it plays:

- Mastication is perhaps the most important role of a good, intact occlusion because of its impact on nutrition. Early loss of primary teeth can disturb mastication and consequently the child's nutrition, which is important during this period of rapid growth.
- Speech development is another important process happening at this age, and early loss of primary teeth can cause sustained speech defects, such as anterior lisping.
- Esthetics is another important consideration for children at this age. Loss of anterior teeth can have a profound psychologic effect on children.
- Maintenance of arch length for permanent teeth is another important function of primary teeth, and early loss of primary teeth can have a profound effect on developing occlusion.

Exfoliation of the primary teeth

Exfoliation of the primary dentition is a physiologic event that occurs during the transitional dentition through the resorption of primary tooth roots. The cells responsible for dental tissue resorption are odontoclasts, but knowledge regarding how the precursors of the odontoclasts appear, how they differentiate, and what gives them the signal to start resorbing the root at specific areas and times, as well as how they are activated in pathologic root resorption, is limited. It is still unclear whether odontoclasts, the cells that resorb the dental hard tissues, are different from osteoclasts, the cells that resorb bone.

The stellate reticulum and the dental follicle of the underlying permanent tooth root are responsible for the initiation of root resorption by the secretion of stimulatory molecules, cytokines, and transcription factors.

Harokopakis-Hajishengallis[37] stated that the primary root resorption process is

regulated in a manner similar to bone remodeling, involving the receptor-ligand system known as *receptor activator for nuclear factor* κ*B* (RANK) and *ligand for receptor activator for nuclear factor* κ*B* (RANKL). The RANK-RANKL system was discovered in the mid-1990s as a regulating factor of bone resorption, which has led to major advances in scientific understanding of how bone modeling and remodeling are regulated. She added that the consistency of symmetric exfoliation and eruption between the right and left sides of the arch suggests that shedding of primary teeth and eruption of the permanent teeth are coupled and may be programmed events.[37]

Sahara[38] investigated the cellular events at the onset of physiologic root resorption in rabbit primary teeth. He stated that the pressure of the erupting permanent tooth is believed to play a contributory role in the resorption of primary roots. Primary teeth without a permanent successor eventually exfoliate, but with some delay.

Fukushima et al[39] stated that shedding of human primary teeth is mediated by osteoclast-like cells (odontoclasts). They used immunocytochemistry and reverse transcriptase polymerase chain reaction to examine the expression of RANKL and osteoprotegerin (OPG), a decoy receptor that prevents RANKL from binding to RANK in human PDL cells during physiologic root resorption. The effect of RANKL on root resorbing activity of odontoclasts was evaluated by measuring the size of the dissolved area on calcium phosphate–coated cover slips. The results indicated that PDL cells during the root resorbing state express RANKL but decrease OPG expression. Expression of RANKL likely participates in odontoclastogenesis and activates physiologic root resorption.

Regarding the effect of the erupting permanent tooth and the resorption process of primary roots, some researchers emphasize the role of the dental follicle and stellate reticulum. It is believed that the pressure of the erupting permanent tooth causes differentiation and activation of the odontoclasts.

Marks and Cahill[40] conducted an experimental study in dogs evaluating the effect of the permanent tooth crown. They removed the developing tooth crown and inserted substitute teeth, such as silicone or metal replicates, that were placed in dental follicles. They found that the tooth substitutes erupted successfully, indicating that the dental follicle, rather than the tooth itself, regulates and coordinates the resorption events of the overlying bone and presumably of the primary predecessor tooth. They also showed that removing the dental follicles and leaving the tooth germs prevented their eruption.

In another experimental study, Cahill[41] prevented the eruption of the permanent tooth bud by using transmembrane wires to inhibit eruption of the permanent tooth while leaving the dental follicle intact. He observed normal resorption of the overlying bone and resorption of the primary roots.

Timing of exfoliation

Exfoliation of the primary dentition is a physiologic event that must occur at the proper time during the transitional dentition to ensure the normal sequence of eruption of the successor teeth and to establish normal occlusion.

Careful monitoring and control of these events by longitudinal panoramic radiograph monitoring, which the author always recommends, can prevent many abnormal exfoliation and eruption disturbances (see chapter 3).

Early exfoliation

Variations in the time of eruption or time of exfoliation of primary teeth are frequently observed in children, and it is commonly believed that a 6- to 10-month early exfoliation or delay in exfoliation can be considered normal. However, this pattern must be consistent with other aspects of the dental development.

Dean et al[42] indicated that exfoliation of primary teeth in the absence of trauma in children younger than 5 years of age merits special attention because it can be related to pathologic conditions of local and systemic origin or the result of local or systemic factors (see chapter 10).

Local factors. Untreated and severe caries and mouth injuries from sports or accidents may be a factor in early tooth loss. The most frequent causes of premature loss of teeth are accidents, especially in children. The teeth lost with the greatest frequency are the maxillary central incisors, especially in children having severe overjet due to severe incisor protrusion.

Other local factors that can cause early exfoliation of primary teeth are longstanding periapical abscesses (Fig 2-24) and periodontitis, which cause early shedding of primary teeth and early resorption of the bone covering the permanent successor.

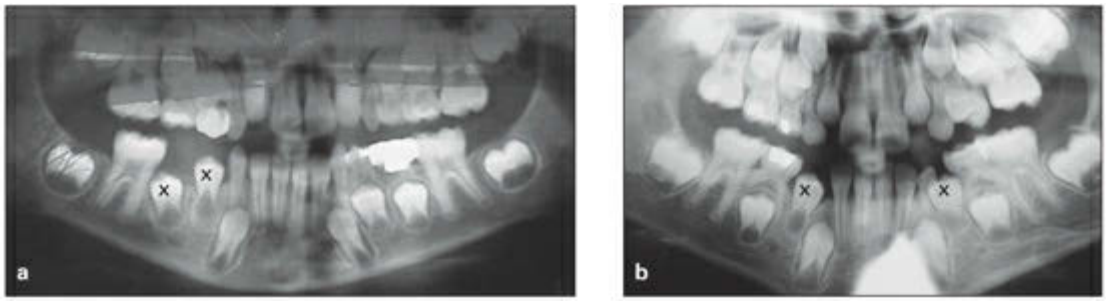


Fig 2-24 *(a)* Panoramic radiograph of a 7-year-old child whose primary right molars have been lost due to long abscess. *(b)* Loss of the molars caused early eruption of the premolars, which have short roots, are mobile, and require maintenance, such as a lower holding arch, splinting, or bonding.

Systemic factors. Examples of systemic factors that cause early exfoliation of primary teeth are agranulocytosis, hypophosphatasia, periodontitis, acrodynia, and radiation therapy (see chapter 10).

Delayed exfoliation

Delayed exfoliation of primary teeth also can be problematic for eruption of their successors and consequently causes problems for normal development of occlusion. Careful serial, longitudinal monitoring with panoramic radiographs can help in the early detection of and intervention in this problem. This anomaly also can result from local or systemic factors.

Local factors. Delayed exfoliation, or overretained primary teeth, can result from *(1)* congenital absence of the permanent follicle, *(2)* ankylosis, or *(3)* trauma. Congenital absence of the permanent follicle is a common cause of delayed exfoliation; in some conditions, the primary tooth can stay for a long period of time. Depending on the patient's occlusion and the situation of the primary tooth, it can be left in place or extracted and replaced by an implant or prosthetic. In some conditions, the best option can be extraction of the primary tooth and space closure by orthodontic treatment.

Ankylosis of primary or permanent teeth is an anomaly of fusion between cementum and alveolar bone that can occur during tooth eruption. Primary tooth ankylosis is frequently seen during the transitional dentition and can cause problems for permanent eruption and vertical alveolar growth. This, in turn, can result in problems for occlusion, such as deflection or impaction of the successor tooth, lateral open bite, anterior crossbite, deep curve of Spee, and overeruption of the opposing tooth (see chapter 10).

Trauma to primary or permanent incisors is common in children. Trauma to the primary incisors can result in tooth germ displacement, delayed eruption or absence of eruption, or hypoplastic enamel. The traumatized primary tooth can be lost or exfoliated early or, due to rupture of the PDL, can become ankylosed and retained, causing problems for successors such as maxillary incisor crossbite (Fig 2-25).

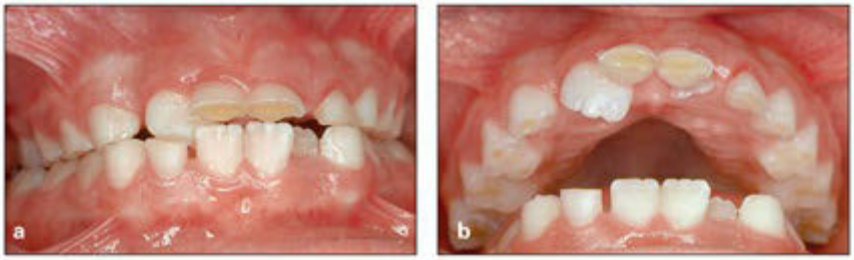


Fig 2-25 *(a and b)* Overretained primary incisors causing eruption problems for the permanent successors.

Systemic factors. Some factors that can have a general effect on the eruption and exfoliation of primary teeth include familial patterns, endocrine disturbances, and syndromic or congenital defects such as cleidocranial dysostosis.

Relationship of the permanent crown and primary root resorption

The vicinity and location of the permanent tooth germ are important factors in primary tooth exfoliation that must be watched during the transitional stage of occlusal development. Resorption of primary tooth roots starts at the site that is closest to the permanent crown; for example, the eruption pathway of permanent incisors is labial and incisal, and the crowns of permanent incisors are located in the lingual side of the apical third of primary roots.

Resorption starts at the lingual surface of the primary root and continues to the labial surface. When the labial surface is also resorbed, the permanent tooth is located at the root apex and resorption proceeds horizontally, until the primary tooth sheds and the permanent tooth erupts.

There are instances when the permanent incisors will not move enough labially over the apex of the primary root, and this causes incomplete and delayed root resorption of the primary root, lingual eruption of the permanent tooth, and delayed exfoliation of the primary tooth.

Occasionally the permanent tooth buds can be displaced due to trauma to the primary teeth. This results in overretained primary teeth and lingual eruption of the permanent incisors (Fig 2-26).

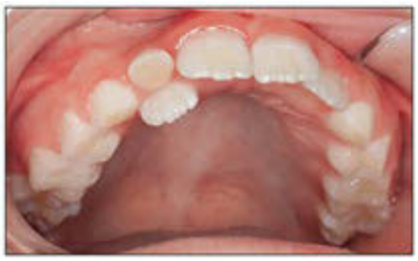


Fig 2-26 Abnormal path of eruption of the permanent lateral incisor and overretained primary lateral incisor.

Careful observation and monitoring of the dental transition can ensure early detection of these issues. Routine clinical testing of the mobility of overretained incisors and radiographic evaluation can aid early detection and determine the optimal time for extraction of the primary tooth for guidance of eruption (see chapter 4).

The developing premolar follicles are also located lingual to their predecessors and gradually move toward the divergent roots of primary molars. Thus, the resorption pattern of primary roots depends on the position and size of the permanent follicle. Uneven resorption can be observed between different roots of some primary molars.

Transitional Dentition

The transitional dentition is a long stage that starts with eruption of the permanent first molars and terminates with exfoliation of the last primary tooth. During this long interchange of primary and permanent teeth, many local or systemic factors can interfere with and influence the normal transition and consequently the normal development of occlusion. As explained earlier, this stage requires careful longitudinal radiograph monitoring.

The transitional dentition during the mixed dentition stage has been arbitrarily divided into three phases:

1. Phase I: Eruption of the permanent first molars
2. Phase II: Exfoliation of the primary incisors and eruption of the permanent incisors
3. Phase III: Exfoliation of the primary canines and molars and eruption of the permanent canines and premolars

Phase I (eruption of permanent first molars)

The mandibular permanent first molar, as an accessional tooth (with no primary predecessor), is the first permanent molar to erupt; this is followed by eruption of the maxillary permanent first molar in a normal transition.

The position of the distal surfaces of maxillary and mandibular primary second molars governs the eruption path of permanent molars; in other words, the permanent first molar follows the condition of the terminal plane. The position of the distal surface of the primary second molars is not functionally important at the stage of primary dentition, but it can greatly influence the position of permanent first molar relations and consequently the final permanent occlusion.

As was explained earlier in the chapter, the distal surfaces of maxillary and mandibular primary second molars form a plane, the so-called terminal plane, which can have one of three types of relationship:

1. Mesial step
2. Flush terminal plane
3. Distal step

Depending on the type of terminal plane and the rate and pattern of mandibular growth, four different relationships between permanent first molars may develop:

1. Class I relationship
2. End-to-end relationship
3. Class II relationship
4. Class III relationship

With a flush terminal plane, when the distal surfaces of primary second molars are in the same vertical level, the position of the permanent first molar varies according to jaw growth. When growth of the mandible and maxilla are normal, the permanent molars end up in a normal relationship. However, they may stay in an end-to-end relationship when growth of the mandible is insufficient or end up in a full Class II relationship when there is severe mandibular deficiency or maxillary overgrowth (Fig 2-27).

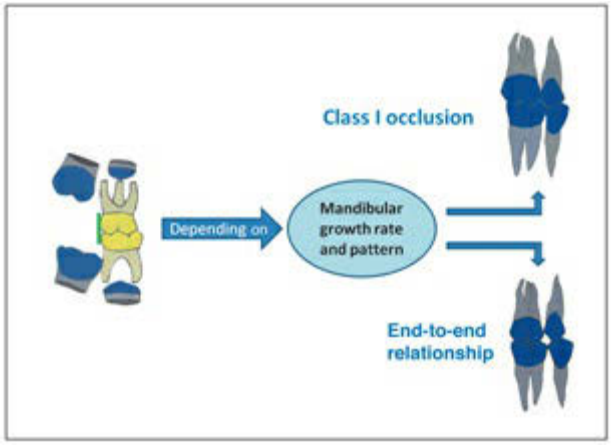


Fig 2-27 Flush terminal plane in the primary dentition. When the distal surfaces of primary second molars are flush, the final position of the permanent first molar varies depending on jaw growth. If growth of the mandible and the maxilla is normal, the permanent molars will end up in a normal relationship. If, for example, growth of the mandible is insufficient, the molars may develop an end-to-end relationship.

A mesial step terminal plane, depending on jaw growth, can result in a Class I molar relationship or a Class III malocclusion when there is excess mandibular growth (Fig 2-28).

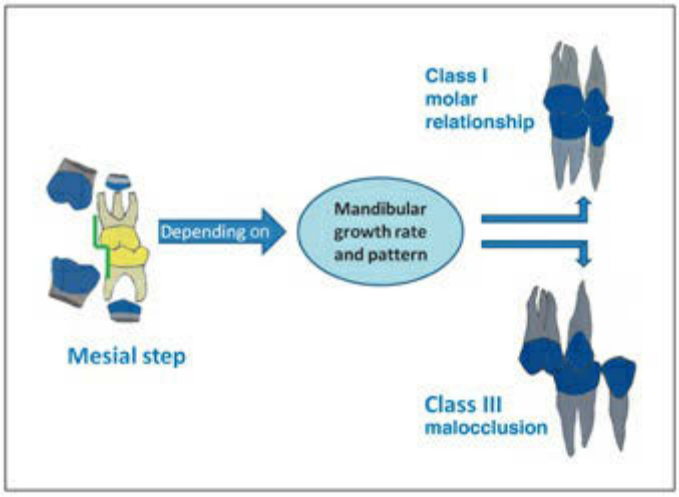


Fig 2-28 Mesial step terminal plane in the primary dentition. The permanent molars may develop a Class I molar relationship or, if mandibular growth is excessive, a Class III malocclusion.

With a distal step, depending on the growth pattern, the molars may stay in a Class II relationship or may develop an end-to-end molar relationship. However, the molars will never end up in a normal molar relationship, and thus this terminal plane deserves attention for interceptive treatment (Fig 2-29).

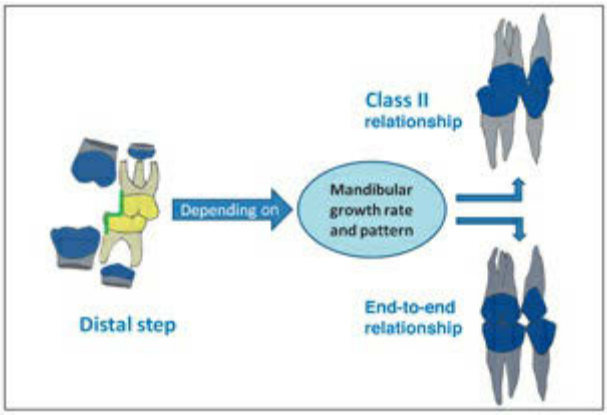


Fig 2-29 Distal step terminal plane in the primary dentition. Depending on the growth pattern, the permanent molars may stay in a Class II relationship or may develop an end-to-end relationship, but they will never develop a normal molar relationship without interceptive treatment.

Incisor liability

There is a difference between the mesiodistal width of permanent incisors and that of their primary incisors; this difference is referred to as the *incisor liability*. Maxillary permanent incisors are around 7.6 mm larger than their predecessors, and mandibular permanent incisors are around 6 mm larger than their predecessors.

Several mechanisms accommodate the permanent incisors and compensate for these differences:

- General dental spacing
- Intercanine width increase, which occurs during lateral incisor eruption
- Some distal movement of the mandibular primary canines to the primate spaces because of lateral pressure
- More labial inclination of the maxillary and mandibular permanent incisors relative to the primary incisors (Fig 2-30)

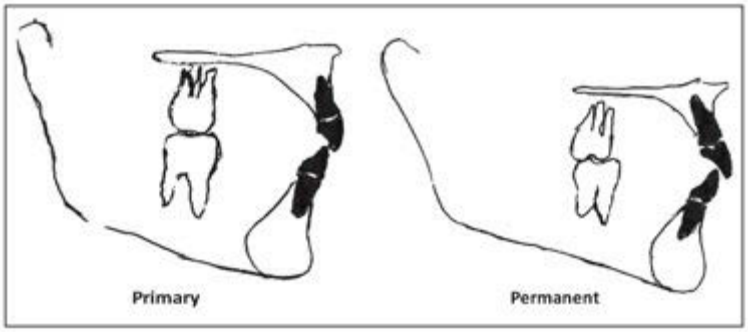


Fig 2-30 Change of incisor inclination. The permanent incisors are more proclined.

As mentioned before, interdental spacing of the primary incisors is one of the

factors favoring incisor liability. Baume31–34 showed that, around 40% of the time, absence of spacing in the primary dentition is followed by crowding in the permanent dentition. The average tooth accommodation derived from interdental spacing is about 3.8 mm in the maxillary arch and 2.7 mm in the mandibular arch. The intercanine arch width increases about 3.0 mm in each arch. Anterior positioning of maxillary permanent incisors provides increases of about 2.2 mm to the maxillary dentition and about 1.3 mm in the mandibular arch. These mechanisms together result in total increases in space availability of around 9.0 mm in the maxillary dentition and 7.0 mm in the mandibular dentition.

Phase II (eruption of permanent incisors)

This stage of the transition is relevant because the eruption of the maxillary and mandibular permanent incisors is a very complex and critical situation. This is due to the larger size of the permanent incisors relative to their predecessors and many other local factors that may interfere with incisor transition. Because different complications can occur during maxillary or mandibular central or lateral incisor eruption, each stage is explained separately.

Eruption of mandibular permanent central incisors

Sometimes mandibular permanent central incisors erupt lingual to the primary incisors. When the primary central incisors are examined, they are not loose. Periapical radiographs indicate delayed resorption of primary roots. Early extraction of the primary central incisors is recommended for this condition. Tongue pressure usually can improve the lingual position of the erupting permanent central incisors (Fig 2-31).

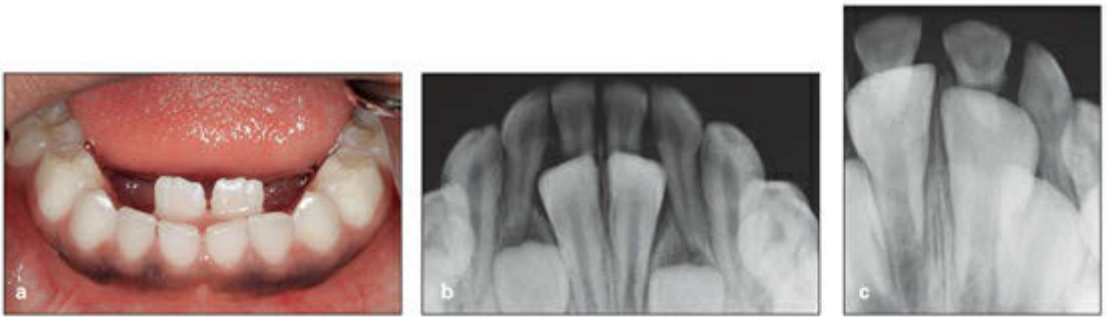


Fig 2-31 *(a to c)* Delayed resorption of the primary central incisor roots, causing lingual eruption of the mandibular permanent central incisors.

Eruption of mandibular permanent lateral incisors

Mandibular lateral incisors also can erupt lingual to resorbing roots of the primary lateral incisors, which are then pushed labially to exfoliate. The permanent lateral incisors also push primary canines to the primate spaces to gain some room for eruption.

When mandibular lateral incisors are disproportionately wide, they may stay in their lingual position or remain unerupted. Often lack of space for the mandibular lateral incisor may cause early exfoliation of mandibular primary canines. This type of early root resorption of the primary canine sometimes shows a crescent moon–shaped resorption on the mesial surface of the primary canine root (Fig 2-32). If this is the case, urgent space analysis and proper intervention are required. Some cases with early loss of the primary canine and severe incisor crowding might fall in the serial extraction category (see chapter 5).

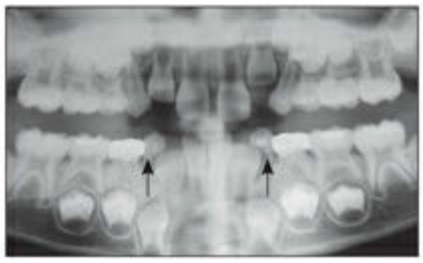


Fig 2-32 Mandibular lateral incisor eruption causing crescent-shaped resorption *(arrows)* of the primary canine roots.

When mandibular primary canines are lost prematurely, mandibular permanent incisors are in a less stable situation and may tip lingually and increase overjet as well as diminish available space for the permanent canines. Sometimes early loss of the primary canine can be unilateral, which causes immediate midline shifts toward the exfoliated canine.

In some instances of early loss of primary canines, especially if it is concomitant with a hypertonic lower lip or lip dysfunction, besides severe lingual tipping of incisors and increasing overjet, the mandibular incisors will continue overeruption until reaching and impinging on the palatal mucosa and produce severe overbite and a deep curve of Spee.

Early intervention in any of the aforementioned situations can prevent future problems. Accurate space analysis and immediate insertion of a lower holding arch is the best option. In some cases of unilateral exfoliation, in addition to use of a lower holding arch, extraction of the contralateral primary canine is recommended for prevention of midline shift.

Eruption of maxillary permanent central incisors

Maxillary incisors also develop lingual to primary incisors. Figure 2-33 shows the relationship of the developing permanent tooth buds to the primary teeth. The permanent incisors are positioned lingual to the roots of the primary incisors, while the canine is more labially placed.

Fig 2-33 Dissected skull of a child of approximately 5 years showing the relationship of the developing permanent tooth buds to the primary teeth.

The maxillary incisors erupt with a greater labial inclination and more flaring than the mandibular incisors. Most of the time, some spacing is present between the maxillary central incisors, while the mandibular incisors usually erupt with some degree of crowding.

The maxillary central incisor crowns are usually tipped slightly distally after eruption.

Normal eruption of the maxillary lateral incisors generally reduces the size of the diastema and the distal crown tipping of central incisors. After permanent canine eruption with normal inclination, the diastema between the central incisors should be completely closed (Fig 2-34).

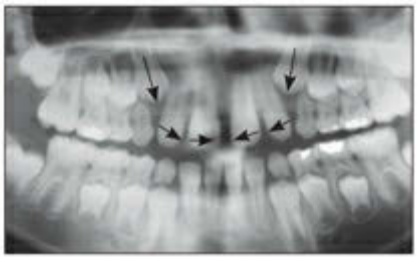


Fig 2-34 Normal pattern of canine and incisor movement *(arrows)* and space closure after canine eruption.

The total width of the maxillary permanent incisors is about 7.6 mm greater than that of their predecessors. Mechanisms to compensate for the incisor liability include interdental spacing, intercanine width increase, primate spaces, and the more labial inclination of the maxillary incisors.

Eruption of maxillary permanent lateral incisors

Maxillary permanent lateral incisors are also located behind the tip of the primary lateral incisors. In comparison to central incisors, maxillary lateral incisors have a more mesial inclination (see Fig 2-37). The mesial inclination of the lateral incisors moves the central incisors more mesially and reduces the size of the central diastema.

After eruption of the maxillary permanent lateral incisors, sometimes their crowns tip distobuccally because of pressure of the maxillary canine crowns on the lateral roots. Broadbent[43] named this normal transitional change the "ugly duckling" stage (Fig 2-35). Careful radiographic monitoring and palpation of the canine bulge is recommended during this stage; the more upright and lower position in the mouth of the permanent canines during eruption will reduce the force to lateral incisors, and the distobuccal position of the lateral incisors will correct spontaneously.

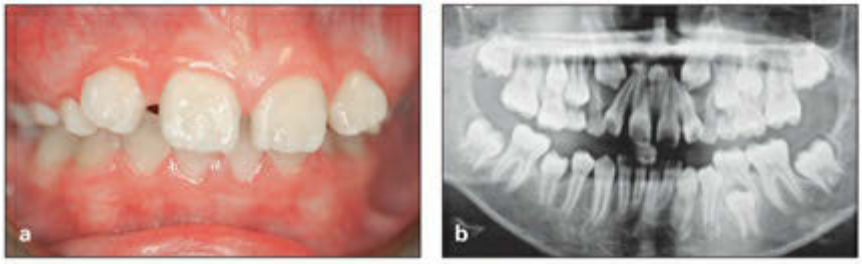


Fig 2-35 *(a and b)* Ugly duckling transitional stage.

Any orthodontic force applied to the lateral incisors during the ugly duckling stage may cause severe resorption of the lateral incisor roots.

During careful monitoring of this stage, if the inclination of the permanent canine does not change and the canine bulge is not palpable, there might be a possibility of canine impaction that requires early intervention (see chapter 10).

Another problem that may arise during the eruption of maxillary lateral incisors is the persistence of the central incisor diastema. This can cause space deficiency and crowding, rotation, or even crossbite of the lateral incisors. Persistence of the diastema can result from several factors, such as mesiodens, missing lateral incisors, abnormal frenum attachment, impinging bite, and abnormal habits. Careful monitoring, early elimination of the cause, and closure of the diastema will facilitate eruption of the lateral incisors and prevent the aforementioned problems.

Another problem after eruption of the lateral incisors is severe proclination accompanied by interdental spacing. Incisors with this condition are prone to fracture, especially in children of this age, and need early intervention for prevention

of teeth fracture (Fig 2-36).

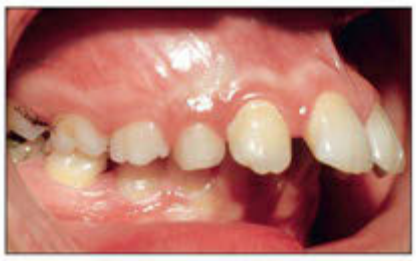


Fig 2-36 Severe maxillary incisor protrusion. These teeth are prone to fracture and require early intervention.

Secondary spacing. Secondary spacing is another mechanism that can counteract the maxillary incisor liability. According to Moorrees,[44] secondary spacing is a process that facilities maxillary lateral incisor eruption. This process occurs when mandibular lateral incisors emerge and push mandibular primary canines laterally and distally to the primate spaces, causing the maxillary primary canines to move laterally by force of occlusion. This process creates more space for the maxillary lateral incisors (Fig 2-37).

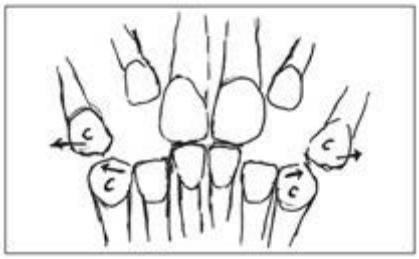


Fig 2-37 Process of secondary spacing, in which eruption of the mandibular lateral incisors forces the primary canines (C) to move laterally and distally *(arrows)*. The maxillary canines are moved laterally by the forces of occlusion, creating additional space for eruption of the maxillary lateral incisors.

Early extraction or early exfoliation of the mandibular primary canines during mandibular lateral incisor eruption will prevent this natural phenomenon from happening and will prevent secondary spacing. Disruption of this phenomenon may even convert a nonextraction case to an extraction case. This is why early extraction of mandibular primary canines is not recommended; many eruption problems for mandibular incisors in cases of mild crowding can be resolved by stripping the mesial side of mandibular primary canines with diamond-coated disks.

Common problems during incisor eruption

During the transition of permanent incisors, many external and internal factors can

interfere with normal eruption patterns and the position of permanent incisors. Careful clinical and paraclinical examination and monitoring of this process can detect problems early, and early intervention can prevent or lessen the sequelae.

A common problem in the early and middle mixed dentition stages during incisor eruption is asymmetric eruption, which requires careful evaluation. If the time of emergence between the left and right incisors is more than 6 months, it indicates an abnormal eruption process.

Many local or environmental factors can cause asymmetric eruption. Box 2-1 provides a summary of the disruptive events that can happen during the transition of permanent incisors. Diagnosis and management of these problems are discussed in part 2 of this book, "Early-Age Orthodontic Treatment of Nonskeletal Problems" (see chapters 4 to 10).

Box 2-1 Common problems during the incisor transition

- Abnormal diastema and spacing
- Abnormal path of eruption
- Crowding
- Mesiodens
- Congenitally missing lateral incisor
- Ectopic eruption
- Delayed eruption or absence of eruption
- Asymmetric eruption
- Early primary tooth loss
- Overretained primary tooth
- Overeruption (deep bite)
- Undereruption (open bite)
- Anterior crossbite
- Midline shift
- Severe protrusion
- Crowding

Phase III (eruption of permanent canines and premolars)

Eruption of the permanent canines and premolars occurs around 10 to 12 years of age. This stage is one of the most critical stages of the transitional dentition and needs careful observation during the exchange of canines and premolars.

The favorable development of occlusion in three dimensions (sagittal, transverse, and vertical) in this region is highly dependent on five factors: *(1)* the availability of

space, *(2)* a favorable sequence of eruption, *(3)* a normal permanent first molar relationship, *(4)* a favorable transverse relationship of the maxilla and mandible to the alveolar processes, and *(5)* normal exfoliation of the primary molars.

Availability of space

One of the important factors that provides normal eruption and normal interdigitation in this region is the presence of normal leeway space. The mesiodistal widths of the primary canines and the primary first and second molars are relatively larger than those of their successors (canines and first and second premolars). This difference is approximately 3.4 mm in the mandible and 1.8 mm in the maxilla.

Leeway space plays an important role in the availability of space for successor teeth and, through the late mesial shift, in the final occlusal relationships of the permanent first molars. Furthermore, the availability of sufficient leeway space can be a useful opportunity to apply interceptive treatment to alleviate mild incisor crowding and to guide eruption (see chapter 5). Therefore, careful monitoring and maintenance of the leeway space during eruption of the permanent canines and premolars are important steps in promoting the normal development of occlusion.

Favorable sequence of eruption

The sequence of eruption is an important process necessary for normal development of occlusion. This phenomenon plays an important role in phase III of the transitional dentition, during the transition of canines and premolars. In the mandible, the canine erupts before the premolars; in the maxilla, the canine is the last tooth to erupt (excluding the third molar).

The most favorable eruption sequence in the mandible is canine, first premolar, second premolar, and then second molar. Eruption of the mandibular canines before the premolars is important, because mandibular canines with strong roots and slight mesial inclination play an important role in maintaining the integrity of the incisors in the anterior region of the mandible. Because of the overlapping of the maxillary incisors over the mandibular incisors and lip force, mandibular incisors have a strong tendency to become retroclined, thus increasing the overjet, and overerupt, causing deep bite and in some cases impinging bite.

Eruption of the mandibular first premolars before the mandibular canine is another kind of abnormal sequence that can follow the early loss of the primary canine or the transposition or impaction of the permanent canine. This abnormal sequence can

cause problems such as retroclination of the incisors, deep bite, midline shift, labioversion, and canine impaction.

An abnormal sequence of canine eruption in the maxilla, such as eruption of the canine before the premolars, can cause space problems and impaction of the maxillary premolar.

Another abnormal sequence in phase III transitional dentition is the eruption of the permanent second molar before eruption of the maxillary or mandibular premolars. This situation can cause mesial shifting of the permanent first molar and loss of space for the premolars. Early detection and insertion of a lower lingual arch or maxillary Nance or transpalatal arch can prevent mesial movement of the first molars, premolar crowding, and impaction.

Normal permanent first molar relationship

Normal maxillary and mandibular permanent first molar position is another important factor that can influence the normal eruption patterns of phase III transition. Permanent first molars with an end-to-end relationship have a higher tendency to tip mesially and cause problems for premolar eruption. Maxillary and mandibular permanent first molars with normal position and good intercuspation and interdigitation have less tendency to tip and lose less mesial space following primary tooth loss.

Favorable transverse relationship of the maxilla and mandible to the alveolar processes

Another factor that can disturb the normal eruption of canines and premolars and development of occlusion is an anomaly in the transverse relationship of the basal bones and the alveolar processes. For example, the presence of posterior crossbite, especially buccal bite or Brodie syndrome, affects the buccolingual inclination and relationship of maxillary and mandibular premolars.

Normal exfoliation of the primary molars

Primary molar ankylosis, which is a common finding during phase III of the transitional dentition, can prevent or deflect premolar eruption. In a submerged condition, ankylotic molars prevent alveolar vertical growth and cause severe damage to the occlusion. Asymmetric eruption between the left and right sides of the posterior segments of the arch, like any other area of the dental arch, is a sign of

primary ankylosis.

Other problems during phase III

Other abnormalities that are common during phase III transition and require careful monitoring and early intervention include early loss of primary molars, the presence of supernumerary teeth, and the congenital absence of teeth. The presence of any of these problems during canine and premolar eruption indicates the necessity for longitudinal panoramic radiograph monitoring to ensure early detection and proper intervention.

Mesial shift

Mandibular permanent first molars shift mesially twice during the transitional dentition. The first shift takes place during the early eruption stage of the permanent first molars and the second during the late mixed dentition.

Early mesial shift. Early mesial shift is the first mesial movement of the permanent first molars, to occupy interdental spacing or the primate spaces, which occurs during eruption of the permanent first molars (around 5 to 6 years of age). In individuals with Baume class II primary dentition, which has no spacing, no mesial shift occurs.

Late mesial shift. Late mesial shift is the second mesial movement of the permanent first molars. This happens after exfoliation of the primary second molars (around 11 to 12 years of age). The molars shift to occupy leeway space and improve the permanent molar relationship. Depending on the amount of leeway space, mandibular permanent molars usually move more mesially than do maxillary molars to establish a normal molar relationship.

Problems during the transition of the permanent canines and premolars

Phase III of the transitional dentition is one of the most critical stages of transition, and several genetic and environmental factors (systemic or local) can influence the eruption pattern or position of canines and premolars and consequently the occlusion (Box 2-2). Diagnosis and management of all these problems are discussed in part 2 of this book, “Early-Age Orthodontic Treatment of Nonskeletal Problems” (chapters 4 through 10).

Box 2-2	Common problems during the canine and premolar transition
• Overretained primary teeth (ankylosis) • Abnormal sequences of eruption • Early loss of primary teeth • Habits, such as a lateral tongue thrust • Asymmetric eruption	• Delayed eruption or absence of eruption • Transposition • Impaction • Hypodontia • Hyperdontia

Dimensional arch changes during the transitional dentition

During transitional dentition, some changes happen to the arch length from the distance of labial incisors to mesial permanent first molars. The first change is a slight decrease in arch length that happens when the first molar erupts, forcing primary molars to use interdental spaces and primate space. The second change is a small increase when the maxillary and mandibular incisors erupt with more proclination. The third change is another decrease that occurs during exfoliation of the primary first and second molars, during the second mesial shift. Therefore, in general the arch is shorter at 18 years of age than at 4 years of age, especially in the mandible.

Crowding and third molars

Crowding of mandibular incisors is a common event that can occur in orthodontically treated or untreated patients; most of the time, crowding by third molars is blamed. Besides the aforementioned decrease in length, there are several reasons for late crowding of mandibular incisors:

- Arch perimeter decrease
- Late mandibular forward growth
- Mesial migration of teeth due to proximal surface wear and mesial force of occlusion
- Improper mechanical movement of the mandibular canine after retraction

An understanding of transitional changes that occur during the different stages of

dentition between the primary and permanent dentition is crucial for any clinician involved in early orthodontic treatment. Careful clinical and radiographic monitoring during the transitional dentition allows early detection of problems. In this way, it is possible to eliminate the cause and prevent, or at least reduce the severity of, the developing abnormalities. Box 2-3 lists some general problems that may arise during development of the dentition.

Box 2-3	General problems during development of the dentition
• Presence of preprimary teeth (natal and neonatal teeth) • Troublesome primary tooth emergence • Dental caries • Discoloration, decalcification, and structural hypoplasia • Hereditary defects of teeth (amelogenesis imperfecta and dentinogenesis imperfecta) • Early loss of primary teeth • Injury and loss of primary or permanent teeth • General interdental spacing • Crowding • Abnormal oral habits • Anomalies of tooth number (hypodontia and supernumerary teeth)	• Anomalies of tooth formation (fusion and malformed teeth) • Anomalies of tooth eruption (delayed or early eruption, abnormal sequence, ectopic eruption, impaction, transposition, and ankylosis) • Soft tissue problems (frenum, tonsils, adenoids, tongue, and lips) • Serious genetic and congenital anomalies (clefts, ectodermal dysplasia, Down syndrome, and Pierre Robin syndrome) • Dental and skeletal malrelationships (sagittal, vertical, and transverse malocclusions), such as Class I, Class II, and Class III occlusions alone or in combination with other abnormalities such as crowding, spacing, deep bite, open bite, and crossbite

Acknowledgment

The author would like to thank Dr Estepan Alexanian for providing the histologic slides in this chapter.

Summary

- The foundation of craniofacial structures, including the dentition, forms during the embryonic period (the third to eighth weeks of intrauterine life). This period is the most critical stage of development.
- Most developmental processes involve a precise coordination and interaction of genetic and environmental factors.
- Most of the facial structure is ultimately derived from migration of neural crest cells during the embryonic period. Any interference with this

migration can cause different craniofacial anomalies.

- During this long process, craniofacial bone growth, neuromuscular functions, and soft tissue structures surrounding the dentition are also interacting; these processes govern the final tooth relationships and lead to occlusal interdigitation.
- This long formative period makes it possible for many environmental and genetic influences to affect the morphology of the dentition and face. These influences include primary tooth loss, early or delayed exfoliation, deleterious oral habits, craniofacial dysfunctions, and cranial posture.
- Development of the dentition is a basis for the clinical application of early-age orthodontic treatment. An understanding of the complex processes of occlusal development not only is helpful for understanding the structural components but also can guide recognition of developing problems at initial stages and intervention at the proper time.
- Review of the prenatal stages of development, especially those occurring during the embryonic period, clarifies the normal relationships of adult body structures and the causes of congenital anomalies.
- An understanding of the complex processes of odontogenesis elucidates the genesis of different anomalies. Disturbances during the initiation stage cause hypodontia and hyperdontia; disturbances during histodifferentiation cause abnormal structure of the dentin or enamel (amelogenesis imperfecta or dentinogenesis imperfecta); aberrations during morphodifferentiation lead to abnormal forms and sizes such as peg-shaped lateral incisors, microdontia, and macrodontia; and systemic or local disturbances during enamel formation cause enamel hypoplasia.
- Development of the dental occlusion during postnatal life is a long, complex process beginning around 6 months of age with the eruption of the mandibular primary incisors and ending at 18 to 20 years, after eruption of all third molars.
- For better understanding, this long process can be divided arbitrarily into six stages of dentition: *(1)* primary dentition, *(2)* first intertransitional period, *(3)* first transitional period, *(4)* second intertransitional dentition, *(5)* second transitional dentition, and *(6)* permanent dentition.
- During all transitional stages of dentition, delicate biologic events occur. Only profound clinical and paraclinical observations can detect problems if they arise.
- Several structural components and normal interaction between them is necessary for normal occlusal development. These important structural

components, such as normal relationship of primary second molars, interdental spacing (primate space), leeway space, normal exfoliation of primary teeth, normal permanent root development, normal sequence of eruption, normal tooth number, and normal muscle balance and function can be evaluated by careful clinical and radiographic assessment.

- All dental practitioners, especially general practitioners, pediatric dentists, and orthodontists, are responsible for understanding and monitoring these developmental changes, detecting problems at an early stage, and intervening or referring patients to a qualified specialist.

References

1. Massler M, Schour I. Growth of the child and the calcification pattern of the teeth. Am J Orthod Oral Surg 1946;32:495–517.
2. ElNesr NM, Avery JK. Development of the teeth: Root and supporting structures. In: Avery JK (ed). Oral Development and Histology, ed 3. New York: Thieme, 2002:108–122.
3. Nanci A. Ten Cate's Oral Histology: Development, Structure, and Function, ed 8. St Louis: Mosby, 2012.
4. Berkovitz BK, Holland GR, Moxham BJ. Oral Anatomy, Histology and Embryology, ed 4. St Louis: Mosby, 2009.
5. Vaahtokari A, Aberg T, Jervvall J, Keränen S, Thesleff I. The enamel knot as a signaling center in the developing mouse tooth. Mech Dev 1996;54:39–43.
6. Coin R, Haikel Y, Ruch JV. Effects of apatite, transforming growth factor b-1, bone morphogenetic protein-2 and interleukin-7 on ameloblasts differentiation in vitro. Eur J Oral Sci 1999;107:487–495.
7. Kettunen P, Laurikkala J, Itäranta P, Vainio S, Itoh N, Thesleff I. Associations of FGF-3 and FGF-10 with signaling networks regulating tooth morphogenesis. Dev Dyn 2000;219:322–332.
8. Tompkins K. Molecular mechanisms of cytodifferentiation in mammalian tooth development. Connect Tissue Res 2006;47:111–118.
9. Neubuser A, Peters H, Balling R, Martin GR. Antagonistic interactions between FGF and BMP signaling pathways: A mechanism for positioning the sites of tooth formation. Cell 1997;90:247–255.
10. Burzynski N, Escobar V. Classification genetics of numeric anomalies of the dentition. Birth Defects 1983;13:95–106.
11. Nieminen P, Arte S, Pirinen S, Peltonen L, Thesleff I. Gene defect in hypodontia: Exclusion of MSX-1 and MSX-2 as candidate genes. Hum Genet 1995;96:305–308.
12. Luke DA. The structure and functions of the dentogingival junction and periodontal ligament. Br Dent J 1992;172:187–190.
13. Enlow D. Handbook of Facial Growth, ed 2. Philadelphia: Saunders, 1982.
14. Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. Am J Orthod 1972;62:339–383.
15. Björk A. Variations in the growth pattern of the human mandible: Longitudinal radiographic study by the implant method. J Dent Res 1963;42(1)pt 2:400–411.
16. Sassouni V. A classification of skeletal facial types. Am J Orthod 1969;55:109–123.
17. Horwitz SL, Shapiro HH. Modification of mandibular architecture following removal of temporalis muscle in rat. J Dent Res 1951;30:276–280.
18. Moss ML. The functional matrix hypothesis revisited. 1. The role of mechanotransduction. Am J Orthod

Dentofacial Orthop 1997;112:8–11.
19. Ingervall B, Thilander B. Relation between facial morphology and activity of the masticatory muscles. J Oral Rehabil 1974;1:131–147.
20. Ingervall B, Helkimo E. Masticatory muscle force and facial morphology in man. Arch Oral Biol 1978;23:203–206.
21. Takada K, Lowe AA, Freund VK. Canonical correlations between masticatory muscle orientation and dentoskeletal morphology in children. Am J Orthod 1984;86:331–341.
22. Solow B, Kreiborg S. Soft tissue stretching: A possible control factor in craniofacial morphogenesis. Scand J Dent Res 1977;85:505–507.
23. Houston WJ. Mandibular growth rotations—Their mechanism and importance. Eur J Orthod 1988;10:369–373.
24. Kantomaa T. The shape of the glenoid fossa affects the growth of the mandible. Eur J Orthod 1988;10:249–254.
25. Rönning O, Väliaho ML, Laaksonen AL. The involvement of the temporomandibular joint in juvenile rheumatoid arthritis. Scand J Rheumatol 1974;3:89–96.
26. Rönning O, Väliaho ML. Involvement of the facial skeleton in juvenile rheumatoid arthritis. Ann Radiol (Paris) 1975;18:347–353.
27. Friel S. The development of ideal occlusion of the gum pads and the teeth. Am J Orthod 1954;40:196–227.
28. Sillman JH. Relationship of maxillary and mandibular gum pads in the newborn infant. Am J Orthod Oral Surg 1938;24:409–424.
29. Leighton BC. A preliminary study of the morphology of the upper gum pad at the age of 6 months. Swed Dent J Suppl 1982;15:115–122.
30. Chawla HS. Management of natal/neonatal/early infancy teeth. J Indian Soc Pedod Prev Dent 1993;11:33–36.
31. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 1. The biogenetic course of the deciduous dentition. J Dent Res 1950;29:123–132.
32. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 2. The biogenesis of accessional dentition. J Dent Res 1950;29:331–337.
33. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 3. The biogenesis of the successional dentition. J Dent Res 1950;29:338–348.
34. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 4. The biogenesis of overbite. J Dent Res 1950;29:440–447.
35. Arya BS, Savara BS, Thomas DR. Prediction of first molar occlusion. Am J Orthod 1973;63:610–621.
36. Bishara SE, Hoppens BJ, Jakobsen JR, Kohout FJ. Changes in the molar relationship between the deciduous and permanent dentitions: A longitudinal study. Am J Orthod Dentofacial Orthop 1988;93:19–28.
37. Harokopakis-Hajishengallis E. Physiologic root resorption in primary teeth: Molecular and histological events. J Oral Sci 2007;49:1–12.
38. Sahara N. Cellular events at the onset of physiological root resorption in rabbit deciduous teeth. Anat Rec 2001;264:387–396.
39. Fukushima H, Kajiya H, Takada K, Okamoto F, Okabe K. Expression and role of RANKL in periodontal ligament cells during physiological root-resorption in human deciduous teeth. Eur J Oral Sci 2003;111:346–352.
40. Marks SC Jr, Cahill DR. Experimental study in the dog of the non-active role of the tooth in the eruptive process. Arch Oral Biol 1984;29:311–322.
41. Cahill DR. Eruption pathway formation in the presence of experimental tooth impaction in puppies. Anat Rec 1969;164:67–77.
42. Dean JA, Avery DR, McDonald RE. Dentistry for the Child and Adolescent, ed 9. St Louis: Mosby, 2010.
43. Broadbent BH. The face of the normal child. Angle Orthod 1937;7:183–208.
44. Moorrees CFA. The Dentition of the Growing Child: A Longitudinal Study of Dental Development between 3 and 18 Years of Age. Cambridge, MA: Harvard University Press, 1959.

Examination, Early Detection, and Treatment Planning

In the past, orthodontics was focused mainly on juvenile and adult treatment. Some types of early intervention procedures, such as orthopedic treatment, were not as well known and accessible as they are today. The major focus was limited to aligning irregularities after complete development of malocclusions.

Regardless of whether therapy involves the early-age or the traditional orthodontic approach, sometimes more emphasis is placed on the mechanisms of treatment than on the important element of diagnosis. Every day, new orthodontic products come to the market, and orthodontic residents and new orthodontists are influenced by the manufacturer's advertising.With so much emphasis on knowledge of bands, brackets, archwire configurations, and "systems," practitioners sometimes get the mistaken impression that orthodontic care is primarily a mechanical procedure.

Early orthodontic treatment is now more generally accepted as a means of gaining the greatest possible control over form and function and changes that happen with time. The main objectives of early orthodontic treatment are to detect problems at early stages of development, to eliminate or control the disturbing effects of these factors, and to intervene at the proper time. To achieve these objectives as well as efficient treatment and excellent results, a thorough diagnosis and sophisticated treatment plan are required.

Treatment planning is strategy, the treatment itself is the tactic, and all of these processes must be organized on a sequential basis. In other words, the clinician must follow this systematic procedure:

1. Examine the patient

2. Diagnose the problem
3. Classify the problems
4. Plan the treatment
5. Treat the patient

Diagnosis is the most critical part of orthodontic treatment. The goal of the diagnostic process is to prepare a comprehensive list of the patient's problems in order to synthesize the various treatment options into a rational treatment plan for the best outcome.

A comprehensive orthodontic diagnosis does not merely focus on the relationships of the maxillary and mandibular dentition. It requires a thorough evaluation of the patient's general health and occlusal situation and a consideration of the relationship of the dentition to the basal bone, other skeletal components, and the neuromuscular and soft tissue environments.

All treatment planning must be based on the elimination or control of etiologic factors and use of the patient's growth potential to guide occlusion toward normality, especially in early orthodontic treatment.

Diagnostic procedures must follow a series of organized and sequential steps (Fig 3-1). The first step toward a thorough and accurate diagnosis in orthodontic treatment is the creation of an accurate and adequate diagnostic database. This is necessary for assessment and recognition of dentoskeletal anomalies. The second step is the design of a treatment plan, based on accurate findings, that both addresses the present situation and allows for future changes in dentition and jaw growth that will take place during the early treatment.

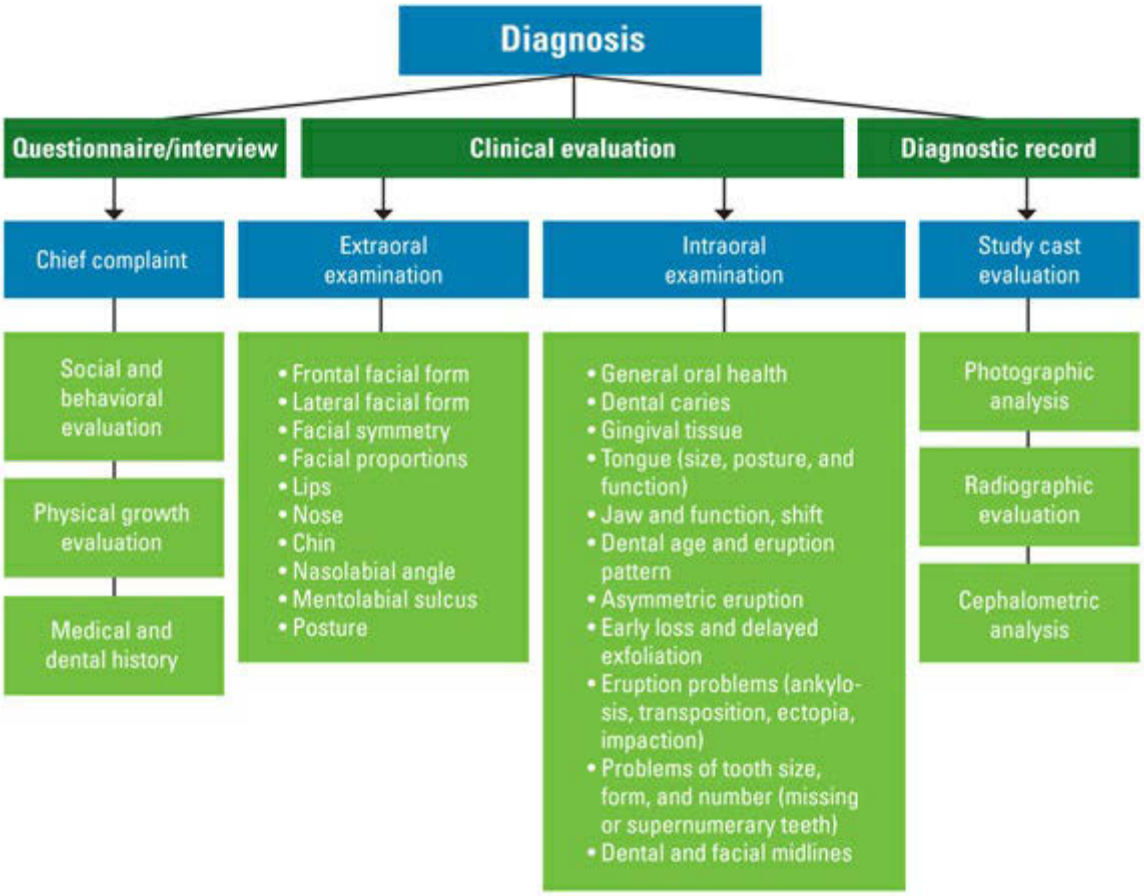


Fig 3-1 Elements of a complete diagnostic database.

Orthodontic problems almost always result from improper developmental processes that take place during development of the occlusal system; they are not the result of a pathologic process. It is often difficult to be certain of etiologic factors, but it is important to establish and rule out the causes of malocclusion. The etiology of malocclusion and the relative contributions of genes and environment have been a matter of controversy over the past century (see chapter 1). Studies in more recent decades indicate that during embryonic life, genetic mechanisms have more influence on the morphogenesis of craniofacial structure. The development of occlusion, in contrast, seems to be influenced strongly by the environment, especially during the early postnatal period.[1]

For orthodontic purposes, all diagnostic databases, whether clinical or paraclinical, are derived from three major sources: *(1)* the questionnaire and interview, *(2)* the clinical evaluation, and *(3)* the paraclinical evaluation (diagnostic record).

Questionnaire and Interview

The interview is an important appointment for the patient and the provider. The main purpose is to evaluate the patient's and parents' desires and their social and behavioral statuses. Many orthodontic practices use questionnaires, which can be completed by nurses or orthodontists.

This type of form can be filled out by patients or parents in advance to determine the patient's wishes, expectations, and goals for orthodontic treatment.

The questionnaire can be broken down into four sections: *(1)* patient's chief complaint, *(2)* social and behavioral evaluation, *(3)* physical growth evaluation, and *(4)* medical and dental history.

Patient's chief complaint

The first step of the interview is to find out the patient's chief compliant and expectations from treatment. The best way to ascertain this information is for the orthodontist to directly question the parent or patient, especially if facial esthetics is a concern. Some example questions include:

- What brought you here?
- What changes are you expecting to achieve?
- What is your chief complaint?

The provider may or may not agree with the patient's wishes; this judgment must be explained after the record is completed, when the clinician explains the problem and offers the best treatment options. The main purpose at this stage is to determine the patient's exact goal, whether it is entirely esthetic or a health concern. Confirming this information makes the practitioner aware of the patient's or parents' knowledge and understanding of the situation. In addition, it makes it easier for the provider to explain the problem, the treatment options, treatment limitations, and the dependability of the results. The results of some early-age treatments are dependent on the patient's cooperation and the parents' care. Reasons for early intervention and the possibility of a second phase of treatment must be clearly explained.

Social and behavioral evaluation

The social and behavioral histories are another important part of the first interview. Although these factors are important for treatment success, some parents may be reluctant to speak of their child's emotional or behavioral problems. In these situations, the practitioner must cautiously explain that treatment success depends on patient compliance.

For this reason, questioning about school progress or the patient's attitude with friends or siblings could be helpful. Recognition of the patient's emotional or learning disabilities will help to modify the treatment approach according to the patient's ability. Treatment can be designed in such a way to reduce the responsibility and cooperation needed from the patient in the use of some orthodontic devices such as headgear or removable appliances.

Pretreatment evaluation of the social and behavioral statuses of the patient is also important for understanding the patient's expectations, particularly for adult patients. Sometimes, after minor interceptive treatment, the patient or parents may expect a great change in facial appearance before full eruption of the permanent dentition. It is the practitioner's responsibility to explain the limitations and the possible final results of treatment.

Physical growth evaluation

The general physical growth of the child is another important element to assess in the general evaluation for any type of early orthodontic treatment. The growth status of a child is an important part of treatment planning; this includes the amount of growth that can occur during treatment and the potential for growth that remains after treatment. The best growth modification strategy is achieved during the growth spurt of children.

The growth status of children can be explored by asking questions such as:

- How rapidly has the child grown recently?
- Has there been a change of clothing size?
- Are there signs of sexual maturation?

While these questions can be helpful in estimating the child's current growth status, other more accurate techniques are available for evaluation of the growth potential, including evaluation of hand-wrist radiographs, neck vertebrae, and cephalometric radiographs.

Medical and dental history

The main purpose of seeking the medical and dental history of the orthodontic patients is to establish the causes of malocclusion, if possible. Although it is sometimes difficult to be certain of etiologic processes, it is still important to conduct a thorough examination and prepare good records. This will clarify problems and allow the best treatment planning. The orthodontist must base the design of the treatment strategy on the nature of the problem and the findings obtained from recorded data.

The patient's medical history consists of two parts, the family's history and the patient's history.

Family's medical history

The most important points in the evaluation of family medical history are:

- Genetic background
- General health of the parents and siblings
- Facial and dental resemblances between the parents and the patient
- History of orthodontic treatment for the parents or siblings
- Dental health of the parents
- Condition of the mother during pregnancy (diseases, medications, accidents) as well as length of term and type of delivery

The family and genetic history can be evaluated by finding resemblances between the facial characteristics and dental occlusions of the parents and the patient. Previous orthodontic treatment of family members and the nature of these issues should also be considered.

The parents' general and dental health is a good indicator of the patient's susceptibility to periodontal disease and caries. The mother's general health during pregnancy can have a direct effect on the patient's health and dental occlusion. Some medications or diseases (eg, viral infections and endocrine disease) and accidents during pregnancy are examples of history items that should be assessed. Reports indicate that length of term and premature birth can also affect development of occlusion by disturbing normal jaw growth or dental structures.[2,3] The use of forceps in difficult deliveries can affect the temporomandibular joint (TMJ), and long-term

effects could detrimentally influence growth and development of the jaws.[4]

Patient's medical history

As discussed in chapter 2, the exact contribution of genetic and environmental factors associated with specific types of malocclusion is not always easy to verify. Therefore, the orthodontist or assistant must ask several important questions to aid assessment.

When the patient's health is evaluated prior to treatment, it is important to note whether the patient has previously taken or is currently taking any long-term medication and for what purpose. This information can help to determine if the patient has any systemic or metabolic disease that may contraindicate orthodontic treatment or postpone the time of treatment. For example, orthodontic treatment for children with diabetes, even under medical control, requires special and careful monitoring, because these individuals are susceptible to periodontal breakdown following application of orthodontic forces. In children with mitral valve prolapse or heart problems caused by rheumatic fever, premedication and antibiotic prophylaxis might be required prior to invasive procedures such as banding.

Another area to assess when gathering the patient's history is any previous occurrences of accidents or trauma to the dentition, jaws, and joints. Early fractures of the condylar neck should not be overlooked, because they can cause significant developmental defects at older ages. For example, childhood condylar jaw fracture is the most likely cause of asymmetric mandibular deficiency in children.[5,6]

Other destructive processes in the TMJ that can cause mandibular problems include rheumatoid arthritis and congenital absence of tissue, as is seen in patients with hemifacial microsomia.

Considering the phenomenon of "form related to function" and the possibility that one might affect the other, any imbalance in muscle function and any disturbed physiologic functions such as respiration, deglutition, mastication, and speech must be carefully evaluated. Assessment of a previous or present history of mouth breathing in growing children is important. This condition can have profound effects, including enlarged tonsils or adenoids, oronasal obstruction, and allergies.

Eliciting information regarding allergies is also important because many patients are allergic to clinical and orthodontic materials such as latex or have sensitivity to nickel, which can be a component of wires and brackets.

Any history of blood transfusions must be considered because of the risk of

immunodeficiency after exposure to viruses such as hepatitis and human immunodeficiency virus (HIV).

The important areas to ask about prior to orthodontic treatment can be summarized as follows:

- History of long-term medication use
- Last visit to a physician and dentist
- Hospitalizations: when and why
- Allergies, especially latex or nickel sensitivity
- History of blood transfusion (to assess the chance of hepatitis or HIV infection)
- Heart problems, such as mitral valve prolapse or problems associated with rheumatic fever (to assess need for antibiotic prophylaxis)
- Accidents or any traumatic injuries to the teeth or jaws (the effects of previous trauma to teeth and apical involvement without any clinical signs may be intensified by orthodontic force and tooth movement)

Clinical Examinations

The orthodontic clinical examination is a thorough evaluation of orofacial structure, both in stasis and in function. The health of oral and perioral hard and soft tissue structures must be carefully assessed before any orthodontic treatment. Pathologic problems such as dental caries, periodontal disease, inadequate attached gingiva, and apical involvement must be treated first. After the problem is under control, bonding and tooth movement can be started.

Clinical examinations involve a series of tests that can be performed by visual observation, digital inspection, and functional analysis of all of the extraoral and intraoral structures. It is important not to concentrate so closely on one aspect of the patient's overall condition that other significant problems are overlooked.

Extraoral Examination

The extraoral clinical examination includes evaluation of facial esthetics and morphology, including structures, proportion, typing, and symmetry. The facial form, in both the frontal and lateral views, is now a major determinant of orthodontic

diagnosis and treatment planning. Facial and dental appearances are of major concern to almost all patients seeking orthodontic treatment. Evaluation of esthetics is an important part of the clinical examination. Graber and Vanarsdal[17] pointed out that malocclusion is a disability with a potential to affect physical and mental health, not a disease, and that appropriate treatment can improve the well-being of the patient.

Assessment of dentoskeletal hard tissue relationships alone without a thorough knowledge of the condition and changes of soft tissue that take place during growth and orthodontic treatment is entirely inadequate. Facial esthetics is an extremely complex issue that must be carefully assessed in the extraoral examination.

Gugino and Dus[8] stated that the human face as a whole is anatomically and functionally one of the most complex regions of the human body. A sound understanding of embryology and physiology of the occlusal system and the stomatognathic system is required to understand and recognize the problem of malocclusion in order to provide the best orthodontic treatment for each patient's occlusion.

Many factors, including eyes, hair, skin, lips, teeth, nose, chin, and jaws, play an important role in the composition of an esthetic face. These components impact our perception of the face and must be carefully evaluated during diagnosis and treatment planning for orthodontic treatment.

Esthetic evaluation of the patient's frontal and lateral appearances and detection of any deformity before treatment, prediction of future growth changes in early orthodontic treatment, and design of a treatment plan that takes these elements into consideration are majors goal of the extraoral examination.

Esthetic evaluation of the patient's frontal and lateral appearances deserves special attention, especially in early-age orthodontic treatment. This evaluation is important at the time of examination, and all potential growth changes during and after treatment must be considered.

The extraoral examination should include assessment of the following important features (see Fig 3-1):

- Vertical and transverse frontal facial morphology (dolichocephalic, mesocephalic, or brachycephalic)
- Facial profile (straight, convex, or concave), to identify any adverse sagittal (anteroposterior) growth patterns and occlusal disharmonies
- Facial proportions, that is, the upper and lower facial dimensions, to identify any

adverse vertical growth patterns
- Upper lip–lower lip height ratio
- Facial symmetry, to identify any adverse transverse growth patterns including asymmetries of maxillary and mandibular bones
- Development, tonicity, and form of facial muscles and soft tissue
- Nose size and proportion and symmetry to the rest of the facial structures
- Chin size, shape, symmetry, and position related to other facial structures
- Lip size, tonicity, and position related to profile and incisors, at rest and in function
- Nasolabial angle
- Mentolabial sulcus
- Patient's posture

Frontal facial evaluation

At the beginning of a systematic facial examination, the child should be seated in an upright position with the practitioner at a distance looking straight at the child's face. Evaluation of the patient's frontal view allows the examiner to identify the patient's facial form, facial symmetry, and proportion between different parts of the face.

Other factors must be considered in treatment planning besides cephalometric measurements. Lines, angles, and numbers cannot be the only diagnostic tools used. The things an observer's eyes reveal about the patient's facial characteristics in three dimensions, both in stasis and in function, are also very important to consider. These elements include facial typing, facial symmetry, and facial proportion.

Facial typing

When the face is viewed from the front, the facial form can be identified as long, wide, or medium. According to En-low's classification,[9] these three types of facial pattern are termed *dolichocephalic* (long face), *brachycephalic* (broad face), and *mesocephalic* (medium face), respectively. According to Enlow,[4] the facial complex attaches to the cranial base, which dictates the dimension, angle, and topographic characteristics of the face.

The dolichocephalic head shape yields a narrow, long, and protrusive face and a mostly convex profile. Patients with Class II malocclusions with a tendency toward

a vertical pattern show this type of facial pattern. The brachycephalic head shape yields a broad, less protrusive, straight profile, a short face, and a tendency toward a horizontal growth pattern. Class II division 2 malocclusions are usually associated with this type of facial pattern. The mesocephalic head shape is an intermediate head form with intermediate facial features. Usually patients with normal or Class I occlusions show this type of configuration. Different head forms are more prominent in different geographic regions. The type of facial morphology, especially in growing patients, can have some impact on growth prediction, and these considerations may impact treatment tactics such as expansion.

Facial symmetry

Facial symmetry in the frontal and lateral views is another important aspect to evaluate because this will reveal asymmetric growth patterns of the maxilla and mandible and/or lateral deviations of the mandible. All faces show a minor degree of asymmetry, but marked asymmetry is not normal and needs careful evaluation, especially in young children.

Facial symmetry can be assessed by viewing the face from the frontal view or while the patient is reclined in the dental chair and the practitioner is seated behind the chair. A piece of dental floss can be stretched from the middle of the bridge of the nose, down the nose and lower face, to the midpoint of the chin to establish the facial midline. Next, facial symmetry can be judged by comparing the left and right sides of the face from the facial midline plane (Fig 3-2).

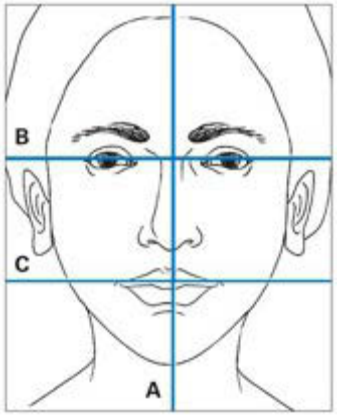


Fig 3-2 Evaluation of facial symmetry. A—facial midline; B—bipu-pillary line; C—lip line.

This evaluation must be performed with the patient both at rest and at maximal intercuspation or centric occlusion to clarify if the asymmetry is due to mandibular shift and chin deviation or to structural asymmetry (Fig 3-3). Facial asymmetry can

be the result of many morphologic abnormalities of different parts of the dentoskeletal structures in congenital misconfiguration.

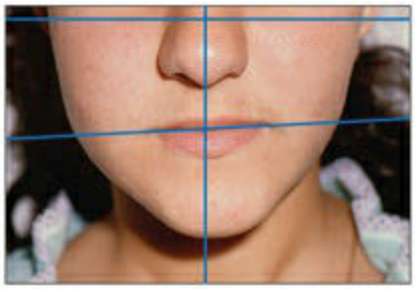


Fig 3-3 Asymmetric growth of the mandible (right condylar hyperplasia) in a 16-year-old girl.

As Subtelny indicated,[10] except in cases of congenital deformity, the maxillary bone is a fixed, stable bone, and the midpalatal suture (raphe) is the true midline indicator of the maxilla. The maxillary midline, except in cases where the maxillary dental midline has shifted because of local factors, coincides with the facial midline and can be a good point of reference in evaluation of facial symmetry.

The causes and the morphologic configurations of facial asymmetry can be determined through the use of diagnostic aids such as posteroanterior cephalometric and lateral jaw radiographs, examination of the dental occlusion on the left and right sides, and observation of occlusal level (curve of Wilson). Patients with significant skeletal asymmetry (those patients with asymmetry not resulting from only a functional shift of the mandible) always fall into the severe problem category.

Early detection of any asymmetry, especially in young children, could be very important for early intervention. The timing of intervention and the treatment plan depend on the cause of the problem, the type of asymmetric mandibular growth, and whether the asymmetry is caused by deficient or excessive growth of the condyle.

Analysis of facial symmetry is discussed further in the section on frontal photography.

Facial proportion

Evaluation of facial proportion is another important aspect of the clinical and paraclinical evaluation. When the anterior facial proportion is evaluated in the vertical dimension, three segments of the face can be assessed: *(1)* from forehead hair line to glabella, *(2)* from glabella to subnasale, and *(3)* from subnasale to soft tissue menton. In a normal, balanced ratio, these three segments are equal (Fig 3-4).

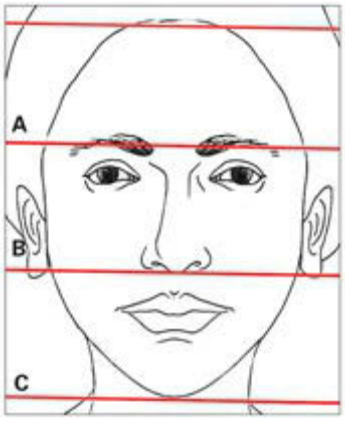


Fig 3-4 Frontal evaluation of facial proportions. In a balanced ratio, the segments from forehead hairline to glabella (A), from glabella to subna-sale (B), and from subnasale to soft tissue menton (C) are equal.

The upper lip–lower lip height ratio is another frontal evaluation of facial proportion. The area from subnasale to stomion should be approximately one third of the total lower third of the face (subnasale to menton)[11] (Fig 3-5).

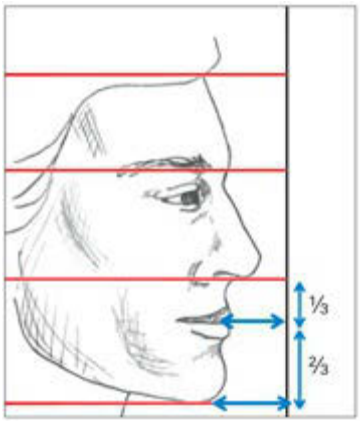


Fig 3-5 Upper lip–lower lip height ratio. The area from subnasale to stomi-on should be approximately one-third of the total lower third of the face.

Another frontal evaluation that can be considered is the sagittal facial proportion, proposed by Sarver,[12] also called the *rule of fifths*. This method is discussed in more detail in the section on facial photography.

There are other important points to be evaluated in both the facial frontal and lateral views, including facial form, symmetry, nose, and chin and lip relationship in three spatial planes: the sagittal, vertical, and transverse dimensions.

Lateral (sagittal) facial evaluation

During the systematic lateral facial examination, the child should be seated in an

upright position with the practitioner looking from a distance at three points: *(1)* the bridge of the nose, *(2)* the base of the upper lip, and *(3)* the chin. This examination gives the observer an idea of the patient's profile.

A straight profile is indicated when all three points are at the same level and form a straight line. If the midpoint is anterior, the patient has a convex profile; if the midpoint is posterior, the patient's profile is concave (Fig 3-6). Each of these three profiles can be caused by abnormalities of one, two, or three point combinations. For example, a convex profile can result from maxillary protrusion, mandibular retrusion, or a combination of both.

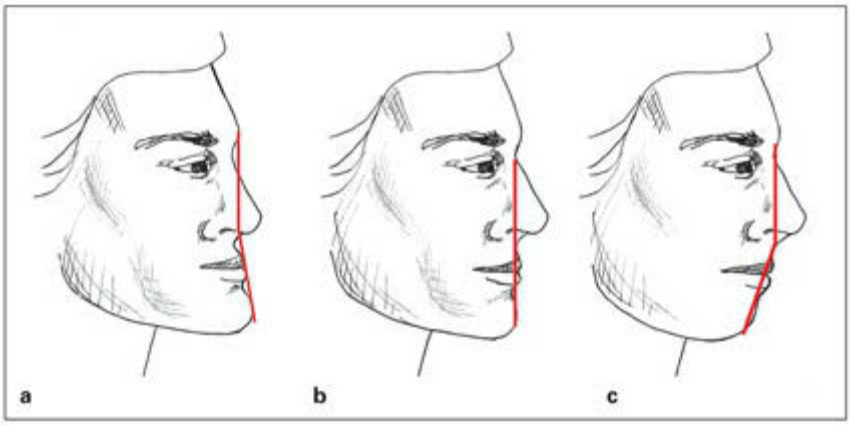


Fig 3-6 Profile types: *(a)* concave; *(b)* straight; *(c)* convex.

Slight retrusion of the mandible before puberty can be considered normal. This dimension will catch up to the maxilla with normal growth of the mandible. Slight convexity of the profile at this age can also be considered normal.

A well-balanced profile usually indicates the presence of a normal or Class I occlusion. An underlying normal skeletal relationship that has no skeletal discrepancy in the anteroposterior relationship, molars and canines with a normal relationship, and primary second molars with a terminal plane that is flush or has a mesial step can be considered normal.

Some children may have normal basal bone relationships but disturbed profiles. These situations may be due to abnormal inclination of the incisors such as that resulting from abnormal, nonnutritive oral habits. Correction of incisor protrusion will improve the profile.

Children with a severely convex profile usually have a Class II malocclusion, and those with a straight or concave profile can have a Class III malocclusion. The clinical and paraclinical examinations can clarify the exact type of malocclusion, whether it is the result of dental or skeletal factors (or a combination of both) or the result of maxillomandibular or dental discrepancies.

The morphology of the patient's profile is also another important area that can change the treatment plan. For example, tooth extraction can be considered in children with a convex profile if other analyses confirm extraction, while extraction should be avoided in children with a straight or concave profile.

Divergence of the face is a termed used by Hellman.[13] Divergence indicates if a profile slopes either anteriorly (anterior divergence) or posteriorly (posterior divergence). Divergence is influenced by the patient's racial and ethnic background. For example, while people of American Indian and Asian descent tend to have an anteriorly divergent face, white people of northern European descent are likely to show posterior divergence.[14]

The upper-lower facial proportion, nose, chin, and lips as well as the interrelationships of these components must also be examined during the evaluation.

Intraoral Examination

Intraoral examinations for orthodontic patients, as for any other type of patient, require a thorough evaluation of the health of all oral hard and soft tissue structures. Any problem, disease, or pathologic condition, including medical problems, dental caries, and pulpal or periodontal involvement, must be treated and under control before orthodontic treatment begins.

The orofacial structure and occlusal system consist of three basic tissues:

1. Dental tissues, consisting of enamel, dentin, cementum, pulp, and the periodontal ligament
2. Skeletal tissues, consisting of bone, cartilage, and ligaments
3. Soft tissues, consisting of neuromuscular tissue, epithelial tissue, glands, the circulatory system, mucous membranes, and connective tissue

The oral cavity and all of the tissue components have several interrelated physiologic functions that directly or indirectly play some role in the development of dental occlusion. Therefore, treatment planning must be based on a thorough examination of all of these structures, in both static and functional states.

For example, evaluating the dental arch relationship in centric occlusion and looking at the relationship of maxillary and mandibular first molars are not sufficient means to classify the type of occlusion. It is necessary to assess all types of mandibular movement, such as protrusive, retrusive, and lateral excursions, and the

dental and facial midline relationships at wide open, rest, occlusion, and path of closure. Assessment of the relationships between rest position and occlusion can differentiate between a functionally true and a forced-bite malocclusion caused by a mandibular shift.

Rest position is the result of a myotatic antistretch reflex in response to gravity; it alters with the position of the head, and natural head position must also be determined for each patient.

To emphasize the important role of orthodontic diagnosis, especially in early treatment, important areas of this assessment are discussed in static and functional evaluations.

The following are important features to consider in the static evaluation of the oral cavity (see Fig 3-1):

- Oral health
- Lips (position relative to each other and to incisors)
- Caries susceptibility and restorations
- Dental age (tooth eruption and sequence)
- Tooth size, forms, and number
- Periodontal and oral mucosal conditions
- Tongue size, posture, and function
- Frenum and oral mucosa
- Adenoids and tonsils
- Type of occlusion (classification, overjet, overbite, midline, urve of Spee)

The following are important features to consider in the functional evaluation of the oral cavity (see Fig 3-1):

• Perioral musculature, including the tonicity and function during mastication, deglutition, respiration, and speech

- Lip competency, tonicity, and function during rest, swallowing, speech, and respiration
- Dental and facial midline relationships at wide open, rest, occlusion, and path of closure

- Mandibular movements and protrusive, retrusive, and lateral excursions
- Freeway space
- TMJ function and dysfunction and condylar movements
- Mode of breathing

Evaluation of the dentition

The first step of occlusal development starts when primary first molars reach occlusion. Numerous reports indicate that many malocclusions develop during primary and early mixed dentition and are recognizable and preventable.[15] Evaluation and classification of the primary dentition are important steps for early detection and intervention. Inspection of a child's dental occlusion must be performed first during the late primary dentition and then before eruption of the permanent first molars.

At this age, occlusion must be carefully examined in three dimensions (sagittal, vertical, and transverse) in both centric occlusion and centric relation. The interdental spacing (Baume classification),[16–19] crowding, primate space, terminal plane, primary molar and canine relationship, overjet, overbite, and midline must be evaluated. The child's age and tooth eruption pattern are also very important to evaluate.

The development of the childhood dentition and resulting occlusion is a dynamic process, occurs in all three planes of space, and may undergo considerable change during childhood and early adolescence. With periodic evaluations, the practitioner can intercept and favorably influence undesirable changes. After more than 40 years of orthodontic practice, especially involving young children, the author believes that many eruption problems can be prevented if the problem is detected early and intervention is started early. The best way to detect these problems is longitudinal monitoring of panoramic radiographs, which is discussed later in this chapter.

Eruption that is early or delayed by a few months is not harmful unless local or systemic problems prevent normal eruption. Any early or late eruption that is detected clinically, especially if it is asymmetric, must be evaluated radiographically to determine if a problem exists.

Asymmetric eruption between the right and left sides of the dental arch lasting more than 6 months is another problem that may require early intervention. Likewise, an abnormal sequence of eruption can be very harmful to occlusal development and deserves careful assessment during the intraoral examination. Early loss and delayed

exfoliation of primary teeth are etiologic factors in some malocclusions. These must be detected at an early stage through a complete intraoral examination.

Other eruption problems, such as ankylosis, tooth transposition, ectopic eruption, and tooth impaction, must be evaluated clinically and radiographically (see chapter 10).

Problems of tooth size, form, and number (missing or supernumerary teeth) are other factors that can interfere with normal eruption and tooth displacement. The absence of a mandibular incisor or the presence of a supernumerary mandibular incisor is easily overlooked; counting the teeth during the examination can prevent these problems (see chapters 6 and 7).

The intraoral examination should include assessment of the following aspects of the dentition (see Fig 3-1):

- Primary occlusion (interdental spacing, terminal plane, and mandibular shift)
- Dental age
- Eruption pattern of the dentition
- Asymmetric eruption
- Early loss and delayed exfoliation of primary teeth
- Eruption problems such as ankylosis, tooth transposition, ectopic eruption, and impaction
- Problems of tooth size, form, and number (missing or supernumerary teeth)
- Dental and facial midlines

Evaluation of soft tissues

Another area of inspection during intraoral examination is evaluation of the soft tissues. The buccal tissues, lips, floor of the mouth, palate, and gingiva should be carefully inspected and palpated. The gingival tissue in young children might be redder and smoother than that of adults because of increased vascularity and thinner epithelium.

Early detection of periodontal involvement before treatment is important; careful probing can be very helpful. Bleeding during probing is indicative of active disease, which must be brought under control before other treatment.

Crowded mandibular incisors must be carefully assessed for tissue recession, especially when attachment to the gingiva is inadequate. Extraction and

nonextraction options must be carefully evaluated.

Recent findings suggest that periodontal disease may have its origins in childhood. A noted increase in the frequency of gingival inflammation in children and progressive periodontal conditions causing primary and permanent tooth loss led the American Academy of Pediatric Dentistry to place greater emphasis on prevention, early diagnosis, and treatment of gingival and periodontal disease in children.[20]

Other areas of the soft tissues that can play an important role in diagnosis and treatment planning in early-age orthodontic treatment are the tongue, frenum, lips, and adenoid and tonsillar tissues. Growth and development of maxillofacial morphology and oral function are closely interrelated.

The tongue has several necessary functions for life and at the same time can play an important role in development of the occlusion. The tongue has three main functions. First, it carries on its surface the taste buds, which send information to the brain about the nature of the food being eaten. Second, the tongue plays an important part in the process of mastication and deglutition. Third, the tongue is involved in speech, assuming different positions in the mouth and altering the shape of the air passage through which sounds made by the vocal cords will pass.

Most tongue postures and movements during normal function exert some forces on the dentition that are in balance with other neuromuscular forces exerted by the perioral musculature. Any imbalanced force, especially at early stages of the dentition, can cause morphologic changes in the occlusal system. Therefore, this structure must be evaluated, in both static and functional states, for the purpose of orthodontic diagnosis and treatment planning. This assessment is based on three important conditions of the tongue: *(1)* tongue size, *(2)* tongue posture, and *(3)* tongue function.

Tongue size

The interaction between tongue size and volume and craniofacial skeletal growth is essential for understanding the mechanism of specific types of malocclusion and treatment planning.

Harvold et al,[21,22] using rhesus monkeys, demonstrated that reducing tongue volume by partial glossectomy caused the dental arch to collapse lingually and produce crowding.

One of the factors that plays an important role in the development of the occlusion is the balance between the tongue and the perioral chain of muscle. Any imbalance

between these two factors can cause irregularities in occlusion. For example, in patients with acromegaly, macroglossia causes many types of irregularities, such as interdental spacing, anterior open bite, dental protrusion, and Class III malocclusion (Fig 3-7).

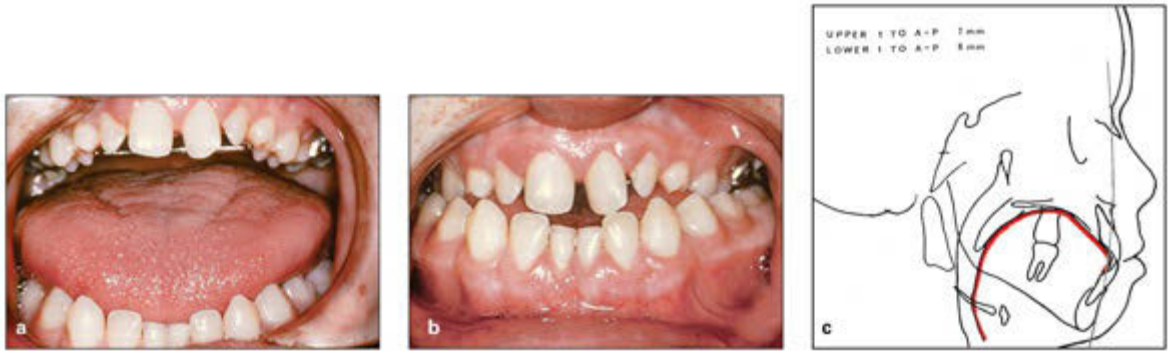


Fig 3-7 *(a to c)* Macroglossia causing a large mandibular arch, anterior and posterior crossbites, and anterior open bite. (Courtesy of J. Daniel Subtelny.)

Another example is the effect of Pierre Robin syndrome, which is a condition present at birth. The infant has a smaller-than-normal mandible, a tongue that falls back in the throat, and difficulty breathing because the tongue is smaller and retropositioned. Patients with this condition have severe crowding.

Tongue posture

Tongue posture also plays an important role in development of some malocclusions, including open bite, interdental spacing, and anterior crossbite. Tongue position can be altered by other conditions, including large tonsils or adenoids and chronic nasal inflammation that can contribute to tongue thrusting and poor tongue posture (Fig 3-8).

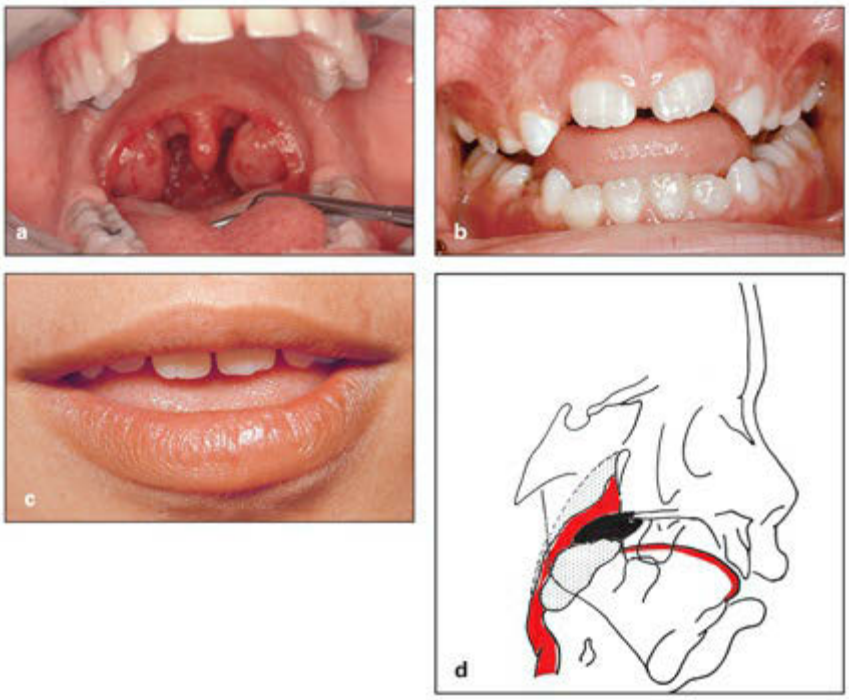


Fig 3-8 *(a to d)* Large tonsils causing forward positioning of the tongue and open bite. (Courtesy of J. Daniel Subtelny.)

Prolonged low tongue position during critical growth periods in children may initiate a sequence of events resulting in excessive molar eruption, a clockwise rotation of the growing mandible, a disproportional increase in lower anterior facial height, retrognathic mandible, and open bite (see chapter 5).

Tongue function

Growth and development of maxillofacial morphology and oral function are closely interrelated; tongue activity and forces during physiologic functions such as deglutition, mastication, and speech can play a role in developing malocclusion if it functions improperly (Fig 3-9). Myofunctional disorder of the tongue has been described in various ways, including *deviate swallow, infantile swallow,* and *abnormal swallow,* and the terms *tongue thrust* and *tongue thrusting* have greater usage among authors.

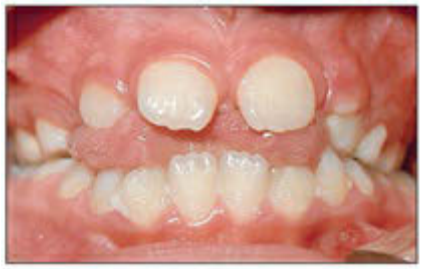


Fig 3-9 Abnormal tongue function.

Atypical swallowing patterns are considered important factors in the etiology of posterior crossbite. Ovsenik[23] examined 243 patients aged 3, 4, and 5 years. She found that atypical swallowing patterns were more common among children with posterior crossbite than those without crossbite. She concluded that the orofacial functions of children in the primary dentition stage with sucking habits should be assessed at each examination. Special attention should be paid to the swallowing pattern, which was found to be an important etiologic factor in the development of posterior crossbite.[17]

Production of certain sounds can be difficult for some patients with severe malocclusion, such as anterior lisping in patients with anterior crossbite. The relationship between malocclusion and speech problems is another aspect of the patient's situation that must be evaluated before treatment planning. Determining whether the malocclusion is contributing to the speech problem or the speech defect has contributed to the malocclusion is complicated and controversial. Neuromuscular problems that cause speech problems can also be causative agents for malocclusion.

Anterior open bite with a large gap between the incisors can cause lisping because it affects the position of the lips in pronunciation of sibilants such as */s/* and */z/*, */sh/*, */ch/*, or */th/*. Irregular incisors or retroclination of maxillary incisors impedes production of linguoalveolar sounds such as /t/ and /d/. In patients with skeletal Class III relationships, labiodental sounds such as */v/* and */f/* are sometimes disturbed.

Evaluation of these three aspects of the tongue to determine whether they represent an etiologic factor in the malocclusion is important for treatment planning and to ensure stability of the treatment result (see chapter 5).

Assessment of TMJ function

Evaluation of the TMJ is an essential part of the clinical evaluation before any type of orthodontic treatment, especially in routine clinical examination of young patients.

The first thing to evaluate is the range of mandibular movement in protrusive, retrusive, and lateral excursions. If these mandibular movements are normal, the function is usually normal. However, if a restrictive movement is present, it usually indicates a functional problem.

Other functional evaluations of the TMJ, such as maximum opening, palpation of the muscles of mastication and head of the condyle, and palpation of the patient's neck and submandibular area, must also be carefully performed. The goal is to look for any signs of TMJ problems, such as limitation of opening, clicking, pain, noise, and crepitus, that may require further evaluation and treatment.

Palpation of the muscles must be performed bilaterally, while the clinician is standing in front of or behind the patient and assessing any soreness or discomfort for the patient on either side. The lateral and medial pterygoid muscles must be palpated intraorally and the temporalis and masseter muscles extraorally.

During jaw movement, any lateral or anterior mandibular shift must also be recognized. Differentiating true mandibular asymmetry from mandibular shift is of special importance in growing patients. Posterior crossbite can cause lateral mandibular shift, and anterior crossbite causes anterior shift. "Sunday bite" is another abnormal position of the mandible that can be seen in some patients with skeletal Class II malocclusion. In this type of bite, also known as "dual bite," a lack of proper fit between the maxillary and mandibular teeth causes the mandible to slide forward to compensate for the maxillary protrusion.

The following are important aspects to examine during evaluation of the TMJs:

- Range of mandibular movement, including protrusive, retrusive, and lateral excursions
- Maximum opening
- Pain during movements
- Crepitus and clicking
- Path of closure of the mandible from resting position
- Freeway space
- Prematurities and point of initial contact
- Displacement or tooth guidance
- History of pain
- TMJ tenderness to palpation
- Muscle tenderness to palpation

Paraclinical Evaluation (Diagnostic Records)

Dental abnormalities or malocclusions are common orthodontic problems that are almost always the result of improper developmental processes. These abnormalities are the result of many genetic and environmental factors acting during prenatal or postnatal life. The result is a multifactorial entity that has many different characteristics and different morphologic patterns.

The first step in treating this multifactorial and multi–characteristic entity is to diagnose, classify, and clarify the problem by careful investigation.

Orthodontic diagnosis involves the study and interpretation of information collected from the clinical examination and data accumulated through the paraclinical evaluation. The paraclinical evaluation includes analysis of all data accumulated from different diagnostic tools, such as study cast, photographs, intraoral and extraoral radiographs, and cephalometric radiographs. For comprehensive diagnosis and treatment planning, these findings are combined with previous information gathered from questionnaires and the clinical examination.

Dental Casts

The orthodontic study cast is one of the most useful tools in orthodontic treatment. Preparation of a good set of casts requires a good impression that covers the complete area of the patient's dentition with maximum displacement of soft tissues, created by maximum extension of the impression to the deepest parts of the sulcus and all retromolar areas. A good cast must show as much as possible of the alveolar process and teeth and must be able to display the inclination of the teeth, not just the location of the crown.

For young children with no previous experience of having impressions taken, the procedures, including the type of impression maneuver, the child's head position, and tongue placement must be explained and demonstrated. It is recommended that the mandibular impression be taken first, because it is easier for children than a maxillary impression.

To prevent posterior flow of alginate, the tray should be seated first on the retromolar area in the mandible, or on the palate for the maxilla, and then on the anterior part of the arch, before it is forced down toward the occlusal surface. At this time, the child's lips should be separated from the teeth, and alginate should fill the sulcus area.

The wax bite taken in centric occlusion is used to register the exact position of the maxillary and mandibular dentition and orient the maxillary and mandibular casts for

proper trimming. After it is properly trimmed and polished, the study cast usually includes patient information such as name, date of birth, and date taken.

A set of good study casts is a part of the patient's record and is a useful tool for occlusal assessment and analysis in diagnosis and treatment planning. The casts can be used to help determine the type of occlusion, arch form, arch symmetry, and curve of Spee, to perform space analysis, and to demonstrate findings to the patient and parents.

Type of occlusion

The patient's exact type of occlusion in three dimensions can be easily verified by casts, while it cannot be done easily through intraoral examination. Examination of each arch and single tooth will reveal displacement or rotation of any single tooth or group of teeth. Any abnormal intercuspation or interdigitation can be recognized in the sagittal, transverse, and vertical evaluations.

Sagittal evaluation

With the sagittal evaluation of the casts, the anteroposterior relationship of canines and molars, in both the permanent and primary dentition (terminal plane), and Angle classification can be accurately established. Intercuspation and interdigitation can be assessed easily from the buccal and lingual views. The anteroposterior relationship of the incisors and the amount of overjet and overbite also can be accurately measured from the cast.

Transverse evaluation

Transverse evaluation of the cast can reveal midline discrepancies and posterior crossbites. Evaluation of the dental midline relative to the maxillary midline suture can reveal any dental midline shift. Therefore, if the maxillary midline is at a normal relationship with the facial and palatal midlines, the problem is from the mandibular midline. This discrepancy can be caused by mandibular dental midline shift or mandibular jaw shift, which can be confirmed by functional evaluation of the mandible during the clinical assessment of resting position and centric occlusion. These are questions that can be answered by combining clinical findings and cast evaluation and by assessing cast symmetry.

Posterior crossbite can be the cause of lateral mandibular shift (see chapter 12).

Vertical evaluation

Vertical evaluation of study casts involves assessment of the vertical overlap of incisors, which is measured and recorded either in millimeters or as a percentage of the total height of the overlapping maxillary incisor over the mandibular incisor crown. Because of variations in the crown length of incisors in different people, it is better to use percentages instead of millimeters.

In a normal bite, the maxillary incisors usually overlap the mandibular incisor crowns by about 25%. In *deep bite*, the mandibular incisors are completely covered (100%), and in some situations the mandibular incisors impinge on the palatal gingiva. The vertical relationship of the posterior segment of dentition must also be evaluated, because in some kinds of buccal bite or Brodie syndrome there is increased incisor overlapping.

An absence of vertical overlap of incisors is called *open bite*, which is also measured in millimeters. Anterior open bite is most commonly caused by a sucking habit in the young age group. There are many different types of open bite, such as dental, dentoalveolar, skeletal, or combination open bite. Open bite can be caused by the maxillary or mandibular teeth, either jaw, or a combination of factors and therefore needs a careful differential diagnosis for proper intervention and treatment planning (see chapter 13).

All of these conditions can be determined by a combined cast evaluation, clinical findings, and cephalometric evaluation.

Arch form and symmetry

The ability to assess arch form and detect any asymmetry within the dental arches is another advantage of having good study casts. To detect arch asymmetry more accurately, a graded plastic tool called a *symmetrograph* can be used. The symmetrograph is placed over the occlusal surface of the maxillary arch, and the central line of the grid is superimposed over the midpalatal suture. This position reveals whether teeth are equidistant from the midpalatal raphe or a distortion is present in the arch.

With this technique, buccolingual and mesiodistal distances of the left and right sides of the dentition can be accurately analyzed and compared with the midpalatal suture to detect if any asymmetry is present (Fig 3-10). Application of this analysis can differentiate whether posterior crossbite is caused by arch constriction or overexpansion (see chapter 12).

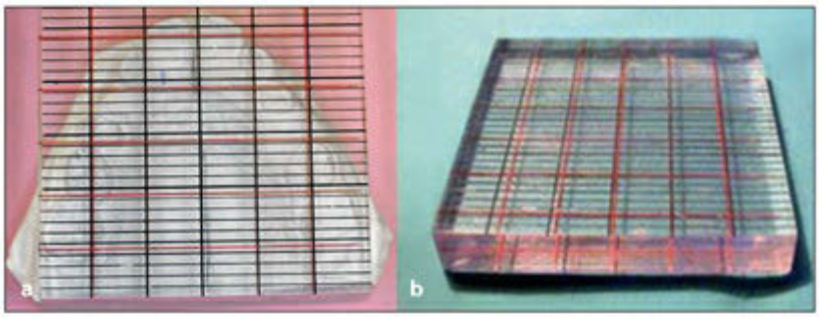


Fig 3-10 *(a and b)* Two-surface ruled grid of thick, transparent plastic for evaluation of arch symmetry (Dentaurum).

Early loss of primary teeth and dental shift may cause a unilateral asymmetry within the dental arch, either from distal drift of incisors or from mesial drift of posterior teeth. This kind of asymmetry can be distinguished from arch asymmetry by an accurate evaluation with the symmetrograph, which can also reveal the mesiodistal shift of the dentition.

Mixed dentition space analysis

Study casts can also be used to determine the availability of space in the arch in crowding cases. The amount of crowding and available space are important factors in determining whether the treatment plan will include tooth extraction. Space analysis is a comparison between the amount of space available in the arch and the amount of space needed for the alignment of the dentition.

Space analysis can be performed either directly on casts and radiographs or with computerized methods after appropriate digitization of the arch and tooth dimensions. The latter methods are easier and more practical.

Arch length, that is, the distance from the mesial surface of one permanent first molar to the one on the other side, can be measured two different ways: It can be measured with a divider or Boley gauge, segment by segment, or with a piece of soft brass wire contoured from the mesial surface of one permanent first molar, over the contact points of the posterior teeth and incisal edges (not including the displaced incisors), to the mesial surface of the molar on the other side. The straightened wire is measured as the amount of available arch length.

Required space is the amount of space needed for the canines and premolars present in the arch. This value can be measured directly from the mesiodistal diameter of all erupted teeth on the cast (contact point to contact point), or indirectly from radiographs. The sum of the mesiodistal widths of all individual teeth indicates

the space needed for the dentition. Good periapical radiographs or lateral jaw radiographs can be helpful for this measurement. For correction of any magnification error, the exact mesiodistal width of an erupted tooth on the cast can be calculated and compared with the width of the same tooth on the radiograph.

A comparison between the available space and the total measurement of tooth material will establish whether space is sufficient to accommodate the dentition, there is a need for more space, or there is excess space that will result in interdental spacing. In all space analysis and application of the measurements, six important factors must always be considered before the final decision is made for space creation or tooth extraction:

1. Incisor inclination
2. Profile
3. Relationship of incisors to lip position
4. Growth pattern
5. Mesial shift of permanent molars
6. Curve of Spee

Incisor inclination

The anteroposterior position of incisors can affect treatment decisions in space management. If incisors are crowded and already proclined, the required space is greater than indicated by the measurements. If the mandibular incisors are retruded because of some muscular dysfunction or habits and the incisors can be brought to a more normal inclination, this would provide more space for crowded incisors.

Profile

The patient's profile and lip position also can affect the treatment decisions. In a patient with a convex profile and procumbent lips, the possibility of tooth extraction would change the result of space measurement.

Relationship of incisors to lip position

Lips that are procumbent relative to the chin and nose may necessitate incisor retraction, which needs more space.

Growth pattern

The patient's growth pattern is another important factor to be considered in the analysis of space availability. Björk and Skieller[24] stated that there is little or no tendency for the dentition to be displaced during growth in a child with a well-proportioned face, while the teeth often shift anteriorly or posteriorly in a child with jaw discrepancy (such as Class II, Class III, long face, or short face relationships) so that space analysis would be less accurate.

Mesial shift of permanent molars

Early loss of primary molars and mesial tipping of permanent molars and space loss is another factor that must be considered in space analysis. The uprighting and distalization of molars (if possible) will reduce space deficiency. In some patients with moderate incisor crowding, taking advantage of leeway space and preventing molar mesial movement could be the treatment of choice (see chapter 4).

Curve of Spee

The study cast is also a useful tool in the measurement of curve of Spee, which is a measurement (in millimeters) of the deepest part of the occlusal plane of the mandibular arch. This can be measured by resting a straight horizontal plane from the tip of the most distobuccal cusp of the last molar to the tip of the mandibular canine. The distance from this plane to the tip of the tallest buccal cusp of the premolars or primary molars shows the amount of curve of Spee. Correction of the curve of Spee also requires extra space, which must be considered in space analysis.

Methods of analysis

One purpose of space analysis during the mixed dentition stage is an estimation of the total mesiodistal width of unerupted permanent teeth to determine the space needed for a well-aligned dentition. Measurement of unerupted permanent teeth has been achieved through three basic approaches:

1. In the Nance[25] technique, the mesiodistal width of the unerupted teeth can be measured directly on radiographs while the arch length is measured with soft brass wire. As mentioned earlier, good periapical or lateral jaw radiographs are required, and the possibility of magnification errors must be considered.

2. The width of unerupted permanent teeth can be estimated with proportionality tables. Studies have shown that there is a reasonably good correlation between the size of the permanent incisors and canines and the premolars.[15,26] This method can be used without radiographs.
3. Staley and Kerber[27] proposed a combination of radiographic and prediction table methods. They used the size of permanent incisors, measured on dental casts, and the size of unerupted premolars, measured on radiographs, to predict the size of unerupted canines.

Another type of space analysis, the Bolton analysis,[28] compares the total mesiodistal widths of the maxillary and mandibular teeth to assess for tooth mass discrepancies that would prevent good occlusion. The greatest mesiodistal dimension of all teeth (except second and third molars) is measured on each cast. The ratio of the 12 mandibular measurements to the 12 maxillary measurements is considered the overall ratio, and the ratio of the 6 mandibular anterior measurements to the 6 maxillary anterior measurements is the anterior ratio.

Other methods of space analysis have been proposed by different investigators; see chapter 4 for more details.

The following are important uses for study casts in orthodontic treatment:

- Preservation of a record of the patient's original occlusion
- Analysis of the original occlusion
- Demonstration to parents and patients
- Transfer of the patient's case records to another practitioner
- Evaluation of treatment progress
- Assessment of arch form and symmetry
- Space analysis
- Evaluation of tooth size discrepancy
- Legal protection

Photographic Evaluation

Photography plays an important role in diagnosis and treatment planning in orthodontic treatment. Because the patient is still growing, many changes occur

during early orthodontic treatment. Initial photographs and longitudinal evaluations during and after treatment can be very useful for assessing treatment results. Clinical photography is usually applied for the following purposes:

- As a permanent record and as documentation to show the initial status of the patient's dental and facial morphology before treatment.
- As an important aid in diagnosis and treatment planning for evaluation and analysis of facial form and proportion in three dimensions.
- As a means of education and demonstration for patients and parents.
- As a means of legal protection.
- As an invaluable source for education, research, patient referral, conferences, and publication, if a complete set of photographs is maintained from the first visit, including photographs during treatment and after treatment.
- For accurate measurement of important points and proportions of the face in three dimensions on tracings of the photographs and comparison of the initial assessment with progress photographs and final results. These assessments cannot be applied clinically.

Photography for orthodontic patients consists of two types of image: extraoral photographs and intraoral photographs.

Extraoral photography

Extraoral photographs are taken while the patient is standing in a relaxed position, assuming a natural head position with the mandible at rest position, and looking straight into the camera or at his or her eyes in a mirror. Extraoral photographs for typical orthodontic cases usually include the frontal view, left and right profiles, 45-degree view, and the smile (Fig 3-11). If patients have asymmetry or craniofacial deformity, extraoral photographs of the affected areas are also needed.

Fig 3-11 Standard series of extraoral facial photographs. *(a)* Frontal smiling view. *(b)* Frontal

resting view. *(c)* Profile view. *(d)* Oblique view.

Frontal view

Figure 3-11 shows four standard facial photographs, including frontal and lateral views of the same patient. Depending on the existence of other facial deformities, other photographs with different angulations or focused on special areas of the face might be needed. For example, Fig 3-12 shows two views of a patient with incompetent lip closure caused by severe dental protrusion and overjet. For this patient, retracting the incisors would improve both lip function and facial esthetics. In patients with prominent lips that are not separated and do not exhibit strain on closure, incisor retraction would have little effect on lip position.

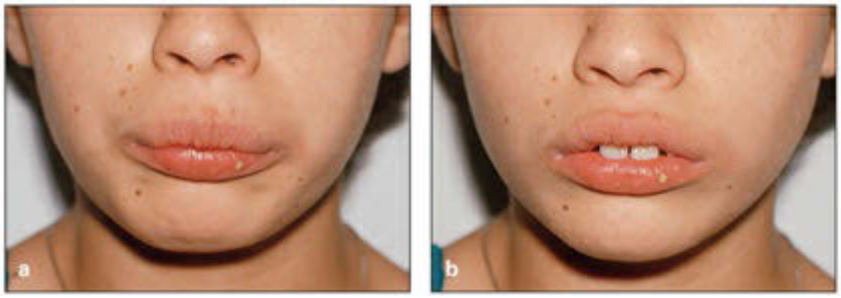


Fig 3-12 *(a and b)* Lip strain.

Lateral view

Profile photographs are also taken in the patient's natural head position, while the patient is in a relaxed position looking straight forward or into a mirror. A lateral photograph is taken from the left and right sides of the patient's profile with the hair pulled behind the ear to permit visualization of the entire face. The lateral view must show the whole head, chin, and base of the neck to permit visualization of the contours of the chin and neck area (see Fig 3-11c).

Oblique (45-degree) view

The 45-degree lateral photograph is a three-quarter view of the face that can help visualization of the midface and any deformities, such as nasal deformities. This view represents the view of the patient most frequently seen by others (Fig 3-11d).

Photographic evaluation of facial esthetics

An improved facial and dental appearance is perhaps the main objective for every

orthodontic patient seeking orthodontic treatment. The major objective of treatment is achieving a normal dentoskeletal relation for better function and stability. Overcoming the psychosocial difficulties that can result from facial and dental appearance is also considered an important esthetic goal in treatment planning.

Clinical facial examination is a direct observation of the patient's face in three dimensions. Facial photographs should be viewed as an adjunct to what the eyes reveal clinically. Printed photographs allow more accurate analysis of the facial form, symmetry, and proportion.

Acetate tracing paper can be placed over different facial photographs, and the structures of the face can be traced and analyzed by drawing specific lines. The tracing can be kept as documentation for the patient's permanent records and for comparison with the treatment progress records and final results.

Evaluation of lateral photographs. The patient's profile is an important part of esthetics and has a profound effect on treatment planning. No treatment planning can be complete and designed without consideration of the patient's profile. There are special areas of concern that must be assessed in the photographic evaluation: profile form (straight, convex, or concave), facial proportion, lip size, lip position, nasolabial angle, and mentolabial sulcus.

Profile. The bridge of the nose, base of the upper lip, and the chin are used to evaluate the patient's profile proportion. Once identified, these three points are connected with straight lines to form a complete drawing of the patient's profile. Figure 3-6 shows an illustrated analysis of the lateral view and delineates the appearance of concave, straight, and convex profiles.

Lateral facial proportion. The lateral vertical proportions of the patient's profile can also be traced and measured with more accuracy on a printed lateral photograph. The vertical proportions can be assessed using four points: the hairline, the bridge of the nose, the base of the upper lip, and the chin. In patients with normal vertical proportions, the upper face, middle face, and lower face are equal (Fig 3-13).

Fig 3-13 Equal vertical proportions. In a balanced proportion, the upper, middle, and lower facial segments are equal.

Lip proportion. The same technique can also be used to trace and measure the vertical lip proportions. The relative normal proportion between the upper lip and the lower lip and chin segment is one-third to two-thirds (Fig 3-14).

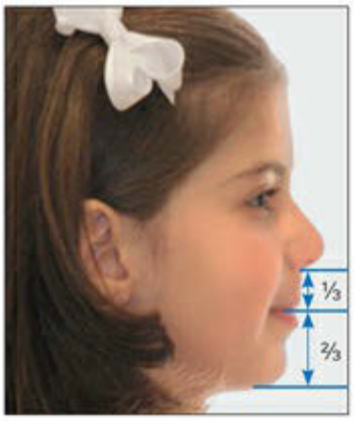


Fig 3-14 Vertical lip proportions. The normal ratio of upper lip height to lower lip height is one-third to two-thirds.

Lip position. The relative position of the lips to the nose and chin plays an important role in the patient's profile and in esthetics. Basal bone position and incisor position and inclination have a direct effect on the position of the lips; therefore, assessment of the lip position is an important aspect of lateral photograph evaluation.

Several analyses have been proposed for evaluation of this lateral soft tissue.[29–32] One of the simplest and most practical ways to evaluate the lip position is Ricketts' esthetic line (E line), which is drawn from the tip of the nose to the most anterior point on the soft tissue chin or soft tissue pogonion. In normal situations in the 3- to 6-year-old child, the upper lip should lie about 3 to 4 mm behind the line, whereas the lower lip should touch or lie slightly in front of this line (Fig 3-15).

Fig 3-15 Relationship of lips to nose and chin (Ricketts E line). In 3- to 6-year-old children, the upper lip usually lies about 3 to 4 mm behind the line, and the lower lip touches or lies slightly in front of the line.

Nasolabial angle. The nasolabial angle shows the relationship of the nose and upper lip. This angle can be traced with two lines, one from the base of the nose and the other parallel to the frontal surface of the upper lip. The value of the angle can range from 90 to 110 degrees (Fig 3-16).

Fig 3-16 Nasolabial angle. The normal range of the angle is 90 to 110 degrees.

The inclination of incisors has a direct effect on this angle; greater incisor protrusion reduces the size of the angle. For example, for an acute angle, caused by maxillary incisor protrusion, treatment involves camouflage and extraction of the maxillary primary first molar. An obtuse nasolabial angle can be caused by dental retrusion, as in patients with a Class II division 2 malocclusion and retruded incisors. Torquing of incisors can correct this angle. An obtuse angle is also observed in patients with maxillary anterior crossbite; proclination of the maxillary incisors can correct this angle.

However, the nasolabial angle may also be out of normal range if the patient has

an elevated nasal tip; this possibility must be considered in treatment planning.

Mentolabial sulcus. The mentolabial sulcus is the fold of soft tissue between the lower lip and the chin; it varies greatly in form and depth among patients.

The mandibular incisor position and inclination and the lower facial height affect the degree of this angle (Figs 3-17 and 3-18). Proclination of mandibular incisors deepens this angle. Because of a lack of lower lip projection, retroclination of mandibular incisors results in a shallow mentolabial sulcus.

Fig 3-17 Normal mentolabial sulcus angle.

Fig 3-18 Acute mentolabial sulcus angle.

In patients with short lower facial height and Class II relationships, the lower lip is usually everted against the maxillary incisors.

Evaluation of frontal photographs. Extraoral frontal facial photographs are a useful tool in the orthodontic diagnostic record and can be used to assess various frontal measurements, such as vertical and lateral proportions and facial symmetry.

Facial symmetry. In almost all normal faces, a small degree of asymmetry exists between the left and right sides.

Proffit and Fields[14] illustrated this asymmetry when they compared the real full face with composites of two right or two left sides. Minor asymmetry can be ignored, but any significant asymmetry, especially in young children, must be detected and treated early.

Asymmetry can be detected more accurately with photographic tracing than with visual observation. However, evaluation of frontal and 45-degree cephalometric radiographs can detect the problem much more accurately.

There are different techniques for evaluation of facial symmetry on frontal facial photographs; one involves drawing the facial midsagittal plane on tracing paper. The midsagittal plane is a vertical line drawn from the center of the nasal bridge, or soft tissue nasion, to subnasale, extending to soft tissue pogonion. Two parallel horizontal lines also are drawn: the bipupillary plane, which is drawn between the left and right pupils, and the lower horizontal line, the stomion plane.

In a symmetric face, the sagittal plane intersects at all three points (soft tissue nasion, subnasale, and soft tissue menton), and the two horizontal lines are parallel and perpendicular to the facial midline. Both sides of the face relative to the facial midline are vertically and horizontally equal and symmetric (Fig 3-19).

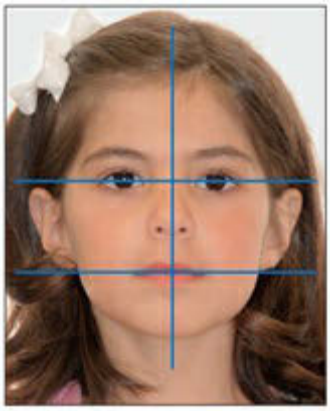


Fig 3-19 Evaluation of facial symmetry. Both sides of the face must be vertically and horizontally equal and symmetric relative to the facial midline.

Rule of fifths. Another technique for assessing frontal facial symmetry was presented by Sarver,[7] who termed it the *rule of fifths*. In this evaluation, six vertical planes are drawn to divide the face sagittally into five equal parts from helix to helix of the outer ears. In this way, the right and left portions of the face can be compared. According to this analysis, the ideal face comprises equal fifths, all approximately equal to the width of one eye (Fig 3-20).

Fig 3-20 Rule of fifths. According to this standard, in the sagittal plane the ideal face is composed of equal fifths, all approximately equal to the width of one eye.

Facial proportion. Evaluation of the balance between the upper, middle, and lower thirds of the face can also be assessed by drawing four parallel lines over the frontal photograph: at the hairline, the glabella or supraorbital ridge line, the base of the nose or subnasale line, and soft tissue menton (Fig 3-21). This divides the face into three segments, which can then be compared. In a normal, balanced ratio, these three portions of the face should be equal.

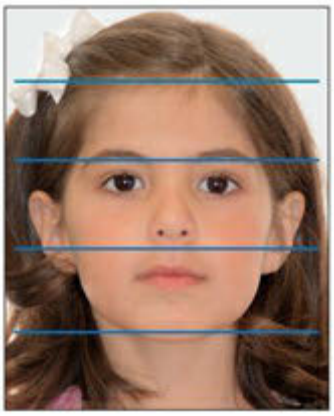


Fig 3-21 Evaluation of frontal facial proportions (upper, lower, and middle facial heights). In a balanced face, these three segments are equal.

Intraoral photography

Intraoral photography is also an important part of the diagnostic record. The series of intraoral photographs routinely includes five views of the occlusion: lateral left, lateral right, frontal, and maxillary and mandibular occlusal views. The occlusal views must show the occlusal surface of the entire dentition and can be taken using a front surface mirror.

Occasionally, other special views with different angulations might be needed,

such as for patients with abnormal frenum attachment, palatal impaction, impinging bite, or severe overjet. In patients with mandibular shift, two photographs, one taken in centric occlusion and one at rest, can show the difference in the midline position.

Intraoral photographs also have many advantages as part of the patient's record. One important purpose of the intraoral photograph is to record the condition of the hard and soft tissues before treatment. The photograph can then be referred to during treatment and compared with the final results. Another important advantage of a good, complete set of intraoral photographs is that it can be used in case presentations, seminars, education, and publications. Furthermore, a good set of intraoral photographs is a perfect tool for showing different views of the dental occlusion and any soft and hard tissue defects, such as white-spot lesions of the enamel, hyperplastic areas, gingival problems, and other pathologic soft tissue conditions. It can also serve as an important form of treatment documentation.

Radiographic Examinations

Radiography is a useful tool in diagnosis and treatment planning for the patient's orthodontic problems and can play an important role in prevention, interception, and early detection of many types of eruption problems.

Depending on the existing dentoskeletal anomalies and malocclusions, different radiographic techniques can be used. The radiographic techniques used in orthodontic treatment can be classified into two major types: intraoral radiography and extraoral radiography. The most common intraoral radiographs used in orthodontics are the periapical, bitewing, and occlusal views. Common extraoral radiographs include panoramic radiographs, lateral jaw radiographs, hand-wrist radiographs, and different types of cephalometric radiographs (lateral, posteroanterior, and 45-degree views). In recent years, computed tomography (CT) has had an increasing role in dentistry and orthodontic diagnosis.

Intraoral radiography

Periapical radiographs

Periapical radiographs can be used to evaluate tooth structure, pulp, periodontium, and supporting bone prior to orthodontic treatment. Any caries, periodontal involvement, bone loss, and periapical lesions must be treated or under control

before tooth movement.

Good periapical radiographs can also be used for accurate measurement of the mesiodistal crown width of unerupted canines and premolars for different space analyses.

Bitewing radiographs

Bitewing radiographs are helpful in detection of proximal caries. A set of two bitewing radiographs, taken before the start of treatment and before molar banding, is recommended.

Occlusal radiographs

Occlusal radiographic projection is a useful technique that reveals the entire maxillary or mandibular arch. These radiographs can also reveal supernumerary teeth, cysts, or any pathologic lesions present in these areas. Occlusal radiographs also can be used in evaluation of the buccopalatal relationships of supernumerary teeth or odontomas to other teeth in the arch.

The most common application of occlusal radiographs in orthodontics is the evaluation of impacted and ectopic eruption to determine the relationships to adjacent teeth. However, the use of CT scanning is much more accurate for this purpose.

Extraoral radiography

Common extraoral radiographs used in orthodontic treatment are lateral jaw projections, hand-wrist radiographs, CT scans, panoramic radiographs, and different cephalometric radiographs (lateral, posteroanterior, and oblique or 45-degree views). The lateral cephalometric radiograph is usually taken at centric occlusion but, depending on the type of occlusal problem and possibility of mandibular shift, it can be taken with the patient at rest and in wide-open positions too.

It is not the intention of this book to discuss all aspects of radiographic application or the different techniques and analyses used in orthodontic diagnostic procedures; there are many books, articles, and reports on these subjects. The main objective of this chapter is to refresh the reader's memory regarding the availability of techniques and tools and to emphasize the need for careful pretreatment investigation before any treatment planning, especially for preventive and

interceptive considerations. The following are the most common extraoral radiographic techniques used in routine orthodontic diagnosis and treatment planning.

Lateral jaw radiographs

The lateral jaw radiograph is taken from each side of the jaw while the cassette film is held on one side of the face. Because there is minimal image superimposition in this type of radiograph, it shows a clear view of one side of the jaw.

Lateral jaw radiographs are useful during the mixed dentition to evaluate the relationship and position of primary teeth and their successors, third molar position, and their supporting bone and status of eruption. This type of radiograph is also useful in space analysis for accurate measurement of the mesiodistal width of unerupted canines and premolars during the mixed dentition.

Hand-wrist radiographs

The most important goal in early orthodontic and especially orthopedic treatment is establishment of a normal and balanced correlation between basal jaw growth and development of occlusion in patients with malocclusions that are progressing with a skeletal discrepancy. This goal must be achieved before skeletal growth is finished. Obviously, the best and fastest result can be achieved when the skeletal growth rate is at its highest (peak height velocity [PHV]). General skeletal maturity is usually used as an indicator to predict the timing of PHV in jaw growth.

Skeletal maturity is a stage of bone development based primarily on the size, shape, and degree of mineralization of the epiphyses and degree of closure of the epiphyseal plates. It has also been recognized that individual chronologic age does not necessarily correlate with skeletal maturation age.[33,34] Effective orthopedic treatment cannot be determined by chronologic age or the stage of dental development, because neither of these two is reliable. Therefore, all orthopedic orthodontic treatment, such as headgear, chin cap, face mask, and functional appliances, must correlate with maximum jaw growth.

Attempts to determine general skeletal maturity have included measurements of body height, sexual maturity, cervical vertebrae, bone age, and hand-wrist growth to find out whether the pubertal growth has started, is occurring, or has finished. Hand-wrist radiographs in particular have been used in many ways by many different investigators.[34–37] Several human growth studies have also shown that the timing of

the pubertal peak growth velocity in statural height (PHV), as well as in growth of jaws, is closely related to specific ossification events observed in the hand-wrist area.[33–35] Fishman[33] developed the Skeletal Maturation Assessment (SMA), a method for the evaluation of skeletal maturity using hand-wrist radiographs. This system is based on four stages of bone maturation (epiphyseal widening, ossification of adductor sesamoid of the thumb, capping of selected epiphyses over their diaphysis, and fusion of selected epiphyses and diaphysis), all located at six anatomical regions of the hand and wrist (thumb, third finger, fifth finger, and radius). In this system, 11 skeletal maturation indicators are found in the six aforementioned sites.

The adolescent growth spurt usually starts around the age of 12 years for girls and 14 years for boys. Pubertal growth acceleration lasts 2.0 to 2.5 years in both sexes. PHV at the apex of the incremental curve occurs for 1 year (6 months before and 6 months after), that is, around 10.3 cm for boys and 9.0 cm for girls, which correspond with SMA values 4 to 7. It has also been shown that pubertal maximum facial growth occurs slightly later than peak growth of general body height. The SMA system has been used for several years in the orthodontic department at the Eastman Institute for Oral Health.

CT scans

Computed axial tomography (CAT) is the process of using computers to generate a three-dimensional image from a flat two-dimensional radiographic image, one slice at a time. CT scans, sometimes called *CAT scans*, show the body's interior structures, such as bone, teeth, and soft tissues, as a three-dimensional image. These scans provide greater clarity and reveal more details than regular radiographic examinations.

This type of radiography can be a very useful tool to localize and show unerupted and superimposed teeth in three dimensions in the jaw (eg, to detect and localize supernumerary or impacted teeth). CT scans also play an important role in identifying problems in the bones of the face as a whole, including tumors and fractures, in guiding difficult extraction procedures, and in evaluating bone support for placement of dental implants. Figure 3-22 is a composite of different CT views of a patient with oligodontia.

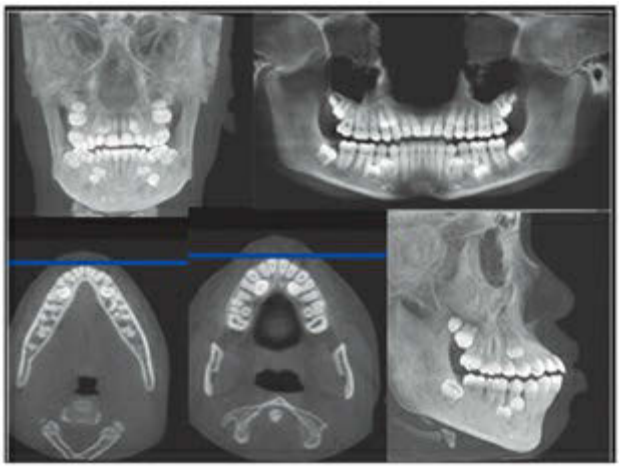


Fig 3-22 Different CT scans of a patient with oligodontia.

Digital imaging

Digital imaging is a recent technology replacing radiographic film in different areas of medical and dental technology. It must be stressed that the image obtained on a digital radiograph is not necessarily any better than one taken using standard radiographic film; however, digital technology does require substantially less radiation than film.

Another advantage of digital radiographs is the ability to enhance the images, for example, by enlarging the image or changing the contrast to achieve better diagnostic quality. Digital imaging can play an invaluable role in treatment and treatment planning for orthodontic and orthognathic surgery by facilitating cephalometric evaluation, hard and soft tissue assessments, and facial photography superimposition.

Panoramic radiographs

The panoramic radiograph is a common diagnostic tool in today's dental practice. It is a kind of radiograph that provides a full picture of the dentition and the complete maxilla and mandible.

Panoramic radiographs do not show the fine detail captured on intraoral radiographs and are not as specific as other intraoral radiographs, but in a single radiograph it provides a useful general view of all dentition, the maxilla and mandible, the sinuses, and both TMJs. This type of radiograph is very useful, especially during the mixed dentition, for early detection and prevention of all problems disturbing the normal development of occlusion.

Especially during the mixed dentition as a diagnostic tool for early-age orthodontic treatment, the following are important aspects that should be carefully evaluated on a panoramic radiograph before any orthodontic treatment:

- Position and pattern of fully emerged as well as emerging permanent teeth
- Sequence of permanent tooth eruption
- Asymmetric eruption
- Comparison of crown height levels on the left and right sides
- Obstacles preventing eruption
- Abnormal tooth malformations (gemination, fusion, dens in dente, or dilaceration)
- Exfoliation and pattern of primary teeth root resorption
- Tooth number and supernumerary teeth or congenitally missing teeth
- Eruption problems, such as impaction, ectopic, transposition, or ankylosis
- Bone density and trabeculation
- Cysts, odontomas, tumors, and other bone defects or pathologic lesions
- Third and second molar positions, inclinations, and relationships to the first molars and ramus edge
- Shape of the condylar head and ramus height
- Comparison of the left and right condylar heads and rami

The characteristics and management of these problems are discussed in their related chapters in part 2 of this book. Chapter 10 introduces a simple and practical technique for application of panoramic radiographs to assess canine impaction.

Longitudinal Panoramic Radiograph Monitoring

Over many years of teaching and practice, in both pediatric dentistry and orthodontic departments, the author became interested in conducting a retrospective evaluation of patients who were referred for some type of orthodontic problem and who had previous panoramic radiographs available. This retrospective evaluation led to the conclusion that the longitudinal monitoring of panoramic radiographs during the mixed dentition is a very valuable, easy technique that enables detection of developmental anomalies during the transitional dentition. Today the author strongly recommends this easy and very useful technique to all practitioners, especially

pediatric dentists and orthodontists.

The transitional dentition is one of the most critical stages of the dentition, and many eruption problems, whether hereditary or environmental, emerge during this stage. Longitudinal panoramic radiograph monitoring is a careful serial monitoring technique that any practitioner can perform for young patients during transitional dentition to watch for developmental anomalies that may arise at these ages.

The technique the author recommends is to take one panoramic radiograph when the patient is around the age of 6 years (during the eruption of the permanent first molar) and then two more panoramic radiographs at 8 and 10 years of age. Careful comparison of two or three consecutive radiographs of a patient at this stage of the dentition can easily reveal any abnormal developmental processes emerging between radiographs and therefore can enable early detection and intervention. The following three cases illustrate the advantages of longitudinal monitoring of panoramic radiographs and proper intervention.

Case 3-1

This case confirms the importance of longitudinal radiographic evaluation, indicating how early intervention could have helped this little girl. Figures 3-23a to 3-23c are three consecutive radiographs found in her record. A periapical radiograph reveals the first sign of a problem, that is, asymmetric eruption of the central incisors at age 7 years. A panoramic radiograph taken about 15 months later shows the eruption of both central incisors and the asymmetric position of the lateral incisors. A third radiograph, a panoramic radiograph taken about 7 months later, reveals that the left lateral incisor had erupted while the right lateral incisor remained unerupted.

The important, detectable abnormal sign in this radiograph is the abnormal position of the maxillary permanent right canine in relation to the unerupted lateral incisor; unfortunately, no intervention was performed at this point, and the patient did not return until 3 years later. Figures 3-23d and 3-23e present the last panoramic and occlusal views, showing the complete resorption of the permanent lateral incisor root.

Possible intervention:
Assessment of the available serial radiographs indicates that the best treatment option was early intervention and extraction of the maxillary primary right canine when the first (see Fig 3-23b), or even the second (see Fig 3-23c), panoramic radiograph was taken. Extraction of the maxillary primary right canine would have facilitated and accelerated eruption of the permanent lateral incisor, moving this tooth away from the canine forces and preventing root resorption (see Figs 3-23d and 3-23e).

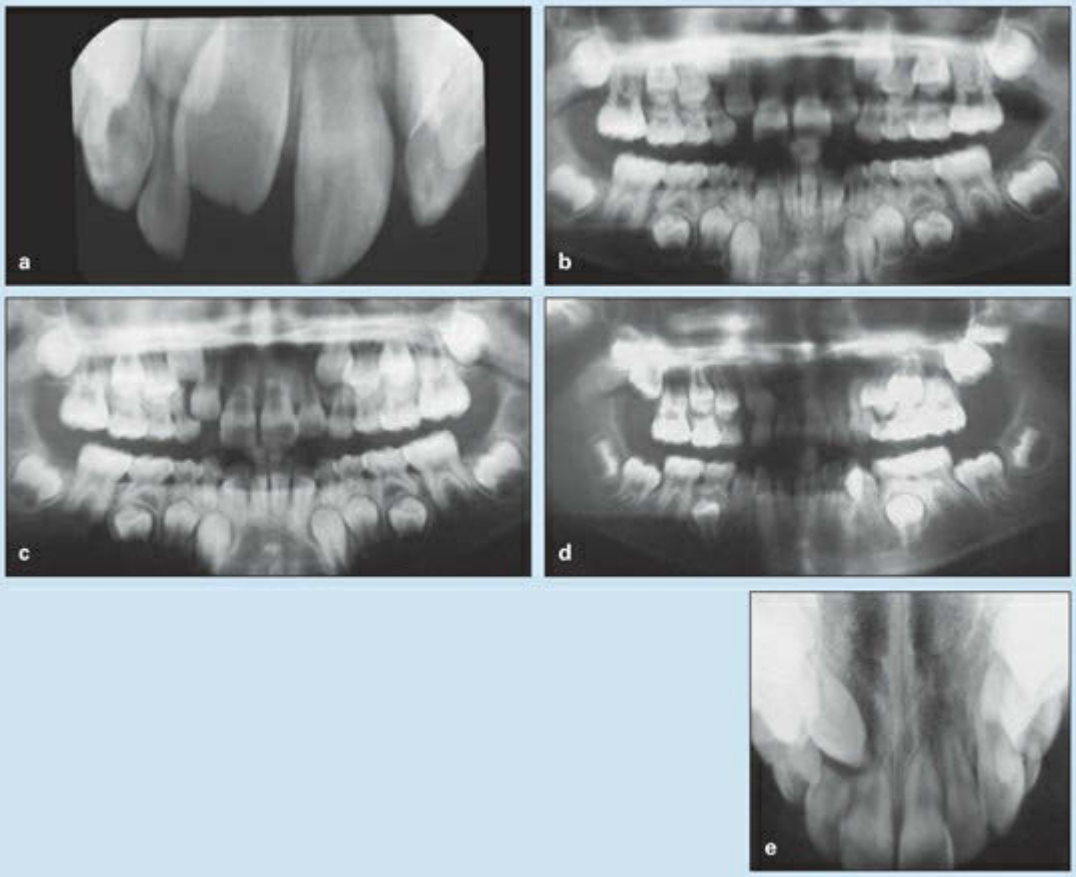


Fig 3-23 *(a)* Periapical radiograph showing asymmetric eruption of the maxillary central incisors. *(b)* Panoramic radiograph taken about 15 months later, showing the eruption of both central incisors and the asymmetric position of the lateral incisors. *(c)* Panoramic radiograph taken 7 months after the first panoramic radiograph, revealing that the right lateral incisor remains unerupted. Panoramic *(d)* and occlusal *(e)* radiographs taken 3 years later. In the absence of treatment, the permanent lateral incisor has undergone complete root resorption.

Case 3-2

A 10-year 5-month old girl exhibited Class II molar relationships, some space deficiency in the maxillary canine area, and two impacted maxillary canines. The maxillary left canine was also ectopic. Figure 3-24a is the first panoramic radiograph, taken by her dentist. Figure 3-24b is the second panoramic radiograph, taken by the same dentist 18 months later, before referral for orthodontic treatment. No treatment had been conducted between radiographs. There was some root resorption of the maxillary left lateral incisor.

Treatment plan:

- Use of maxillary cervical headgear to retract the maxillary molars to Class I molar relationships and to gain space for the canines
- Extraction of the maxillary left primary molar to accelerate eruption of the maxillary left first premolar, which in turn would facilitate eruption of the maxillary left canine
- Placement of a semiremovable lower holding arch and extraction of all mandibular primary molars

to take advantage of leeway space

Result:

Figure 3-24c shows the status during treatment. All mandibular canines and premolars had erupted, the maxillary molars were in a Class I relationship, and sufficient space was available for both canines. The maxillary right canine changed its path of eruption and was erupting spontaneously, but the left canine was still positioned over the apex of the lateral incisor. Therefore, the next step was surgical exposure and traction of the left canine.

Figure 3-24d shows the conditions 1.5 years later, after completion of active treatment. Good results were achieved, but some root resorption of the maxillary left lateral incisor was present. Figure 3-24e, taken another 1.5 years later, without a retainer, shows that lateral root resorption was unchanged.

Discussion:

Figures 3-24f and 3-24g present close-ups of the first and second panoramic radiographs, which were taken at an 18-month interval. Comparison of the two images seems to indicate that retraction of the molars and extraction of the maxillary primary molar, performed immediately after the first radiograph, could have provided sufficient space to change the path of eruption of both canines or that even earlier surgical traction could have prevented more damage to the lateral incisor root.

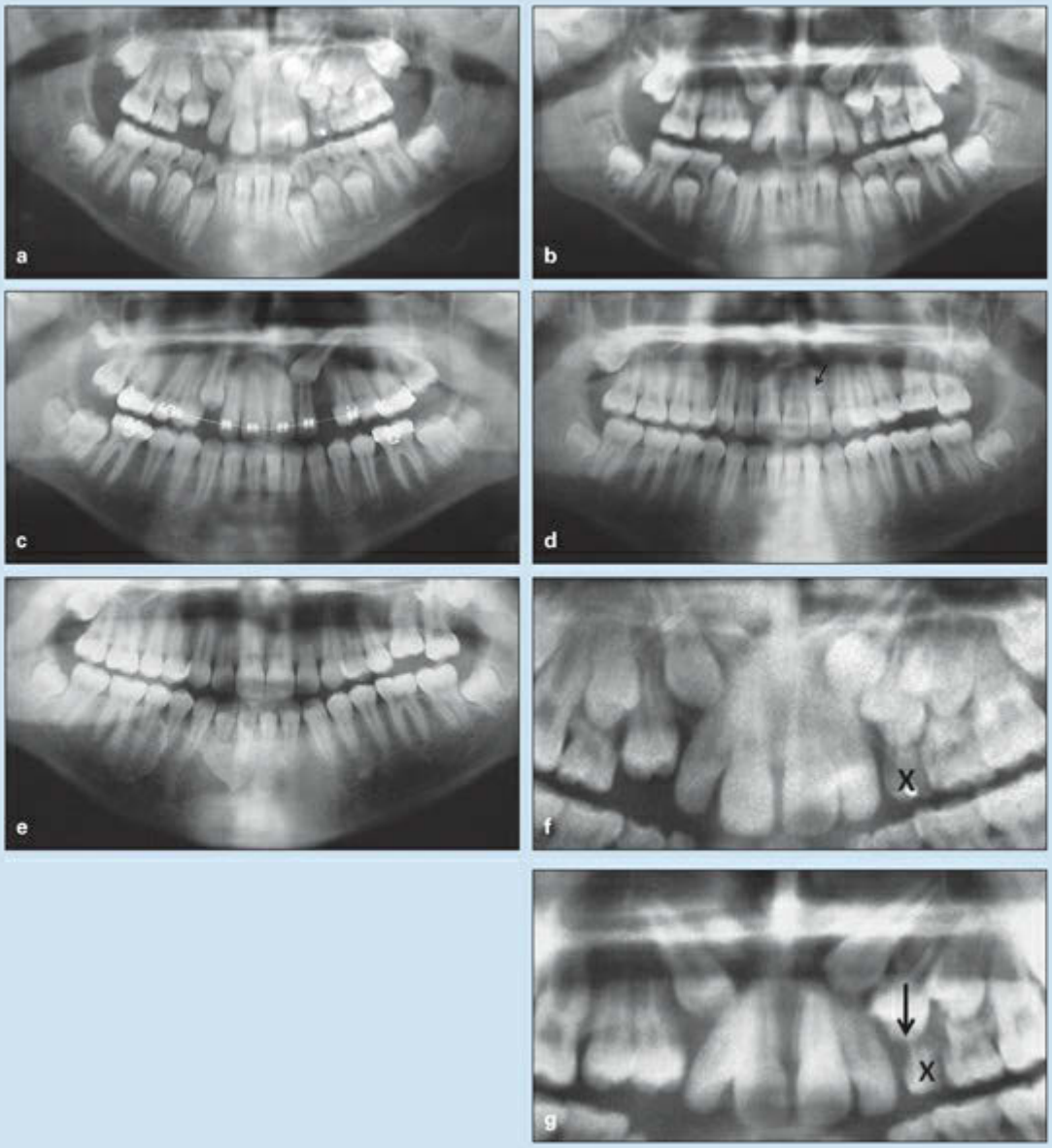


Fig 3-24 *(a)* Initial panoramic radiograph of a 10-year-old girl with a Class II occlusion, space deficiency in the maxillary canine area, impacted maxillary canines, and an ectopic maxillary left canine. *(b)* Panoramic radiograph taken 18 months later, before referral for orthodontic treatment. No treatment has been conducted. The maxillary left lateral incisor shows signs of resorption. *(c)* Panoramic radiograph taken during treatment. After molar retraction, the maxillary right canine has erupted spontaneously, but the left canine will need surgical exposure and orthodontic traction. *(d)* Panoramic radiograph taken after treatment. The maxillary left lateral incisor shows root resorption *(arrow)*. *(e)* Panoramic radiograph taken 18 months later. No retainer has been used. The root resorption of the lateral incisor has not progressed. *(f and g)* Immediate intervention at the time of the initial or second panoramic radiograph by molar distalization and primary teeth extraction could have accelerated eruption of the premolar (marked by an x) and guided left molar

movement *(arrow)*.

Case 3-3

A 12-year-old boy (at the time of referral) had a serial set of panoramic radiographs taken by the dentist, but intervention was neglected (Figs 3-25a to 3-25c). The third radiograph was taken when the patient was referred for orthodontic treatment.

Several problems are visible in these radiographs. As shown in the first radiograph, both maxillary canines were impacted, the maxillary left canine and first premolar were transposed, and the maxillary permanent right lateral incisor was missing. The second radiograph, taken 1 year later, shows the following changes: The maxillary permanent left lateral incisor was erupted, the maxillary left first premolar was erupting, and both maxillary canines were horizontally impacted. The final panoramic radiograph, taken 1 year after the second, shows the following changes: The primary maxillary right lateral incisor and canine roots were resorbing, the maxillary right canine was moving down, and the maxillary left canine was still horizontally impacted.

Discussion:
These three panoramic radiographs indicate the importance of longitudinal radiographic evaluation and reveal the significant changes during transition that can require early detection and proper intervention. The questions are:

1. What caused this significant difference between the right and left canines?
2. Was there any chance of early intervention in this case?

The answers are:

1. The different patterns of eruption for the left and right canines can be attributed to the following: the missing maxillary right lateral incisor and resorption of the primary lateral incisor and canine, the transposition of the maxillary permanent left canine and first premolar, and delayed exfoliation of the primary maxillary left lateral incisor and canine, causing delayed eruption of the maxillary permanent left lateral incisor.

2. Yes. Interceptive treatment was possible.

Possible intervention:
Careful observation of the first panoramic radiograph reveals a problem on the left side—that is, the transposition of the left canine and the first premolar—that was not present on the right side (Fig 3-25d). This situation caused the differences in the eruption patterns of the right and left canines (Fig 3-25e).

The following intervention, if it had been carried out after the first radiograph was taken, could have helped maxillary left canine guidance:

- Early extraction of both primary canines and the primary left lateral incisor to accelerate eruption of the maxillary permanent left lateral incisor and permanent right canine
- Early extraction of the maxillary primary left and right first molars to accelerate eruption of the maxillary left and right first premolars

Accelerating eruption of the maxillary left premolar, an obstacle for the left canine, could have facilitated maxillary left canine eruption.

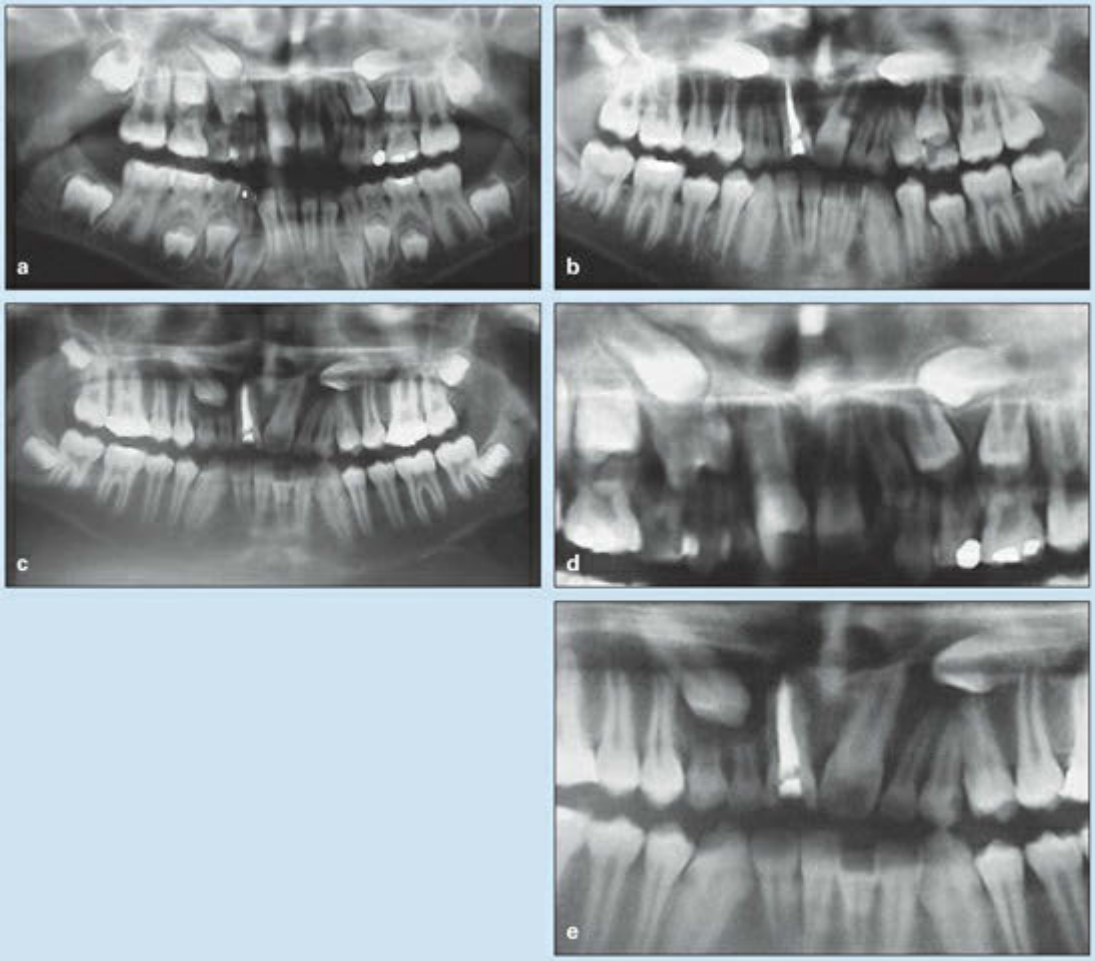


Fig 3-25 *(a)* Initial panoramic radiograph of a 10-year-old boy with impacted maxillary canines, transpositioned maxillary left canine and first premolar, and missing permanent right lateral incisor. *(b)* Panoramic radiograph taken 1 year later. No treatment has been performed. *(c)* Panoramic radiograph taken before orthodontic referral, when the patient was 12 years old. The maxillary right canine is moving down, while the maxillary left canine is still horizontally impacted. *(d and e)* Close-up views of the initial and second panoramic radiographs, revealing the cause of the continued left canine impaction: the transposition of the canine and first premolar. Early intervention at the time of the initial radiograph could have provided left canine guidance.

Cephalometric radiography

Different cephalometric radiography techniques and different analyses have been introduced by numerous investigators over the past 80 years, and the array of techniques used by different practitioners around the world is so extensive that any discussion in this regard would be incomplete. The discussion of this subject in this chapter is intended only to draw the reader's attention to the importance of using cephalometric analysis as a tool in orthodontic diagnosis and treatment planning,

especially for early diagnosis and early treatment.

From the time that this technique was introduced by Broadbent et al[38] in the United States and by Hofrath in Germany to the present, the cephalometric radiograph is the most frequently used radiograph in orthodontic treatment. Cephalometry is a radiographic technique for head measurement. This technique is a further development of the craniometric technique, which was used by anthropologists for direct measurement of the dried skull.

The essence of cephalometric radiography is standardization. The patient's position and the orientation of the x-ray beam are established by mechanical devices in such a way that repeated exposures at other times have essentially the same conditions.

Before 1931 and the introduction of the cephalometric radiograph, orthodontic diagnosis and treatment planning were primarily based on observation of the facial form and dental relationships and a set of plaster study casts. The cephalometric technique became more common because advancements in understanding of the biology and morphology of dentoskeletal anomalies and improvement in cephalometric technology motivated practitioners to use this technique.

Downs[39] analysis, introduced in 1948, was the first analysis based on skeletal and facial proportions in a group of 25 untreated white adolescents with ideal dental occlusions. Comparisons were made only with patients with excellent occlusion and facial proportions.

Now orthodontists are more aware of the significance of the underlying jaw and other bony structures of the skull, the relationships of dental occlusion, and the importance of differentiating these relationships of the teeth to each other, to the jaw base, and to other bony structures of the skull.

The original purpose of cephalometric analysis was to enable research of craniofacial growth changes. Very soon it was recognized that the cast alone cannot identify the type of occlusion and whether the malocclusion is the result of dental or dentoskeletal problems. Two dental casts may seem to show a similar malocclusion, while cephalometric evaluation may reveal that the two malocclusions have a different basis.

Uses of cephalometric radiography

Since the cephalometric radiograph was introduced, the importance of this technique has been widely understood. The following discussion briefly summarizes the

benefits and applications of this radiograph.

Longitudinal growth evaluation. As mentioned earlier, the first purpose of this technique was research on craniofacial growth changes. Before this technique, all information regarding growth and development of the skull was based on anthropometric evaluation of dried skulls or investigation in animals, including vital staining, metallic implantation, surgical approaches, and histologic evaluation.

The application of cephalometric radiographs by Broadbent et al[38] brought this technique to the highest level of attention regarding longitudinal evaluation in children from 3 to 18 years of age.

Growth prediction. Another advantage of the cephalometric technique is prediction of the patient's growth pattern before treatment planning in early orthodontic treatment. All craniofacial bones, including the maxilla and mandible, grow under three mechanisms: increase in size, change in proportion, and change in position. All of these mechanisms are under the control of genetic and environmental factors. Normal occlusal development depends on the correlation and harmony of these structures, which are controlled by growth rate, amount of growth, and direction of growth.

Orthodontic treatment planning in growing patients cannot be completed without predicting the facial growth pattern. Successful prediction requires specification of both the amount and the direction of growth. Cephalometric data and the morphology of the basal bone can identify the growth pattern of each individual growing patient to enable proper timing and sequencing of the treatment plan.

One of important advantages of early orthodontic treatment is the possibility of observing the actual treatment response in growing children during treatment. By carefully monitoring the patient's response at all stages of treatment, the provider can determine appropriate adjustments to the original plan according to variations in response.

Identification of type of malocclusion. Another important role of cephalometry in orthodontics is identification and classification of the patient's type of malocclusion. So many kinds of malocclusions present with different etiologic factors, and different morphologic and structural combinations can be identified and classified by accurate cephalometric measurement and analysis to allow proper treatment planning.

Soft tissue evaluation. Besides the identification of type of malocclusion and

dentoskeletal malrelationships, evaluation of the soft tissue (lips, nose, and chin) relationships to each other and to dentoskeletal structures is another important part of cephalometric evaluation, as well as evaluation of other soft tissues, such as the tongue, soft palate, tonsil, and adenoid tissues.

Reevaluation of orthodontic progress. Reevaluation of patients during treatment is another important application of this technique. Especially in early-age treatment, cephalometric reevaluation of treatment progress and growth changes is highly recommended.

Orthodontic treatment planning. Another important application of cephalometry is planning the treatment tactics that take place after analysis of the dentoskeletal and soft tissue relationships and recognition of the types of anomalies and classification of malocclusion.

Evaluation of treatment results. After the completion of orthodontic treatment, posttreatment reevaluation through superimposition of different dentoskeletal segments for comparison of the treatment results is another important role of the cephalometric evaluation technique.

Clinical application of cephalometric analysis

Since the introduction of the cephalometric radiographic technique to medicine and dentistry, many articles, books, techniques, and analyses have been presented. It is not the intention of this book to recommend one special method or analysis to be used for diagnosis and treatment planning in early orthodontic treatment. The main purpose of this discussion is to remind the reader of some important biologic facts for early-age orthodontic diagnosis and treatment planning:

- Dental occlusion is not a single entity; teeth and alveolar processes are not the only foundations of the functional component of occlusion. Other craniofacial structures that can influence the occlusion through their growth rate and pattern include the cranium, cranial base, nasomaxillary complex, and maxillary and mandibular basal bones.
- The malformation and disharmony of many factors and structures can influence development of the occlusion. Therefore, differential diagnosis and recognition of the involved structures and morphology of the malocclusion is essential. Cephalometric evaluation and analysis can clarify the morphology of

malocclusion and differentiate the structures involved in dentoskeletal abnormalities.

Dentoskeletal components to evaluate by cephalometric analysis

Many different points and analyses can be used in a routine cephalometric approach, depending on the practitioner's desire. From a practical point of view, the elements introduced here are those important components of occlusion that absolutely must be evaluated for early orthodontic treatment. These areas can be classified according to structures and according to measurement data (angles and linear measurement). The main structures that must be evaluated for young patients are:

- The relationships of the incisors and the molars to each other, to basal bone, to the cranial base, and to the profile
- The relationships of the basal bones, maxilla, and mandible to each other and to other craniofacial structures
- The relationships of the cranial base to other structures, including length, and angulation

Over the past 80 years, hundreds of angles and linear measurements have been developed for orthodontic evaluation. Box 3-1 shows the measurements and relationships that are especially important in early orthodontic treatment.

Box 3-1	Cephalometric measurements relevant to early-age orthodontic treatment
Maxillary skeletal measurements • Sella–nasion–point A • Lande angle (nasion–Frankfort horizontal) • N perpendicular • Maxillary length (anatomical) • Maxillary length (functional) ***Mandibular skeletal measurements*** • Facial angle • Sella-nasion–point B • Y-axis • Mandibular plane angle • Gonial angle • Occlusal mandibular angle • Mandibular plane to palatal plane • Mandibular length (anatomical) • Mandibular length (functional)	***Maxillary dental measurements*** • Interincisal angle • Maxillary incisor to palatal plane • Maxillary incisor to Frankfort horizontal • Maxillary incisor to sella–nasion • Maxillary incisor to point A to pogonion (degrees) • Maxillary incisor to point A to pogonion (mm) • Maxillary incisor to nasion–point A (degrees) • Maxillary incisor to nasion–point A (mm) • Maxillary incisor to N perpendicular ***Mandibular dental measurements*** • Interincisal angle

• Mandibular shape ***Soft tissue evaluation*** • Lips: size, position, and proportion to each other, to incisors, and to profile • Tongue: size, position, and proportion • Tonsils and adenoids: size and airway passage • Nose: size, shape, and relationship to profile • Chin: size, shape, and relationship to profile	• Mandibular incisor to Frankfort horizontal • Mandibular incisor to occlusal plane • Mandibular incisor to point A–pogonion (degrees) • Mandibular incisor to point A–pogonion (mm) • Mandibular incisor to N perpendicular

Application of frontal cephalometric radiographs

Frontal (anteroposterior) cephalometric evaluation has several indications in orthodontic treatment, especially in early treatment. Orthodontic diagnosis and treatment planning for young children have some specific considerations that deserve special attention in growing children, including facial asymmetry, maxillary dental and skeletal constriction, and mandibular functional shift. In addition, a lateral cephalometric radiograph describes only two planes of space, whereas patients must be treated in all three planes. Anteroposterior cephalometric radiographs allow evaluation of the transverse dimension. Frontal cephalometric analysis allows evaluation of the following features:

- Facial type (dolichocephalic, mesocephalic, or brachycephalic)
- Facial proportion
- Facial symmetry (dental and skeletal)
- Maxillomandibular basal arch width in crossbites
- Maxillomandibular molar relationships in crossbites
- Occlusal plane disturbances
- Mandibular functional shift
- Unerupted, impacted, or ectopic teeth
- Palatal expansion
- Nasal passage constriction
- Congenital deformities
- Orthognathic surgery needs

Summary

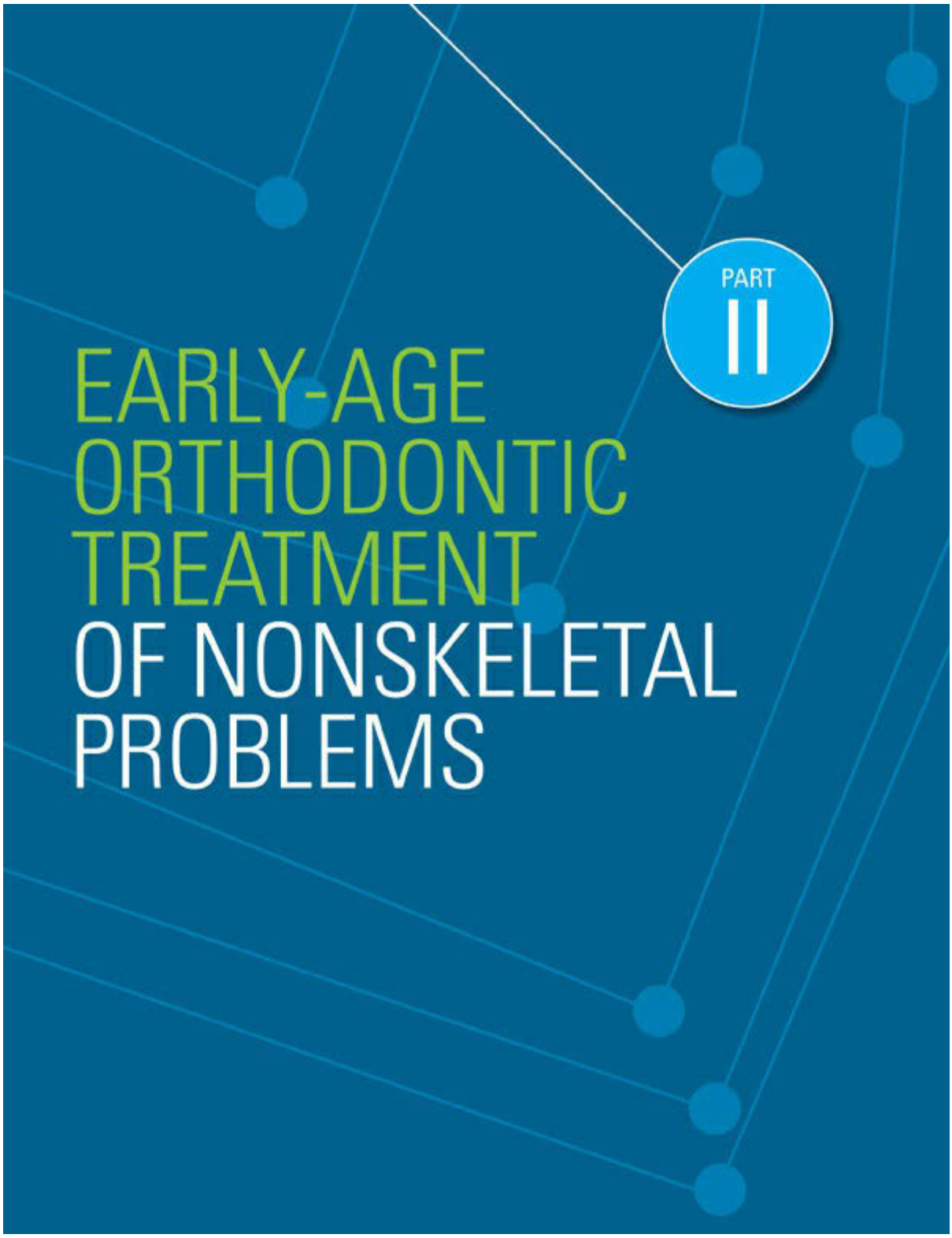
- Diagnosis is the most critical part of orthodontic treatment. The goal of the diagnostic process is to prepare a comprehensive list of the patient's problems in order to synthesize the various treatment options into a rational treatment plan for the patient's best benefit.
- Treatment planning is strategy, the treatment is the tactics, and this entire process has to be organized on a sequential basis: Examine the patient, diagnose the problem, classify the problems, plan the treatment, and then treat the patient.
- A comprehensive orthodontic diagnosis does not merely focus on the relationships of the maxillary and mandibular dentition. It requires a thorough evaluation of the patient's general health and occlusal situation and considers the relationships of the dentition to basal bone and other skeletal, neuromuscular, and soft tissue environments.
- All this information, whether clinical or paraclinical, is derived from three major sources: the questionnaire and interview, clinical evaluation, and paraclinical evaluation (diagnostic record).
- Paraclinical evaluations include diagnostic tools such as study casts, intraoral and extraoral photographs, and intraoral and extraoral radiographs, including cephalometric radiographs.
- Careful collection of data from clinical and paraclinical assessments enables practitioners to detect the type of malocclusion, to clarify the problems list, to classify the problems, and to design a proper treatment plan according to the individual patient's malocclusion and growth pattern.

References

1. Harris EF, Johnson MG. Heritability of craniometric and occlusal variables: A longitudinal sib analysis. Am J Orthod Dentofacial Orthop 1991;99:258–268.
2. Paulsson L. Premature birth—Studies on orthodontic treatment need, craniofacial morphology and function. Swed Dent J Suppl 2009;(199):9–66.
3. Rythén M. Preterm infants—Odontological aspect. Swed Dent J Suppl 2012;(224):1–106.
4. Germane N, Rubenstein L. The effects of forceps delivery on facial growth. Pediatr Dent 1989;11:193–197.
5. Van den Bergh B, Heymans MW, Duvekot F, Forouzanfar T. Treatment and complications of mandibular fractures. J Craniomaxillofac Surg 2012;40:e108–e111.
6. Chrcanovic BR. Open versus closed reduction: Mandibular condylar fractures in children. Oral Maxillofac Surg 2012;16:245–255.
7. Graber LW, Vanarsdall RL Jr, Vig KWL. Orthodontics: Current Principles andTechniques, ed 5. St Louis: Mosby, 2011.
8. Gugino CF, Dus I. Unlocking orthodontic malocclusions: An interplay between form and function. Semin Orthod 1998;4:246–255.

9. Enlow D. Handbook of Facial Growth, ed 2. Philadelphia: Saunders, 1982.
10. Subtelny JD. Early Orthodontic Treatment. Chicago: Quintessence, 2000.
11. Jacobson A, Jacobson RL. Radiographic Cephalometry: From Basics to 3D Imaging. Chicago: Quintessence, 2006.
12. Sarver DM. Esthetic Orthodontics and Orthognathic Surgery. St Louis: Mosby, 1998.
13. Krogman WM. The contributions of Milo Hellman to physical anthropology. Am J Orthod 1947;34:61–82.
14. Proffit WR, Fields HW Jr, Sarver DM. Contemporary Orthodontics, ed 5. St Louis: Mosby, 2012.
15. Moyers RE. Handbook of Orthodontics, ed 4. Chicago: Year Book Medical, 1988.
16. Baume LG. Physiological tooth migration and its significance for the development of the occlusion. 1. The biogenetic course of the deciduous dentition. J Dent Res 1950;29:123–132.
17. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 2. The biogenesis of accessional dentition. J Dent Res 1950;29:331–337.
18. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 3. The biogenesis of the successional dentition. J Dent Res 1950;29:338–348.
19. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 4. The biogenesis of overbite. J Dent Res 1950;29:440–447.
20. American Academy of Periodontology Research, Science and Therapy Committee. Periodontal diseases of children and adolescents. Pediatr Dent 2008–2009;30(7 suppl):240–247.
21. Harvold EP. The role of function in the etiology and treatment of malocclusion. Am J Orthod 1968;54:883–898.
22. Harvold EP, Chierici G, Vargervik K. Experiments on the development of dental malocclusions. Am J Orthod 1972;61:38–44.
23. Ovsenik M. Incorrect orofacial functions until 5 years of age and their association with posterior crossbite. Am J Orthod Dentofacial Orthop 2009;136:375–381.
24. Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. Am J Orthod 1972;62:339–383.
25. Nance HN. The limitations of orthodontic treatment. 1. Mixed dentition diagnosis and treatment. Am J Orthod 1947;33:177–223.
26. Tanaka MM, Johnston LE. The prediction of the size of unerupted canines and premolars in a contemporary orthodontic population. J Am Dent Assoc 1974;88:798–801.
27. Staley RN, Kerber PE. A revision of the Hixon and Old-father mixed-dentition prediction method. Am J Orthod 1980;78:296–302.
28. Bolton WA. The clinical application of a tooth-size analysis. Am J Orthod 1962;48:504–529.
29. Ricketts RM. Planning treatment on the basis of the facial pattern and an estimate of its growth. Angle Orthod 1957;27:14–37.
30. Steiner CC. Cephalometrics for you and me. Am J Orthod 1953;39:729.
31. Merrifield LL. The profile line as an aid in critically evaluating facial esthetics. Am J Orthod 1966;52:804–822.
32. Holdaway RA. A soft-tissue cephalometric analysis and and its use in orthodontic treatment planning. Part I. Am J Orthod 1983;84:1–28.
33. Fishman LS. Chronological versus skeletal age; an evaluation of craniofacial growth. Angle Orthod 1979;49:181–189.
34. Todd TW. Atlas of Skeletal Maturation. St Louis: Mosby, 1937.
35. Fishman LS. Radiographic evaluation of skeletal maturation; A clinically oriented method based on hand-wrist films. Angle Orthod 1982;52:88–112.
36. Greulich WW 2nd, Pyle SI. Radiographic Atlas of Skeletal Development of the Hand and Wrist, ed 2. Stanford, CA: Stanford University Press, 1959.
37. Pike JB. A serial investigation of facial and statural growth in 7 to 12 year old children. Angle Orthod 1968;38:63–73.
38. Broadbent BH Sr, Broadbent BH Jr, Golden WH. Bolton Standards of Dentofacial Developmental Growth.

St Louis: Mosby, 1975.
39. Downs WB. Variations in facial relationships: Their significance in treatment and prognosis. Am J Orthod 1948;34:812–840.



Space Management in the Transitional Dentition

Development of the dentition is a long process that occurs in an orderly fashion under the control of genetic and environmental factors, from the primary dentition through the transitional stage to the permanent dentition, resulting in a functional, esthetic, and stable occlusion. During this long process, many local or systemic factors can influence development of the occlusal system, and any disturbances can prevent the progression to normal occlusion. General practitioners, pediatric dentists, and orthodontists have the highest responsibility to monitor this process carefully during developmental stages and to detect and manage disruptions of normal occlusal guidance.

Every tooth is maintained in its socket over the basal bone in a certain relationship to the dental arch, with adjacent and opposing teeth under a continuous balanced force resulting from the action of different environmental forces. If one of these forces is altered or removed, the relationships with adjacent teeth will change, causing drifting of teeth and initiating space problems. Early loss of primary teeth is one of the most common problems during the transitional stage of dentition, resulting in loss of space.

Fundamentals of Space Management

Before treatment planning and designing any appliance for space management, practitioners must understand the nature of the problem and the fundamental bases governing the biology and mechanism of occlusal development. The following are important processes to consider in this regard:

- Etiology of early primary tooth loss
- Effects of primary molar extraction on succedaneous teeth
- Incidence and nature of space closure
- Prediction of tooth emergence
- Factors influencing time of eruption
- Factors influencing mesial or distal drift

Etiology of early primary tooth loss

Local or systemic factors can cause early loss or early exfoliation of primary teeth:

- Extraction due to extensive caries
- Traumatic injury
- Early exfoliation due to abnormal root resorption (eg, ectopic eruption)
- Systemic disorders or hereditary syndromes, such as hypophosphatasia, rickets, acrodynia, leukemia, juvenile periodontitis, and Papillon-Lefèvre syndrome (see chapter 10)

Effects of primary molar extraction on succedaneous teeth

Fanning[1] studied the lateral jaw radiographs of four boys and four girls who had undergone early and unilateral extraction of primary molars at different ages. The longitudinal study evaluated the tooth development and eruption of the antimere teeth, which were used as controls, and the rate of formation and the speed and time of eruption of the succedaneous teeth. No change was observed in the rate of root formation of the premolars after extraction; however, an immediate spurt occurred in the eruption of the premolar, regardless of its stage of development and the stage at which the primary molar was extracted. Premolar eruption was accelerated in the presence of longstanding necrosis of the primary tooth, especially if accompanied by loss of surrounding bone. Extraction of the primary tooth during active eruption of a developed premolar caused early clinical emergence of the premolar.

Early extraction of the primary tooth, before complete formation of the premolar crown (approximately 4 years of age), resulted in an initial surge, but then the tooth remained stationary and eruption was delayed. Therefore, the question arises

whether it is possible to estimate individual eruption patterns by considering root development and skeletal development in addition to chronologic age. The effects of primary molar extraction on formation and eruption of their successors can be summarized as follows:

- The rate of formation of premolars does not change.
- An immediate surge in eruption occurs, regardless of the dentition stage and the patient's age.
- Eruption is delayed if extraction occurs before half of the root formation is completed.
- Eruption is accelerated if extraction occurs when half of the root is developed.

Posen[2] examined the complete records of 350 children at the ages of 4 and 5 years. Of these children, 62 patients, 34 girls and 28 boys, were selected who had undergone unilateral extraction of primary molars. The accumulated records included plaster casts and annual oblique cephalometric radiographs.

Posen[2] concluded that eruption of premolars was delayed in these children. After the age of 5 years, a gradual decrease in delayed eruption of premolars was noted in these patients, and, at ages 8, 9, and 10 years, eruption of premolars greatly accelerated. This study confirmed delayed eruption of premolars and acceleration at ages 8 to 10 years after early extraction of primary molars.

Incidence and nature of space closure

Premature loss of primary teeth can cause functional disturbances and, because of the possibility of drifting of adjacent teeth, can result in space loss, difficulty in eruption of their successors, and, consequently, occlusal problems. Thus, premature loss of primary teeth needs careful attention during development of the dentition.

The consequences of premature extraction of primary teeth have been the subject of discussion for many years. In 1742, French physician Pierre Fauchard spoke of the inevitability and undesirability of removing primary teeth before their natural shedding.

The consequence of premature loss of primary teeth has been evaluated in several longitudinal and cross-sectional studies. In a review of previous studies, Owen[3] revealed a large controversy in opinions on space management in the past. Some authors, such as Lundström,[4] Linder-Aronson,[5] and Seipel,[6] believe that space

maintainers are usually a waste of time and even are harmful 19% of the time. On the other hand, many researchers believe that premature loss of primary teeth usually results in space closure and causes harmful effects in the permanent dentition.[3,7–10] Owen[3] concluded that the following general trends are evident:

- The incidence of space closure increases with time. For all experiments, at least 96% of premature losses result in some closure within 12 months.
- The highest and earliest incidence of closure happens after premature loss of the maxillary primary second molar.
- Some mandibular extraction spaces show no closure.
- The rate of space closure is higher in the maxilla than in the mandible.
- Rates of closure in the mandible are more varied.
- The greatest amount of space closure in a given unit of time occurs at the maxillary primary second molars and then the mandibular primary second molars.
- There is complete agreement that space closure in the maxilla is predominantly caused by mesial movement of the teeth posterior to the extraction space. In the mandible, space closure is predominantly caused by distal movement of the teeth anterior to the space.
- There is evidence that the tooth mesial to the affected molar can drift distally into the space. Therefore, loss of space or arch length can occur from both directions.[4]
- Reports of space opening are only associated with late extraction.

The rates of the space lost after primary tooth loss differ. Northway et al[10] stated that the space loss was greatest in the first 6 months and occurred in smaller amounts after that, with the least occurring 1 year after tooth loss.

Johnsen[7] evaluated primary first molar loss in 10 white children during the mixed dentition stage (8 had bilateral and 2 unilateral premature primary first molar loss). At the initial visit, alginate impressions, photographs, and radiographs were taken. In all patients, the primary canine was present at the initial impression, and the primary second molars were present at the final impression. For six of the patients extraction was within 1 month of the initial casts; for one it was within 3 months; and for the remaining three it was more than 6 months after the casts were prepared.

Final measurements of the distances between the permanent first molars and permanent incisors were taken on the initial and final casts and compared. Spaces at the extraction site were measured from teeth immediately adjacent to the extraction

site. Johnsen[7] reported no significant mesial movement of the mandibular permanent molars after mandibular primary first molar loss but some space loss at the maxillary molars, especially if the permanent molars were erupting. Space loss in the mandible after premature loss of the primary first molar was the result of distal movement of the anterior teeth, especially in the presence of mandibular anterior crowding.

Northway et al[10] digitized 107 dental casts from University of Montreal growth samples. Longitudinal data were collected annually from the age of 6 years, for an average observation period of 5.9 years. Trying to determine different aspects of early loss of primary teeth, Northway et al[10] reported the following:

- Regarding dimensional loss due to caries or primary tooth loss (the first molar [D], the second molar [E], or both the first and second molars [D + E]), they found that in both sexes significantly greater amounts of space were lost in the mandible, with an average loss of 1.7 mm (ranging from 0.9 to 3.7 mm), than in the maxilla, with an average loss of 1.2 mm (ranging from 0.7 to 3.0 mm).
- The maxillary D loss group experienced a later increase in the amount of maxillary D + E loss.
- In both arches, the greatest loss of space occurred as a result of mesial molar movement; maxillary canine migration was significant only when the maxillary primary molars were lost as early as 9 years of age.
- In the mandible, maximum canine migration occurred after mandibular primary first molar loss.
- More space was lost in the first year following an extraction than in the successive years.
- In the maxilla, the rate of loss was age related (more space loss in younger children); in the mandible, the rate of loss was not age related.
- There was no significant difference in age for space opening during the emergence of the succedaneous teeth in the mandible; the opening occurred during second premolar eruption.
- Permanent molar mesial drift occurred in the maxilla after both E and D + E loss.
- Mesial movement of the mandibular permanent molar occurred only after E loss.
- Maxillary D loss affected maxillary canine relationships, while mandibular D + E loss affected mandibular canine relationships.

Prediction of tooth emergence

The timing of tooth eruption in orthodontic treatment is sometimes a decisive factor, whether in space management or extraction strategy. Unfortunately, individual variation in the age of eruption, especially in teeth emerging through the gingiva, is very high.

Grøn[11] assessed tooth formation and emergence in 874 children (347 boys and 440 girls) by taking radiographs of the emerging tooth and its antimere as well as a left hand-wrist radiograph. Tooth formation was estimated by intraoral radiographs and rated in four stages of root length: one-quarter, one-half, three-quarters, and full root length (with open apex). Findings indicated that tooth emergence is more closely associated with the stage of root formation than chronologic age or skeletal age. The majority of teeth studied had three-quarters of the root complete at the time of clinical emergence; the mandibular central incisors and first molars had less root development, while the mandibular canines and second molars had just past three-quarters' root development at emergence.

Hägg and Taranger[12] studied tooth emergence in 212 randomly selected urban Swedish children who were followed from birth to 18 years of age. The study found that emergence of the primary teeth in boys is less than 1 month ahead of girls until the eruption of the primary second molars. Girls are consistently ahead of boys from eruption of the primary second molars to eruption of the permanent second molars (difference from 3 to 11 months).

Demirjian,[13] studying a genetically homogenous French Canadian group of children 2.5 to 19 years of age, evaluated 5,437 panoramic radiographs and found no difference in the timing of dental development between boys and girls from 5 to 6 years of age. In contrast, among the older children girls were always more developed than boys.

Smith and Buschang[14] examined growth of the mandibular canine and premolar roots in a mixed longitudinal sample of orthodontic patients (77 females and 74 males). They found that as a percentage of tooth length, roots grow with decreasing velocity between 7 and 14 years of age. More complex patterns appear for absolute growth in root length; for example, girls show an earlier maximum growth rate for the canine than for the premolars. Substitution of dental age for chronologic age reduces between-subject variation (assessed at age 11 years), especially for boys. A better understanding of dental maturation, including root length growth, should allow improved prediction models.

Factors influencing time of eruption

To summarize the literature, the following are some guidelines for predicting tooth emergence:

- Tooth emergence appears to be more closely associated with stage of root formation than chronologic age or skeletal age.
- There are variations between individuals.
- There is a much larger variation in the stage of root development at the time of clinical emergence than there is at the time of alveolar emergence.
- Sex differences in the timing of eruption are smallest for mandibular first molars and greatest for mandibular canines.
- The majority of teeth had attained three-quarters of their root length at clinical emergence and one-half at alveolar emergence.
- In normal tooth emergence, the root apices are always open and the root length is never less than one-quarter of normal size.
- Mandibular first molars and central incisors had one-half of their root developed at the time of emergence.
- Mandibular canines and second molars had more than three-quarters' development of their roots.
- Emergence of the primary teeth in boys is around 1 month ahead of that in girls.
- In emergence of the permanent dentition, girls are ahead.
- There is a tendency for earlier tooth eruptions in the mandible.
- Usually there is no significant difference in age of eruption between the right and left sides of the mouth. (The right and left side difference in development is never more than one-quarter of the root). A variation in the emergence between the left and right sides can be considered normal if it persists for less than 6 months. Any asymmetric pattern of eruption that lasts for more than 6 months must be evaluated carefully.
- Most asymmetric eruption occurs in the premolar area.

Factors influencing mesial or distal drift

Principal factors that have the greatest influence on the rate and extent of mesial and distal drift of teeth are the degree of crowding in the dental arch, the type of primary

tooth that is extracted, the type of occlusion, the age of the patient, the presence of abnormal oral musculature, the existence of abnormal oral habits, and the stage of dentition.

Degree of crowding

Degree of crowding in the dental arch is an important factor in space loss following premature loss of primary teeth.

Mandibular incisor crowding and extraction of the mandibular primary canine or primary first molar may cause a quick movement of adjacent teeth, such as midline shift or retraction of the incisors; increase overjet; and cause overeruption of mandibular incisors and impinging bite. On the contrary, in an uncrowded arch, there may be little or no movement of teeth following extraction. The author has observed no space loss in patients with a large mandible (mandibular prognathism). Much less space loss occurs in a primary dentition with interdental spacing (Baume class I) than in a primary dentition with no spacing (Baume class II).

Type of extracted tooth

Space loss and drift of adjacent teeth also depend on the type of primary tooth. For example, premature loss of the primary second molar, whether in the maxilla or mandible, causes mesial shift of the permanent first molar. Early loss of primary first molars can cause mesial drift of the posterior teeth and distal drift of the anterior teeth (in the mandible mostly distal and in the maxilla mostly mesial). Extraction of a primary canine allows permanent incisors to drift distally, but mesial drift of teeth is minimal.

Type of occlusion

Patients with deep bite have a greater tendency for space loss, especially in mandibular dentition, than do patients with open bite. Class II division 1 malocclusion usually becomes more severe after loss of mandibular primary teeth.

Permanent first molar occlusion has some influence on the rate of space loss. For example, maxillary and mandibular permanent first molars with good intercuspation have less tendency for mesial shift than do those with an end-to-end relationship.

Age of the patient

The age of the patient, or the dental age of the patient, plays a strong role in space loss following primary tooth extraction. The earlier a primary tooth is extracted, the greater the amount of drift. Research has shown that the highest rate of space loss happens in the first year after extraction, especially the first 6 months.[15]

If a primary molar is extracted before eruption of the permanent first molar, then the severity of mesial drift and space loss is much greater, even in arches that are not crowded.

Abnormal oral musculature

A strong mentalis muscle and high tongue position can damage occlusion after the loss of a mandibular primary molar or primary canine. In these conditions, collapse of the mandibular dental arch and distal drifting of the anterior segment will result.

Abnormal oral habits

Thumb or finger habits that exert abnormal forces on the dental arch, if accompanied by early loss of primary teeth, can aggravate arch collapse.

Stage of dentition

The stage of dentition and occlusal development can influence the rate and amount of space loss. In general, if the teeth near the space left by the premature loss of primary teeth are in an active stage of eruption, more space loss is likely to occur.

Planning for Space Management

Besides all of the aforementioned treatment planning considerations, such as the incidence and nature of space loss, prediction of tooth emergence, mesial and distal drift of adjacent teeth, and local and systemic factors, including type of occlusion, age of the patient, location of tooth loss, and time elapsed after extraction, the following specific points must be considered in order to determine the appropriate treatment plan, type of management, and type of device to be applied:

- Space availability (determined via space analysis)
- Amount of space loss

- Severity of the problem
- Nature of the problem
- Crowding or spacing in other areas of the arch
- Chronologic and dental ages of the patient
- Stage of occlusal development
- Presence or absence of a permanent successor
- Condition of the permanent successor and eruption potential (delayed or retarded eruption)
- Patient's profile and any skeletal disharmony
- Relation of adjacent and opposing teeth
- Dental and periodontal conditions
- Time elapsed since loss of the primary tooth
- Status of permanent first molar eruption
- State of emergence and type of occlusion of the permanent first molar
- Position of the teeth and stage of eruption in the opposing arch
- Amount of overlying bone
- Sequence of eruption of the permanent teeth
- Presence of any abnormal muscle dysfunction
- Presence of any oral habits
- Patient's occlusion and existing malocclusion
- Prognosis of the proposed management

Planning for any type of space management, like any other orthodontic treatment or tooth movement, requires a series of diagnostic procedures.

Diagnostic procedures

Diagnostic aids such as clinical evaluation, cast analysis, and radiograph and cephalometric evaluation enable the practitioner to reach proper decisions about space management that are linked to individual conditions and variations. After careful evaluation, the decision then will be made whether to maintain, regain, create, or eliminate space for each patient according to his or her dentoskeletal structural composite.

Diagnostic procedures, like any other treatments, require tools for data collection, analysis, and measurements. After the necessary diagnostic aids are collected, the first step in space management is recognition of the available space relative to the space needed for the permanent successor and occlusion.

Space evaluation and size prediction

The space available in the arch can be determined with space analysis. Space analysis is the evaluation of spacing or crowding within the dental arches, achieved through a comparison of the amount of space available and the amount of space required to align the teeth properly. In other words, the purpose of this type of analysis is a comparison of arch length to tooth material. Several types of analysis are available:

- Nance analysis[16]
- Moyers analysis[9]
- Tanaka and Johnston analysis[17]
- Staley and Kerber analysis[18]
- Merrifield analysis[19]
- Bolton analysis[20]

Nance analysis. In a comprehensive study, Nance[16] concluded that the length of the dental arch from the mesial surface of one mandibular permanent first molar to the mesial surface of the opposite side is always shortened during the transition from the mixed to the permanent dentition.

Nance[16] also reported an average leeway of 1.7 mm between the combined mesiodistal widths of the mandibular primary canine and the primary first and second molars and the mesiodistal widths of the corresponding permanent teeth in each side of the arch, with the primary teeth being larger. In the maxillary arch, the difference between the primary and corresponding permanent teeth is 0.9 mm per side.

Nance's method[16] compares a direct measurement from the periapical radiograph of unerupted teeth to available space. Available space is calculated by measuring arch length with a soft brass wire from the mesial surface of one permanent first molar to the mesial surface of the first molar on the other side. A sharp divider or a piece of 0.010-inch brass wire is used to measure the mesiodistal width of

unerupted teeth on complete-mouth periapical radiographs (the tube is aimed directly toward the contact points). For accuracy and to assess for distortion in the film, the width measured on the radiograph is compared with the actual cast width.

Besides leeway space, two other dimensions are measured: the outside measurement, which is the perimeter of the arch from the mesiobuccal surface of one mandibular permanent first molar around to the mesiobuccal surface of the first molar of the opposite side, taken with a brass wire, and the inside measurement, which is taken with a divider from the mesiolingual surface of the mandibular permanent first molar on one side, at the point where the lingual tissues touch the crown of the tooth, to the gingival tissues between the mandibular central incisors on their lingual aspect. Nance[16] indicated that the outside measurements do not increase from the time of the mixed dentition to the time when all permanent teeth are present but decrease during the transition; likewise, the inside measurement also decreases.

Moyers mixed dentition analysis. The mixed dentition analysis advocated by Moyers[9] is a measurement taken from the four mandibular incisors to find the estimated size of maxillary and mandibular canines and premolars from probability tables that he calculated for girls and boys. The mandibular incisors were used because they would provide greater accuracy than maxillary lateral incisors, which have more variation in size.

The purpose of this analysis is to evaluate the amount of space available in the arch for succeeding permanent teeth and necessary occlusal adjustment. This method has some advantages: The analysis can be applied directly in the mouth as well as on casts on both arches; it is not time-consuming; and it requires no special equipment or radiographic projection.

Tanaka and Johnston analysis. The Tanaka and Johnston[17] analysis is a variation of Moyers's method[9] except that the probability chart is not needed. In this technique, the total width of the four mandibular permanent incisors is measured and then divided by 2. The result plus 10.5 mm gives the estimated width of the mandibular permanent canine and premolars and the result plus 11.0 gives the estimated width of the maxillary canine and premolars. According to Dean et al,[8] the estimated width, in millimeters, of unerupted canines and premolars measured with the Tanaka and Johnston method[17] corresponded to the 75% level of probability in Moyers's prediction table.[9]

For example, if the width of the mandibular incisors is 23.0 mm, one would divide by 2 and add 10.5 mm for the mandibular arch. The result is 22.0 mm,

compared with 22.2 mm obtained from Moyers's table. The corresponding values for the maxillary arch are 22.5 mm for the Johnston and Tanaka analysis and 22.6 from Moyers's table. The Tanaka and Johnston analysis provides significant clinical acceptability with a minimal amount of time and effort.

Staley and Kerber analysis. Staley and Kerber[18] introduced a combination method of space analysis in which the size of permanent incisors is measured directly on the dental casts and the size of unerupted premolars is measured on radiographs to predict the mesiodistal width of unerupted canines, which cannot be accurately measured on a radiograph.

Merrifield analysis. Merrifield's "total space analysis" is based on craniofacial analysis and Tweed's triangle together with total dentition analysis.[19] In this analysis, the dentition is divided into three areas: anterior, midarch, and posterior. This division is made for two reasons: *(1)* simplicity in identifying the area of deficient or surplus space and *(2)* the possibility of arriving at a more accurate diagnosis, according to the mandibular incisor position and inclination and the patient's skeletal pattern.

Bolton analysis. The Bolton[20] analysis is another kind of cast analysis used for a comparative evaluation of the mesiodistal widths of the maxillary and mandibular dentition. The greatest mesiodistal dimension of all teeth (except second and third molars) is measured on each cast. The ratio of the 12 mandibular measurements to the 12 maxillary measurements (sum of mandibular 12 divided by sum of maxillary 12, multiplied by 100) is considered the overall ratio. The ratio of the 6 mandibular anterior measurements to the 6 maxillary anterior measurements (sum of mandibular 6 divided by sum of maxillary 6, multiplied by 100) is the anterior ratio. Bolton[20] reported that the mean overall ratio is 91.3 (± 1.91). The mean anterior ratio is 77.2 (± 1.65).

This analysis addresses discrepancies in tooth mass between the maxillary and mandibular arches. Proportional sizes of the dentition are a prerequisite for good occlusion; for example, if teeth are large in one arch and normal or small in the opposing arch, some irregularity in occlusion will occur, and there is no way to achieve an ideal occlusion.

Another example of disproportion is when large maxillary incisors combined with normal mandibular incisors result in increased overjet. Large mandibular incisors and normal maxillary incisors cause mandibular incisor crowding (Fig 4-1). Thus,

these discrepancies deserve attention before treatment planning.

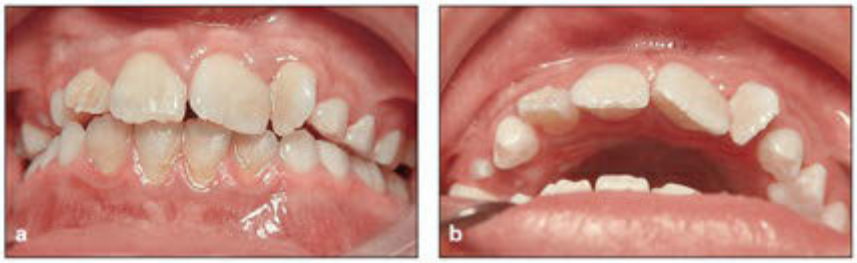


Fig 4-1 *(a and b)* Bolton discrepancy (oversized maxillary incisors).

Other considerations determining space adequacy

Space management is a general strategy of different procedures applied during the primary or mixed dentition to prevent or intercept problems of occlusal development. The purpose of all mixed dentition analysis is to estimate as accurately as possible the amount of space required for permanent canines and premolars before eruption. Careful evaluation is an important step in achieving proper space management during the primary or mixed dentition.

Regardless of the method used in arch length analysis, which is a linear comparison of arch length and tooth size, the following factors also must be considered:

- Inclination of the mandibular incisors
- Patient's profile
- Patient's growth pattern
- Incisor shape and degree of crowding
- Curve of Spee
- Interdental spacing
- General eruption patterns and problems of eruption
- Amount of leeway space

Inclination of mandibular incisors. The anteroposterior position of incisors can affect decisions about space management. If incisors are crowded and already proclined, the space requirement would be greater than the analysis measurement indicates. If the mandibular incisors are retruded because of muscular dysfunction or habits, there is a possibility of bringing the incisors to a more normal inclination and gaining some space. This also must be considered in space calculation.

Nance[16] asserted that overprotruding incisors to gain space is a big mistake; he claimed that "it is a suicide" because it is not stable, it damages alveolar bone support, and it disturbs the patient's profile. Treatment planning for any type of space creation must be based on the position and inclination of incisors and the patient's profile.

Patient's profile. The patient's profile and lip position are another important consideration in space analysis. A lip that is procumbent relative to the chin and nose may require incisor retraction, which needs more space. Likewise, correction of incisor crowding while the profile is convex and the lips are procumbent requires more space than the analysis indicates, and the possibility of extraction would change the results of space measurement.

Patient's growth pattern. The patient's growth pattern is another important factor to be considered. As Björk and Skieller[21] stated, in a child with a well-proportioned face there is little or no tendency for the dentition to be displaced during growth, while in a child with jaw discrepancy the teeth often shift anteriorly or posteriorly. Space analysis would be less accurate in children with problems such as Class II or III malocclusion, long face, or short face).

In addition, the growth pattern informs whether distalization of posterior segments is possible for space regaining or space creation. Distalization can be very critical in vertical growth and advantageous in deep bite conditions.

Incisor shape and degree of crowding. During evaluation of anterior crowding and measurement of the amount of crowding, the amount of incisor overlap must be carefully determined. Generally every overlapped contact will require 1 mm or more of space for correction. Incisors with wide edges have a higher tendency for overlap and more need for stripping and the result is more stable.

Curve of Spee. According to Andrews,[22] the ideal occlusion will have a nearly flat curve; correcting the curve of Spee and bringing the curve to a flat or nearly flat level requires some space, which must also be considered in space analysis.

Study casts are a useful tool in the measurement of the curve of Spee, which can be measured, in millimeters, from the deepest part of the curve to the occlusal plane, which is a straight horizontal plane resting from the tip of the most distobuccal cusp of the last molar to the tip of mandibular canine of the mandibular arch. Correcting the curve of Spee also requires some extra space that must be considered in space analysis.

It has been generally believed that, for each 1 mm of curve, 1 mm of space is required for correction on each side of the arch.

Interdental spacing. The presence of any interdental spacing in the arch must be carefully measured and considered to ensure accuracy of the overall space analysis.

General eruption pattern and problems of eruption. The presence of any obstacle disturbing the eruption process must be considered in management planning. This includes hypodontia and hyperdontia, abnormal sequence of eruption, retarded eruption, abnormal root development, resorption, infection, ankylosis, or any pathologic lesions.

All of these problems can change final decisions about space management, and careful radiographic evaluation of eruption patterns is essential.

Amount of leeway space. Many investigators, including Nance,[16] Moorrees and Chadha,[23] and Ricketts,[24–26] have shown that leeway space and arch circumferences decrease during the transitional dentition as the molars shift mesially. Arch length also decreases through the proximal wear of molars and premolars; Moorrees and Chadha[23] reported that the average arch length of an individual is smaller at 18 years of age than at 3 years of age.

Considering these events, the presence of good leeway space is another important point to be considered in space analysis. Moderate space deficiency of the incisors during the mixed dentition can be solved in some situations in the presence of a good amount of leeway space (see space supervision in the following section).

The best tools for accurate evaluation of all the aforementioned points and completion of accurate space analysis are the clinical evaluation, study casts, radiographic examination, and cephalometric analysis.

Treatment Options for Space Management

After careful space analysis and evaluation of the important points mentioned, and depending on the specific conditions of the patient's problem, the following five options may be considered for space management:

1. Space maintenance
2. Space regaining

3. Space creation
4. Space closure
5. Space supervision

Space Maintenance

Space maintenance is a procedure performed in the primary and mixed dentitions to preserve the available space before any closures occur in the arch length. The use of a space maintainer appliance or restoration of a proximal caries lesion in the primary dentition at the right time can obviate the consequences of loss of arch length and the need for complex orthodontic treatment at a later stage.

Appropriate space maintenance usually is a process to maintain a space when adequate space is available and all unerupted teeth are present and at the proper stage of development. There are also other situations when maintaining the space is indicated even without the presence of a successor.

Indications

- When the available space is sufficient for the permanent successor and there is more than 6 months' time before the permanent successor erupts, space maintenance is recommended.
- When the permanent successor is missing but the patient has a perfect occlusion, space must be maintained to prevent occlusal disturbances and allow for a future implant or fixed partial denture.
- When hypodontia in one arch causes constriction and arch length deficiency and there is crossbite, space must be maintained to preserve arch length for a better arch relationship; an example is when there is a missing maxillary lateral incisor and maxillary deficiency.
- If the space has already decreased some and is not sufficient for the succedaneous tooth but overall arch evaluation indicates adequate arch length, there is no need for space regaining; space maintenance is sufficient.

Prevention of space loss during the primary or mixed dentition is not limited to premature loss of primary teeth; there are other situations that need early intervention, such as the presence of proximal caries and primary molar ankylosis.

Early maintenance is necessary to preserve space for the interproximal contact until proper restoration. Ankylosis of primary teeth, especially when they are below the level of occlusion, can result in tipping of adjacent teeth and space loss as well as overeruption of the opposing tooth. These problems are more destructive when the ankylosed tooth is submerged.

Contraindications

There are situations when space maintenance is not the proper management and other options such as space regaining, space creation, space closure, or other choices must be applied. Examples of these situations include the following:

- When the space is not sufficient for the permanent successor
- When the space is sufficient but the space analysis indicates an overall arch length deficiency and comprehensive orthodontic treatment and extraction is required
- When no permanent successor is present but overall space analysis in the arch indicates a need for space closure
- When the primary dentition is widely spaced
- When space loss is not predicted
- When the succeeding tooth is expected to erupt in less than 6 months
- When the opposing molars are locked into a desirable and stable relationship

Failure to maintain space

Early loss of primary teeth can cause many different problems for the dentition. Two cases can best explain this principle (Figs 4-2 and 4-3). Both patients were at almost the same stage of dentition and similar occlusion, and each had lost one of the maxillary primary lateral incisors. The child shown in Fig 4-2 had lost the maxillary right primary lateral incisor, and the child shown in Fig 4-3 had lost the maxillary left primary lateral incisor. Both patients showed a midline shift to the place of loss; however, a severe diastema was created in the second patient because she had also prematurely lost her left primary canine.

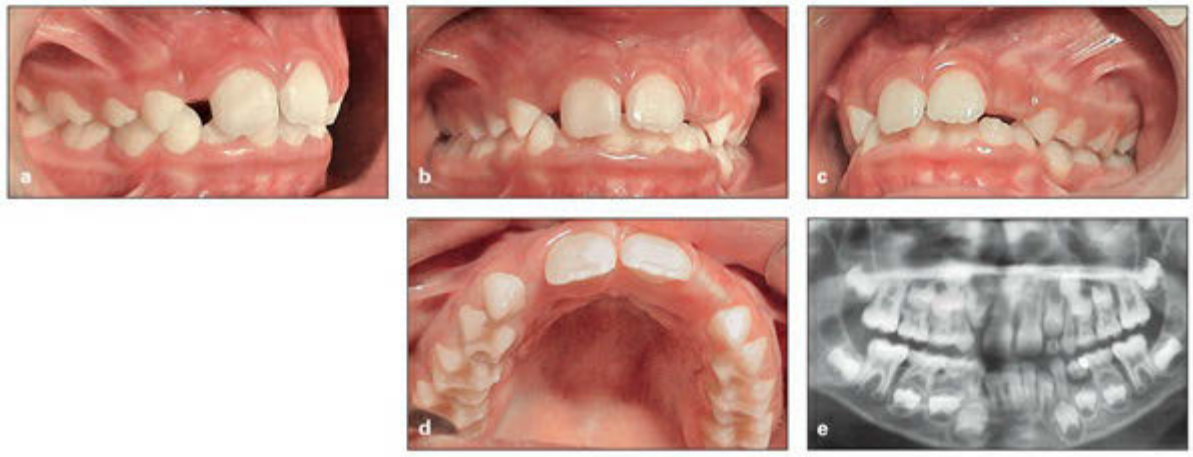


Fig 4-2 *(a to e)* Early loss of the right primary lateral incisor, resulting in a midline shift and space loss.

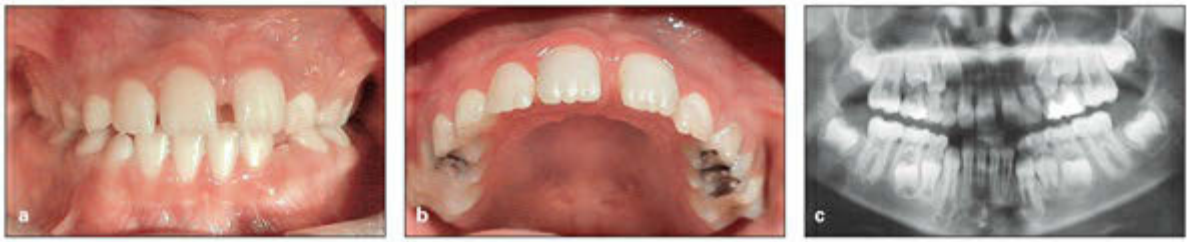


Fig 4-3 *(a to c)* Early loss of both the maxillary left primary lateral incisor and canine, resulting in a diastema, severe midline shift, and interdental space.

Qualities of a good space maintainer

A good space maintainer must have the following qualities:

- Maintain the entire mesiodistal available space created by premature primary tooth loss
- Prevent overeruption of the opposing tooth
- Not irritate or damage oral tissues
- Provide masticatory function
- Improve esthetics in anterior teeth
- Assist speech development of children
- Control abnormal tongue function
- Not inhibit or deflect normal growth changes
- Be strong enough to withstand functional forces
- Not exert excessive stress on opposing teeth

- Permit maintenance of oral hygiene

Types of space maintainer

Several treatment techniques and several different kinds of devices can be used successfully for space maintenance, depending on the specific situation. Space maintaining appliances can be classified:

- *According to structure*. They can be fixed, such as the band and loop or lingual holding arch; removable, such as the Hawley appliance; or semiremovable, such as the Ellis lingual arch.
- *According to design*. They can be unilateral, such as the band and loop, or bilateral, such as the lower holding arch (LHA), transpalatal arch (TPA), or Nance appliance.
- *According to function*. They can be functional, such as the band and bar or crown and bar, or nonfunctional, such as the band and loop, lingual arch, or transpalatal arch.

Nonfunctional fixed unilateral space maintainers

Band and loop. The band and loop is a commonly used unilateral fixed nonfunctional space maintainer (Fig 4-4). The band and loop can be used after unilateral loss of primary molars anywhere in the maxillary or mandibular posterior segments. It is usually applied in cases of unilateral loss of one tooth, but it can be used when two primary teeth are lost; in that case, a rest is created on the occlusal surface of the anterior tooth. This appliance has a number of advantages: It is easy to construct; easily adjustable; noninvasive; inexpensive; and comfortable for patients. In addition, it does not interfere with permanent tooth eruption.

Fig 4-4 Band and loop space maintainer. This is a commonly used unilateral fixed, nonfunctional space maintainer.

The band and loop also has some disadvantages: It cannot prevent overeruption of the opposing tooth, and it does not performing any functions.

Distal shoe. The distal shoe is a unilateral fixed space maintainer that is applied after early loss of primary second molars to guide eruption of the permanent molar. Construction and application of this type of space maintainer require careful evaluation with radiographs prior to insertion and cementation. The length and position of the device and relationship to the unerupted permanent first molar must be carefully evaluated.

Nonfunctional fixed bilateral space maintainers

All of these kinds of maintainers have bilateral construction and play a role in maintaining space on the left and right sides of the arch. The most common forms of this type of space maintainer are the LHA, the Nance appliance, and the TPA.

Lower holding arch. The LHA is the most common type of bilateral fixed maintainer. It consists of two bands or crowns on the permanent first molars (sometimes primary molars) connected with a palatal or lingual bar of 0.032- or 0.036-inch stainless steel wire. In special cases, 0.040-inch stainless steel wire is soldered to the molar bands, touching the mandibular incisors (anterior stop on the cingulum of the mandibular incisors).

The inclusion of two U-loops, mesial to the left and right bands, allows for minor adjustment (Fig 4-5). Adjustment and activation of the loop make it possible to achieve some expansion, torquing, or uprighting of the molars. The LHA can be used as good anchorage in mandibular canine retraction after first premolar extraction.

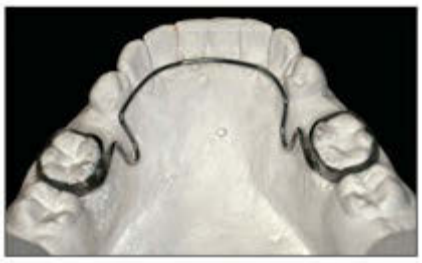


Fig 4-5 Lower holding arch. The inclusion of two U-loops, mesial to the left and right bands, allows minor adjustment.

Another important function of the LHA is prevention of molar eruption, which has many applications in treating vertical problems (see chapter 13). The LHA is also used when there is bilateral posterior tooth loss, when more than two unilateral units

of posterior teeth are missing, or in situations of anterior tooth loss.

When early loss of the primary first molars has occurred in the mandible, soldering of a spur distal to the canine or lateral incisor on each side is recommended to prevent distal shifting of the primary canine and lateral incisor (Fig 4-6). Other modifications of the LHA include the Ellis semiremovable arch and the active LHA.

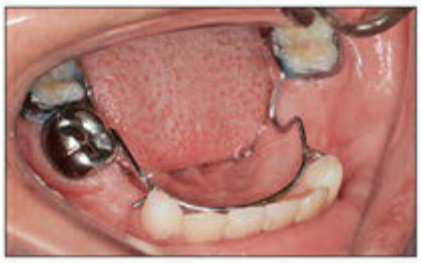


Fig 4-6 Lower holding arch with spurs to prevent distal shifting of the primary canine and lateral incisors.

Note: After prolonged use of an LHA, the stainless steel wire has a tendency to expand gradually and cause molar crossbite. To prevent expansion, heat treatment of the archwire prior to cementation is very important.

Ellis lingual arch. The Ellis lingual arch is a semiremovable holding arch. The lingual bow is not soldered to the bands. Instead, the arch has a post to be inserted to a prewelded lingual tube on the molar bands. The advantage of this holding arch is the ability to easily remove the arch without removing the band for any activation and readjustment (Fig 4-7).

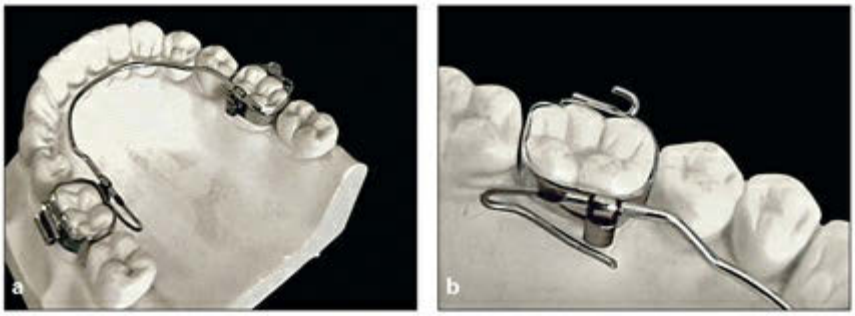


Fig 4-7 *(a and b)* Ellis semiremovable lingual arch. The advantage of this holding arch is its easy removal for activation and readjustment.

Active holding arch. Attachment of two light wire springs to the lingual arch can make the LHA an active appliance that can be applied for minor uprighting, alignment of incisors, and correction of moderate incisor crowding (Fig 4-8).

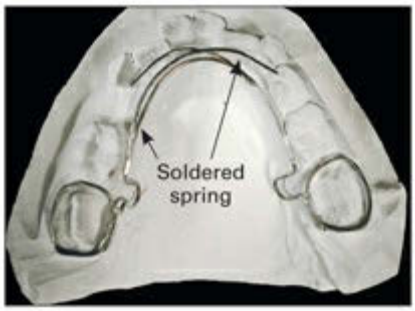


Fig 4-8 Active lower holding arch. The light wire springs allow minor uprighting, alignment of incisors, or correction of moderate incisor crowding.

Transpalatal arch. The TPA is a bilateral holding arch used in the maxilla. It can be fixed or semiremovable. It consists of two bands for the maxillary molars and a palatal bar made of 0.036-inch stainless steel wire that is soldered to the molar bands (Fig 4-9). It can also be semiremovable like the mandibular Ellis arch with a horizontal tube welded to the molar bands.

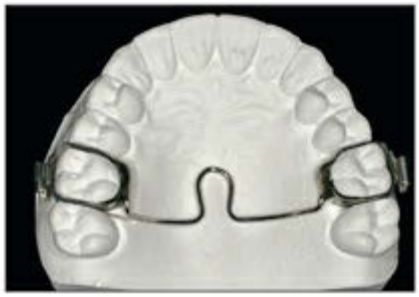


Fig 4-9 Simple transpalatal arch.

Both fixed and removable arches can be made with or without omega loops. Omega loops allow several adjustments, such as molar expansion, molar rotation, and molar torquing.

There are two kinds of TPA: the regular and the reverse TPA, which has an extra U-loop before the molar bands and gives the appliance more flexibility for adjustment (Fig 4-10).

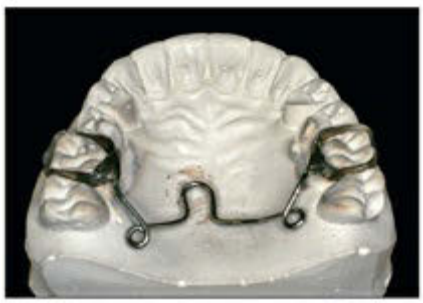


Fig 4-10 Reverse transpalatal arch with extra loops for molar movement.

The TPA can be used as a holding arch to maintain space in the maxillary dentition following multiple primary tooth loss. The lack of acrylic resin button (like that found in the Nance appliance) makes the TPA less tissue irritating and more cleansable, and the TPA is especially recommended for young patients.

The TPA is a bilateral fixed maintainer for the maxilla, but because of the lack of anterior stop (acrylic resin button) it is not as strong an anchorage device as the Nance appliance.

With adjustment, it has the potential to provide expansion. A TPA with omega loops can rotate the permanent molars and gain space. The addition of extra coil to the TPA wire results in more flexibility and the possibility of more molar movement (see Fig 4-10). All of these advantages can be achieved more effectively with a reverse TPA.

Another important indication for the TPA is the ability to prevent molar extrusion in children who have lost a mandibular permanent molar.

With some modification, the TPA has the ability to intrude molars (see chapter 13).

Nance holding arch. The Nance holding arch is also a bilateral holding arch applied in the maxillary arch (Fig 4-11a). It is constructed of two molar bands and a palatal bar of 0.036- to 0.040-inch stainless steel wire soldered to the molar bands. The anterior part of the bar is located in the deepest part of the palate. The bar should be separated from the tissue with acrylic resin material, and a V-bend is created in the wire for acrylic resin retention.

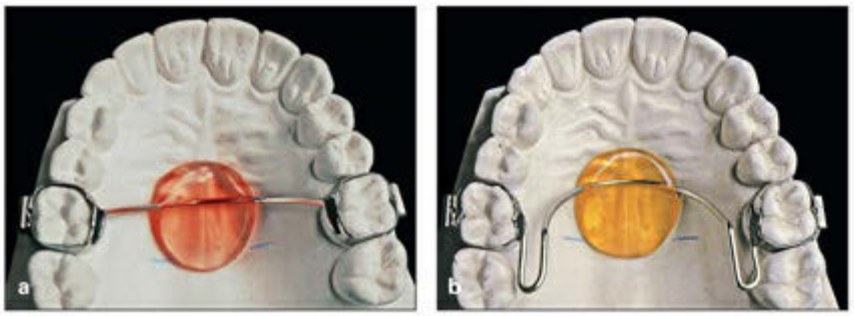


Fig 4-11 *(a)* Nance holding arch. *(b)* Reverse Nance holding arch. The extra loops allow for expansion and rotation of the molars.

There are two kinds of Nance appliance: the regular Nance and the reverse Nance. The reverse Nance has an extra U-loop before each joint soldering the wire to the bands. Besides holding the molars, the reverse Nance can serve as an active appliance for expansion and rotation of the molars (Fig 4-11b).

The Nance appliance is a good space maintainer in the maxillary arch to prevent mesial molar movement in cases of multiple primary tooth loss.

The acrylic resin button provides an additional stop, and it plays an important role in providing strong anchorage in extraction cases.

Early loss of the maxillary primary second molar usually causes mesial migration of the molars and rotation around the palatal root. Both the Nance holding arch and the TPA can prevent these events. However, because of hygiene problems and the possibility of irritation, the Nance appliance is not recommended for a long period of time in early-age treatment, unlike the TPA.

Functional fixed space maintainers

There are many types of fixed or removable, unilateral or bilateral functional appliances that can maintain space and prevent overeruption of opposing teeth. These appliances can play a role in functions such as mastication, speech, and esthetics. The following are some of the different types of fixed functional space maintainers:

- Band and occlusal bar
- Band and pontic
- Crown and bar
- Crown and pontic
- Anterior provisional fixed partial denture

Posterior unilateral functional maintainers have the same indications as band and loop appliances, along with some extra indications.

Band and occlusal bar. This two-band space maintainer with an occlusal bar is a unilateral fixed space maintainer similar to the band and loop appliance. It is designed to maintain space after primary tooth loss; it can also prevent supereruption of the opposing teeth, and it allows patients to easily maintain their oral hygiene. This maintainer cannot be used if the adjacent molars are tipped, unless both sides are uprighted (Fig 4-12a).

Band and pontic. A functional space maintainer with an occlusal pad or tooth, this appliance has the same function as the band and bar appliance but is more esthetically pleasing, provides better masticatory function, and allows better oral

hygiene (Fig 4-12b).

Fig 4-12 *(a)* Band and bar functional space maintainer. *(b)* Band and pontic functional space maintainer, which has the same function as the band and bar but is more esthetically pleasing, provides better masticatory function, and allows better oral hygiene. (Courtesy of Great Lakes Orthodontics.)

Crown and bar or crown and pontic. These maintainers are also unilateral functional space maintainers. They can play the same role as band and bar or band and pontic appliances, but because of the crown they are stronger. The best indication for a crown and bar or crown and pontic maintainer is for patients with deep caries and weak crowns.

Anterior provisional partial denture. Anterior tooth loss occurs primarily as a result of trauma and secondarily as a result of caries. Although the prevalence of dental caries appears to be declining, children suffering from nursing bottle caries and rampant caries exhibit early tooth loss in the anterior and posterior segments. Early loss of anterior teeth causes drift and space problems for occlusal development and can cause functional disturbances in speech and chewing as well as some psychologic issues in young children.

Early intervention in cases of missing primary incisors may be necessary for several reasons, namely space maintenance, function, speech, and esthetics.

Space maintenance. Some believe that early loss of primary incisors does not always result in drifting of the adjacent teeth.[4–6] This appears to be true in some clinical situations with adequate interdental spacing. Nevertheless, patients with severe space deficiency have a higher tendency for drifting; this is more evident in the presence of mandibular incisor crowding and gets even more complicated in deep bite cases.

Preservation of function. The child's nutrition, especially at an early age, is an important factor to be considered in early loss of primary incisors. Improving

masticatory function is another reason for replacing missing primary incisors.

Prevention of speech problems. Some investigators contend that the altered speech development following early incisor loss is a justification for replacing missing maxillary incisors. Many sounds are made by touching the tongue to the lingual side of the maxillary incisors (so-called linguodental sounds), and early loss of incisors can cause inappropriate speech compensations, especially at ages when speech development is critical. This defect can produce lisping.

Esthetics. Esthetics is also an important factor to be considered, especially when it is a matter of concern for the child or parents. A fixed lingual arch or a removable partial denture with attached primary teeth can serve as a prosthetic replacement. These types of appliances must be monitored, adjusted, and possibly replaced over a longer period of time.

Prosthetic maintainers can be fixed or removable. Appliances designed for the incisor region have special specifications. The fixed type, also called a *pedo temporary bridge*, is formed by two bands on the permanent first molar and a strong lingual archwire soldered to molar bands. Each anterior tooth (up to four teeth) is fitted to the wire or a spur soldered to the base arch for more strength (Fig 4-13).

Fig 4-13 Pediatric anterior fixed partial denture (pedo temporary bridge). (Courtesy of Summit Co.)

When more than one pontic is included in the prosthesis, acrylic resin material is also applied to the lingual surfaces of the teeth to bond them into a single unit. This type of anterior maintainer can provide very pleasant esthetics but requires extra care because it is vulnerable to distortion and breakage.

The Groper fixed anterior prosthesis is a pediatric fixed prosthesis that is attached to each tooth separately by a specially designed, stainless steel pad. Each unit is then welded and soldered to the archwire. Placement of a rest seat on each side gives more strength to the appliance.

Early loss of primary incisors in the mandible may not affect esthetics or function as much as their loss in the maxilla, but the possibility of space loss and midline shift is higher, especially in patients with crowding. Another consequence of early loss in the mandible is the possibility of retroclination and overeruption of the permanent incisors (increasing overjet and overbite). Early intervention to prevent the mandibular anterior teeth from moving lingually or mesiodistally is recommended. Placement of a lingual arch after the permanent incisors erupt may be appropriate to prevent the midline shift and lingual movement.

Figure 4-14 shows a functional removable partial denture that can be used to maintain space, function, and esthetics.

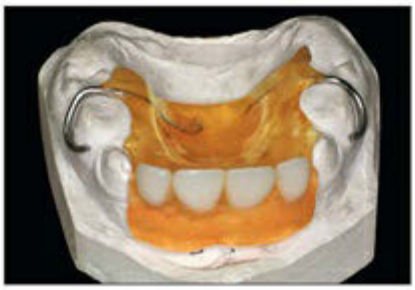


Fig 4-14 Removable functional space maintainer.

Removable space maintainers

Removable appliances also can be applied for different kinds of space management. These appliances are typically used when more than one tooth has been lost in a quadrant. If abutment teeth and a distal cantilever design would be visible, or the band and loop appliance is too weak to withstand occlusal forces over a two-tooth span, the removable appliance is the only alternative appliance. Furthermore, inclusion of pontics in the appliance can help occlusal function.

Removable maintainers can be simple maintainers, unilateral or bilateral, for the posterior or anterior segment, and functional or nonfunctional. Removable maintainers have several advantages:

- They are easier to fabricate.
- They are easier to remove, allowing patients to maintain oral hygiene.
- They can be used in cases of multiple tooth loss, bilaterally (anteriorly and/or posteriorly).
- They can be used in the absence of abutment teeth.
- They can be designed in such a way as to have multiple functions at the same time,

for example, as a maintainer and retainer, for control of habits, as an active plate for different purposes such as a bite plate, or as an expander.

Removable appliances also have some disadvantages that must be considered during treatment planning and appliance design:

- They require the patient's compliance.
- They are more breakable.
- When several teeth have been lost, retention is a problem.
- They are easily lost by children.
- If cleaning is not done properly, caries, tissue irritation, and hyperplasia are inevitable.

Hawley appliance. Hawley appliances can be used in the maxillary and mandibular arches to maintain space in cases of multiple primary tooth loss (Fig 4-15). Hawley appliances can fulfill several functions during the mixed dentition, serving as both a simple maintainer and a functional device to preserve esthetics and prevent overeruption of opposing teeth in the posterior segment.

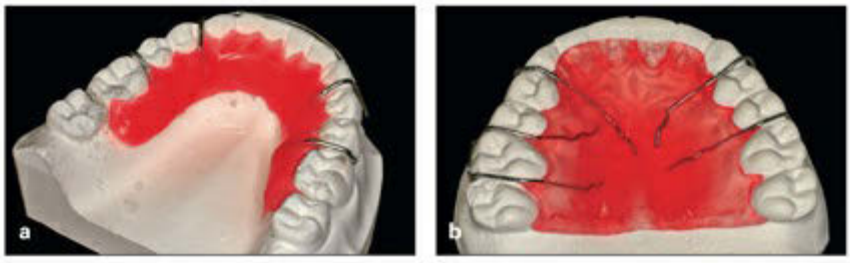


Fig 4-15 *(a)* Mandibular Hawley appliance. *(b)* Maxillary Hawley appliance.

Figure 4-16 shows a Hawley appliance with primary incisors embedded in acrylic resin as a flipper, or provisional partial denture. The regular Hawley labial bow provides good retention. This kind of construction can be used to maintain space and replace maxillary and mandibular incisors lost after an accident.

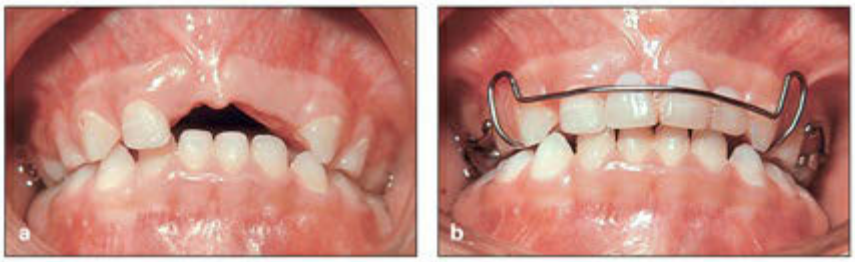


Fig 4-16 *(a and b)* Hawley appliances fabricated with three replacement primary incisors to serve as a functional space maintainer.

For all kinds of pediatric fixed prostheses and partial dentures, fabrication of an opposing cast and completion of a bite registration are necessary tools for proper construction.

Space Regaining

Space regaining is a procedure to regain the lost space or improve the eruptive position of the permanent succedaneous teeth; it is an important treatment modality in the management of developing occlusions. Space regaining actually represents a tooth movement, and, like any other active procedure in orthodontic treatment, requires consideration of numerous factors during treatment planning and a special mechanical design. There are sometimes serious limitations in tooth movement in certain directions that cannot be recognized except by clinical observation. For example, tight contact between the permanent first and second molars usually precludes repositioning and distalization of the first molar.

Lack of proper anchorage control in some space-regaining appliances may have an adverse effect on other parts of the dentition, such as distalization of molars without proper anchorage preparation that can cause incisor flaring.

The timing of space regaining is another condition that must be considered before the procedure is started. To determine the time, it is necessary to distinguish if the space loss is stable or progressive. If it is progressing, immediate action is needed after all diagnostic and pretreatment assessments are completed. If the present space is static, such as in cases where the mandibular first molar is tipped and the extrusion of the maxillary primary second molar serves as a stop against the mesial surface of the mandibular permanent first molar, early regaining may not be practical in a 7- or 8-year-old child because after space regaining the space would have to be maintained for a long time.

After the necessary space is regained, it should be maintained until the adjacent permanent tooth is completely erupted.

Success in regaining the necessary space depends on recognition of all relevant factors, such as the severity of the problem, position of the adjacent teeth, and the general situation of the occlusion; pretreatment assessment cannot be focused only on the dental segment containing the lost space. There are limitations and problems that must be considered before space opening is attempted:

- Tight contact between first and second molars

- Crowding in the molar area
- Possible anterior tooth flaring
- Timing and whether the space loss is static or progressive
- Nature of the crowding (acquired or hereditary)
- Patient's profile

The position of the second molars in relation to the first molar and crowding of all molars in general are important factors to consider in retraction or uprighting of the permanent first molars.

Another factor that must be considered in all types of space regaining is the phenomenon of action and reaction. When a space is opened or a tooth or a segment of dentition is moved, there might be negative effects on other areas of dentition, such as flaring of the incisors. Thus, incisor position and the patient's profile are important factors to consider in space regaining.

Indications

Space regaining is indicated in the following situations:

- When space is decreasing and there is not enough space for a permanent successor
- When no permanent successor is present but maintenance of good occlusion requires space opening and uprighting of adjacent teeth

Note: Space loss in the molar area, especially the maxillary molar area, is sometimes the result of molar rotation and mesial tipping; therefore, correction of the rotation will create some space too.

Contraindications

There are situations where space opening or space regaining is not the proper option for space management:

- When space analysis indicates an overall space deficiency and extraction of other areas of the dentition is needed
- When space opening is difficult and the results of space creation will not be stable

In these situations, the proper option might be premolar extraction.

Types of space regainer

Appliances used for space regaining can be classified into three types: fixed, removable, and semiremovable. Any of these can be unilateral or bilateral.

Fixed unilateral and bilateral regainers

Sliding loop regainer. The sliding loop is a good, effective appliance for posterior segments. It is designed with one band on the permanent molar and a loop, similar to the space maintainer, but the loop is not soldered to the band. Rather, two 0.036-inch buccal tubes are welded to the molar band. The loop is made with 0.036-inch stainless steel wire (Fig 4-17).

Fig 4-17 Fixed unilateral sliding loop space regainer.

Space is regained by activating the sliding loop inside the molar tubes and inserting push coil springs larger than the available space.

This regainer is especially recommended in cases where both the first molar and first premolar have tipped into the primary second molar space. The setup applies a constant force to move the first premolar mesially and, with some reciprocal distal movement, move the permanent molar distally.

Gurin lock regainer. The Gurin appliance is also a unilateral fixed space regainer. It has the same indications as the sliding loop and less chance of premolar rotation. It consists of bands on the first premolar and molar and a sliding bar soldered to the premolar band. The bar slides into a buccal tube on the molar. A piece of nickel-titanium coil spring is placed between the tube and a Gurin lock, which can be readjusted for coil activation on each visit (Fig 4-18).

Fig 4-18 Gurin lock space regainer.

Band and U-loop. The band and U-loop is another type of fixed unilateral expander. It is designed with two bands and a U-shaped wire that is soldered to both bands. It can be activated by slight opening of the loop to upright the adjacent teeth (Fig 4-19).

Fig 4-19 Band and U-loop space regainer. (Courtesy of Great Lakes Orthodontics.)

All of the aforementioned regainers (sliding loop, Gurin lock, and band and U-loop) are used when it is desirable to deliver force to both sides of the space and upright both abutment teeth. When only distalization of the molar, and not force to the anterior abutment, is required, anchorage must be included in the design of the appliance.

Anchored regainers (distalizer). These types of regainers are designed in such a way to deliver force only to the posterior tooth and to prevent force to the anterior abutment.

Molar distalizer with Nance anchorage. This is an anchored, fixed space regainer used in maxillary molar distalization. It prevents application of force to the anterior teeth through a palatal acrylic resin button (similar to that in the Nance holding arch), thereby facilitating molar distalization without having an adverse effect on the anterior component (Fig 4-20).

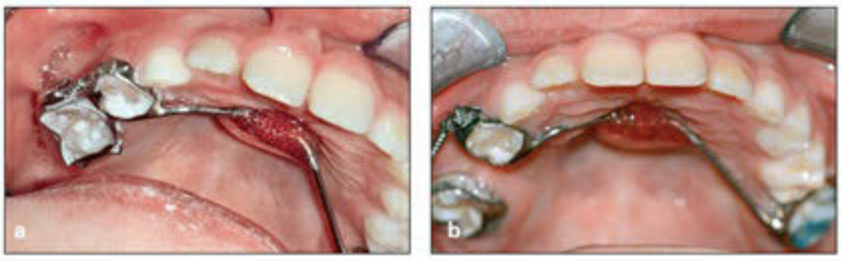


Fig 4-20 Molar distalizer with Nance anchorage. *(a)* Space loss at the time of appliance placement. *(b)* Space regained at the end of treatment.

This type of unilateral regainer is recommended in cases where the force is to be directed only to the molar in the maxillary dentition.

Sliding loop and lingual arch. This appliance is designed similarly to the sliding loop regainer, but it includes a lingual holding arch connected to the opposite molar band to provide anchorage and prevent adverse effects on the anterior component (Fig 4-21).

Fig 4-21 Mandibular molar distalizer. (Courtesy of Great Lakes Orthodontics.)

Pendulum appliance (molar distalizer). The pendulum appliance is a fixed bilateral or unilateral molar distalizer. It is designed with two bands cemented to the primary first molars or the premolars and an acrylic resin button touching the palate to provide good anchorage. One end of a β-titanium spring is embedded in acrylic and the other end is inserted in the palatal tube, making the spring removable (Fig 4-22). The appliance can be activated at each appointment. This type of distalizer is indicated for the permanent dentition, in cases of space loss or Class II molar correction.

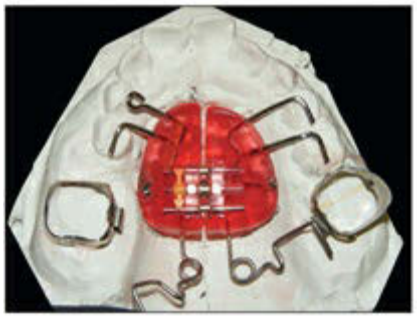


Fig 4-22 Pendulum distalizer with spring activation on the right molar. The distalizer in this image also includes a screw for expansion.

Distal jet appliance. The distal jet appliance is also a fixed unilateral or bilateral distalizer with an acrylic resin button for anchorage. Bands are cemented to the anterior abutment, and two bars with open coil spring slide to embedded tubes for activation. The bars connected to the molar palatal tube can be removed, and the push coil can be reactivated (Fig 4-23).

Fig 4-23 Distal jet appliance for molar distalization. (Courtesy of Great Lakes Orthodontics.)

2 × 4 bonding. Molar distalization and space regaining can be achieved as a part of 2 × 4 bonding in patients who need incisor alignment (such as space closure, crossbite correction, or midline shift) during the early or middle mixed dentition. A light force can be applied to molars by a push coil inserted between lased incisors and the permanent molar tube (Fig 4-24).

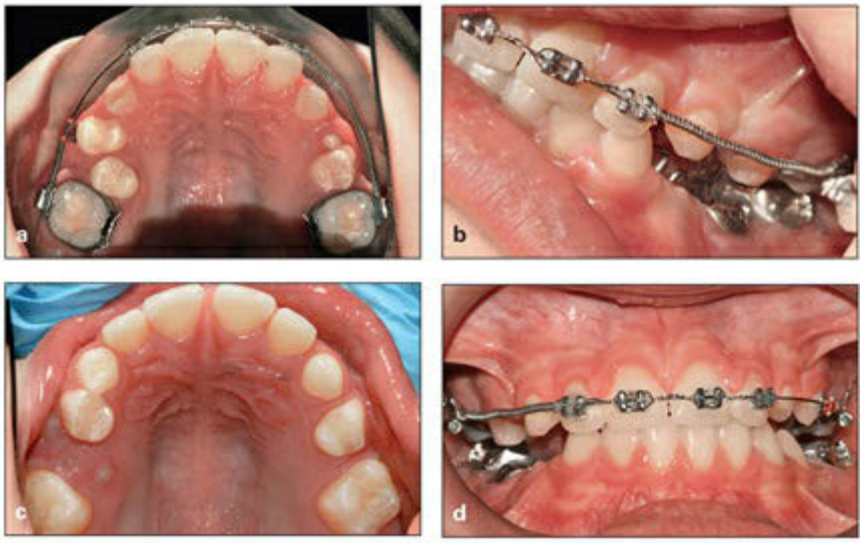


Fig 4-24 *(a to d)* Push coil and 2 × 4 bonding to regain space for the maxillary second premolars.

Sectional bracketing. In patients with normal occlusion and space loss in one quadrant, minor tooth movement and space regaining can be achieved by sectional bracketing. Figure 4-25 shows a patient with a good Class I mandibular and maxillary left dentition. The problem is space loss at the maxillary right second premolar site that has resulted from mesial tipping of the molar and distal tipping of the first premolar. Sectional bracketing of this segment, leveling with a sectional archwire, and placement of a push coil between the tipped molar and premolar can open space and upright the adjacent teeth.

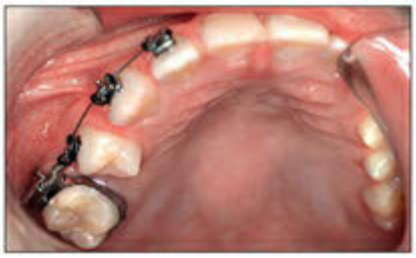


Fig 4-25 Sectional bracketing to open space for the maxillary right premolar.

Removable space regainers

Removable appliances can also be used for space regaining as well as space maintenance. This can be accomplished by incorporating different springs or screws in the appliance, either unilaterally or bilaterally. A Hawley appliance with different modifications is a simple, effective appliance that can be used for all of these purposes (Fig 4-26).

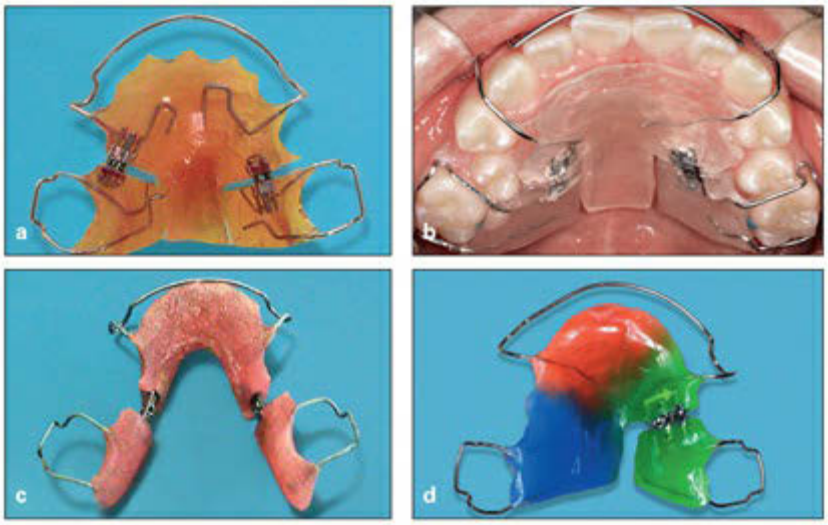


Fig 4-26 Hawley removable space regainers with jackscrews. *(a and b)* Bilateral removable regainers for the maxilla. *(c)* Bilateral removable regainer for the mandible. *(d)* Unilateral removable regainer for the maxilla.

The two main advantages of removable regainer appliances are that they facilitate cleansing and maintenance of good oral hygiene and they allow patients to open the screw outside the mouth. Another advantage is that these appliances can be designed to have multiple actions, such as concomitant space maintenance, space regaining, and tooth movement.

The major disadvantage of removable space regainers, like all other removable appliances, is the need for patient compliance in wearing of the appliance. Breakage and loss of the appliance are other disadvantages.

Space Creation

Space creation is another type of space management that can be performed in cases of moderate space deficiency. This indication is different from cases in which the patient has experienced space loss and requires space regaining. In situations that require space creation, the patient exhibits moderate crowding in the arch, and space analysis indicates that there are some overall space deficiencies.

Space creation is a tactic that can be applied in certain cases, especially during the mixed dentition as an early intervention procedure. Generally, creation of space is much more complex than space maintenance and regaining, and it involves more sophisticated application of biomechanics and appliance therapy. The patient's age and growth potential are important factors that must be considered in treatment planning for space creation, as in any other early treatment.

Space creation can be achieved in the following ways:

- Tooth extraction
- Sequential selective enamel stripping
- Sagittal expansion
- Transverse expansion
- Combination of the aforementioned methods

Tooth extraction

Tooth extraction is a general strategy applied in cases of severe crowding that need more than 7 to 8 mm of space, where extraction combined with comprehensive orthodontic treatment is the only option (for more details, see chapter 5).

Sequential selective enamel stripping

In some cases of moderate crowding, a tooth size discrepancy in a segment of dental arch may be the cause of crowding while the rest of the dentition shows normal relationships. An example is mandibular incisor crowding resulting from oversized mandibular incisors (Bolton discrepancy).

The best treatment option in these situations is interproximal stripping to alleviate the crowding.

Removal of minor amounts of hard tissue with a disk does not cause patient discomfort; some postoperative sensitivity may occur, and fluoride therapy helps. It is possible to gain as much as 3 to 4 mm of anterior space through this procedure.

Sagittal expansion

Another option for space creation is an increase in the anteroposterior arch length, or sagittal expansion. The amount of space that can be created through this kind of expansion is limited, and the technique is more complicated and sometimes very difficult to achieve in some cases. Therefore, careful clinical and paraclinical evaluations must be completed for each patient.

With this strategy, the arch length can be increased through protrusion of anterior teeth, by distalization of posterior teeth, or by a combination of both. Protrusion of

anterior teeth and distalization of posterior teeth, if applied in the wrong situation, are unstable and can result in other complications.

Protrusion must be applied to the proper patients at the proper time and with careful mechanics. The results are not only unstable but also can disturb the patient's profile and cause alveolar bone resorption. Molar distalization, especially in the mandible, is not only a difficult process but also unstable and can result in many complications, such as second molar impaction, vertical dimension problems, and disturbances of the patient's profile.

Nevertheless, careful creation of space in the anteroposterior dimension of the dental arch during the mixed dentition in suitable cases is the kind of early interceptive treatment that can obviate extraction in the permanent dentition.

Case selection

Treatment planning and selection of appropriate cases are the first steps in this type of treatment. The following are important points to consider before sagittal expansion:

- Age of the patient and growth potential
- Patient's growth pattern
- Molar position and inclination
- Incisor position and inclination
- Roots and periodontal condition
- Patient's soft tissue profile
- Intraoral and extraoral muscle balance
- Periodontal condition of the incisors
- Lip size and position relative to incisors
- Lip dysfunction
- Deleterious oral habits

Patient's growth pattern. One of the tactics for space creation through sagittal expansion is distalization of the permanent molars. Application of this technique in some cases can be very harmful to the occlusion, such as in patients with a vertical growth pattern, in whom molar distalization will result in additional mandibular rotation, greater anterior open bite, and more vertical problems. In contrast, molar distalization in patients with some horizontal growth patterns not only creates space

but also can be helpful in reducing anterior overbite and lower facial height.

Molar position and inclination. Another important point that must be considered before distalization of the first molars is the position and condition of other molars; these considerations include tight contact between the first and second molars, crowding in the molar area, and the possibility of second molar impaction (Fig 4-27). Distalization of the first molar often causes impaction of the second molar; in some cases, preventive extraction of third molar buds might be the only choice.

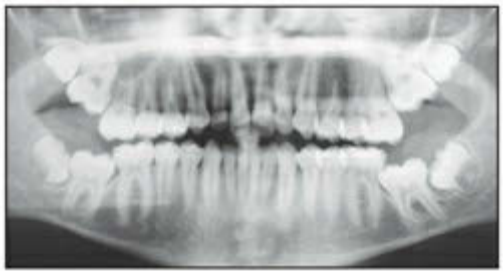


Fig 4-27 Molar crowding that is unsuitable for first molar distalization because of severe crowding in the second and third molars.

Incisor position and inclination. In certain patients with moderate anterior crowding, sagittal expansion can be the treatment of choice, provided that the inclination of incisors allows this movement; otherwise the result will be flaring of anterior teeth.

As Nance[16] indicated, labial movement of the incisors can be extremely detrimental in some cases. Therefore, careful clinical and paraclinical assessments, including the incisor inclination relative to basal bone and other bony and soft tissue structures, must be completed before this movement is attempted.

Patient's soft tissue profile. Labiolingual movement of the incisors has a direct effect on the patient's soft tissue profile that must be evaluated before sagittal expansion.

Intraoral and extraoral muscle balance. The tonicity and function of the perioral musculature also play an important role in the position and inclination of incisors. Labial movement that results in overproclination and places the incisors under excessive muscle force and dysfunction would not have a stable result.

Periodontal condition of the incisors. The periodontal condition and bone support in the incisor area are also important factors that must be considered before any labiolingual movement of incisors is attempted, even if evaluation of the incisor

position and inclination support application of minor protrusive forces to the incisors as part of sagittal space creation.

Treatment options

After careful case selection, the options available for increasing the sagittal dimension of the dental arch are molar distalization, incisor proclination, and a combination of both.

Molar distalization. Maxillary and mandibular molar distalization can be achieved with many different appliance therapies (see chapter 11). The present discussion will be limited to space creation and the advantages and disadvantages related to this procedure.

Certain cases suitable for application of this procedure include all cases of mesially tipped molars or of Class II malocclusions with deep bite, deep curve of Spee, and tipped maxillary and mandibular molars. A lip bumper or a lip bumper combined with cervical headgear is the appliance of choice if used during the mixed dentition and in proper cases (see chapter 11).

In any situation, distalization of the molars requires careful analysis of the molar position and the patient's growth pattern and profile.

Incisor proclination. The other option for increasing arch length in the sagittal direction is proclination of the incisors. This procedure can be the treatment of choice in certain cases, such as retroclined incisors that cause incisor crowding or anterior crossbite. Proclination of incisors for the purpose of space creation in unsuitable cases can also cause several complications, such as profile imbalance, overprotrusion of the incisors, and alveolar recession.

The ideal choices for application of incisor proclination are patients during the mixed dentition stage whose incisors have some degree of crowding and are already retroclined relative to basal bone. Usually these patients have a straight or slightly concave profile. An example of this situation is anterior dentoalveolar crossbite with mandibular mesial shift causing crowding in the anterior region of the maxilla, and sometimes space deficiencies and impaction of the maxillary permanent canines.

Another indication for incisor proclination is treatment of patients with crowded, retroclined mandibular incisors that are causing overjet and overbite. The etiology in these cases can be the early loss of mandibular primary canines, lip dysfunction, and finger sucking. Elimination of the cause and forward positioning of the

mandibular incisors creates space and can correct other related malocclusions, such as overjet, crowding, and profile problems (see cases 4-3 and 4-6).

Mechanotherapy in sagittal expansion

Sagittal expansion can be achieved by many different devices, such as lip bumpers, headgear, all kinds of fixed distalizers, and removable appliances with a spring or screw and extended arch (see the earlier section on space regaining in this chapter and chapter 12).

Lip bumper. The lip bumper is a semiremovable appliance that has several indications in early orthodontic treatment. This appliance consists of two bands cemented to the mandibular permanent first or second molars and a removable bow. An acrylic resin pad is included in the front part of the bow, which is inserted in a buccal tube (Fig 4-28). The lip bumper does not touch the mandibular incisors, and therefore the force of lip musculature is directed to the molars, causing molar uprighting and distalization. At the same time, the absence of force from the lip on the incisors and the concomitant application of forward force from the tongue can procline the incisors. Therefore, the lip bumper can create sagittal space in both directions.

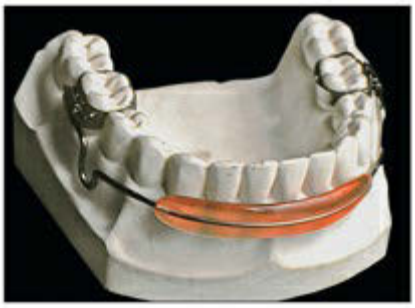


Fig 4-28 Lip bumper for molar uprighting and distalization.

Active lingual arch. The active lingual arch is a fixed LHA with two finger springs soldered to the posterior part of the arch and extended to the lingual surface of the mandibular incisors. This applies a buccal force to the incisors (see Fig 4-8). This appliance can be used to correct moderate crowding and linguoversion of the mandibular incisors.

Pendulum appliance (maxillary molar distalizer). Currently several kinds of molar distalizer are available. One of these, the pendulum appliance, is a good space creator that works by distalizing the maxillary molars. It is a fixed type of appliance

that consists of an acrylic resin button on the palate to serve as anchorage and two extensions soldered to the primary first molar or permanent first premolar bands. Two springs are inserted in the acrylic resin part; the other end of each spring is inserted in the palatal tube of the molar bands; the springs can be removed and reactivated in the mouth.

If a jackscrew is added to the palatal acrylic resin of this appliance, it can function as a rapid palatal expander and therefore achieve sagittal expansion through the springs and transverse expansion through the screw (Fig 4-29).

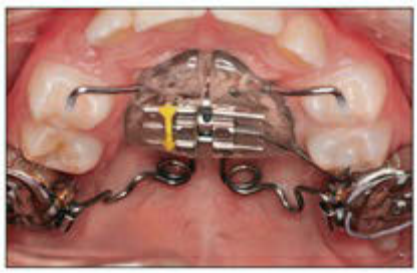


Fig 4-29 Pendulum appliance (molar distalizer).

Removable distalizer. The removable distalizer is a Hawley appliance with an acrylic resin portion and a labial bow that serve as anchorage. A jackscrew is embedded in the acrylic resin and connected to the molars by some clasps (see Fig 4-26).The screw is activated every 3 to 5 days to distalize the molar. The appliance can be unilateral or bilateral, and it can be used in the maxillary or mandibular dentition. Patients can reactivate it themselves and clean it after every meal. Patient cooperation is important for success.

Headgear. Headgear is another kind of molar distalizer that has many other indications in early orthodontic treatment (see chapter 11).

Transverse expansion

Transverse expansion is another type of space creation. Transverse expansion also needs careful evaluation before any determination is made and before treatment planning. Creation of space by transverse expansion depends on the degree of skeletal involvement and whether or not a functional shift exists. Posterior crossbites with mandibular shift need early intervention and maxillary expansion to correct the problems and prevent skeletal deformity. These are ideal cases for space creation by transverse expansion. Posterior crossbites, whether unilateral or bilateral, with or without mandibular shift, are morphologically and etiologically different and need

different treatment options and different mechanotherapy, by slow or rapid expansion. This can be accomplished by a variety of available expanders (see chapter 12).

O'Higgins and Lee[15] reported that a 1-mm increase in the intermolar width will allow approximately 0.3 mm reduction in overjet or creation of 0.6 mm of space within the arch. Transverse expansion could be a treatment of choice in the following situations:

- In a rapidly growing child
- When there is lateral constriction of the dental arch
- When the basal bone accommodates such a movement
- In the mandibular arch, but only distal to the mandibular canines

There are many indications for which proper expansion at the proper time is the best choice for early treatment during the primary or mixed dentition. Lateral expansion can be accomplished in several ways, including dental expansion, dentoalveolar expansion, skeletal expansion, or a combination of these.

McInaney et al[27] showed that early expansion therapy with Crozat appliances eliminated the need for extraction of primary canines or permanent premolars. Lutz and Poulton[28] reported on 13 cases of expansion during the primary dentition, followed for 6 years. Comparing arch perimeter and width at the permanent canines and premolars with those of a control group, they found slightly greater values in the expanded group.

Orthopedic palatal expansion appears to be effective and stable if applied prior to late puberty. The application of expansion must be determined by the specific needs of each patient. Expansion during the primary and mixed dentition for correction of transverse abnormalities (unilateral or bilateral crossbite) is a common treatment option that is discussed in chapter 12.

Another indication for transverse expansion during the mixed dentition is correction of moderate incisor crowding. Sayin and Türkkahraman,[29] studying the possible factors contributing to mandibular anterior crowding during the early mixed dentition, compared the total incisor widths, primary arch widths, and permanent intermolar widths of a group with crowding and a group without crowding. They found significant correlations between crowding and total available incisor space and between primary and permanent arch widths and interalveolar width but no significant correlation between crowding and total arch length.

Radnzic[30] compared arch dimensions in 120 boys (aged 13 years to 15 years 11 months) and reported that boys with crowding had smaller arch widths than did boys without crowding or with spaced arches. He suggested that expansion in carefully selected young patients might be beneficial.

Depending on the dentoskeletal relationships of the dental arch, several kinds of fixed and removable techniques can be applied for transverse expansion. For maxillary arch expansion, several kinds of appliances are available, including the removable jackscrew expander, the W-arch, the quad helix, the reverse TPA, the Hyrax expander, and the Haas expander. For the mandibular arch, a removable jackscrew, a lingual arch with expansion arm, or a lip bumper can be used (see chapter 12).

Fixed expanders

Various fixed expanders can be used during the primary, mixed, and permanent dentitions for different reasons such as posterior crossbite correction and some space creation, including the W-arch, the quad helix, the Haas expander, and the Hyrax expander.

W-arch. The W-arch, or Porter appliance, is a W-shaped arch soldered to molar bands made with 0.036- or 0.040-inch stainless steel wire. Depending on the shape of the arch, the appliance can be activated in different ways (Fig 4-30). If molar expansion is needed, the U-shaped bar in the anterior part can be widened; if expansion at the buccal segments (primary molars or premolars) is required, the U-shaped loop distal to the molar bands can be activated; if expansion for both the permanent molars and primary molars is required, both the anterior and posterior parts of the expander can be activated.

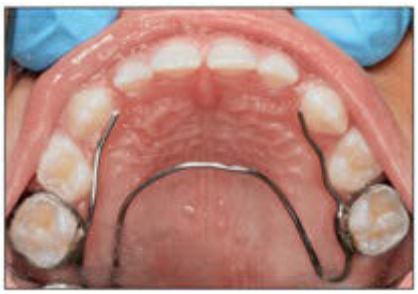


Fig 4-30 W-arch expander (Porter appliance).

The W-arch is an efficient and easy appliance for correction of posterior crossbite during the primary or mixed dentition, especially in 3- to 5-year-old

children. It is well tolerated by children, and it does not need activation by patients or parents. It provides a continuous force and can be reactivated every 4 to 6 weeks. The W-arch also can be used as a reminder appliance for control of finger habits.

Quad helix. The quad helix is another bilateral fixed expander. It has almost the same design specifications as the W-arch but has four coils incorporated in the arch, which give it more flexibility and a greater range of activity (Fig 4-31).

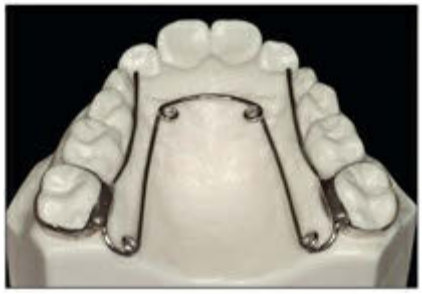


Fig 4-31 Quad helix bilateral fixed expander.

Haas expander. The Haas expander is the first type of expansion appliance introduced by Haas.[31] It is designed for lateral expansion, and because of the palatal acrylic resin it is especially effective for opening and separating the midpalatal suture. It can be used during both the mixed and permanent dentition stages.

This appliance is both tooth and tissue borne, and it is usually fabricated with bands on the premolars and molars; in the mixed dentition, bands can be placed on the primary molars. Two heavy palatal wires are formed to touch both sides of the buccal segments and are soldered to the bands. The wires extend to the palate, where they are embedded in the acrylic resin pads. A jackscrew is located at the center of the acrylic resin (Fig 4-32). The acrylic resin pads and lingual wire apply pressure to both the teeth and palatal mucosa.

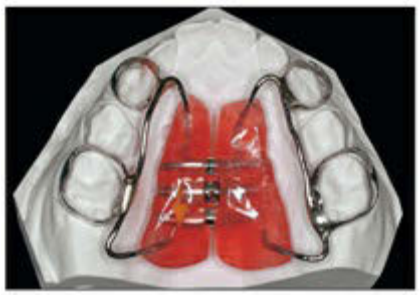


Fig 4-32 Haas rapid expander.

The Haas appliance has several indications for use, such as expansion of constricted maxillary arches in patients with maxillary deficiency, correction of posterior crossbite, and alleviation of some maxillary crowding.

Hyrax expander. The Hyrax expander is a fixed rapid expander made entirely from stainless steel. The appliance is typically fabricated with bands on the first molars and lingual bars extending to the first premolar. A screw located in the center is separated from the tissue, and therefore the appliance is more hygienic than the Haas expander (Fig 4-33).

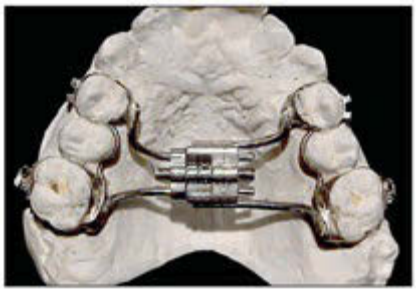


Fig 4-33 Hyrax rapid expander.

Removable expanders

The Schwarz expander is a removable slow expander that has been used in the maxilla and the mandible. The basic parts of this appliance are a jackscrew with some kind of clasp for retention that is embedded in acrylic resin (Fig 4-34). The screw can be opened and reactivated every 3 to 5 days.

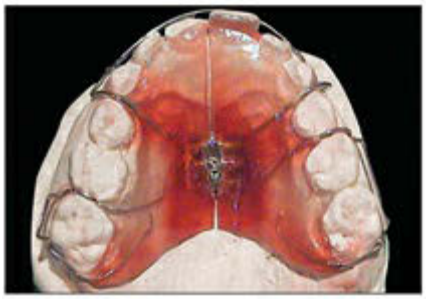


Fig 4-34 Schwarz removable slow expander.

The Schwarz expander is especially useful for dental expansion during the early mixed dentition. It can be removed by the patient for cleaning, and therefore its use requires the patient's compliance.

Space Closure

Another type of space management is elimination or closure of abnormal spacing that can interfere with normal development of the dentition and occlusion. The aims of space closure are to guide eruption and facilitate normal transitional changes during

occlusal development.

Several etiologic factors can produce abnormal spacing in the dentition, such as congenital absence of teeth, microdontia, abnormal habits, and large tongue. Each of these problems has different etiologic factors and different treatment options, which are discussed in their related chapters.

An example of space closure as a space management strategy is during transition of the maxillary incisors. Maxillary permanent incisors usually erupt with a diastema, and during the eruption of the lateral incisor and canine, this space will gradually close. Before their eruption, lateral incisors are slightly behind the central incisor; after the central diastema is closed, the lateral incisor will erupt more labially. If this normal pattern is disturbed and the central diastema remains open, lateral incisors may erupt palatally into a crossbite situation. With early detection and intervention, closure of the diastema provides space for lateral incisors and prevents the development of crossbite.

Another example is when a patient is congenitally missing one or both lateral incisors, resulting in a large diastema. Not only is this situation esthetically unacceptable, but also the probability of canine impaction is high; therefore, closure of the diastema and guidance of canine eruption is recommended.

The following are indications for space closure:

- When extra space is present
- For management of hypodontia, when treatment planning requires a decrease in arch length and space closure is the best treatment option (see chapter 7)
- For closure of the resulting diastema after frenectomy when there is an abnormal frenum attachment
- When an open space such as an abnormally large diastema is preventing eruption and causing malpositioning of adjacent teeth
- For elimination of interdental spacing of maxillary incisors with severe overjet, sometimes seen during the early mixed dentition, to prevent incisor fracture

Space Supervision and Guidance of Eruption

Incisor crowding is a common problem during the mixed dentition. The amount and type of crowding and its management is discussed in detail in chapter 5. In this section, treatment of moderate crowding is discussed within the context of space

supervision.

Space supervision is a term used for space management and correction of moderate crowding during the mixed dentition when no severe arch length deficiency is present. With this tactic, moderate crowding of incisors can be directed to the posterior segment and to leeway space.

The prognosis for space supervision is sometimes questionable, whereas the prognosis is always good for space maintenance and regaining.

As Moyers[9] claimed, space supervision cases are those that will have a better chance of getting through the mixed dentition with clinical guidance than without it. Proper selection of cases and a thorough case evaluation are always necessary for this approach. Misdiagnosed space supervision cases that subsequently require extraction of permanent teeth are more difficult to treat than cases of gross discrepancy.

The following are examples of different kinds of space supervision protocols:

- Sequential slicing of the primary canine will allow spontaneous alignment of the lateral incisors.
- Enamel stripping of the mesial surface of mandibular primary first molars will facilitate eruption of the mandibular permanent canine and reduce pressure on the lateral incisors.
- Disking of the mesiodistal convexity of the primary molars will help normal eruption of adjacent teeth.
- Insertion of an LHA to preserve arch circumferences followed by sequential extraction of primary first molars and then second molars will help anterior crowding.

According to Gianelly,[32] the leeway space provides adequate space to resolve crowding that is present during the mixed dentition in the majority of individuals. He also recommended insertion of a lip bumper after eruption of the first premolars to preserve arch length.

In a study of patients with incisor crowding during the mixed dentition, Brennan and Gianelly[33] preserved arch length and found adequate space to resolve the crowding in 73 (68%) of 107 patients.

Case Reports

The following cases show correction of moderate crowding, whether the problem was space loss that could be regained or moderate space deficiency that could be managed with space creation. As mentioned earlier, space creation is appropriate for cases of moderate space deficiency when the incisor inclination, the patient's profile, and the relationship between the teeth and basal bone permit creation of the necessary space by minor sagittal or transverse expansion. These types of interceptive treatment are especially recommended during the mixed dentition stage and when children are growing.

Case 4-1

A 13-year-old girl in the early permanent dentition stage had a Class I malocclusion, zero overjet, a straight profile, and good mandibular dentition. She presented with maxillary crowding; both maxillary lateral incisors were in crossbite (locked occlusion), and both maxillary canines were out of the arch because of space deficiency (Figs 4-35a to 4-35d).

Treatment:
The treatment plan called for nonextraction management; minor space creation was to be achieved by cross-bite correction of the lateral incisors (sagittal movement) and slight expansion. The treatment began with slow expansion with a maxillary Hawley appliance that had occlusal coverage to disocclude the anterior teeth during correction of the lateral crossbite. This was followed by 2 × 6 maxillary bonding to help lateral incisor proclination and alignment of the canines. Use of the Hawley appliance was stopped, and treatment was concluded with canine alignment. Figures 4-35e to 4-35g show the posttreatment occlusion.

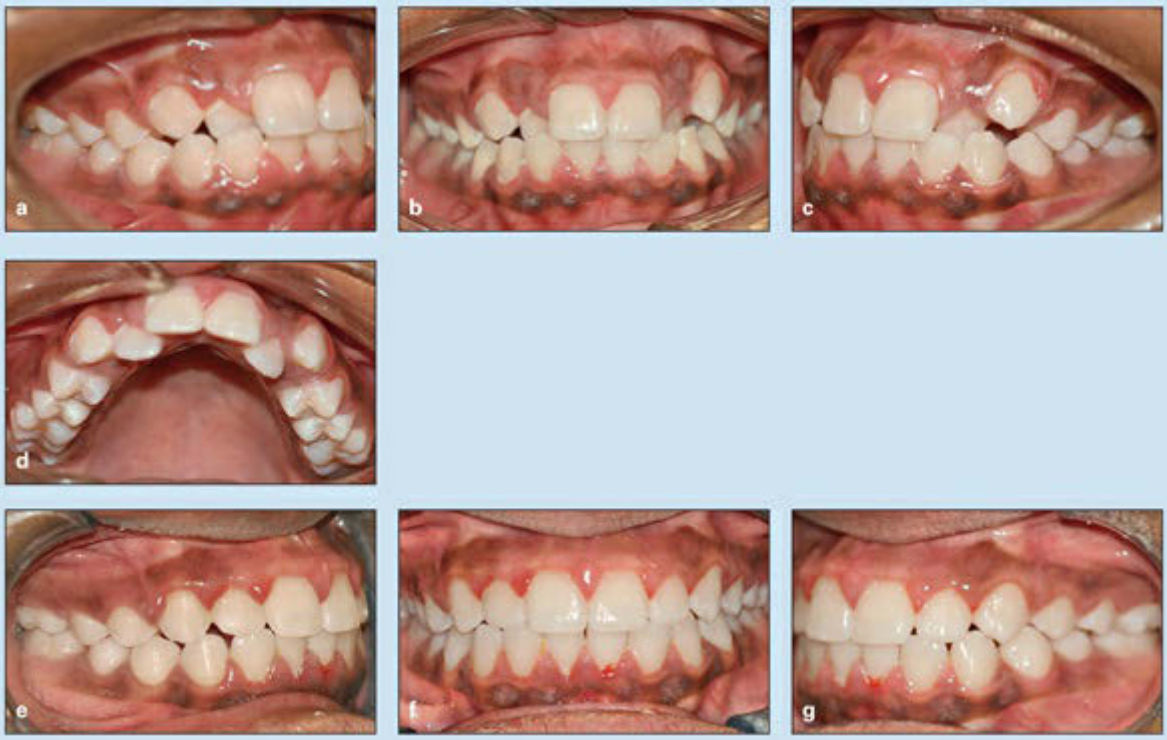


Fig 4-35 Space creation in a 13-year-old girl in the early permanent dentition stage. *(a to d)* Pretreatment occlusion. There is maxillary crowding, both maxillary lateral incisors are in crossbite (locked occlusion), and both maxillary canines are out of the arch because of space deficiency. *(e to g)* Posttreatment occlusion.

Case 4-2

A 10-year-old boy in the late mixed dentition stage had a Class I malocclusion and space deficiency in the maxillary right and mandibular left and right segments as a result of early primary molar loss (Figs 4-36a to 4-36f).

Treatment:
Space was regained by 2 × 4 bonding first and later by bonding of erupted premolars and a push coil mechanism for space opening and uprighting of the tipped molars (Figs 4-36g and 4-36h). Figures 4-36i to 4-36k show the posttreatment occlusion.

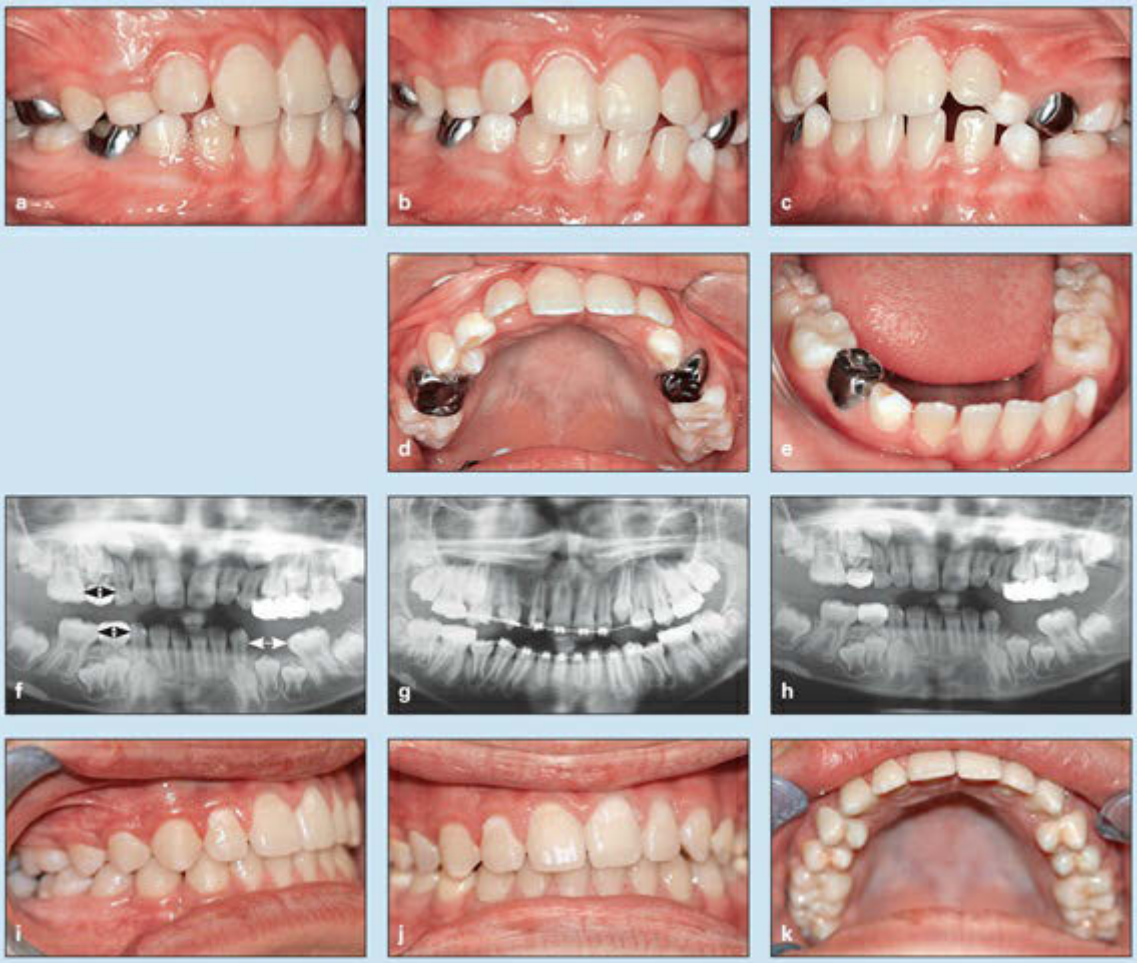


Fig 4-36 Space regaining in a 10-year-old boy in the late mixed dentition stage. *(a to e)* Pretreatment occlusion. *(f)* Panoramic radiograph showing that the maxillary right premolars and mandibular left and right premolars *(arrows)* are affected by space deficiency due to early primary loss. *(g and h)* Panoramic radiographs showing the mechanism of space opening. *(i to k)* Posttreatment occlusion.

Case 4-3

A 13-year-old girl had a Class I malocclusion and right molars with a slight Class II tendency. The maxillary midline had shifted to the right due to early loss of the primary canine. There was no space for the maxillary right permanent canine, and the left canine was slightly out of the arch. She also exhibited mandibular anterior crowding (Figs 4-37a to 4-37g).

Treatment:
The treatment involved space creation (sagittal expansion) through distalization of the maxillary right segment and slight proclination of the maxillary and mandibular incisors (Figs 4-37h to 4-37n).

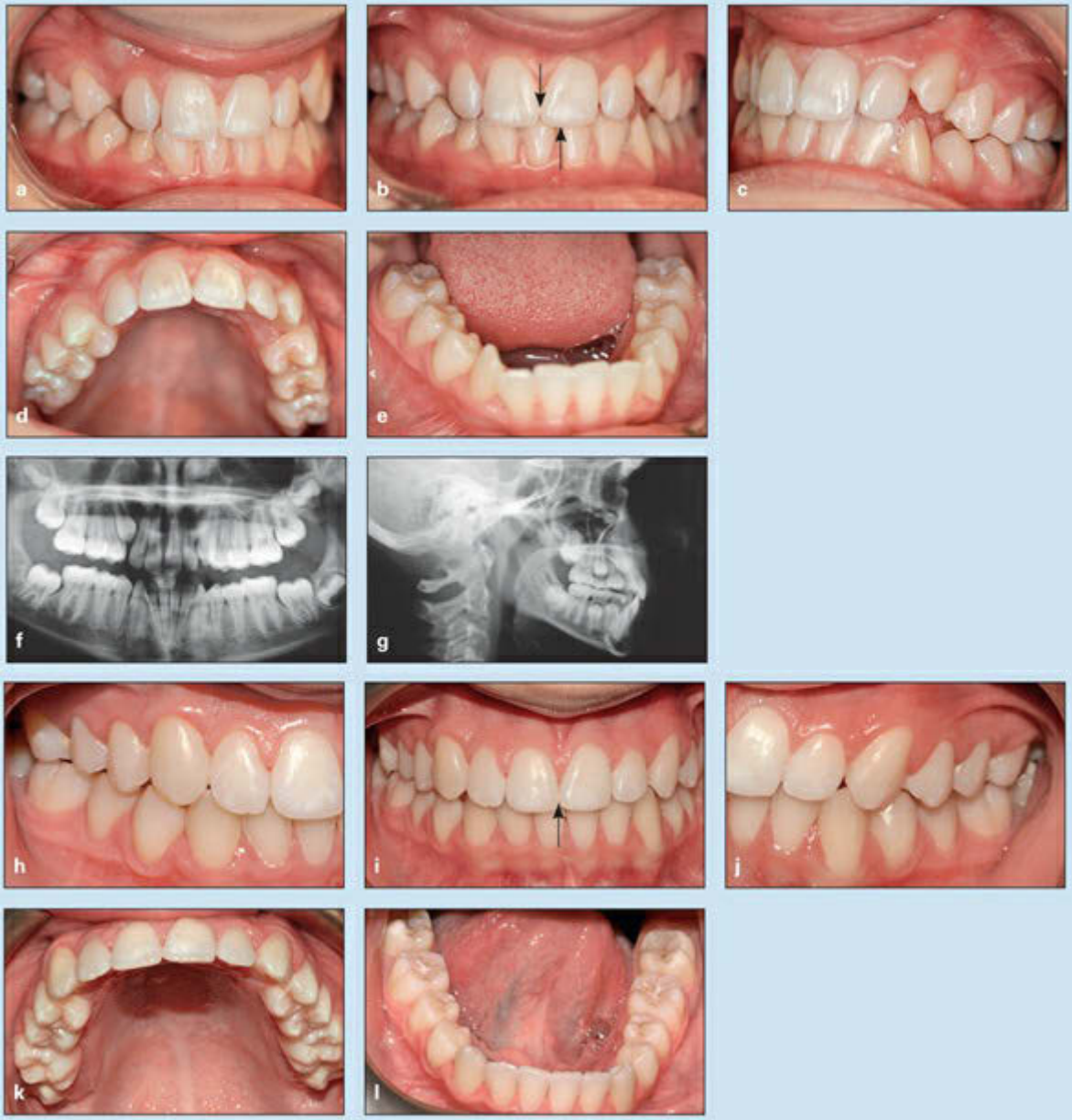


Fig 4-37 Space creation in a 13-year-old girl with early loss of maxillary primary canines, maxillary midline shift *(arrows)*, no space for the maxillary right canine, and mandibular anterior crowding. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g)* Pretreatment lateral cephalometric radio-graph. *(h to l)* Posttreatment occlusion.

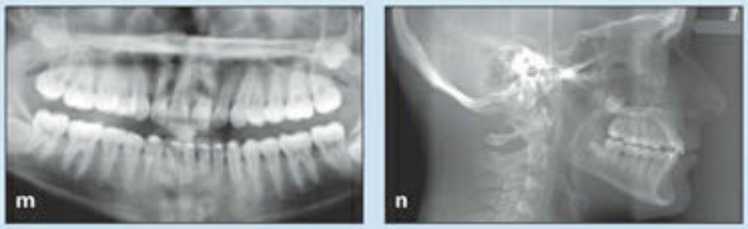


Fig 4-37 *(cont) (m)* Posttreatment panoramic radiograph. *(n)* Posttreatment lateral cephalometric radiograph.

Case 4-4

A 10-year-old boy had a history of multiple primary tooth extractions because of extensive caries. He presented with severe space deficiency because primary tooth extraction was not followed by proper space management. At the time of presentation, he had a Class II molar relationship, normal overjet and overbite, posterior cross-bite, and a 2-mm midline diastema. The midline was shifted to the left, and there was a 12- to 14-mm space deficiency in the maxillary and mandibular arches (Figs 4-38a to 4-38f).

Treatment:
Because of his normal skeletal pattern, normal sagittal basal bone relationship, and straight profile, the treatment plan called for nonextraction space creation through transverse expansion to correct the posterior cross-bite, sagittal expansion by maxillary molar distalization for correction of the Class II molar relationship, space opening for unerupted canines and premolars, and uprighting of erupted permanent teeth (Figs 4-38g to 4-38m).

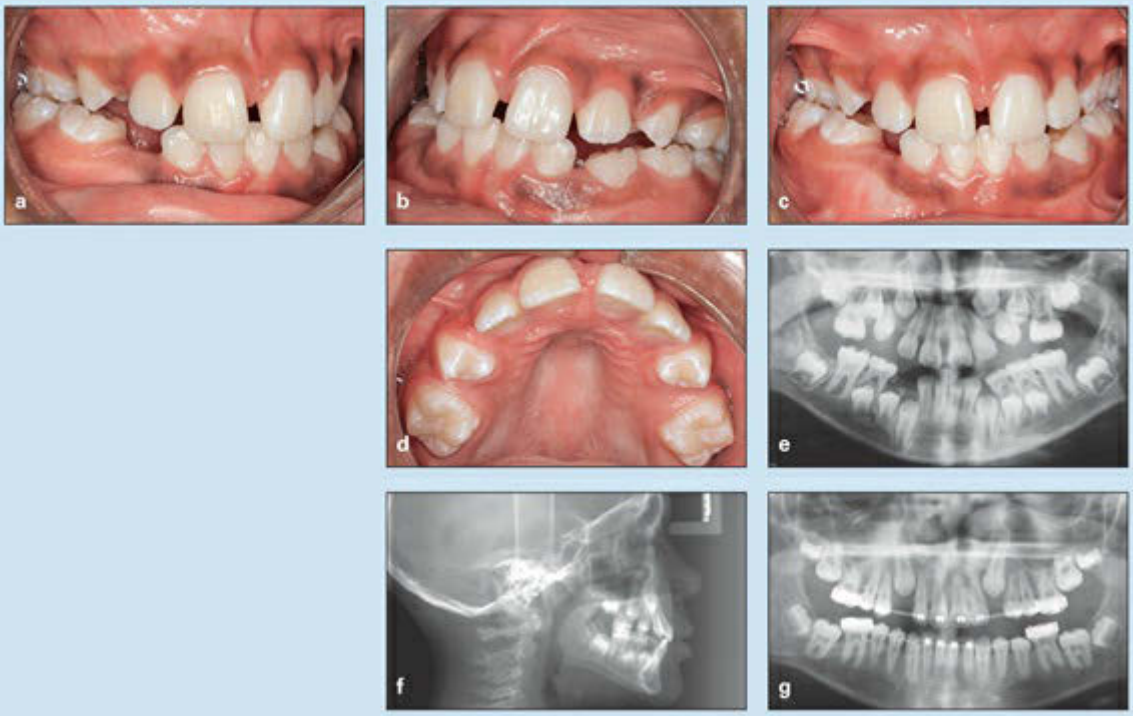


Fig 4-38 Space creation in a 10-year-old boy with severe space deficiency resulting from early loss of primary teeth and the lack of any space management. *(a to d)* Pretreatment occlusion. *(e)* Pretreatment panoramic radiograph. *(f)* Pretreatment cephalometric radiograph. *(g)* Panoramic

radio-graph taken during treatment.

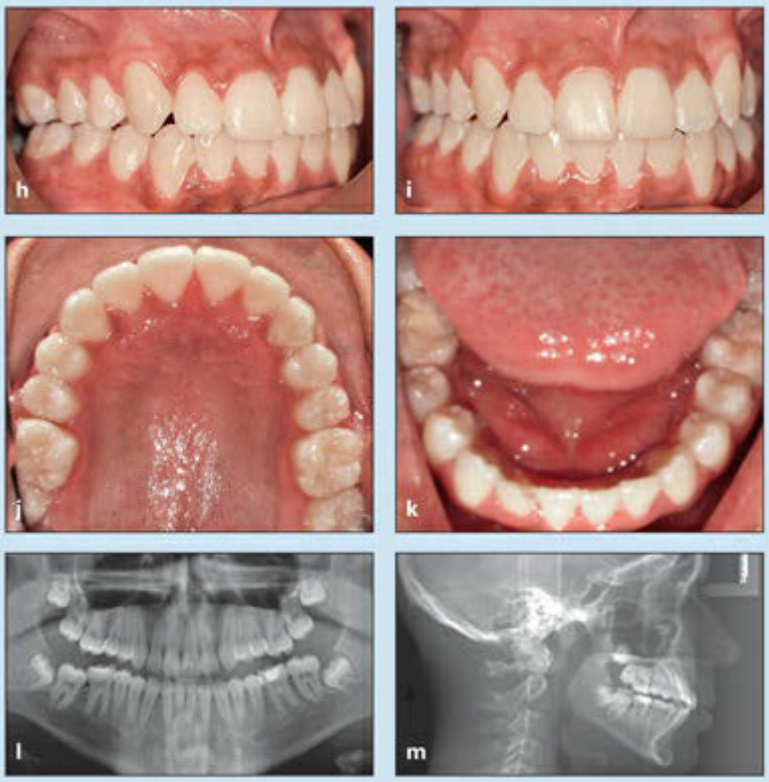


Fig 4-38 *(cont) (h to k)* Posttreatment occlusion. *(l)* Posttreatment panoramic radiograph. *(m)* Posttreatment cephalometric radiograph showing the condition of the dentition and the dentoskeletal relationships.

Case 4-5

This case is an example of space supervision. A 14-year-old girl presented with four submerged primary molars preventing eruption of premolars and normal vertical alveolar growth and consequently disturbing normal occlusal development (Fig 4-39a to 4-39e).

Figures 4-39f to 4-39j reveal the status of the occlusion after extraction of the primary submerged teeth, placement of anchorage appliances (maxillary Nance and LHA), guidance of eruption, and no other treatment or bonding. Figures 4-39k to 4-39n show the posttreatment results.

Fig 4-39 Space supervision in a 14-year-old girl with four submerged primary molars that are preventing premolar eruption and alveolar growth, disrupting the development of occlusion. *(a to c)* Pretreatment occlusion.

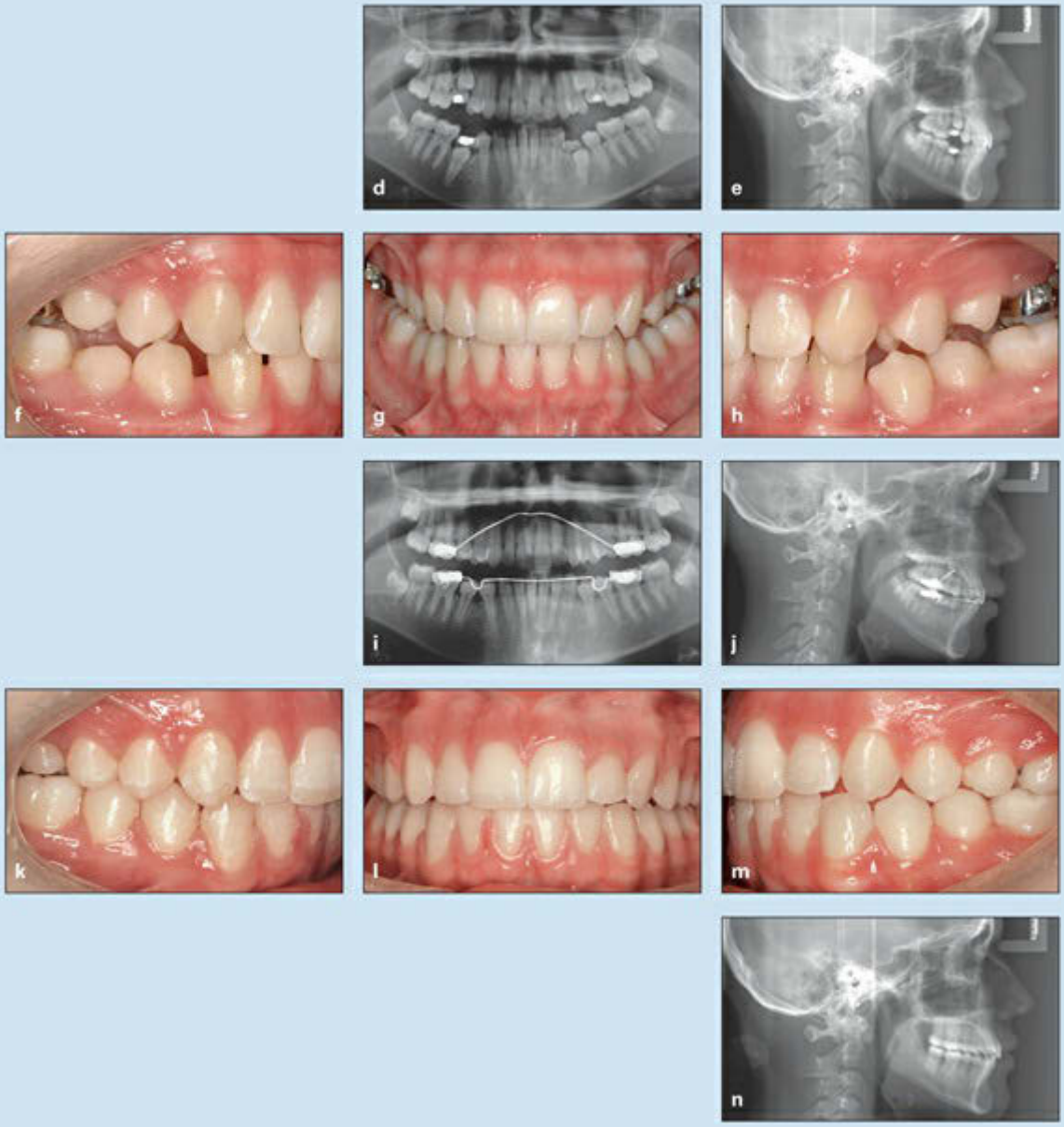


Fig 4-39 *(cont) (d)* Pretreatment panoramic radiograph. *(e)* Pretreatment cephalometric radiograph. *(f to j)* Condition of the occlusion during space supervision, which involved extraction of the primary submerged teeth, anchorage preparation (maxillary Nance holding arch and lower holding arch), and guidance of eruption. *(k to m)* Posttreatment occlusion. *(n)* Posttreatment cephalometric radiograph.

Case 4-6

A 10-year-old girl exhibited a Class I malocclusion (left molars with a Class II relationship) and severe maxillary and mandibular crowding due to space loss. Both mandibular canines were out of the arch, the maxillary midline had shifted to the left, and there was no space for the maxillary right canine, which showed a prominent canine eminence (Figs 4-40a to 4-40f).

Treatment:

Because of the patient's age and skeletal pattern and the etiology of crowding, that is, early loss of the mandibular primary canines and maxillary left primary canine, the plan was designed as nonextraction treatment with space regaining and minor space creation. A lower lingual holding arch was placed to preserve leeway space. Maxillary and mandibular anterior teeth were bonded using push coil springs to distalize the maxillary left molar and shift the midline to the right with minor incisor proclination.This achieved correction of the maxillary midline and space opening for the maxillary left canine. Figures 4-40g to 4-40l show the posttreatment results.

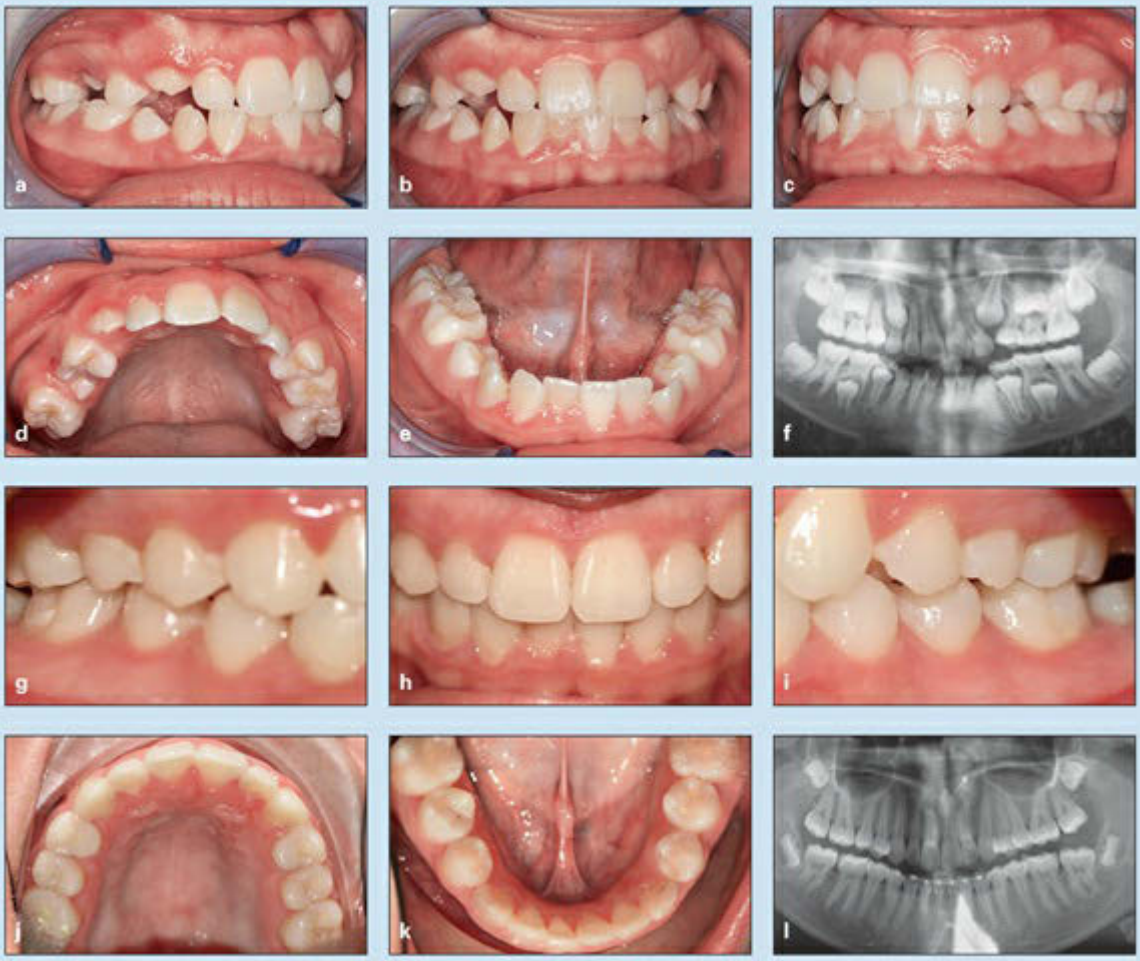


Fig 4-40 Space regaining and minor space creation in a 10-year-old girl with maxillary and mandibular crowding due to space loss. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g to k)* Posttreatment occlusion. *(l)* Posttreatment panoramic radiograph.

Summary

- Every tooth in its socket over the basal bone is maintained under continuous, balanced mesiodistal, buccolingual, and occlusal forces from adjacent and opposing teeth.
- Any change in this balancing force, such as loss of a tooth unit, can cause unfavorable tooth movement of the adjacent and opposing teeth and produce

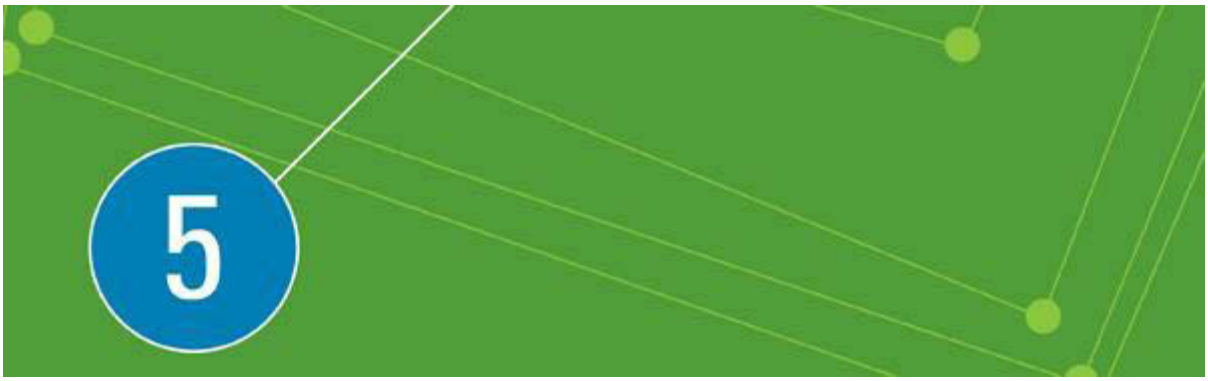
tooth malrelationships. Therefore, any space as a result of tooth loss must be controlled by some kind of space management.

- Management of the space depends on several factors, such as the patient's occlusion, the time elapsed since tooth loss, and the age of the patient.
- Proper space management requires sufficient knowledge of the etiology and morphology of tooth loss, the type of tooth movement, the effects of primary molar extraction on succedaneous teeth, the incidence and nature of space loss, prediction of tooth emergence, and factors influencing mesial and distal tooth drift.
- Depending on these factors, several kinds of management are available: space maintenance, space regaining, space creation, space closure, and space supervision.
- Space maintenance is a preventive type of management to prevent space loss and abnormal tooth movement.
- Space regaining is an interceptive procedure to regain lost space.
- Space creation is a corrective procedure to create some space in patients with moderate space deficiency. This procedure is more complicated than space regaining and requires biomechanical principles and more cautious tooth movement.
- Space closure is an interceptive procedure for guidance of eruption (eg, closure of abnormal diastemas to guide normal eruption of lateral incisors).
- Space supervision is also a kind of interceptive procedure to supervise occlusal development, such as by controlling leeway space to eliminate moderate incisor crowding.

References

1. Fanning EA. A longitudinal study of tooth formation and root resorption. N Z Dent J 1961;57:202–217.
2. Posen AL. The effect of premature loss of deciduous molars on premolar eruption. Angle Orthod 1965;35:249–252.
3. Owen DG. The incidence and nature of space closure following the premature extraction of deciduous teeth: A literature survey. Am J Orthod 1971;59:37–49.
4. Lundström A. The significance of early loss of deciduous teeth in the etiology of malocclusion. Am J Orthod 1955;41:819–826.
5. Linder-Aronson S. The effect of premature loss of deciduous teeth: A biometric study in 14- and 15-years-olds. Acta Odontol Scand 1960;18:101–122.
6. Seipel CM. Prevention of malocclusion. Trans Eur Orthod Soc 1947/1948:203–211.
7. Johnsen DC. Space observation following loss of the mandibular first primary molars in mixed dentition. J Dent Child 1980;47:24–27.

8. Dean JA, Avery DR, McDonald RE. McDonald and Avery's Dentistry for the Child and Adolescent, ed 9. St Louis: Mosby, 2010.
9. Moyers RE. Handbook of Orthodontics, ed 4. Chicago: Year Book Medical,1988.
10. Northway WM, Wainright RL, Demirjian A. Effect of premature loss of deciduous molars. Angle Orthod 1984;54: 295–329.
11. Grøn AM. Prediction of tooth emergence. J Dent Res 1962;41:573–585.
12. Hägg U, Taranger J. Dental development, dental age and tooth counts. Angle Orthod 1985;55:93–107.
13. Demirjian A. Cranio-maxillofacial and somatic growth and oral health in French Canadian children [in French]. J Can Dent Assoc (Tor) 1968;34:79–78.
14. Smith SL, Buschang PH. Growth in root length of the mandibular canine and premolars in a mixed-longitudinal orthodontic sample. Am J Hum Biol 2009;21:623–634.
15. O'Higgins M, Lee RT. How much space is created from expansion or premolar extraction? J Orthod 2000;27:11–13.
16. Nance H. Limitations of orthodontic treatment in the permanent dentition. 2. Diagnosis and treatment in the permanent dentition. Am J Orthod 1947;33:253–301.
17. Tanaka MM, Johnston LE. The prediction of the size of unerupted canines and premolars in a contemporary orthodontic population. J Am Dent Assoc 1974;88:798–801.
18. Staley RN, Kerber PE. A revision of the Hixon and Old-father mixed-dentition prediction method. Am J Orthod 1980;78:296–302.
19. Merrifield LL. Differential diagnosis. Semin Orthod 1996;2:241–253.
20. Bolton WA. The clinical application of a tooth-size analysis. Am J Orthod 1962;48:504–529.
21. Björk A, Skieller V. Normal and abnormal growth of the mandible: A synthesis of longitudinal cephalometric implant studies over a period of 25 years. Eur J Orthod 1983;5:1–46.
22. Andrews LF. The six keys to normal occlusion. Am J Orthod 1972;9:296–309.
23. Moorrees CF, Chadha JM. Available space for the incisors during dental development—A growth study based on physiologic age. Angle Orthod 1965;36:12–22.
24. Ricketts RM. Early treatment. 1. J Clin Orthod 1979;13:23–38.
25. Ricketts RM. Early treatment. 2. J Clin Orthod 1979;13:115–127.
26. Ricketts RM. Early treatment. 3. J Clin Orthod 1979;13:181–199.
27. McInaney JB, Adams RM, Freeman M. A nonextraction approach to crowded dentitions in young children: Early recognition and treatment. J Am Dent Assoc 1980;101:251–257.
28. Lutz HD, Poulton D. Stability of dental arch expansion in the deciduous dentition. Angle Orthod 1985;55:299–315.
29. Sayin MO, Türkkahraman H. Factors contributing to mandibular anterior crowding in the early mixed dentition. Angle Orthod 2004;74:754–758.
30. Radnzic D. Dental crowding and its relationship to mesiodistal crown diameters and arch dimensions. Am J Orthod Dentofacial Orthop 1988;94:50–56.
31. Haas JA.The treatment of maxillary deficiency by opening the midpalatal suture. Angle Orthod 1965;35:200–217.
32. Gianelly AA. Leeway space and the resolution of crowding in the mixed dentition. Semin Orthod 1995;1:188–194.
33. Brennan MM, Gianelly AA. The use of the lingual arch in the mixed dentition to resolve incisor crowding. Am J Orthod Dentofacial Orthop 2000;117:81–85.



Management of Incisor Crowding

Incisor crowding is one of the most frequently observed dental phenomena in young children during the early and middle mixed dentition periods. Incisor crowding has been a subject of increasing concern for children and their parents, and it is among the most common reasons patients seek orthodontic treatment.

Because of the discrepancy between primary and permanent incisors, some degree of incisor crowding is very common during the early mixed dentition. Some of this crowding can be transitional and needs no intervention, some can be corrected with occlusal guidance and space supervision, and some can be critical and develop to a severe malocclusion and therefore must be managed with tooth extraction at the proper time.

Overall space availability for all dentition varies, depending on the growth of the jaws and relative variation in the mesiodistal crown diameters of the permanent teeth and their predecessors. Children who have little or no developmental spacing between the primary incisors and occasionally some crowding in the primary dentition develop severe crowding in the permanent incisors. As Baume[1] showed, children with no spacing during the primary dentition (Baume class II) have a 40% chance of developing crowding in their permanent incisors.

Mandibular anterior crowding is initially identified as a discrepancy between the mesiodistal tooth widths of the four permanent incisors and the available space in the anterior part of basal bone. However, many other variables must be considered prior to early intervention and treatment, such as the direction of mandibular growth, early loss of primary molars, incisor and molar inclinations, and oral and perioral musculature balance. All of these factors can play a role in and be associated with incisor crowding.

Because not all incisor crowding during the mixed dentition is alike, the treatment procedure must vary depending on the etiology and morphology of the crowding. However, this common problem sometimes is a cause of great concern for parents and patients, so practitioners must be prepared to address this issue with parents. The most appropriate answer to their questions is not, "This is not a problem," or "Wait until all permanent teeth erupt." Rather, the wise and proper answer is, "This might be a sign of other problems, so the situation should be evaluated now."

The next question for practitioners is how to identify and differentiate different situations of incisor crowding to enable optimal management of each case. This chapter will answer this question by examining different aspects of incisor crowding:

- Is there any way to predict this problem?
- What is the common cause of this problem?
- How can different types of crowding be classified and differentiated?
- What kind of intervention can prevent the problem?
- How can the problem be treated?

Prediction

Predicting permanent incisor crowding at an earlier stage has been a goal in several longitudinal studies during the primary, mixed, and permanent dentitions. However, there are conflicting results in the literature evaluating the correlation between crowding and other dentofacial structures. Baume[1] reported that children with no spacing during the primary dentition (Baume class II) have a 40% chance of crowding in their permanent incisors.

Hunter,[2] after analysis of crowding and spacing of the teeth, concluded that estimates of the size of unerupted teeth in the mixed dentition are more accurate than estimates of future spacing or crowding; this is because arch perimeter does not decrease by the same amount in all cases but is rather associated with the amount of crowding in the mixed dentition. The amount of crowding or spacing must always be determined after considering molar, canine, and incisor relationships as well as the condition of lip posture on the dentition.

Sampson and Richards[3] tested the hypothesis that pre-eruptive tooth positions and dental arch parameters might forecast crowding changes in Class I untreated

dentition unaffected by caries or attrition. Crowding scores, radiographic relationships, and dental arch dimensions were evaluated for the mixed dentition. The sample was divided into two groups, depending on whether there was an increase or decrease in incisor and canine crowding between the mixed and the permanent dentitions. Dental arch form and tooth size were important factors in measuring the amount of incisor or canine crowding at stage 1 and stage 2. The researchers found that neither the radiographic nor the dental arch predictors proved useful in forecasting crowding changes.

Howe et al[4] examined the extent to which tooth size and jaw size each contribute to dental crowding. Two groups of dental casts were selected on the basis of dental crowding. In one group, 50 pairs of dental casts exhibited gross dental crowding. The second group consisted of 54 pairs of dental casts that exhibited little or no crowding. Howe et al[4] observed significant differences between the dental arch dimensions of the two groups. The crowded group was found to have smaller dental arch dimensions than the noncrowded group. The researchers suggested that practitioners should consider treatments that increase dental arch length rather than those that reduce tooth mass.

Bishara et al[5] attempted to determine the changes in the maxillary and mandibular tooth size–arch length discrepancy between the primary dentition (mean age of 4.0 years) and second molar eruption (mean age of 13.3 years) to establish whether a tooth size–arch length discrepancy in the permanent dentition can be predicted in the primary dentition. They evaluated the records of 35 boys and 27 girls. The mesiodistal diameters of all primary teeth and their permanent successors, as well as various parameters of dental arch width and length, were measured in the primary and permanent dentitions.

The researchers concluded that additional factors are involved in tooth size–arch size discrepancy and should be considered in explaining this phenomenon. These factors are present in most persons, whether or not they have a malocclusion or have undergone orthodontic treatment. Regression analysis indicated that these changes are related, in part, to tooth size as well as to changes in arch length and arch width. The correlations between the various primary and permanent tooth size–arch length discrepancies are of such a magnitude that they do not allow accurate prediction of discrepancies in the permanent dentition from the available dental measurements in the primary dentition.[5]

Sinclair and Little[6] also found no clinically significant associations between various mandibular parameters and incisor crowding.

Melo et al[7] searched for indicators during the primary dentition of future crowding of the mandibular incisors during the early mixed dentition. For this purpose, they examined the skeletal and dental morphologic characteristics of the primary dentition using dental casts and cephalograms of 23 subjects. They then assessed the degree of crowding in the same subjects at 9 years of age and found 12 normal and 11 crowded dentitions. The size of permanent incisors in the crowding group was significantly greater than that in the normal group. The maxillary and mandibular dental arch lengths and the posterior cranial base length (sella-basion) were also effective discriminators between the two groups.

Common Causes

Conducting an implant study in children, Björk[8] clearly showed that the direction and rotational pattern of mandibular growth have different effects on occlusion, incisor position, and crowding. In fact, the same implant studies revealed that internal jaw rotation also changes the anteroposterior position of the incisor teeth, which in turn has a major influence on changes in arch length. They showed that the rotational pattern of jaw growth obviously influences the magnitude of tooth eruption. When the mandible rotates downward and backward, anterior facial height increases, there is a tendency toward anterior open bite, and the incisors are thrust forward relative to the mandible. When excessive counterclockwise rotation occurs, short facial height develops, and lingual displacement of the incisors relative to the maxilla and mandible increases the tendency toward crowding.

Leighton and Hunter[9] also reported that the angles between the sella-nasion line and the mandibular and occlusal planes were significantly larger in patients with crowding during both the mixed and permanent dentition stages. They also associated downward and deficient growth of the mandible with upright or retroclined mandibular incisors and with crowding.

Sayin and Türkkahraman[10] tried to find factors that can contribute to mandibular anterior crowding. They evaluated 60 dental casts of patients during the early mixed dentition, divided into two groups according to the severity of mandibular anterior crowding. In the mandible, the primary intercanine widths, intermolar widths, permanent intermolar widths, interalveolar widths, space available for the mandibular permanent incisors, and total arch length were all significantly larger in patients with crowding than they were in patients without crowding. No significant correlation was found between crowding and total arch length.

Türkkahraman and Sayin[11] evaluated the association between dentofacial structures and mandibular crowding during the early mixed dentition by analyzing cephalometric radiographs and casts of 60 children. They found that children with crowding had smaller mandibular incisor–nasion–point B angles and shorter maxillary and mandibular skeletal length. They also found greater interincisal angles, overjet, overbite, and Wits appraisal measurements. They concluded that crowding of the mandibular incisors is not merely the result of a tooth size–arch size discrepancy; dentofacial characteristics also contribute to this misalignment.

Intervention and Prevention

Despite much research and many longitudinal investigations to find special characteristics and criteria for early detection and prevention of permanent incisor crowding during the primary dentition, there is still much controversy on the subject. Practitioners still see young children with incisor crowding that was not prevented and highly concerned parents seeking some kind of early intervention. Nevertheless, incisor crowding during the mixed dentition can be managed differently depending on the amount of crowding and other variables involved in this process, and all factors must be considered in treatment planning.

There is no reliable criterion during the primary dentition to predict permanent incisor crowding, but even if it were possible prevention is not possible until all incisors are erupted. However, the author's clinical experience in some early treatment during the primary dentition indicates that some type of early intervention, even before eruption of permanent incisors, can be useful in providing space for the permanent incisors. For example, during rapid palatal expansion in the primary or early mixed dentition for correction of posterior crossbite, some space can develop for incisors and canines, facilitating normal eruption of the anterior segment (Fig 5-1; see the discussion on early correction of posterior crossbite in chapter 12).

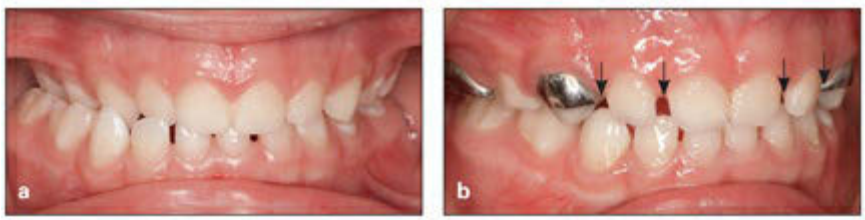


Fig 5-1 *(a and b)* Space opening *(arrows)* after rapid expansion and posterior crossbite correction.

Moreover, clinical observation indicates that an abnormal sequence of eruption

between mandibular canines and first premolars has a tendency to precede some kinds of mandibular incisor crowding, and in such cases early detection and proper intervention can reduce or correct incisor crowding. Other examples of prevention and early intervention for mandibular incisor crowding are control of some types of thumb sucking to retrude the mandibular incisors and control of lower lip dysfunction that can cause mandibular incisor crowding.

Longitudinal monitoring of panoramic radiographs and the three phases of transitional dentition is a prudent measure that can be considered the first step in prevention and early intervention.

Characteristics and Classification

Mandibular anterior crowding is identified as a discrepancy between the mesiodistal tooth widths of the four permanent incisors and the space available between the mesial surfaces of the primary canines. Because crowding can result from different morphologic and etiologic factors, the first step for the purpose of proper treatment planning is to identify the kind of crowding, that is, to determine the amount of permanent incisor crowding during an early stage of the mixed dentition. This can be achieved by careful measurement and analysis of tooth size–arch size discrepancy (see chapter 4).

However, incisor crowding is not merely a tooth size–arch size discrepancy. Many variables, such as direction of mandibular growth, early loss of primary molars, the oral and perioral musculature, and incisor and molar inclinations, can be associated with crowding. Thus, the second issue for proper treatment planning is to search for dentofacial factors that might be associated with mandibular crowding, using all necessary diagnostic tools during the early mixed dentition.

After careful assessment of the aforementioned variables, incisor crowding can be arbitrarily classified into three types of crowding:

1. Minor crowding up to 3 mm of space deficiency
2. Moderate crowding with 3 to 5 mm of space deficiency
3. Severe crowding with more than 6 mm of space deficiency

However, the amount of crowding in millimeters is not the only decisive criterion for treatment decisions; many other factors must be evaluated and considered in treatment planning. The purpose of this classification is to provide the first practical

step of discriminating between different amounts of crowding.

Generally incisor crowding has been classified into two general types, acquired crowding and hereditary crowding, which are discussed in detail later in this chapter.

Treatment

Although incisor crowding is not merely a function of tooth size–arch size discrepancy and many other local and general variables might be involved and must be evaluated, for practical purposes the treatment options have been classified according to the amount of incisor crowding (minor, moderate, and severe crowding).

Minor Crowding

Because of the size difference between primary and permanent incisors (incisor liability), a minor amount of permanent incisor crowding (less than 3 mm) at an early stage of the mixed dentition is considered transient crowding; up to 3 mm of incisor crowding may resolve spontaneously during the transitional dentition without the need for early intervention.

Studies of children with normal occlusion also indicate that transitional changes from the primary to the mixed dentition resolve up to 3 mm of incisor crowding spontaneously without treatment. As was discussed in chapter 2, there are natural mechanisms that can self-correct minor crowding of incisors during the transitional dentition, such as interdental spacing, more proclination of permanent incisors, and intercanine width increase.

Moorrees and Reed[12] studied a series of 184 casts at 3 years and 16 to 18 years of age. They found that, in the mandible, an average of 1.6 mm of crowding in boys and 1.8 mm of crowding in girls will recover to 0.0 mm by the age of 8 years. They attributed this recovery to a phenomenon termed *secondary spacing*, which facilitates lateral incisor eruption. This process occurs when the mandibular lateral incisors emerge and push the mandibular primary canines laterally. This also causes the maxillary primary canines to move laterally by occlusal force, creating spaces for the maxillary lateral incisors.

Extraction of the mandibular primary canines during this natural phenomenon will

prevent secondary spacing and the increase in intercanine width. Prevention of this phenomenon may even convert a nonextraction case to an extraction case. Therefore, no greater relief of spacing in the incisor segment can be expected after complete eruption of the lateral incisors. They also added that the level of dental maturation, that is, tooth formation and emergence, gives decisive clues for diagnosis and treatment planning because it defines the timetable of individual development.[12]

It is also important to remember that physiologic self-correction of minor crowding can occur, provided that the following factors are present:

- Normal growth and development of the jaws
- Normal interdental spacing, especially primate spaces
- Normal labial inclination of the maxillary and mandibular incisors
- Normal increase in intercanine width
- On the other hand, the presence of any of the following disturbances can disrupt self-correction of minor crowding and change the treatment plan:
- Premature loss of primary teeth
- Problems of tooth number, size, or form
- Abnormal habits
- Soft tissue problems, such as lip dysfunction
- Hereditary or congenital disturbances

Therefore, for patients with minor incisor crowding, practitioners must carefully monitor the transitional dentition and maintain arch length integrity against any abnormal factors such as interproximal caries, accidental tooth loss, or abnormal habits.

Moderate Crowding

Moderate mixed dentition crowding is the presence of 3 to 5 mm of crowding during the transitional stage of dentition. Depending on the variable conditions of occlusion, moderate crowding can be treated differently at different stages of transition. Treatment can take place from the time of eruption of the mandibular permanent central incisors (around 6 years) to the late mixed dentition stage (around 11 to 12 years).

Permanent incisor buds are located lingual to the root of primary incisors and,

especially in mandibular dentition, they often erupt lingually. Therefore, monitoring is very important at this stage of transition. For example, delayed exfoliation of primary incisors might be problematic; sometimes permanent central incisors erupt lingually while primary central incisors are still present and immobile. In this situation, immediate radiographic evaluation and extraction of the over-retained primary teeth must be performed (Fig 5-2).

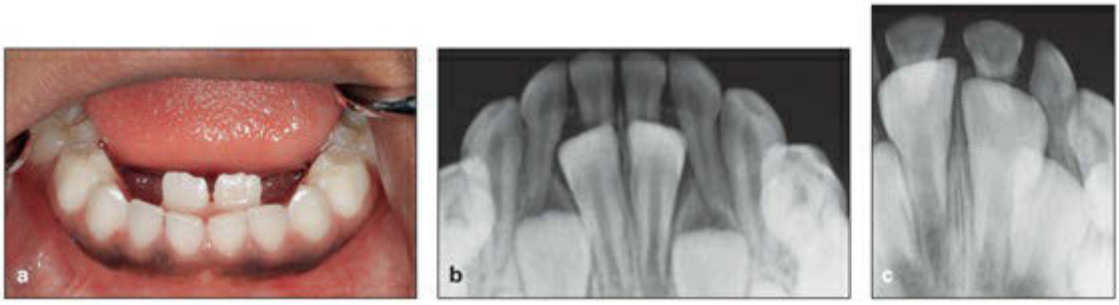


Fig 5-2 *(a to c)* Delayed resorption of the primary central incisor roots, causing lingual eruption of the permanent incisors.

If the primary canine has already exfoliated or been extracted without supervision and some space has been lost, a space regainer such as a lip bumper should be used to regain the lost space. Afterward, use of a lingual holding arch or fixed appliance is recommended (for more detail on space regaining, see chapter 4).

If, on the other hand, space analysis and evaluation of all other perioral factors indicate that the space available in the arch and basal bone can accommodate all incisors, canines, and premolars and there is no evidence of space loss resulting from early loss of the primary dentition or caries, moderately crowded incisors can be guided to better positions by careful space management in the posterior segment of the arch, in a procedure called *space supervision*. In other words, when space analyses indicate that there is "just enough" space, the situation is a borderline case for extraction, and an attempt should be made to correct moderate crowding through supervision of the transitional dentition to prevent loss of leeway space. Moyers[13] described space supervision cases as those that will make it through the mixed dentition better with clinical guidance than without it.

Proper selection of cases and a thorough case evaluation are always necessary for this tactic. In patients with moderate anterior crowding, space supervision can be applied through four different tactics:

1. Sequential stripping of primary teeth
2. Preservation of E space

3. Space creation
4. Combination approach, using more than one of the aforementioned methods

Sequential stripping of primary teeth

Sequential stripping of primary lateral incisors, canines, or molars can allow spontaneous alignment of incisors and distal eruption of the canines and premolars without any mechanotherapy. Minor amounts of stripping do not cause patient discomfort. Greater amounts of slicing may require administration of local anesthesia and produce some postoperative sensitivity; fluoride therapy helps alleviate the sensitivity. It is possible to gain as much as 4 mm (maximum) of anterior space through this procedure. This procedure can be done by stripping metal tape or a fine flexible diamond disk.

Space supervision and slicing of primary teeth can be applied during eruption of the mandibular permanent lateral incisors, which usually have more difficulty in erupting than central incisors because of their size. Disking of the mesial surface of the primary canine can resolve the problem. In some cases of severe space deficiency, it may be necessary to extract the primary canines, but immediate insertion of a lower holding arch with spurs is indicated to prevent any space loss that can result from lingual movement of mandibular incisors (Fig 5-3).

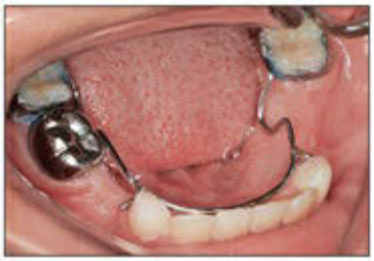


Fig 5-3 Lower holding arch with spurs.

When disking or extraction of the mandibular primary canine is planned, the normal transitional process of secondary spacing must be considered. The disking or extraction must be timed so as not to disrupt the development of secondary spacing.

Another example of sequential stripping is disking of the mesial surface of the primary first molar to provide space for the mandibular permanent canine and guide distal eruption of the canine helping spontaneous correction of moderate incisor crowding. Likewise, the mesial surface of the primary second molar can be stripped to facilitate and guide first premolar eruption. With this technique, the moderate incisor crowding is actually transferred to the posterior segment, taking advantage of

leeway space, without use of any appliances.

Another example of selective grinding is disking of the distal surface of the mandibular primary second molar. This procedure is indicated when the maxillary permanent first molar has erupted before the mandibular molar. Distal stripping of the primary second molar accelerates mesial migration of the mandibular molar to achieve a Class I molar relationship.

Preservation of E space

Preservation of E space, or guidance of eruption in moderate incisor crowding, is another kind of space supervision that can be applied after careful evaluation of dentofacial structures and muscles, space analysis, and confirmation of a moderate space deficiency.

Gianelly[14] stated that the late mixed dentition stage of dentition, after the eruption of the first premolars, is a favorable time to start treatment to resolve incisor crowding. Moyers[13] and Gianelly[15] suggested that in the majority of individuals with moderate crowding in the early mixed dentition, this technique provides adequate space to resolve the moderate incisor crowding.

The author suggests that the optimum time to apply this procedure is before exfoliation of both primary first and second molars. Insertion of a proper lingual holding arch connected to the permanent first molars before exfoliation of the primary first and second molars will prevent any mesial shift of the first permanent molar to leeway space. This tactic will preserve 2 to 4 mm of space for crowded incisors.

After insertion of a good lower holding arch to prevent any mesial shift of the permanent molars, extraction of the primary first molar will actually guide the first premolar eruption more distally. Subsequently, the canine can move distally to facilitate spontaneous correction of incisor crowding.

Preservation of E space and guidance of eruption of premolars can be applied in patients with primary molar ankylosis and tilted eruption of premolars.

The best appliances to preserve E space or prevent mesial movement of permanent molars are the lower holding arch in the mandible and the transpalatal arch in the maxilla (Fig 5-4).

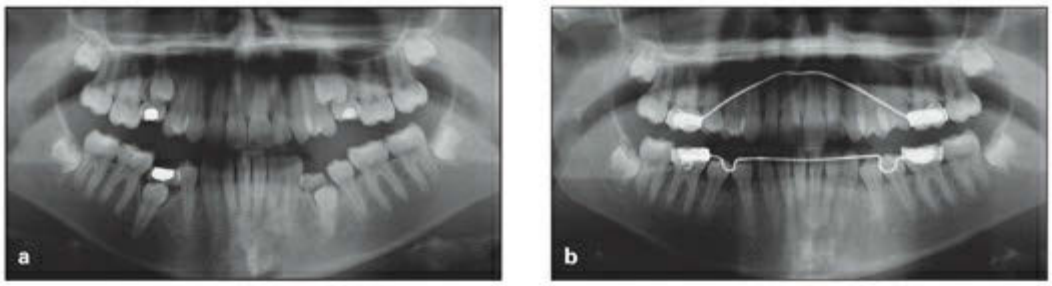


Fig 5-4 *(a and b)* Preservation of E space to allow normal eruption of premolars.

Space creation

Space creation is another means of correcting moderate incisor crowding (3 to 5 mm of crowding) during the mixed dentition. The procedure of space creation is different from space regaining, which is the procedure to apply when existing space has been lost due to early loss of primary teeth. Rather, space creation is a procedure to create needed space. Space creation is a more complex procedure and needs much more evaluation and analysis before any treatment planning. This procedure can be performed in the following ways:

- Transverse expansion
- Sagittal expansion (anteroposterior arch length increase)
- Selective stripping
- Combination of the aforementioned methods

For more details about space creation, see chapter 4.

Combination approach

Incisor crowding during the transitional dentition is a common problem that worries patients and parents. In many patients, the problem can be treated with nonextraction procedures at an early age, while tooth extraction will be required if the treatment is postponed to the permanent dentition.

Clinical experience indicates that this kind of crowding usually develops during incisor transition as a result of a combination of etiologic factors such as early primary tooth loss, trauma, supernumerary teeth, missing teeth, Bolton discrepancy, abnormal habits, abnormal muscle function, abnormal basal bone relationships such as deep bite, or anterior crossbite. Thus, the practitioner must take advantage of all

the examinations and diagnostic tools available to search for dentofacial factors in the early mixed dentition that might be associated with mandibular crowding.

As discussed earlier, Björk and Skieller[16] reported that the mandibular growth pattern and mandibular rotation affect incisor position and crowding. When the mandible rotates downward and backward, anterior facial height increases, there is a tendency toward anterior open bite, and the incisors are thrust forward relative to the mandible. When excessive counterclockwise rotation occurs, a short facial height results, and lingual displacement of the incisors relative to the maxilla and mandible increases the tendency toward crowding. Therefore, these types of crowding with multifactorial causes cannot be managed by space supervision or space creation alone. However, elimination or prevention of the causes, especially through early detection and proper intervention, can result in a stable and balanced occlusion. The following cases are examples of this type of crowding that can be managed at an early age.

Case 5-1

A 10-year, 8-month-old boy presented with a Class I malocclusion, locked anterior crossbite (without mandibular shift), maxillary incisor crowding, displacement, and a diastema (Figs 5-5a to 5-5e).

A panoramic radiograph taken about 1 year after an accident (Fig 5-5f) revealed the effect of trauma to the permanent incisors (early resorption of the left primary central and lateral incisors and delayed resorption of the right primary lateral and central incisors), causing delayed eruption of the right permanent central and lateral incisors, tilting to crossbite, and mandibular crowding (Fig 5-5g to 5-5j).

Treatment:
Treatment was started with 2 × 4 maxillary bonding and placement of composite resin on the occlusal surfaces of the mandibular molars to disocclude the anterior segment. A nickel-titanium archwire was also placed for leveling, followed by a stainless steel archwire with open loop for correction of incisor crossbite.

After crossbite correction was completed, mandibular bonding was performed for correction of incisor crowding. Figures 5-5k and 5-5l show the dentition during treatment. Figure 5-5m reveals the change of canine inclination. This patient was then treated only with 2 × 6 maxillary and mandibular bonding. Figures 5-5n to 5-5r show the final posttreatment occlusion.

Fig 5-5 Treatment of a 10-year, 8-month-old boy with a Class I malocclusion, locked anterior crossbite, maxillary incisor crowding, displacement, and a diastema. *(a to e)* Pretreatment occlusion.

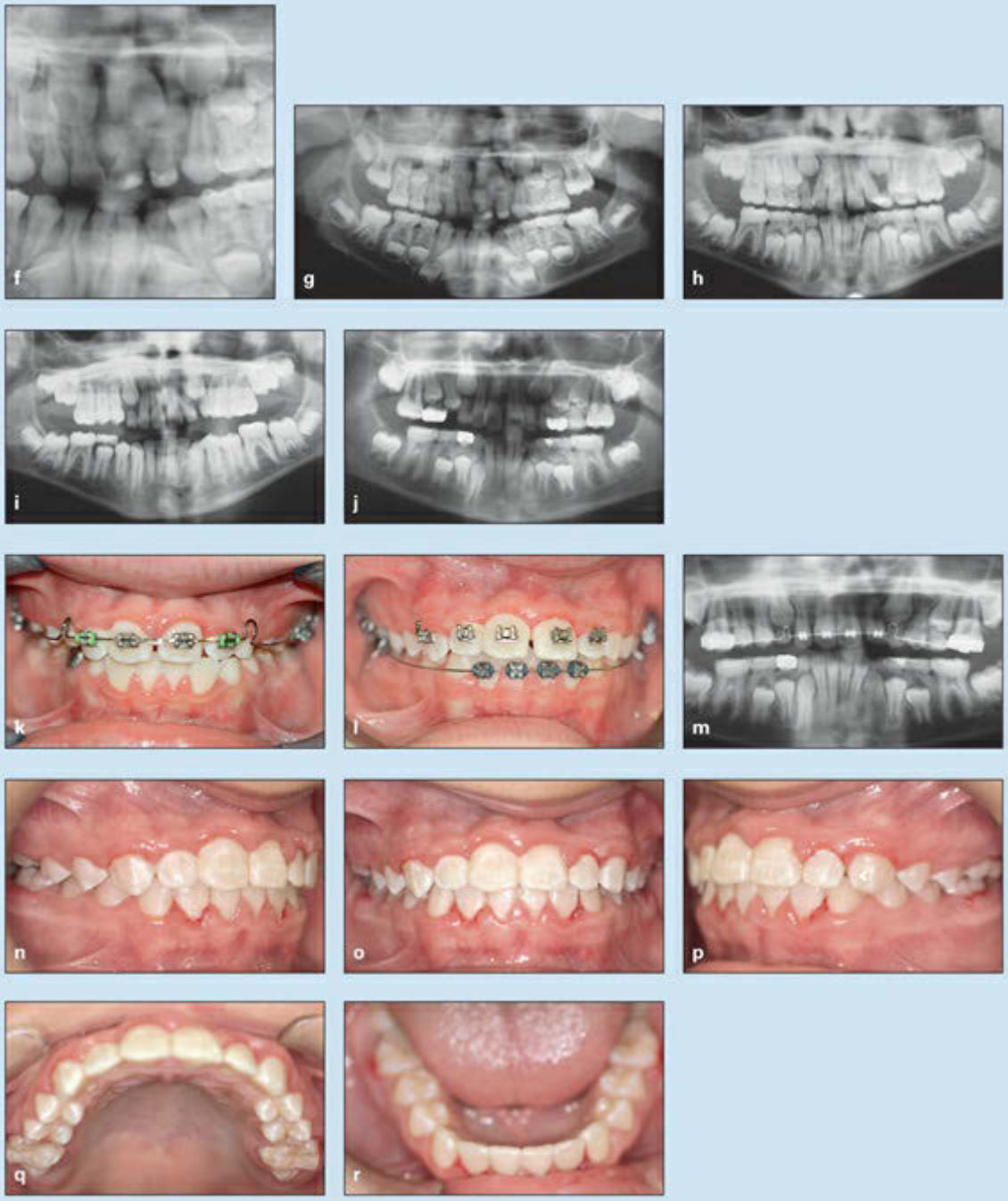


Fig 5-5 *(cont) (f)* Magnified panoramic radiograph, taken about 1 year after trauma, reveals displacement of the maxillary permanent right lateral incisor. *(g to j)* Consecutive pretreatment panoramic radiographs. *(k and l)* Dentition during treatment. *(m)* Panoramic radiograph during treatment, revealing the change in canine inclination. *(n to r)* Posttreatment occlusion.

Case 5-2

A 10-year-old girl presented with an anterior locked occlusion that caused severe crowding and displacement of the mandibular incisors (Figs 5-6a to 5-6f).

Treatment:

A nonextraction treatment procedure was planned. Treatment was started with mandibular 2 × 6 bonding and application of composite resin on the occlusal surfaces of the mandibular first molars to disocclude the anterior segment. After correction of crossbite and proclination of all of the maxillary incisors, mandibular 2 × 3 bonding was used first to align three incisors and create space for the mandibular right lateral incisor. The final step was traction of the severely displaced right lateral incisor into the arch and alignment (Figs 5-6g to 5-6l).

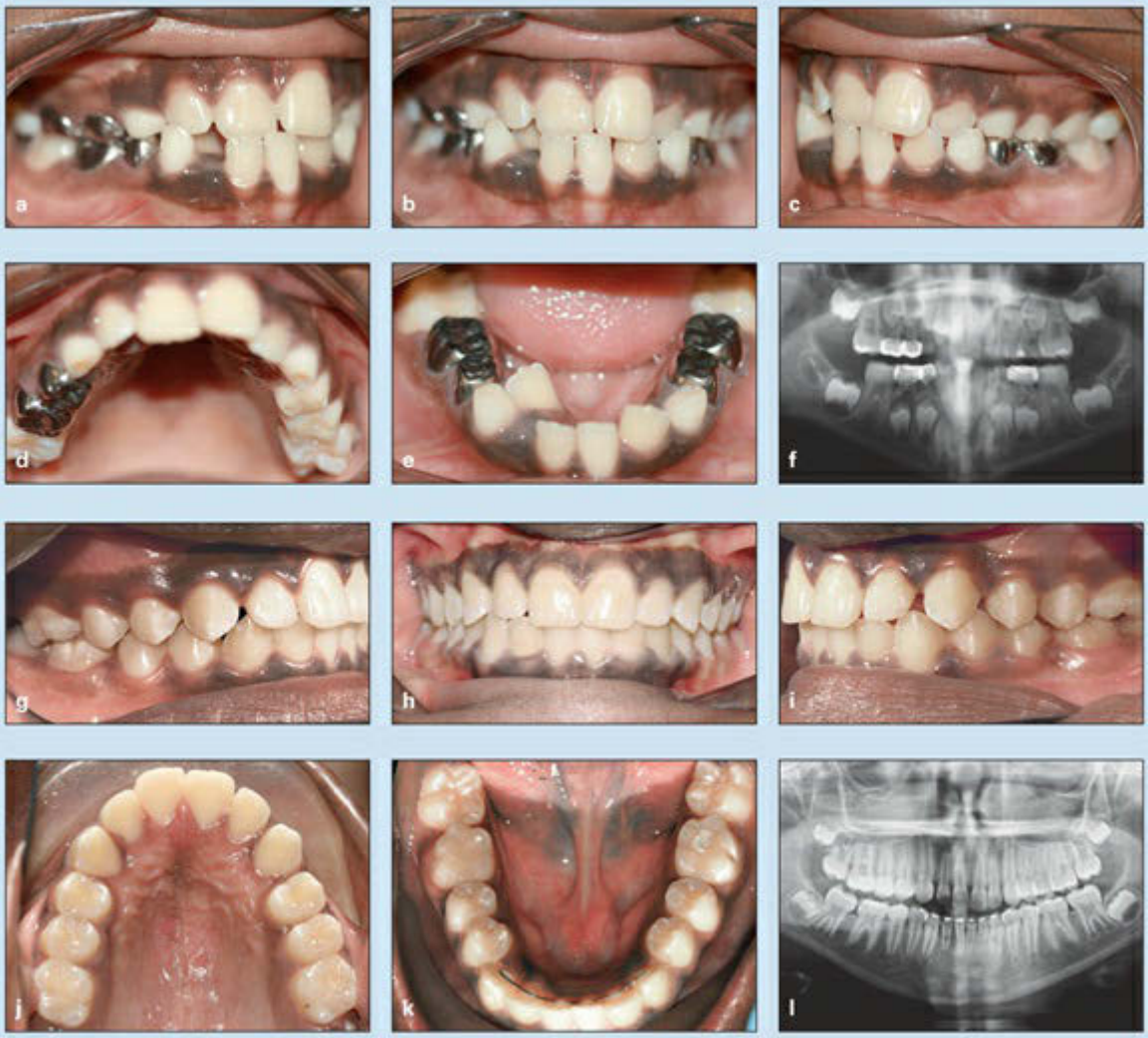


Fig 5-6 Treatment of a 10-year-old girl with severe anterior crowding. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g to k)* Posttreatment occlusion. *(l)* Posttreatment panoramic radiograph.

Case 5-3

A 10-year-old boy presented with a Class I very deep bite (impinging) and retroclined maxillary and mandibular incisors. He had severe mandibular incisor crowding, and the mandibular right lateral incisor was completely displaced lingually. Early loss of the mandibular primary incisors and deep bite resulted in mandibular anterior collapse and some retrusion of the maxillary incisors (Fig 5-7a to 5-7f).

Treatment:

The treatment plan involved correction of the deep bite, slight proclination of the incisors (space creation), and correction of crowding without extraction. First, the maxillary first molars were banded and an anterior bite plate was placed to disocclude the posterior segment in order to stimulate tooth eruption and reduce overbite. Then 2 × 4 bonding was used for maxillary incisor alignment. A maxillary utility arch was used to intrude the maxillary anterior segment and extrude the posterior segment. Lowe 2 × 4 bonding was used for mandibular incisor alignment and proclination, to create some space to alleviate mandibular crowding. The final step was to continue with complete-mouth bonding for correction of the deep bite. Figures 5-7g to 5-7k show the posttreatment results, and Figs 5-7l and 5-7m show the pre- and posttretment cephalometric radiographs.

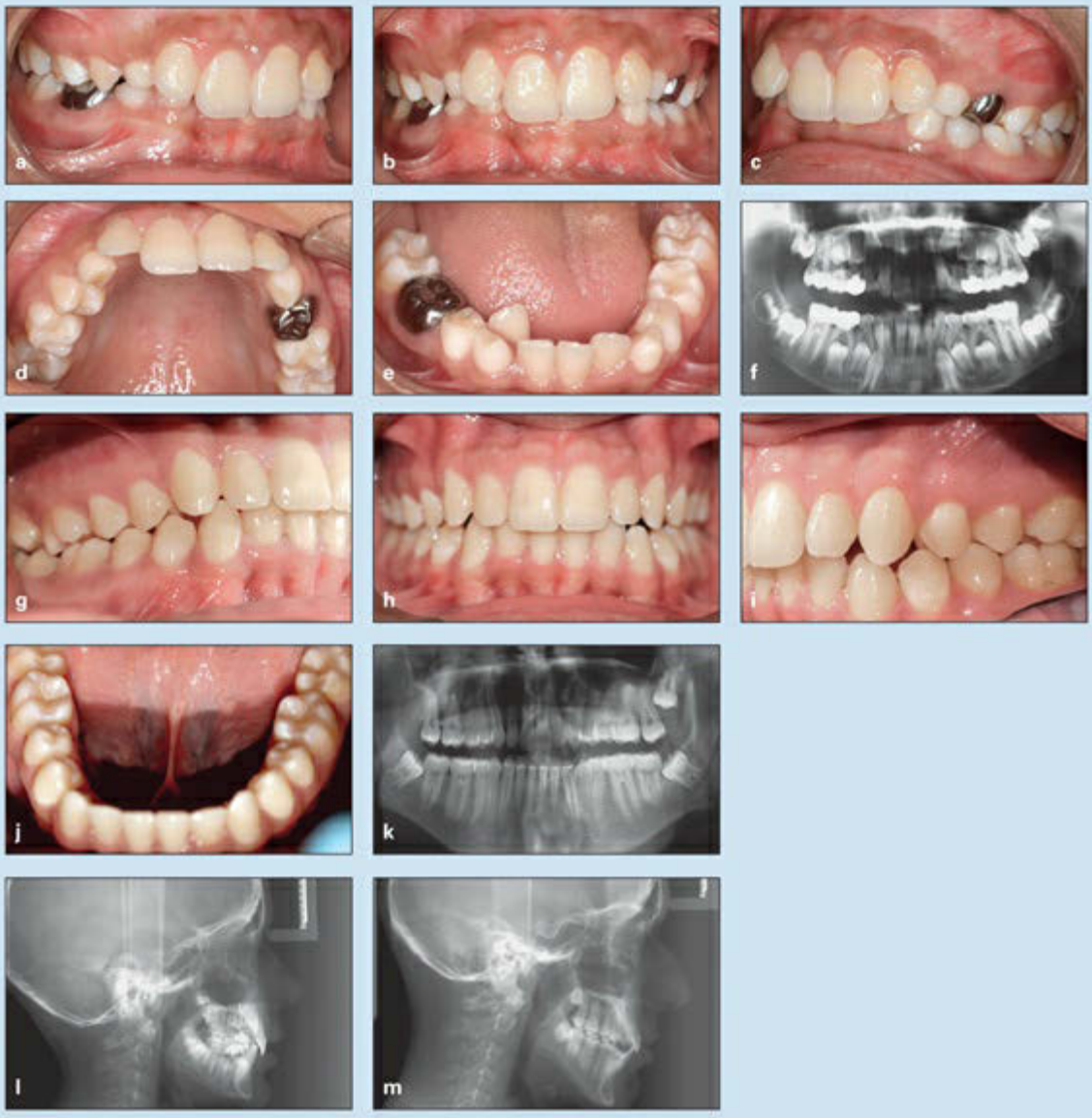


Fig 5-7 Treatment of a 10-year-old boy with a Class I very deep bite (impinging bite) and retroclined maxillary and mandibular incisors. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g to j)* Posttreatment occlusion. *(k)* Posttreatment panoramic radiograph. *(l and m)* Pretreatment and post-treatment cephalometric radiographs.

Severe Crowding

Severe crowding includes incisor crowding of more than 6 mm. As discussed earlier, the number of millimeters of crowding is not the only decisive point for correction of crowding cases. There are many other points, such as inclination of incisors, patient's profile, growth pattern and the type of crowding (acquired crowding or hereditary crowding), that must be considered in treatment planning of crowding cases. These include all kinds of incisor crowding that cannot be treated with space supervision or space creation with a favorable and stable result, with the only remaining option being tooth extraction.

Another kind of mandibular incisor crowding usually seen in the permanent dentition is a kind of Bolton discrepancy between maxillary and mandibular incisors, causing severe crowding in the mandibular incisor segment with normal maxillary incisors and normal interdigitation in the buccal segments. This type of problem can be managed through a completely different procedure[17] discussed in more detail later in this chapter (see case 5-4).

Serial Extraction

The controversy over whether or not to extract has been going on for more than 100 years. Orthodontic theory and practice was based on Angle's idea that extraction should be avoided,[18] which he first expressed early in the 20th century. Extraction versus nonextraction plans have since been debated for several generations. This pendulum has been swinging to either side over the past few decades.

However, the nonextraction treatment philosophy does not have a strong scientific basis; in fact, numerous studies contradict this theory and have shown relapse following nonextraction treatment.[19,20] They advocate that the extraction strategy not only will provide normal alignment of the teeth but also will improve the patient's profile and smile with a stable result.

Considering the controversy regarding extraction, clinicians sometimes are faced with the following question: Does jaw growth solve the crowding of incisors after the completion of eruption? The answer is "no": Extensive research has confirmed that there is a continuous decrease in arch length (about 2 to 3 mm) during the early transition to adulthood.[1,8,12,13,21]

The following processes are responsible for arch length reduction during the transitional dentition:

- General mesial tooth migration because of proximal contact wear

- First and second mesial shifting of permanent first molars
- Forward jaw growth against muscles and incisor uprighting

Malocclusions can be treated in many ways, including methods that involve extraction of some tooth units and methods without extraction. Selection of the best option requires a thorough evaluation of the problem. Therefore, diagnosis and treatment planning must be based on full assessment of the anteroposterior, vertical, and transverse dentofacial patterns and growth status of each individual patient.

When all clinical and paraclinical evaluations show a severe space deficiency and a tooth size–arch size discrepancy is confirmed, the best solution might be elimination of some tooth units.

This kind of treatment during the mixed dentition is termed *serial extraction*. Serial extraction is a sequential extraction of some primary teeth followed by extraction of some permanent teeth to resolve space deficiency, facilitate eruption of remaining permanent teeth, and reduce the need for mechanical tooth movement at a later age.

As Charles H. Tweed[20] said, "Knowledge will gradually replace harsh mechanics," and intervention will be more effective if it is carried out during the mixed dentition period. When the basal bone cannot accommodate 32 teeth and the only option is reduction in the number of tooth units, failure to perform early intervention not only will affect the occlusion and supporting structures but also may have negative effects on the patient's physical and psychologic well-being and coordination of jaw growth and certainly will demand much more complicated treatment at a later age.

The technique of early extraction of some tooth units before complete eruption of the permanent dentition was first introduced by Bunon[22] in 1743. However, a lack of understanding of the biology involved and insufficient knowledge of biomechanics produced disastrous results. Therefore, this technique was considered a failure and practitioners did not have the desire to acquire necessary knowledge to perform it well. Kjellgren[23] reintroduced the concept of serial extraction in the 1940s. Unfortunately, this procedure is still applied sometimes by individuals who have not attained the requisite knowledge of diagnosis, different types of crowding, and proper application of this technique, and therefore even now the results sometimes are disastrous.

The term *serial extraction* may not be the best term for this type of treatment; it represents an oversimplification and sometimes, especially among general

practitioners, implies that only extraction of teeth is involved. Either of the terms used later by Hotz,[24] *guidance of eruption* o r *guidance of occlusion*, is more suitable.

As Dale[25] has said, "Serial extraction is not easy, as so many mistakenly believe." On the contrary, he argues, a comprehensive diagnosis is necessary in order to thwart failure and disappointment. Ignoring the basic principles of diagnosis will cause injury not only to the patient but also to the reputation of the practitioner and ultimately the profession.[23]

Early extraction of teeth can facilitate eruption of the remaining dentition in the dental arch, depending on the available basal bone accommodation and perioral neuromuscular balance, but removal of some teeth does not replace mechanotherapy. However, it certainly can reduce significantly the amount of time and effort of final mechanotherapy.

Diagnostic procedures

Comprehensive orthodontic examination of children during the primary and mixed dentitions is discussed in chapter 3; the following is a brief discussion of the assessments specifically important for the serial extraction procedure.

Clinical examination

Clinical examination of the child for the possibility of serial extraction includes extraoral and intraoral examinations. The extraoral examination is used to evaluate the patient's facial morphology, symmetry, and proportion. The profile evaluation assesses the convexity and concavity of the profile and lower and upper facial heights. The lower mandibular border is examined for any vertical or horizontal growth pattern. The size and position of the lips, the relationship of the lips to the incisors, and the chin-nose relationship are evaluated. The tonicity of the lip musculature is an important factor in determining incisor inclination and position.

The intraoral examination includes assessment of the general health and hygiene of the soft and hard tissues, the patient's occlusion, tooth eruption and exfoliation, and the sequence and pattern of eruption. Different aspects of occlusion, such as crowding, spacing, overjet, overbite, and midline, both at rest and at function, are important points that must be carefully evaluated.

Paraclinical examinations

Paraclinical examinations can be performed with diagnostic tools such as study casts, photographs, and different radiographic techniques.

Study casts. Study casts reveal the type of occlusion, arch form and symmetry, the amount of arch length inadequacy (assessed with different space analyses to verify the severity of arch length discrepancy), and the kind of crowding (minor, moderate, or severe).

Photographs. Photographs can be used to distinguish facial form, symmetry, and proportion, as well as other components such as the lips, chin, and nose, with more accuracy than is provided by clinical evaluation.

Radiographs. Different types of radiographic images can reveal many undetected features that were not shown by clinical examination. A good panoramic radiograph is a very helpful and necessary tool, especially for determination of the serial extraction procedure. This radiograph reveals many abnormalities, obstacles, and pathologic conditions such as missing teeth, supernumerary teeth, odontoma, anterior crowding, bone loss, and many other conditions that can change the entire protocol for serial extraction.

Panoramic radiographs reveal all transitional stages of the dentition, including primary root resorption, stage of permanent root development, situation and condition of permanent successors, prediction of tooth emergence, and exfoliation and eruption patterns. Panoramic radiographs can also reveal radiographic signs of true hereditary crowding, which include palisading maxillary molars and ectopic maxillary molar eruption.

Longitudinal panoramic radiograph monitoring of the transitional dentition is an important step in a preventive protocol that the author always recommends to all practitioners who work with children during the transitional dentition. Longitudinal monitoring can play an especially important role in serial extraction procedures (see chapter 3). In this technique, three panoramic radiographs are taken, at ages 6, 8, and 10 years. By comparing this series of radiographs, the practitioner can discover many eruption problems at their initial stages of development and therefore intervene to prevent many future abnormalities.

Other standard radiographic techniques, such as periapical, bitewing, occlusal, and hand-wrist radiographs, have special indications as pretreatment guides in evaluation for the serial extraction procedure. Before any serial extraction

procedure is planned, the following important aspects must be evaluated radiographically:

- Tooth size–arch size discrepancies
- Tooth structure
- Dental health
- Periapical pathoses
- Tooth fracture
- Bone structure and pathologic lesion
- Dental age of the patient and stage of root development
- Eruption pattern of unerupted permanent teeth
- Maturational age of the patient and bone growth
- Size, shape, and relative positions of unerupted permanent teeth

Cephalometric radiographs. Cephalometric evaluation of dentoskeletal and soft tissue relationships is an important part of orthodontic diagnosis and treatment, especially in growing children who are undergoing serial extraction procedures. Dental occlusion is an integral part of craniofacial structure, and the interaction and coordination of skeletal growth changes during occlusal development are essential for establishing a normal and harmonious arrangement of dental occlusion.

The growth and development of craniofacial structures is not merely a progressive increase in size; instead, growth is a differential process in which some parts enlarge more or less than others and in a multitude of directions. The growth and development of various craniofacial bony structures happens under three essential processes: *(1)* increase in size by new bone formation; *(2)* change in shape by bone remodeling; and *(3)* displacement, which is a movement of whole bones away from one another at their articular junctions, as each undergoes a size increase and remodeling.

In some special areas of craniofacial structure, these three growth mechanisms can affect normal development of the dental occlusion. These areas include the anterior cranial base, posterior cranial base, cranial base angle, ramus of the mandible, corpus of the mandible, gonial angle, nasomaxillary complex, and maxillary and mandibular dentition. Therefore, cephalometric analysis of all craniofacial components, especially in young patients before serial extraction, can guide treatment planning according to the individual patient's growth pattern. The following are important values that have to be clarified by careful cephalometric

measurements before the serial extraction approach is undertaken:

- Dentoskeletal relationships
- Maxillary and mandibular jaw position and proportion in three dimensions
- Facial growth patterns
- Incisor inclination and position relative to basal bone
- Relationship of maxillary and mandibular incisors to profile
- Soft tissue profile, including lips, nose, and chin

Treatment planning

Proper planning for serial extraction requires adequate knowledge of many histologic and biologic aspects of development of the dentition: *(1)* tooth formation, *(2)* tooth eruption, *(3)* tooth exfoliation, *(4)* tooth replacement, *(5)* prediction of tooth emergence, *(6)* effects of premature loss of primary teeth on the permanent dentition, *(7)* arch dimension, *(8)* space loss, *(9)* nature of space closure, and *(10)* available methods of space analysis for tooth size prediction (see chapters 2 and 4).

Before treatment planning and any extraction, it is also important to evaluate many other factors that can affect the final results of treatment:

- Amount of crowding
- Patient's profile
- Type of occlusion
- Incisor inclination
- Dental age and root development
- Skeletal pattern
- Type of crowding

Amount of crowding

Measuring the amount of crowding and the amount of space available is the first step in determination of tooth extraction (see chapter 4 for different space analyses). According to the classification cited, severe crowding is defined as a space deficiency of at least 6 mm, but the amount of deficiency in millimeters alone is not a decisive factor. For example, for a patient with a 7-mm deficiency but retroclined

incisors or a dished-in or flat profile, it may be necessary to avoid extraction or change the type of extraction. In another patient with a 2- or 3-mm space deficiency and dental protrusion and a convex profile, an extraction approach may be the best choice.

Patient's profile

Another important consideration in treatment planning is the patient's soft tissue profile and the relationship of incisors to each other, to basal bone, and to the lips at rest and during a smile. Not only does tooth extraction have a direct effect on the profile, but the patient's profile is an important determining factor in extraction planning. A patient with a convex or slightly convex profile is a good candidate for serial extraction, while serial extraction is contraindicated in a patient with a flat or dished-in face. If extraction is an inevitable option for the latter type of patient, the practitioner must try to extract more posteriorly and design mechanotherapy in a way that will not disturb the profile.

Type of occlusion

Serial extraction should be planned according to dentoskeletal relationship. For example, in one patient with Class II division 1 malocclusion, it may be better to remove two maxillary first premolars and two mandibular second premolars. In another patient with Class II division 1 malocclusion but with severe overjet and a good mandibular dentition, extraction may be limited to the maxillary first premolars.

Incisor inclination

Incisor inclination also plays an important role in serial extraction procedures. Proclined incisors favor tooth extraction while uprighted or retroclined incisors contraindicate extraction.

Dental age and root development

The dental age of the patient and the amount of root development of permanent teeth are other factors to evaluate before serial extraction is planned. For example, extraction of primary molars depends on the sizes and stages of root formation of the premolars. If the primary molars are extracted too early, eruption of premolars may

be delayed. On the other hand, eruption is accelerated if primary molar extraction takes place after formation of half of the premolar root.

Skeletal pattern

The skeletal pattern of the patient is another important factor to evaluate before serial extraction. For example, tooth extraction in patients with a horizontal growth pattern is not the same as that in patients with a vertical growth pattern; mechanotherapy might also be different. In a patient with a vertical growth pattern and anterior open bite tendency, extraction of second premolars might be preferable if other factors allow it. In a patient with horizontal growth (low-angle cases), extraction must be avoided unless the patient has severe hereditary crowding and extraction is compulsory. In this situation, mechanotherapy must be designed in such a way to prevent abnormal incisor uprighting and disturbance of the patient's profile.

Type of crowding

Depending on the etiology and morphology of crowding, there are two general types of crowding: acquired crowding and hereditary crowding. Each kind of crowding can have completely different treatment options, and therefore differentiation of their special characteristics is essential for proper prevention or correction of problems.

Characteristics of acquired or environmental crowding. Acquired or environmental crowding is crowding that is caused by local or environmental factors, such as early loss of primary teeth or abnormal oral habits. In other words, patients with this type of crowding initially did not have discrepancy between the available space and tooth material; however, as a result of some local factor, such as caries, trauma, or early primary tooth loss, and subsequent neglected intervention, their dentition now shows crowding and space deficiency.

Management of environmental crowding most often involves regaining the lost space and providing necessary space for unerupted or crowded teeth through early mechanical intervention. Some environmental crowding, if neglected for a long time, may change to severe crowding that requires extraction for treatment.

There are many environmental factors that can, if detected early, prevent future crowding or, if neglected, produce crowding and other abnormalities. Following is a list of local factors that must be detected and managed at the proper time:

- Overretained primary teeth

- Oversized restorations and crowns
- Congenitally missing teeth (see chapter 7)
- Supernumerary teeth (see chapter 8)
- Trauma
- Aberrant tooth shape
- Tooth transposition (see chapter 10)
- Tooth rotation
- Abnormal sequence of eruption of permanent teeth
- Premature loss of primary teeth (see chapter 4)
- Space loss due to interproximal caries (see chapter 4)
- Uneven root resorption of primary teeth
- Abnormal path of eruption of permanent teeth (see chapter 10)
- Constricted dental arches as a result of habits, airway obstruction, or muscle imbalance (see chapter 6)
- Discrepancy in the size of individual teeth (anterior Bolton discrepancy; see discussion later in this chapter)

Each of these factors, the consequences for occlusion if intervention is delayed or neglected, and their management are discussed in the related chapters.

Characteristics of hereditary crowding. Hereditary crowding, which has a genetic background, is caused by tooth size–arch size discrepancy and most of the time is a candidate for an extraction approach. Dale[25] mentioned some clinical and radiographic signs that can help in early recognition of this type of crowding:

- Premature exfoliation of the primary canines: Ideally, when the permanent mandibular incisors erupt, the mechanisms of interdental spacing between the primary incisors, including primate spaces[1] and secondary spacing,[12] can accommodate the larger permanent incisors. However, when incisors are large and there is some inadequacy of anterior basal bone, the force of lateral incisor eruption causes early root resorption and exfoliation of mandibular primary canines.
- Crescent moon–shaped canine root resorption: This form of resorption on the mesial aspect of the primary canine roots is a sign of hereditary crowding that can be detected by radiograph during eruption of the mandibular lateral incisors (Fig

5-8).

- Displacement of the mandibular midline and blocking out of one lateral incisor: This situation is another obvious sign of hereditary crowding. It occurs after early exfoliation of the primary canine on one side and a rapid midline shift due to space deficiency. In cases of bilateral early exfoliation of the primary mandibular canines, lip pressure can displace the mandibular incisors lingually, resulting in an upright positioning of the permanent mandibular incisors and increasing overjet. Early detection and insertion of lower lingual holding arch can prevent this abnormality.
- Gingival recession at prominent mandibular incisors: In severe mandibular incisor crowding, one or more incisors may be pushed labially and, due to deficiency of bone support, suffer some gingival recession (Fig 5-9).
- Prominent bulging of unerupted canines: Because the maxillary permanent canines are the last permanent teeth to erupt (excluding third molars), the eruption of incisors and premolars in a situation of space deficiency will push the maxillary canines out of the arch. Occasionally, mandibular canines can be out of the arch and exhibit a prominent bulge due to space deficiency, especially if the sequence of eruption is also disturbed (Fig 5-10).
- Splaying of lateral incisors: Crowding of the incisors and canines and a more horizontal inclination of the canines force the lateral incisor root mesially and cause splaying of the lateral incisor crowns. The resulting Class II division 2 relationship of the lateral incisors is another sign of hereditary crowding (Fig 5-11).
- Ectopic eruption of the maxillary permanent first molars and premature exfoliation of the maxillary primary second molars: Ectopic eruption is an alteration of the first molar eruption pathway, evident most often in the maxilla, that causes premature resorption of the primary second molar teeth. This abnormality can have different causes; one is deficient growth of the tuberosity. Therefore, the presence of this abnormality may be a sign of arch length deficiency and thus hereditary crowding (Fig 5-12).
- Vertical palisading of the maxillary molars in the tuberosity area: This palisading relationship can be easily detected by evaluation of panoramic radiographs. This situation also indicates a lack of sufficient growth of maxillary bone (Fig 5-13).
- Impaction of the mandibular permanent second molars: Crowding in the mandibular molar area, especially impaction of mandibular permanent second molars, is another sign of hereditary crowding that indicates growth deficiency of

the mandible and improper bone remodeling of the anterior border of the ramus. This abnormality is also easily detected on panoramic radiographs. Early detection and intervention can prevent many future eruption problems (Fig 5-14; for more details, see chapter 3 for longitudinal panoramic radiograph monitoring).

- Anterior dentoalveolar protrusion without spacing: Regular dental protrusion of incisors in patients with lip dysfunction, a thumb sucking habit, or a Class II division 1 malocclusion is usually accompanied by some interdental spacing. In incisor protrusion caused by hereditary crowding, there is no spacing or there may even be overlapping of the incisors.

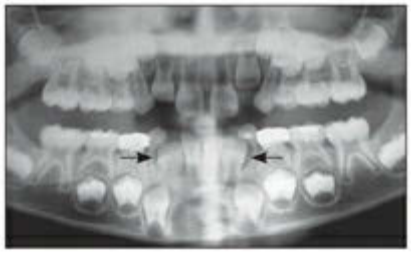


Fig 5-8 Crescent moon–shaped resorption of the primary canines *(arrows)*, a sign of hereditary crowding.

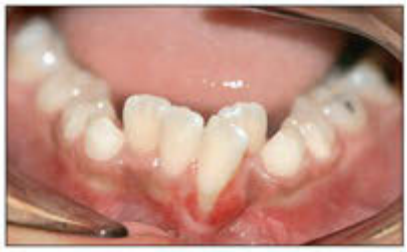


Fig 5-9 Bone recession caused by severe crowding.

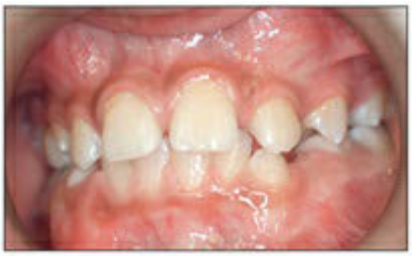


Fig 5-10 Prominent canine bulge.

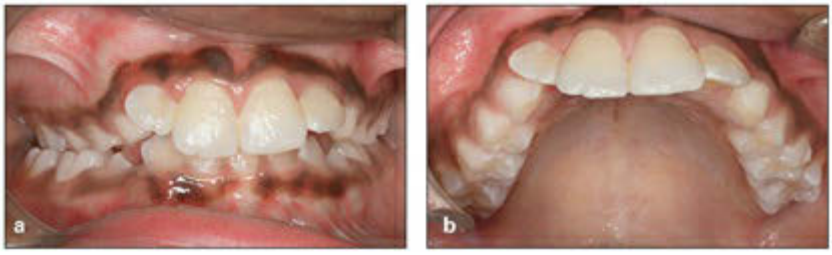


Fig 5-11 *(a and b)* Splayed incisor crowns.

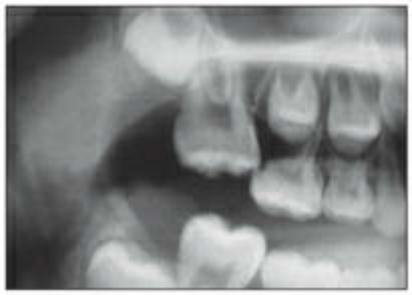


Fig 5-12 Ectopic eruption of the permanent first molar.

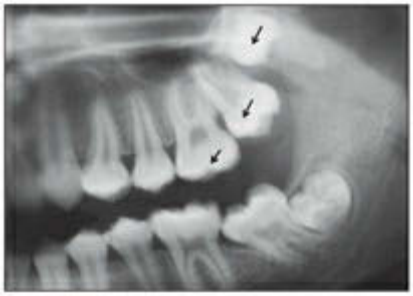


Fig 5-13 Vertical palisading of the maxillary molars (arrows) and molar crowding.

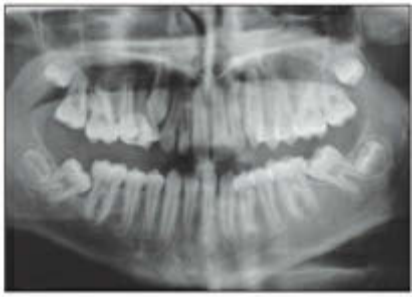


Fig 5-14 Impaction of the permanent second molars.

Serial extraction in patients with Class I malocclusions

After comprehensive examination and analysis and before any treatment planning for serial extraction, two important issues must be verified:

- The exact etiology, morphology, and type of problems, that is, the amount of crowding, the type of crowding, and the dentoskeletal and soft tissue relationships
- The primary and permanent tooth relationships and the location of crowding in the arch

The most ideal conditions for performing successful serial extraction are a Class I malocclusion with severe hereditary crowding, minimal overjet and overbite, normal basal jaw relationships, and an orthognathic facial pattern or a slight maxillomandibular dentoalveolar protrusion. In this type of malocclusion, proper serial extraction would need minimal mechanotherapy.

In everyday practice, however, clinicians are faced with many different types of conditions that, depending on the eruption pattern and place of crowding in the dental arch, may require a different type of extraction sequence between the primary and permanent teeth. In other words, there is no one single prescription to write for all patients undergoing serial extraction procedures.

In patients with marked incisor crowding during the early mixed dentition, when space analysis and all paraclinical examinations confirm a severe space deficiency and tooth extraction is the best choice to relieve the severe arch length discrepancy, two questions arise:

1. When should the serial extraction procedure be started?
2. What sequence of extraction is correct?

Timing of serial extraction

The timing of serial extraction procedures depends on two conditions: *(1)* the eruption of incisors and *(2)* the stage of root development of the premolars. If all incisors are erupted and the premolar roots are half developed, it is time to start the procedure.

There are special conditions under which this procedure can be started earlier. For example, if the incisors are not fully erupted or are erupting lingually because of severe space deficiency, then incisor eruption must be facilitated first by guidance of eruption, as discussed earlier. These procedures can include sequential stripping or extraction of overretained primary incisors or extraction of primary canines and insertion of a holding arch or 2 × 4 bonding to maintain arch circumference and prevent lingual displacement of permanent incisors and space loss. Then, at the proper time, serial extraction is continued.

Sequence of serial extraction

There is no one prescription for the order of serial extractions; it may be started with the primary canines, the primary first molars, the primary canines and first molars

together, or even the primary second molars. The proper sequence depends on *(1)* the location of crowding, *(2)* the sequence of permanent tooth eruption, *(3)* the general amount of crowding, *(4)* the amount of arch space deficiency, and *(5)* the patient's dentoskeletal occlusion.

Extraction of primary canines first. In this situation, the primary goal is to facilitate unraveling of the incisors and influence eruption of the permanent first premolars, to facilitate eruption and distal movement of the permanent canines into the premolar spaces.

Figure 5-15a shows a situation of severe incisor crowding. The first step of treatment is extraction of the primary canines to provide space for the incisors to unravel and to accelerate permanent canine eruption. Figure 5-15b shows the situation after extraction of the primary canines. Extraction has resulted in alignment of the incisors, some root lengthening, and some eruption of the permanent canines and premolars. Figure 5-15c shows the conditions after extraction of the primary first molars. The primary first molars are extracted when one-half to two-thirds of the first premolar root is formed to accelerate eruption of the first premolars. The permanent first premolars are extracted after eruption to facilitate eruption of the canines into the remaining extraction spaces. Figure 5-15d illustrates the final stage of serial extraction and guidance of eruption, which will be continued with a second phase of treatment that includes full bonding for root paralleling, space closure, if necessary, and final occlusal adjustment.

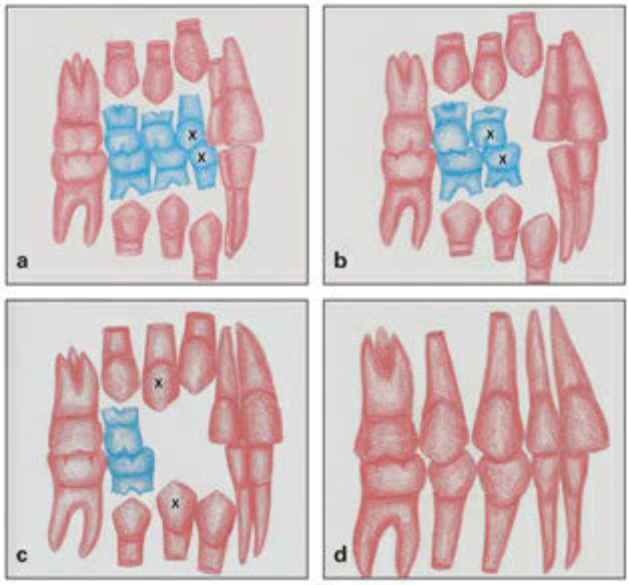


Fig 5-15 *(a)* Extraction of the primary canine to unravel crowded incisors and accelerate canine eruption. *(b)* Condition after extraction of the canines and before extraction of the primary first molars. The incisors have started to unravel, and the canines have erupted further. *(c)* Condition after extraction of the primary first molars, to accelerate premolar eruption, and before extraction

of the first premolars, to facilitate canine eruption. *(d)* Condition after eruption of all permanent teeth and before bonding of appliances for final occlusal adjustment. (Courtesy of Dr Cynthia Wong, Rochester, New York.)

To prevent eruption problems and facilitate automatic alignment of the dentition, the author always recommends use of some kind of temporary anchorage (maxillary transpalatal arch, lower holding arch) during the stages shown in Figs 5-15a to 5-15c. Patients with a Class I malocclusion, without severe incisor crowding, and without muscle dysfunction may not need anchorage; it can be eliminated to allow some automatic space closure.

In patients with a Class I malocclusion, severe incisor crowding (around 10 mm), and no skeletal discrepancy, extraction therapy with good anchorage can result in a very good occlusion with no need for a second phase of treatment or a very limited time of mechanotherapy (see case 5-5).

Extraction of primary molars first. To prevent an abnormal sequence of eruption of the permanent teeth during serial extraction, it may be necessary to change the extraction sequence, that is, extract the primary first molars first and then the canines. The normal sequence of eruption in the maxilla is eruption of first and second premolars before the canines, and usually there is no problem during serial extraction. However, in the mandibular arch, the canines normally erupt before the first premolars. In cases of severe crowding, the canine will often be displaced facially or rarely lingually. To avoid this abnormality, extraction of the mandibular primary first molar when the root of the premolar is at least half formed is recommended to accelerate premolar eruption. Subsequent extraction of the premolars will result in early eruption of the canines and their movement to the premolar space.

Figure 5-16a shows a situation in which the permanent canine is located below the premolar and has the potential to be displaced. Figure 5-16b shows the condition after the primary first molars have been extracted, accelerating the eruption of the first premolars. The primary canines are still present, maintaining the position of the permanent incisors to prevent lingual tipping and development of a deep bite. Figure 5-16c shows the condition after extraction of the primary canines and advanced eruption of the first premolars. This stage will be followed by extraction of the first premolars. Figure 5-16d shows the complete eruption of all permanent canines and second premolars, before full appliance therapy is implemented for final space closure and uprighting.

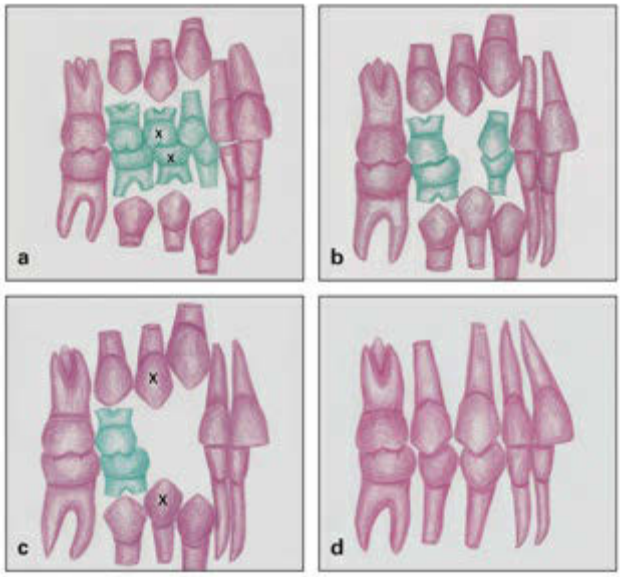


Fig 5-16 *(a)* Extraction of the primary first molars before the primary canines, to prevent crowding of the first premolars in situations where crowding is not severe. *(b)* Condition after primary first molar extraction. Eruption of the first premolars has accelerated. *(c)* Condition before extraction of the first premolars. *(d)* Condition after complete eruption of all permanent canines and second premolars and before full appliance therapy for final space closure and uprighting. (Courtesy of Dr Cynthia Wong, Rochester, New York.)

Figure 5-17 shows another type of serial extraction that can be implemented in some patients with severe incisor crowding that has caused early exfoliation of the primary canines and early eruption of the permanent canines, which occupy the premolar spaces and therefore are causing impaction of the first premolars. The treatment of choice in this situation is extraction of the primary second molars and simultaneous enucleation of the first premolars. This procedure is accompanied by placement of a lower holding arch.

Fig 5-17 Severe incisor crowding, early loss of primary canines, and early eruption of permanent canines, resulting in crowding and impaction of the first premolars. The recommended sequence of extraction is extraction of the primary second molar and enucleation of the first premolar. This should be accompanied by placement of a lower holding arch. (Courtesy of Dr Cynthia Wong, Rochester, New York.)

Wrong serial extraction of primary canines without application of a holding arch

or too early extraction of first primary molars can also cause early eruption of the permanent canines and impaction of the first premolars.

In cases of severe incisor crowding and lack of space for lateral incisor eruption, early exfoliation of primary canines may occur, or early intervention may require early extraction of primary canines; in any of these conditions, inserting a holding arch before primary canine extraction or exfoliation is highly recommended.

The best early intervention in these types of problems is the following protocol:

1. Insertion of a lower holding arch before exfoliation or extraction of primary canines, to hold arch circumferences.
2. Extraction of the primary first molar to accelerate eruption of the first premolar.
3. Extraction of the first premolar, which prevents later surgical removal and facilitates and guides canine eruption.

The perception that serial extraction is an easy procedure and requires no mechanotherapy is incorrect, and mismanagement can result in a complicated situation.

As discussed earlier, the author always recommends anchorage preparation during the early stages of extraction, but it is also important to remember that with serial extraction there is always a tendency toward increasing the overbite.

This tendency might be beneficial in cases of vertical problems and open bite but will certainly cause problems in patients with horizontal growth patterns, leading to more deep bite and requiring more effort in a second phase of treatment. For these patients, application of a Hawley anterior bite plate may be beneficial because it can *(1)* provide anchorage and act as a holding arch in the maxillary dentition and *(2)* prevent overbite increase and correct deep bite.

Serial extraction in patients with Class II division 1 malocclusions

As discussed earlier, the most ideal condition for successful serial extraction is a Class I malocclusion with severe hereditary crowding, minimal overjet and overbite, normal basal jaw relationships, and an orthognathic facial pattern or a slight maxillomandibular dentoalveolar protrusion. Nevertheless, even in patients with Class II and Class III malocclusions, if all evaluations confirm space inadequacy during the mixed dentition, then reducing the number of teeth at the

proper time in conjunction with mechanotherapy not only can solve crowding problems and facilitate eruption but also can help final coordination of occlusion. However, serial extraction in patients with different malocclusions is much more complicated than it is in patients with Class I malocclusion and demands careful management and caution at every step of the procedure.

Serial extraction can be performed in patients with Class II division 1 malocclusion and severe space deficiency, but extreme caution must be taken not to aggravate the Class II relationship by causing lingual collapse of the mandibular incisors. Depending on the type of Class II malocclusion and growth pattern of the patient, the steps of treatment vary greatly. Treatment might involve extractions in one arch or both arches, and it might involve first premolar or second premolar extractions.

For this type of problem, there are usually two phases of treatment and one phase of observation, or interim phase. The initial period of interceptive treatment may extend 1 to 18 months.

Maxilla

1. Initial phase:
   - Anchorage preparation.
   - Extraction of the maxillary primary first molars.
   - Extraction of the maxillary first premolars to provide space for canine eruption.
2. Interim phase, observation:
   - Waiting for canine eruption.
   - Depending on the situation of the maxillary incisors, 2 × 4 bonding can be applied, keeping anchorage or headgear if needed for anterior alignment and retraction if the incisors are spaced and proclined before permanent canine eruption.
3. Second phase:
   - Permanent canine alignment and retraction, if needed.
   - Incisor retraction.

Mandible

1. Initial phase:
   - Anchorage preparation (lingual lower holding arch), which is very important to prevent any uprighting or lingual movement of the mandibular incisors and

thereby to prevent aggravation of the Class II relationships.

- Extraction of the primary canines if the mandibular incisors are crowded or extraction of the mandibular primary first molars and finally the first premolars.
- Banding and bonding of the mandibular permanent first molars and primary second molars and incisors, to level and preserve arch circumferences and progress to an ideal arch.

2. Interim phase, observation:
   - After extraction of the primary first molars, primary canines, and premolars and subsequent alignment of the permanent incisors, if the permanent canines are erupting and ready for bonding, canine alignment can be continued before retraction.
   - If the canines are high and not ready to erupt, an observation or interim phase can be instituted. During this period, retentive appliances are worn, and patients are examined every 3 months for assessment of growth and occlusal development.
3. Second phase, final stage:
   - Multibonding when all the permanent teeth are erupted.
   - Uprighting, final leveling, and space closure.

Serial extraction in patients with Class II division 2 malocclusions

Class II division 2 malocclusions often are characterized by deep bite, a convergent face, prominent pogonion, and a dished-in profile. These patients are not good candidates for serial extraction strategy.

The best choice for patients with this type of malocclusion is early intervention, and every effort should be made to treat with a nonextraction protocol, such as 2 × 4 bonding and an anterior bite plate to reduce overbite and head-gear to distalize the maxillary molars and achieve Class I molar relationships (see chapter 11).

Occasionally clinicians are faced with a patient who has a Class II division 2 malocclusion accompanied by severe space deficiency during the mixed dentition, and extraction of the teeth is inevitable. Every effort should be made to prevent any worsening of the dished-in face by maintaining the mandibular incisors as far mesially as possible and preserving the stability of the force produced by the perioral musculature.

Extraction therapy in Class II division 2 malocclusions should be limited to cases of true, severe hereditary tooth size–arch size discrepancy.

1. Initial phase:

Maxilla

- Cervical headgear and anterior bite plate to correct the molar relationship, reduce overbite, and rotate the mandible to reduce chin prominence. Extraction of primary and permanent teeth can follow the same procedure as in treatment of Class II division 1 patients.
- Bonding of the maxillary anterior segment and primary second molars after correction of molar relationships.
- Leveling and some anterior intrusion, if needed. End use of the cervical headgear, anterior bite plate, and rectangular archwire and start of retraction and torque with a high-pull J-hook.

Mandible

- Banding and bonding of permanent first molars, primary second molars, and the anterior segment.
- Leveling and progression to an ideal edgewise archwire to upright the incisors and maintain arch length.
- Extraction of primary first molars or canines, if needed.

2. Interim phase, observation: Placement of a maxillary Hawley anterior bite plate and lower lingual holding arch or fixed canine to canine retainer to prevent anterior collapse.
3. Second phase, final stage:
   - Full bonding, including permanent second molars, for leveling and uprighting to prevent deep bite.
   - Multibonding when all the permanent teeth are erupted, for leveling, anterior retraction, and maintenance of necessary root torquing.

Serial extraction in patients with open bite

Depending on whether the problem is dental, dentoskeletal, or skeletal, and depending on the severity of crowding, different extraction procedures can be performed. The first or second premolars or even the permanent first molars can be extracted for correction of severe open bite.

For all of these vertical problems, careful analysis, anchorage preparation, and prevention of any posterior extrusion are key to success (see chapter 13).

Serial extraction in patients with Class III malocclusions

Crowding in Class III malocclusions has different characteristics that require different extraction procedures. In patients with a pseudo Class III malocclusion, serial extraction can be performed if hereditary crowding is confirmed but only after correction of anterior crossbite. Any mandibular shift must be eliminated first. Then the position of the maxillary and mandibular incisors relative to the basal bone and soft tissue profile must be reevaluated so that the conditions of crowding and the proper type of extraction can be determined.

The serial extraction approach in Class III malocclusions is different regarding which jaw requires extraction and which tooth must be extracted. In patients with a Class III malocclusion and mandibular dentoalveolar protrusion, extraction only in the mandibular arch is usually desirable to improve the final incisor relationship.

In patients with a true Class III malocclusion with mandibular prognathism, early extraction is not recommended because the mandibular incisors may already be retroclined; comprehensive treatment and even orthognathic surgery may be advised.

Patients with a Class III malocclusion and true hereditary crowding in the maxillary and mandibular arch, mandibular crowding with dentoalveolar protrusion, or severe maxillary crowding might be treated with four premolar extractions (four first premolars or mandibular first and maxillary second premolars).

It should be emphasized that for all skeletal problems, serial extractions facilitate the correction of a true hereditary tooth size–arch size discrepancy. The skeletal problems themselves cannot be corrected by extraction alone.

Early Intervention in Severe Space Deficiency

As discussed earlier, when severe space deficiency is found to be the result of a tooth size–arch size discrepancy, the best solution is to reduce the number of tooth units through serial extraction. The best timing for this procedure is during the middle or late mixed dentition. Early detection of these cases is not difficult, and proper management can reduce the mechanical effort needed during the second

phase. In some cases, a second phase of treatment may not even be necessary.

Case 5-4

A 10-year-old girl exhibited a Class I malocclusion, severe maxillary and mandibular crowding, slight incisor proclination, mandibular molar crowding, and palisading maxillary molars (Figs 5-18a to 5-18f).

Treatment:
The treatment plan called for serial extraction. Treatment was started with anchorage preparation and then extraction of the maxillary primary canines and permanent first premolars, which had already erupted. Subsequently, extraction of the mandibular primary canines and primary first molars was followed by extraction of both mandibular first premolars. Figures 5-18g to 5-18m illustrate the posttreatment results.

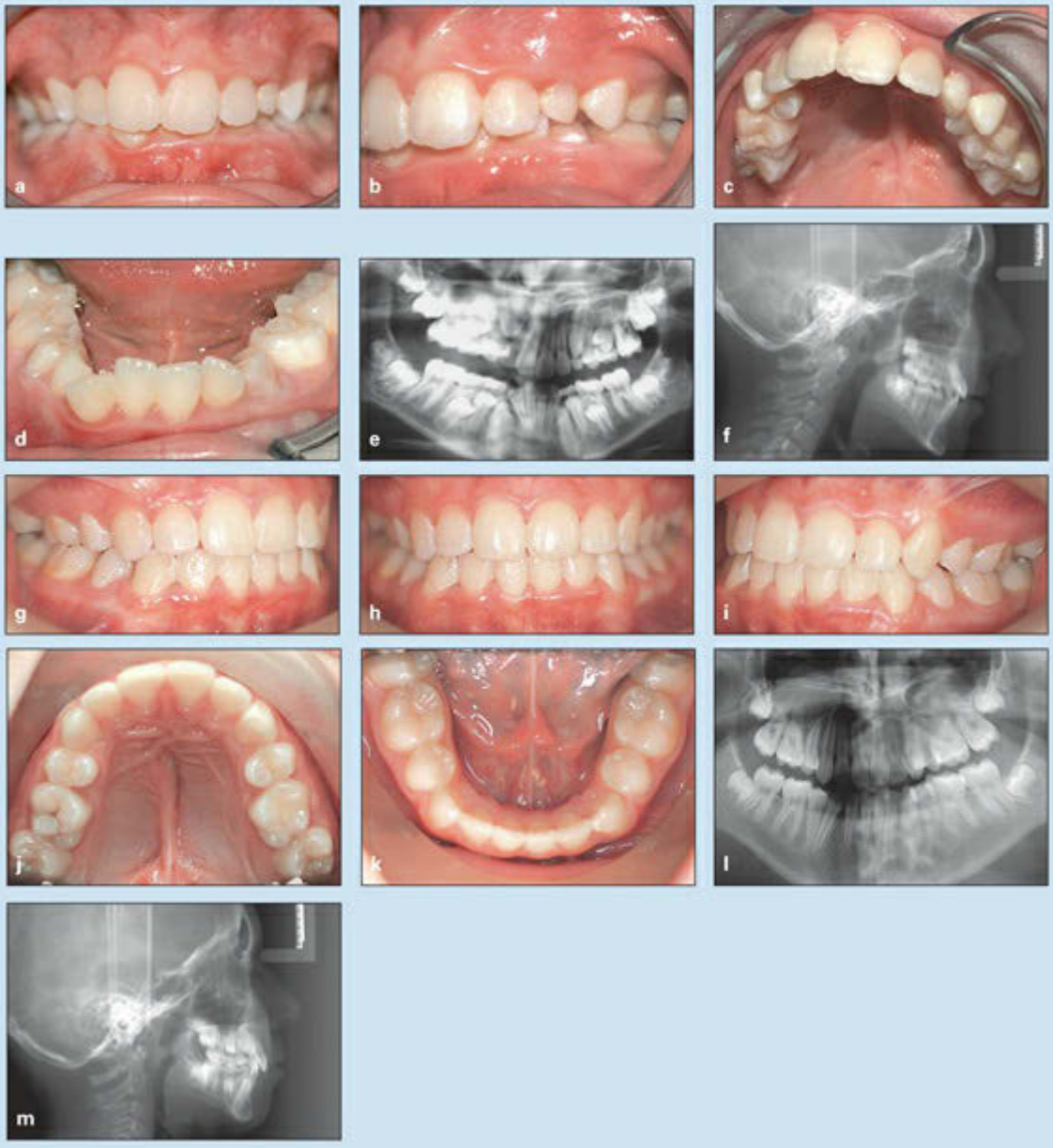


Fig 5-18 Treatment of a 10-year-old girl with a Class I malocclusion, severe maxillary and mandibular crowding, and deep bite. *(a to d)* Pretreatment occlusion. *(e)* Pretreatment panoramic radiograph, revealing palisading and crowded molars. *(f)* Pretreatment cephalometric radiograph, revealing slight incisor proclination. *(g to k)* Posttreatment occlusion after serial extraction of all four first premolars. *(l)* Posttreatment panoramic radiograph. *(m)* Posttreatment cephalometric radiograph.

Case 5-5

A 10-year-old girl with a Class I malocclusion during the middle mixed dentition presented with severe maxillary and mandibular crowding, insufficient space for the permanent canines, and some profile convexity (Fig 5-20a).

Treatment:
Treatment was planned as a two-phase procedure. Phase 1 included anchorage preparation and sequential extraction of primary first molars, primary canines, and permanent first premolars. Phase 2 would have involved full bonding for minor tooth movement and finishing. Unfortunately, because of a lack of insurance coverage, only phase 1 of treatment was accepted by the parents.

Figures 5-19b to 5-19d show different stages of treatment: primary tooth extraction and subsequent placement of anchorage appliances. A reverse transpalatal arch was used to hold the molars and correct rotation and tipping in the maxilla, and a lower holding arch was used in the mandible. Figures 5-19e to 5-19h illustrate the patient's occlusion after eruption of all premolars and before extraction of the first premolars.

Figures 5-19i to 5-19n show the conditions during the final stage of active treatment. All second premolars and canines (except on the maxillary right side) have erupted. Both the transpalatal arch and the lower holding arch have been removed to allow occlusal settling and self-closure of remaining spaces. Considering the limitations of treatment in this case, with only phase 1 treatment allowed and the use of only two holding arches, the results are acceptable.

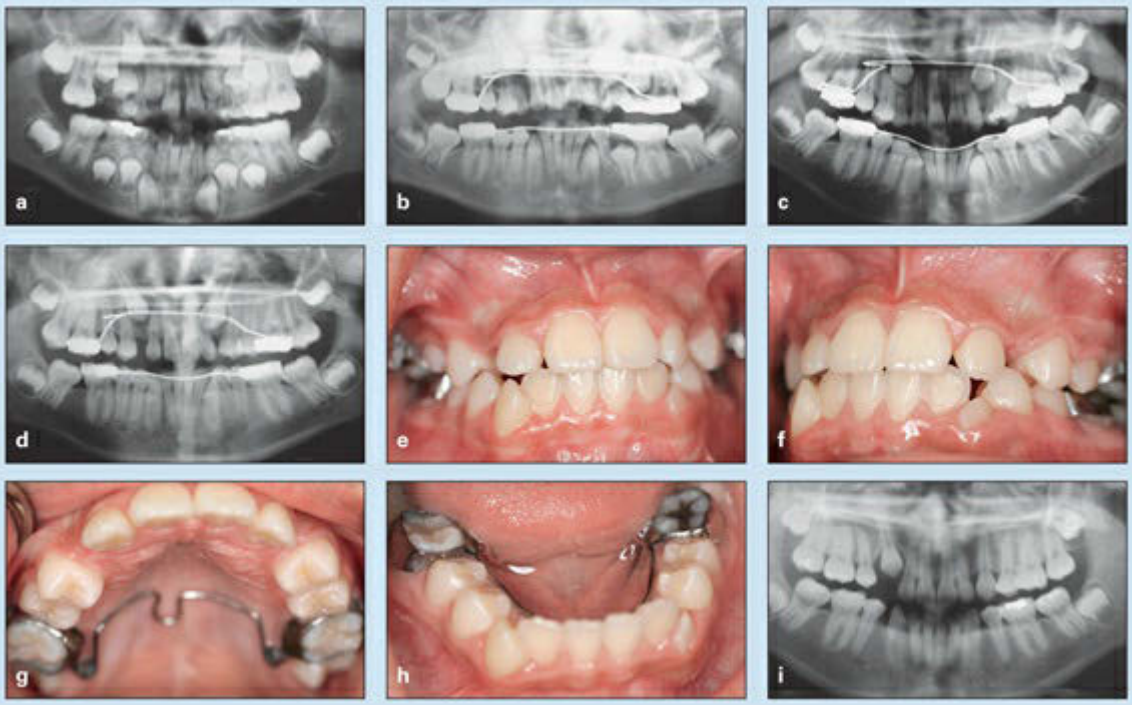


Fig 5-19 Treatment of a 10-year-old girl with a Class I malocclusion during the middle mixed dentition. *(a)* Pretreatment panoramic radiograph, revealing severe and insufficient space for eruption of the permanent canines. *(b and c)* Panoramic radiographs taken during the stages of primary tooth extraction. *(d)* Panoramic radiograph after eruption of the first premolars and before their extraction. *(e to h)* Anchorage preparation before extraction of the first premolars. *(i)* Panoramic radiograph after removal of four premolars, eruption of the permanent dentition, removal of anchorage to allow self-closure of space, and occlusal settling.

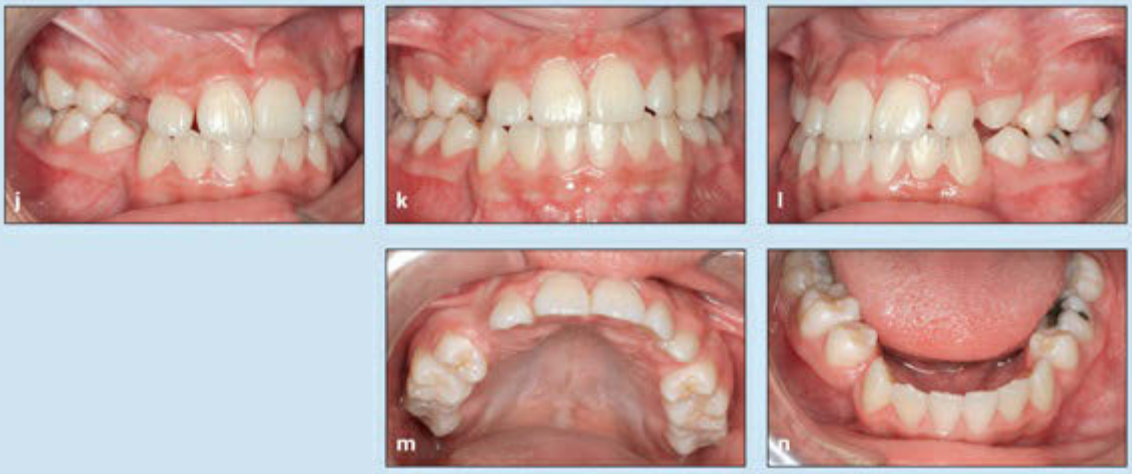


Fig 5-19 *(cont) (j to n)* Occlusion after removal of the transpalatal arch and lower holding arch. All premolars and canines (except the maxillary right side) have erupted. Very minor rotation of the canine is present.

Mandibular Incisor Crowding Caused by Bolton Discrepancy

A rare kind of mandibular incisor crowding is severe crowding caused by mandibular incisor size discrepancy (Bolton discrepancy). The following are specific characteristics of this type of malocclusion:

- Class I dental malocclusion with severe incisor crowding
- Absence of skeletal discrepancy
- Good interdigitation and normal molar and canine relationships
- Good maxillary dentition
- Normal overjet, overbite, and especially anterior end-to-end incisor relationships
- Normal profile

None of the treatment options discussed earlier, including proximal stripping,

sagittal or transverse expansion, or serial extraction, can solve this problem with perfect dental relationships and perfect functional and esthetic results. As a result of clinical experience that led to good results, the author introduced the strategy of extracting one incisor as an ideal technique for treatment of this type of malocclusion.[17] This technique has since been cited by many investigators.[26–29]

Extraction of a single mandibular incisor can be employed as a compromise treatment for certain malocclusions, if the end result will fulfill the requirements for a healthier dentition that is functionally and esthetically harmonious with the surrounding structures. Class I malocclusions with a normal maxillary dentition and good buccal interdigitation in which there is severe mandibular anterior crowding are the best cases for this procedure, provided that the mandibular anterior arch length deficiency is greater than 5 mm and the anterior tooth ratio according to Bolton analysis is more than 83 mm. This procedure also can be applied in selected adults with a tendency toward mild to moderate Class III malocclusion and reduced overjet and overbite.

For patients who exhibit a deep overbite pattern, extraction of the mandibular anterior unit should be avoided. Furthermore, the author does not regard mandibular incisor extraction to be a routine orthodontic procedure but rather a measure of almost last resort, because it involves the most important stabilizing area of occlusion. Careful torque control of all mandibular teeth, particularly the canines, is required throughout the treatment period, as is placement of fixed canine-to-canine lingual retention.

Patients with mandibular incisor crowding resulting from Bolton discrepancy usually are treated during the permanent dentition. The following case reports describe two patients who were treated with extraction of one mandibular incisor.

Case 5-6

A 22-year-old man with a Class I malocclusion exhibited severe mandibular anterior crowding. Both canines were out of the arch because of a Bolton discrepancy. He also had minor maxillary crowding (Figs 5-20a to 5-20c).

Treatment:
In keeping with the patient's wishes, treatment was limited to the mandibular arch. Because the dental midline had shifted to the right and there was more crowding in the left segment, the mandibular left central incisor was extracted. There was no anchorage. Figures 5-20d and 5-20e show the posttreatment occlusion.

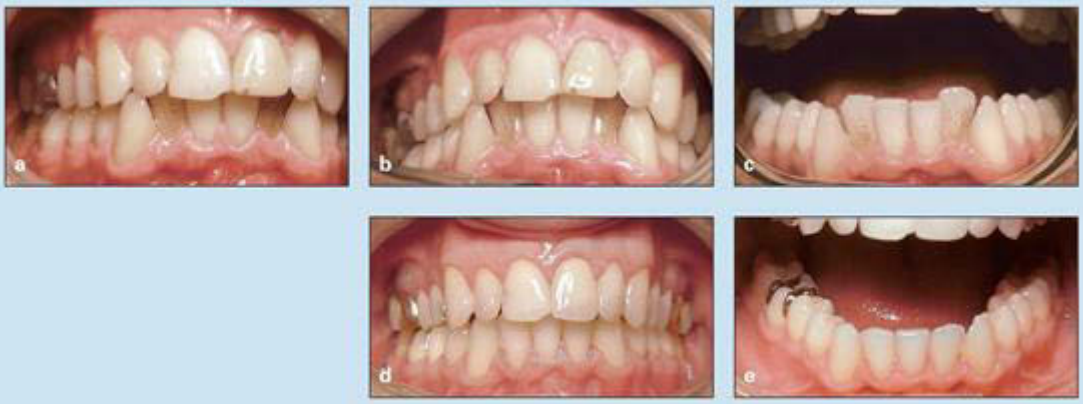


Fig 5-20 Treatment of a 22-year-old man with a Class I malocclusion and a Bolton discrepancy that has resulted in severe mandibular anterior crowding, with both canines out of the arch, and minor maxillary anterior crowding. *(a to c)* Pretreatment occlusion. *(d and e)* Posttreatment occlusion after extraction of one mandibular incisor.

Case 5-7

A 26-year-old woman with a Class I malocclusion had severe mandibular incisor crowding caused by a Bolton discrepancy and minor rotation of the maxillary lateral incisors (Figs 5-21a to 5-21c).

Treatment:
Treatment involved only mandibular bonding and extraction of the mandibular right central incisor. No anchorage was applied. A Hawley maxillary appliance was used to align the maxillary lateral incisors after alignment of the mandibular canines. Figures 5-21d to 5-21f show the posttreatment occlusion.

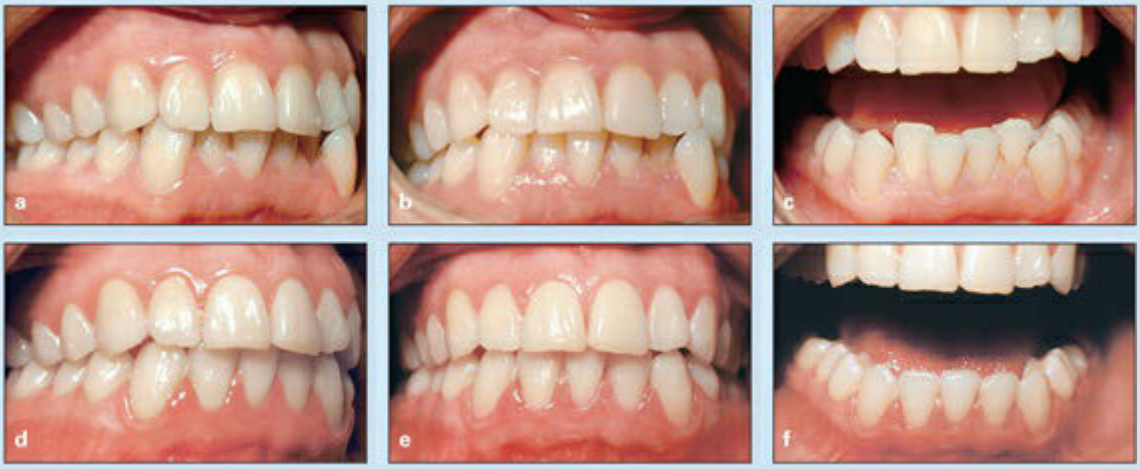


Fig 5-21 Treatment of a 26-year-old woman with a Class I malocclusion and severe incisor crowding resulting from a Bolton discrepancy. *(a to c)* Pretreatment occlusion. *(d to f)* Posttreatment occlusion after extraction of one mandibular incisor.

Summary

- Mandibular incisor crowding is identified as a discrepancy between the mesiodistal tooth widths of the four permanent incisors and the space available between the mesial surfaces of the primary canines.
- Because of size discrepancies between the primary and permanent incisors, some degree of incisor crowding during the early mixed dentition is common.
- Not all incisor crowding is alike; hence, treatment options depend on the amount, etiology, and morphology of crowding and other variables associated with this problem.
- Incisor crowding is not merely the result of a tooth size–arch size discrepancy; many variables, such as early loss of primary molars, direction of mandibular growth, incisor and molar inclination, and oral and perioral musculature, can be associated with crowding.
- Predicting permanent incisor crowding at an earlier stage has been a goal. The literature indicates a relationship of crowding to other criteria such as primary intercanine width, primary intermolar width, permanent intermolar width, and total arch length, all of which are greater in patients without crowding. Other reports have indicated larger angles between sella-nasion and the mandibular and occlusal planes in patients with crowding during both the mixed and permanent dentition stages. Baume[1] stated that children have a 40% possibility of crowding of their permanent incisors if the primary dentition has no spacing.
- Patterns of jaw growth influence tooth eruption. Clockwise rotation causes incisors to thrust forward; counterclockwise rotation causes lingual displacement of the incisors relative to the maxilla and mandible and increases the tendency toward crowding.
- There are also conflicting results in the literature, and there is no precise criterion to predict incisor crowding. In fact, even if crowding could be predicted, there is little that can be done as prevention before incisor eruption.
- Therefore, for proper treatment planning and ideal interception, the first step is to identify the amount and type of crowding.
- Crowding according to tooth size–arch length discrepancy has been classified into two general types: acquired crowding and hereditary crowding.
- The amount of crowding and space availability can be determined by different space analyses. For practical purposes, incisor crowding can be

arbitrarily divided into three types: minor crowding (less than 3 mm), moderate crowding (3 to 5 mm), and severe crowding (more than 6 mm).

- The amount of crowding (in millimeters) is not the only decisive consideration for treatment planning; many other factors must be evaluated and considered, including the patient's profile, the position and inclination of the incisors, the relationship of the incisors to the profile, the tonicity of the lip musculature, and the patient's growth pattern.
- Research reports indicate that minor crowding is usually self-corrected in the presence of other physiologic factors, such as primate spacing and intercanine width increases.
- Moderate crowding can also be corrected by space supervision, such as preservation of leeway space.
- If severe incisor crowding is determined to be the hereditary type of crowding, the serial extraction approach can be implemented.
- Hereditary crowding is caused by a tooth size–arch size discrepancy, which has certain clinical and radiographic characteristics.
- Acquired crowding is the result of environmental factors such as early tooth loss, abnormal habits, and lip dysfunction.

References

1. Baume LJ. Physiological tooth migration and its significance for the development of occlusion. 1. The biogenetic course of the deciduous dentition. J Dent Res 1950;29:123–132.
2. Hunter WS. Application of analysis of crowding and spacing of the teeth. Dent Clin North Am 1978;22:563–577.
3. Sampson WJ, Richards LC. Prediction of mandibular incisor and canine crowding changes in the mixed dentition. Am J Orthod 1985;88:47–63.
4. Howe RP, McNamara JA, O'Connor KA. An examination of dental crowding and its relationship to tooth size and arch dimension. Am J Orthod 1983;83:363–373.
5. Bishara SE, Khadivi P, Jakobsen JR. Changes in tooth size-arch length relationships from the deciduous to the permanent dentition: A longitudinal study. Am J Orthod Dentofacial Orthop 1995;108:607–613.
6. Sinclair PM, Little RM. Maturation of untreated normal occlusions. Am J Orthod 1983;83:114–123.
7. Melo L, Ono Y, Takagi Y. Indicators of mandibular dental crowding in the mixed dentition. Pediatr Dent 2001;23:118–122.
8. Björk A. The use of metallic implants in the study of facial growth in children: Method and application. Am J Phys Anthropol 1968;29:243–254.
9. Leighton BC, Hunter WS. Relationship between lower arch spacing/crowding and facial height and depth. Am J Orthod 1982;82:418–425.
10. Sayin MO, Türkkahraman H. Factors contributing to mandibular anterior crowding in the early mixed dentition. Angle Orthod 2004;74:754–758.
11. Türkkahraman H, Sayin M. Relationship between mandibular anterior crowding and lateral dentofacial

morphology in the early mixed dentition. Angle Orthod 2004;74:759–764.
12. Moorrees CFA, Reed RB. Changes in dental arch dimension expressed on the basis of tooth eruption as a measure of biologic age. J Dent Res 1965;44:129–141.
13. Moyers RE. Handbook of Orthodontics, ed 4. Chicago: Year Book Medical, 1988.
14. Gianelly AA. Crowding:Timing of treatment. Angle Orthod 1994;64:415–418.
15. Gianelly AA. Leeway space and the resolution of crowding in the mixed dentition. Semin Orthod 1995;1:188–194.
16. Björk A, Skieller V. Normal and abnormal growth of the mandible: A synthesis of longitudinal cephalometric implant studies over a period of 25 years. Eur J Orthod 1983;5:1–46.
17. Bahreman AA. Lower incisor extraction in orthodontic treatment. Am J Orthod 1977;72:560–567.
18. Angle EH. Treatment of Malocclusion of the Teeth, ed 7. Philadelphia: S.S White Dental, 1907.
19. DeKock WH. Dental arch depth and width studied longitudinally from 12 years of age to adulthood. Am J Orthod 1972;62:56–66.
20. Tweed CH. Indication for the extraction of teeth in orthodontic procedure. Am J Orthod Oral Surg 1944–1945;44:22–45.
21. Nance H. Limitations of orthodontic treatment in the permanent dentition. 2. Diagnosis and treatment in the permanent dentition. Am J Orthod 1947;33:253–301.
22. Bunon R. Essay sur les maladies des dents; ou I'on propose les moyens de leur procurer une bonne confirmation des la plus tendre enfance, et d'en assurer la conservation pendant tout le cours de la vie. Paris: Briasson, Chaubert, et De Hansy, 1743.
23. Kjellgren B. Serial extraction as a corrective procedure in dental orthopedic therapy. Eur Orthod Soc Trans 1947– 1948:134–160.
24. Hotz R. Guidance of eruption versus serial extraction. Am J Orthod 1970;58:1–20.
25. Dale JG, Brandt S. Dr. Jack C. Dale on serial extraction [interview]. J Clin Orthod 1976;10:44–60.
26. Kokich VG, Shapiro PA. Lower incisor extraction in orthodontic treatment. Four clinical reports. Angle Orthod 1984;54:139–153.
27. Færøvig E, Zachrisson BU, Effects of mandibular incisor extraction on anterior occlusion in adults with Class III malocclusion and reduced overbite. Am J Orthod Dentofacial Orthop 1999;115:113–124.
28. Pinto MR, Mottin LP, Derech CD, Araújo MTS. Extração de incisivo inferior: Uma opção de tratamento. Rev Dent Press Ortodon Ortop Facial 2006;11:114–121.
29. Valinoti JR. Mandibular incisor extraction therapy. Am J Orthod 1994;105:107–116.

Management of Deleterious Oral Habits

Oral function and the growth and development of maxillofacial morphology are closely interrelated. The interplay between form and function is an important mechanism that acts on orofacial structures during the development of occlusion. This mechanism is influenced by the surrounding environment, known as the *functional matrix*. Examples of this phenomenon include:

- The interplay between facial musculature and the tongue, which affects facial and jaw growth and dental occlusion
- The influence of respiration and the capsular matrix on the nasomaxillary complex
- The activity of the muscles maintaining the head position, which can also affect craniofacial growth and occlusion

Abnormal functions such as mouth breathing, tongue thrust swallowing, low tongue position at rest, unilateral chewing, and abnormal postures of perioral musculature can cause malocclusion.

Research has shown that many deformities caused by muscle dysfunction during the primary or mixed dentition are not self-corrected and become worse in older age. In the context of the present discussion, functional problems include all of the etiologic factors that affect development of normal function and can consequently affect the overall health of the stomatognathic system, including dental occlusion.

The human face is, anatomically and functionally, one of the most complex regions of the human body. Important vital senses of vision, hearing, smell, and taste all

reside within the face. Orofacial components of the stomatognathic system include the dental occlusion. These components serve as a portal of entry to the gastrointestinal system, for mastication and deglutition, and have other important functions such as articulation of words and the emotional expression of various moods, which is made possible by extremely delicate movements of the facial muscles. The mouth is also believed to be the first organ of which consciousness is aware.

All of these habits are complex, learned patterns of muscle contraction that serve as stimuli for normal growth patterns and are a part of normal physiologic function of the oropharyngeal structures. Examples include normal lip position and action during speech and normal tongue position and function during deglutition. Any abnormality in the pattern of physiologic habits may interfere with the regular pattern of facial growth and must be differentiated from the desired normal habits.

Non-Nutritive Sucking

Developmental psychologists have produced a number of theories to explain abnormal oral habits. These habits are referred to as *non-nutritive oral habits*.

The habit of sucking is the first coordinated muscular activity of the infant. There are essentially two forms of sucking: the nutritive form, which provides essential nutrients, and non-nutritive sucking, which ensures a feeling of warmth and a sense of security. *Non-nutritive sucking* has been defined as the sucking on an object (digit or pacifier) not related to ingestion of nutrients.

Definitions

Different definitions have been proposed for abnormal oral habits:

- An *abnormal habit* has been defined as an act performed without thinking.[1]
- Bryant[2] described a *habit* as a thing done often and therefore easily and that has become automatic.
- According to Peterson and Schneider,[3] a *habit* is a formed reaction that is resistant to change. Whether it is harmful or useful depends on the degree to which it interferes with the child's physical, emotional, or social function.

Etiology

There are two schools of thought regarding the cause of abnormal oral habits: *(1)* The psychoanalytic schools regard the habit as a symptom of some emotional disturbance, while *(2)* behaviorists view the habit as a simple learned act with no underlying neurosis.

Psychologic views

Early psychologic views of abnormal oral habits were firmly based on classic Freudian theory of personality development, which emphasizes the effects of the sexual pleasure drive on the individual psyche.[4] He claimed that specific body parts are particularly sensitive to sexual, erotic stimulation, these zones being the mouth, the anus, and the genital region.[4]

A child has certain needs and demands at each stage of development, such as the infant's need to nurse. The oral stage therefore begins at birth, and according to Freud the infant, whose primary focus of libidal energy is the oral cavity, cannot separate this sexual activity from the taking of nourishment; thus, thumb sucking provides the same pleasure to the infant as nursing. An abrupt interference with such a basic mechanism would likely lead to the substitution habit.

Behavioral views

After Freud described sucking habits as a manifestation of infantile sexuality, many controversies arose between psychoanalytic and learning theory proponents. Despite extensive discussion in the literature, few studies support the psychologic hypothesis, and learning theory appears more relevant.

Thumb sucking is one of the earliest examples of the neuromuscular learning process in the infant. It has been shown that the fetus sometimes sucks fingers in utero.

Haryett et al[5] strongly supported the theory that digit-sucking habits in humans are a simple learned response and reported that suckers failed to demonstrate any consistent psychologic differences from a control sample. These researchers provided further proof of their idea when they studied the psychologic effects on patients treated through orthodontic intervention.

Inadequate sucking activity. Digit sucking has also been related to inadequate

sucking activity, based on the findings that children who did not suck their fingers were nursed longer.[6,7] According to this theory, the frustration of weaning produces thumb sucking.

Oral drive theory. Sears and Wise[8] presented their oral drive theory, which suggested that the strength of the oral drive is in part a function of how long a child continues to feed by sucking. Prolonged nursing (feeding by sucking) strengthens the oral drive, and sucking increases the erotogenesis of the mouth.

In an experiment with monkeys, Benjamin[9] found far less thumb sucking among those whose nutritive sucking experience had been greatly reduced. Benjamin also proposed that thumb sucking arises simply as a placing reflex, which was tested by covering infants' hands with mittens in the very first weeks to prevent accidental placement of the thumb in mouth.

Time of habit appearance

The time that a habit starts also has etiologic significance:

- Those habits that are typically related to feeding appear during the very first weeks of life.
- Some children begin to suck a finger as a teething device during the difficult period of primary tooth eruption.
- Some children suck for the release of emotional tensions when they are unable to cope.
- Sometimes, a new habit may arise from a desire to attract attention.

Clinical implications

Different theories regarding non-nutritive oral habits are not incompatible with one another. The clinician should view these habits as a behavioral pattern of multivariate nature. In a general view, abnormal habits can be defined as learned functions responding to a drive. It is believed that a neural pathway exists between a stimulus and a response. Thus, all digit sucking habits should be studied for their psychologic and behavioral implications. They may be related to hunger, the sucking instinct, insecurity, or even a desire to attract attention.

Thumb or Finger Sucking

Thumb or finger sucking is one of the most common types of non-nutritive oral habit present in children. Reports indicate that sucking habits are reflexes whose precursors appear during intrauterine life.[10] Ultrasound pictures of intrauterine life have shown fetuses sucking their thumb. Thumb sucking appears to be a natural habit of children in all parts of the world, and it seems that putting objects in their mouths and sucking on them is a way for children to explore their world.

Reported prevalence rates in the first year vary from 50% to 70% and decrease thereafter. Helle and Haavikko[11] reported that two-thirds of finger suckers stop by age 5 years. Persistence beyond this age is considered a chronic non-nutritive habit that needs some type of intervention.

Prolonged digit sucking habits can have a negative impact on dental occlusion, speech, and the physical and emotional development of children. Friman et al[12] found that, among first graders, thumb suckers were considered to be significantly less intelligent, less attractive, and less desirable as friends.

Parents who are under the impression that the habit will stop and there is no harm to the permanent teeth must be informed that many children who continue this kind of habit do so to relieve stress; stress is a powerful stimulus for sucking activity and prolonged sucking habits. The investigation by Kelly et al[13] demonstrated that millions of children continue their habit through the permanent dentition. And, according to Van Norman,[14] stress levels in today's society are probably much higher than they were when Kelly's study was performed in 1973.

Effects on occlusion

The extent of damage caused by any abnormal force to the dentition, such as sucking on a finger or any other object, depends on the duration, frequency, and intensity of the force. *Intensity* is the amount of force applied to the teeth and supporting bone during sucking. *Duration* is the total time children spend sucking. *Frequency* refers to the number of times children practice the habit during the day.

The biologic response of tooth movement to different types of force is not the same; continuous light force has a greater effect on the dentition than high but intermittent force. In other words, the duration of a force is more important than its magnitude. Based on this mechanism, if a habit such as thumb sucking creates pressure against the teeth for a sufficient time, it certainly could move teeth.

According to Profitt,[15] sufficient time must be more than threshold duration, which is 6 hours or more per day. Therefore, if the habit has a shorter duration, little or no effect would be expected, no matter how heavy the pressure. In other words, children who suck intermittently with high intensity may not produce much tooth movement at all, whereas a child who sucks continuously (for more than 6 hours) can create significant dental change.

Sucking habits during the primary dentition usually have little, if any, long-term effect. If sucking stops at this stage, normal lip and cheek pressures soon restore the teeth to their normal positions. However, if these habits persist beyond the time that the permanent teeth begin to erupt, malocclusion will develop.

Clinical signs

The characteristics of malocclusions that develop because of thumb or finger sucking depend on the position of the digit, associated orofacial muscle contractions, the position of the mandible during sucking, and the facial skeletal morphology during the sucking habit. The most common dental signs of an active habit are reported to be:

- Maxillary incisor flaring. When a child places a thumb or finger between the teeth without sucking, the force is directed only to incisors. Depending on the position of the force, the habit can produce maxillary incisor flaring or, if the force is directed to the mandibular incisors, retroclination and crowding of the mandibular incisors, or both (maxillary incisor protrusion and mandibular incisor retrusion).
- Anterior open bite. When the thumb is placed between anterior teeth and force is applied to the maxillary incisors, the result can be anterior open bite due to anterior undereruption, posterior overeruption, or a combination of both. To differentiate between etiologies, the author recommends the evaluation of the occlusal plane in relation to the palatal and mandibular planes (for more detail, see chapter 13). If the jaw is positioned downward and posterior teeth are apart causing posterior tooth overeruption, a clockwise mandibular rotation will exaggerate open bite and cause antegonial notching.
- Posterior crossbite. The tongue is lowered vertically away from the maxillary posterior teeth, and children vigorously suck and contract the cheek. This force can cause a narrow palate due to the change in equilibrium between the oral musculature and the tongue. The orbicularis oris and buccinator muscles continue to exert force on the buccal surfaces of the maxillary dentition, while the tongue

no longer exerts a counterbalancing force from the lingual surface, and the posterior maxillary arch collapses into crossbite.

Finger sucking can cause many different types of deformities in the dentition or the skeleton (Figs 6-1 and 6-2). Box 6-1 summarizes the dental and skeletal effects of finger sucking.

Box 6-1 Effects of chronic thumb or finger sucking

Dental effects
- Increased overjet due to incisor flaring
- Anterior open bite
- Interdental spacing
- Posterior crossbite
- Mandibular incisor crowding
- Lingual tipping of mandibular incisors
- Undereruption of maxillary incisors
- Overeruption of posterior teeth
- Class II molar relationships
- Narrow (V-shaped) anterior maxillary arch

Skeletal effects
- Counterclockwise maxillary rotation
- Clockwise mandibular rotation
- Antegonial bending
- Increased mandibular plane steepness
- Increased Class II relationship (forward positioning of point A)
- Increased anterior facial height

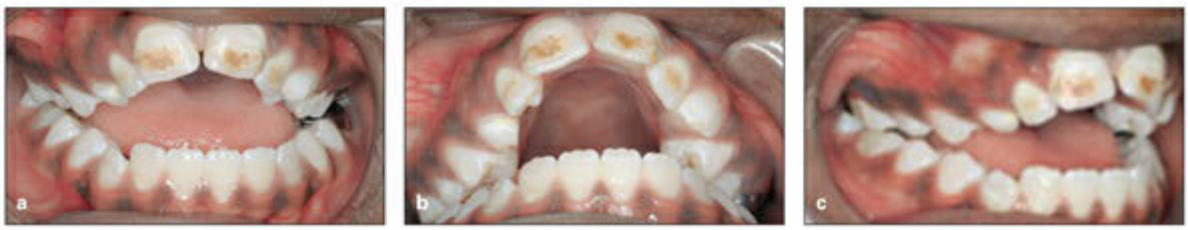


Fig 6-1 *(a to c)* Severe open bite; severe overjet; A-B discrepancy; bilateral crossbite; narrow, deep palate; and Class II malocclusion as a result of thumb sucking.

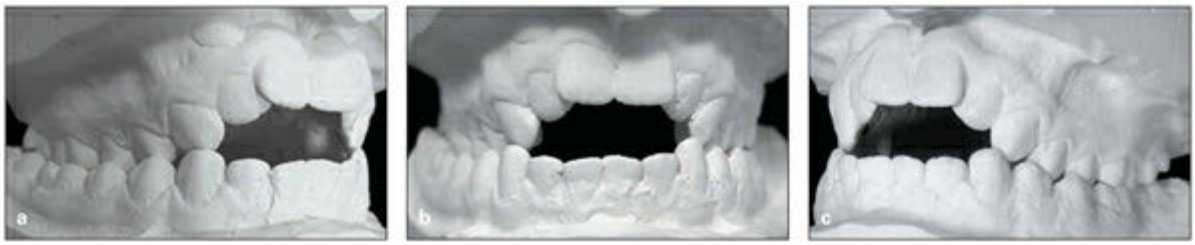


Fig 6-2 *(a to c)* Dentoskeletal effects of thumb sucking: severe open bite, overjet, constricted

maxilla, bilateral crossbite, and Class II malocclusion.

Clinical examination

When the history of a child's habit is assessed, parents are usually the best source of information. However, indirect questions to the child might also be helpful, for example: "When do you suck your finger?" "Which finger do you use while sucking?"

Extraoral examination

The offending digit may be red, wrinkled, calloused (Fig 6-3), or unusually clean. The profile is usually convex because of dentoalveolar protrusion, and the lips are usually apart.

Fig 6-3 Calluses developed as a result of the sucking habit.

Intraoral examination

The positions of the individual teeth and occlusion should be noted, based on the signs noted during the extraoral examination. Cephalometric analysis is very useful in assessing dentoskeletal changes.

Treatment Planning

Because of their high incidence, oral habits should be considered a major health hazard to children. Patients who exhibit signs of harmful effects to occlusion require intervention. Successful treatment requires a multidisciplinary approach to the basic cause of the problem. Regarding the etiology of abnormal oral habits, although the views of psychoanalysts and behaviorists differ, it would be wiser to consider the possibility of both views in treatment planning. The child must be involved in the process; successful control of abnormal habits depends on the child and parents'

cooperation and the child's decision to discontinue the habit.

Before any treatment planning, some important questions should be considered to assess the type, severity, and nature of the problem:

- Is the habit causing or enhancing a malocclusion?
- How long has the child had this habit?
- When does he or she perform the habit? Day? Night? Constantly?
- Does the child do it at school?
- Will cessation of the habit allow for self-correction of minor defects?
- Will cessation prevent worsening of the malocclusion?
- Is the child emotionally mature enough to accept treatment?
- Does anyone ridicule the child about the habit? Mocking, especially in front of others, tends to create a negative reaction in the child.

Age of intervention

The early elimination of digit sucking habits is one of the most important therapeutic services offered by practitioners. Sucking habits during the primary dentition years usually have little if any long-term effect and usually stop around 4 to 5 years of age. Intervention is recommended after 4 or 5 years of age but should be considered earlier if a high-intensity and long duration are involved and there is severe damage to occlusion or to the jaw, such as clockwise rotation of the mandible and antegonial notching.

There are other situations that may necessitate early intervention before 4 years of age. This includes children who, in addition to abnormal oral habits, have other behavioral problems such as hair pulling or parents and children who ask for help in stopping the habit because of developing speech problems or embarrassment.

Treatment Approaches

Depending on the age of the child, type of habit, etiologic background, and willingness of the child to stop the habit, four different approaches to treatment have been advocated:

1. Psychoanalytic methods

2. Behavioral modification techniques
3. Use of orthodontic appliances
4. Combination method

Psychoanalytic methods

Psychologic implications of therapy are less clearly understood and have often been overstated. With this method, the clinician attempts to analyze and understand the psychologic cause of an oral habit. This approach could solve the problem of the child's primary need for the oral habit in a radical manner. However, it is practically impossible to apply these techniques within the practice of dentistry and requires consultation with a specialist.

Behavioral modification techniques

Oral habits have been labeled psychodynamic phenomena by psychologists and psychiatrists. The behavioral modification technique is a method aimed to reinforce the child's positive behavior according to the Skinnerian principle of stimulus-response reward.[16] It has fast results, but it is a conditioned treatment.

Dentists are concerned with oral habits because of the detrimental consequences they have in the orofacial system, and they must be aware of the psychologic background of the patient as well as of the conditions under which the child performs the habit in order to overcome emotional difficulties. The dentist should also search into the child's family to discover the causes of the child's oral habit and to ensure both the child's and the family's cooperation; otherwise, the mechanotherapy alone might not be successful.

Depending on the cooperation of the child and his or her willingness to stop the habit, different approaches to treatment have been advocated.[17] These approaches include reminder therapy, reward therapy, and appliance therapy.

Reminder therapy

Reminder therapy is applied with children who want to stop the habit and need some kind of reminder until termination of the habit. An adhesive bandage can be attached to the involved finger for this purpose. Any type of reminder must be applied with the child's awareness and willingness, not as a punishment. Another device that has

been used as a reminder is a thumb device that covers the thumb and is held on the wrist by straps.

Reward therapy

In this approach, a type of contract is established between the child and parents or between the child and dentist. The contract simply states that the child agrees to stop the habit for a specified period of time and in return he or she will receive a reward if the requirements of the contract are met.

Use of orthodontic appliances

Management of non-nutritive habits should begin with the simplest form of therapy. If reminder therapy and reward therapy fail, appliance therapy can be implemented, with the child's awareness and cooperation. The patient and parents must be taught that the appliance is not a punishment device but rather a permanent reminder.

This method is easy and useful, provided that the child's and parent's acceptance and cooperation are obtained and they are informed that the appliance is visible and can cause speaking and eating difficulties for a short period of time. Otherwise, failure may result, which can increase the child's psychologic need for the habit, or it can be considered by the child as a punishment.

Habit-discouraging appliances usually have positive effects within 2 to 3 months, but they should be left in place for at least 6 months to make sure the habit is completely controlled.

Depending on the patient's cooperation, the kind of habit, and the type of deformity, several kinds of removable, fixed, or semiremovable appliances can be used.

Removable habit breakers

A removable habit breaker is a simple Hawley appliance with a piece of wire embedded in the acrylic resin portion behind the incisors; it can be a useful reminder device and has the advantage of allowing the patient to remove it for eating and maintaining good hygiene. The disadvantages of this type of appliance are that patient compliance is a major factor and the appliance can be easily misplaced or lost (Fig 6-4).

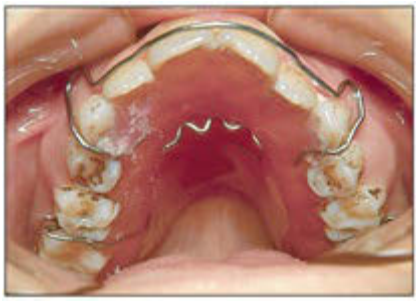


Fig 6-4 Removable habit breaker.

Fixed habit breakers

Fixed habit breakers are advantageous because they do not rely on patient compliance. A fixed device is an intraoral appliance attached to the maxillary teeth by means of two bands fitted to the primary second molars or the permanent first molars. A palatal archwire soldered to the bands forms the base of the appliance, to which a crib or some loop is soldered to the anterior portion of the arch, behind the incisors (Fig 6-5).

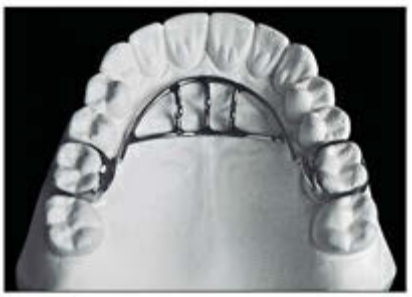


Fig 6-5 Fixed habit breaker (palatal bar with reminder).

Fixed devices serve as reminder and also prevent the patient from putting the palmar surface of the thumb in contact with palatal gingiva, thus preventing the pleasure of sucking.

Many kinds of fixed habit breaker are available, including the Bluegrass appliance, palatal bar, and quad helix.

Bluegrass appliance. This appliance was introduced by Haskell and Mink[18] in 1991 and can be used during the primary or mixed dentition. It is very well tolerated by patients and parents. The Bluegrass consists of a six-sided roller that is slipped over a 0.045-inch stainless steel wire soldered to molar bands. The child is instructed to turn the roller with his or her tongue instead of sucking the digit. The substitution of tongue movement for digit sucking seems to be very effective and encourages the child to stop the habit (Fig 6-6).

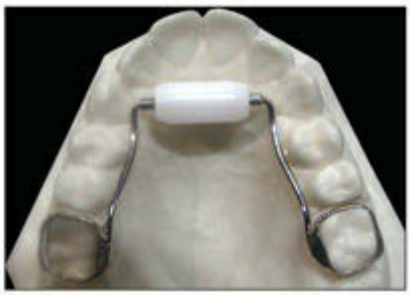


Fig 6-6 Bluegrass habit breaker.

Palatal bar. The palatal bar is constructed of two molar bands and a 0.036-inch stainless steel wire to serve as the base of the appliance. A shield or crib made of 0.030-inch wire is adapted to the deepest anterior part of the palate, separating the thumb or finger from the soft tissue. A short vertical extension in the front serves as a reminder (Fig 6-7).

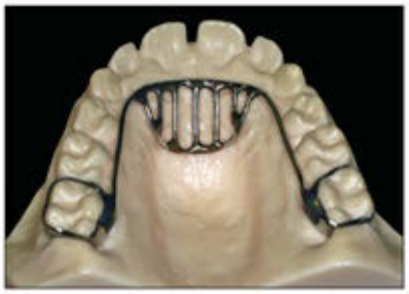


Fig 6-7 Palatal bar habit breaker.

Quad helix. The quad helix is a fixed expander that is applied during the primary or mixed dentition for transverse expansion of the maxillary arch. A combination of the quad helix and habit controller soldered to the anterior part of the helix can be useful in patients who have thumb sucking habits and also need palatal expansion. The quad helix alone, without a habit controller, also can prevent thumb sucking (Fig 6-8).

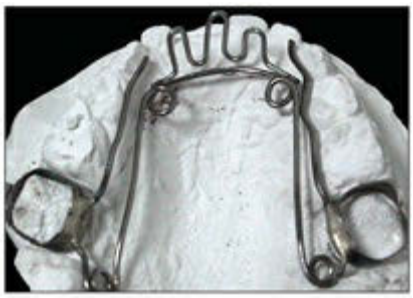


Fig 6-8 Quad helix with habit controller.

Tongue crib–transpalatal arch appliance

The tongue crib–transpalatal arch (TC-TPA) is a combination of tongue guard and transpalatal arch with some modifications that the author designed years ago. The author has used this appliance with habit control to treat many cases of open bite or open bite tendency, whether the problem is dental or dentoskeletal.

This appliance is constructed of two maxillary molar bands and two pieces of 0.036-inch stainless steel wire (Fig 6-9). One wire serves as a transpalatal arch with an omega loop of 10- to 12-mm diameter toward the distal side, where the force of the dorsum of the tongue exerts maximum pressure during swallowing. This arch should be soldered to molar bands so that it is separated by at least 1 mm from the palate to facilitate maxillary molar intrusion without impinging on the palate. The second wire is used to form a tongue guard wide enough to cover the incisor gap. It should be long enough to stay below the level of mandibular incisal edge at occlusion.

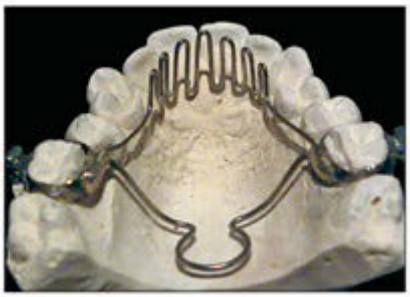


Fig 6-9 Transpalatal arch and tongue guard combination (TC-TPA).

The author applies TC-TPA in cases of severe open bite associated with some degree of facial divergence (steep mandibular plane, clockwise mandibular rotation, and vertical problems) when the patient is a finger sucker or has stopped sucking the finger but continues with tongue thrust. In conjunction with TC-TPA, a heavy lower holding arch should always be applied to prevent eruption of the mandibular molars. The goals of applying this appliance plus a lower holding arch are fourfold:

1. Control of the finger and tongue habit.
2. Facilitation of maxillary incisor eruption and anterior alveolar growth.
3. Intrusion of maxillary molars and prevention of mandibular molar extrusion.
4. Clockwise rotation of the mandible. For more detail, see chapter 13.

Case 6-1

A 7-year, 6-month-old girl in the middle mixed dentition presented with a Class II division 1

malocclusion, a severe overjet (11 mm), and open bite (4 mm); she was a severe thumb sucker who had developed a tongue thrust. Figures 6-10a to 6-10c show the pretreatment intraoral photographs and lateral cephalometric radiograph.

Treatment:
The TC-TPA appliance was provided to control the tongue thrust habit and intrude the maxillary molars. A lower holding arch was applied to prevent overeruption of the mandibular molars. High-pull headgear was used for Class II molar correction and maxillary molar intrusion.

The posttreatment results (Figs 6-10d to 6-10f) show Class I molar relationships with a 1-mm overjet and 10% to 15% overbite.

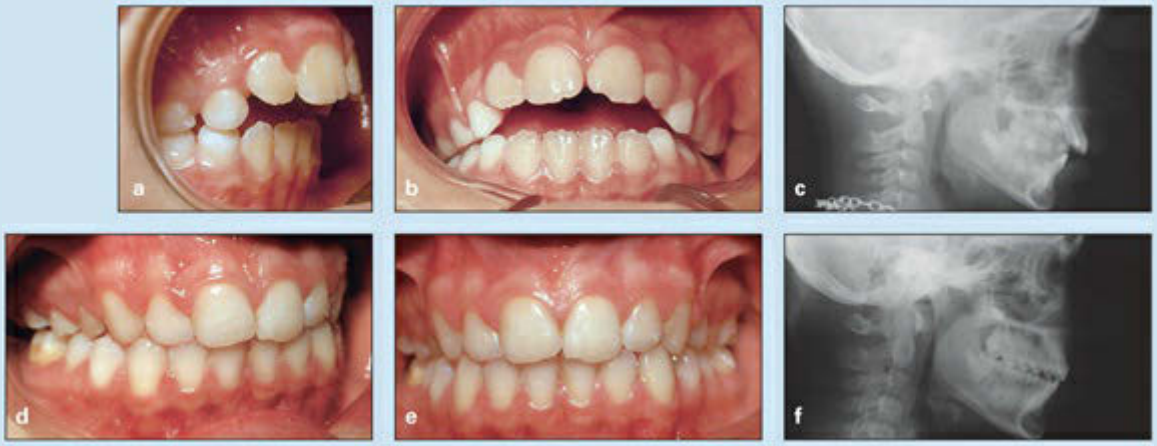


Fig 6-10 Treatment of a 7-year, 6-month-old thumb sucker with a Class II division 1 malocclusion, severe overjet and open bite, a steep mandibular plane angle, and increased lower facial height. *(a and b)* Pretreatment occlusion. *(c)* Pretreatment cephalometric radiograph. *(d and e)* Posttreatment occlusion. *(f)* Posttreatment cephalometric radiograph.

Case 6-2

A 9-year, 10-month-old girl who was an active thumb sucker with a consequent tongue thrust presented (Figs 6-11a to 6-11e). She had a Class I dentoskeletal malocclusion, 5-mm overjet, 3-mm dental open bite, and maxillary and mandibular dental protrusion.

Treatment:
The first phase of treatment included placement of a fixed palatal tongue guard and lower lingual holding arch, preserving leeway space and molar eruption. Within 2 months, finger sucking had stopped. The tongue guard was left in place for another 6 months. The open bite and overjet were more than 60% corrected. The second phase of treatment, after removal of the tongue guard, involved 2 × 4 maxillary and mandibular bonding for some incisor retraction and bite closure (Figs 6-11f to 6-11j).

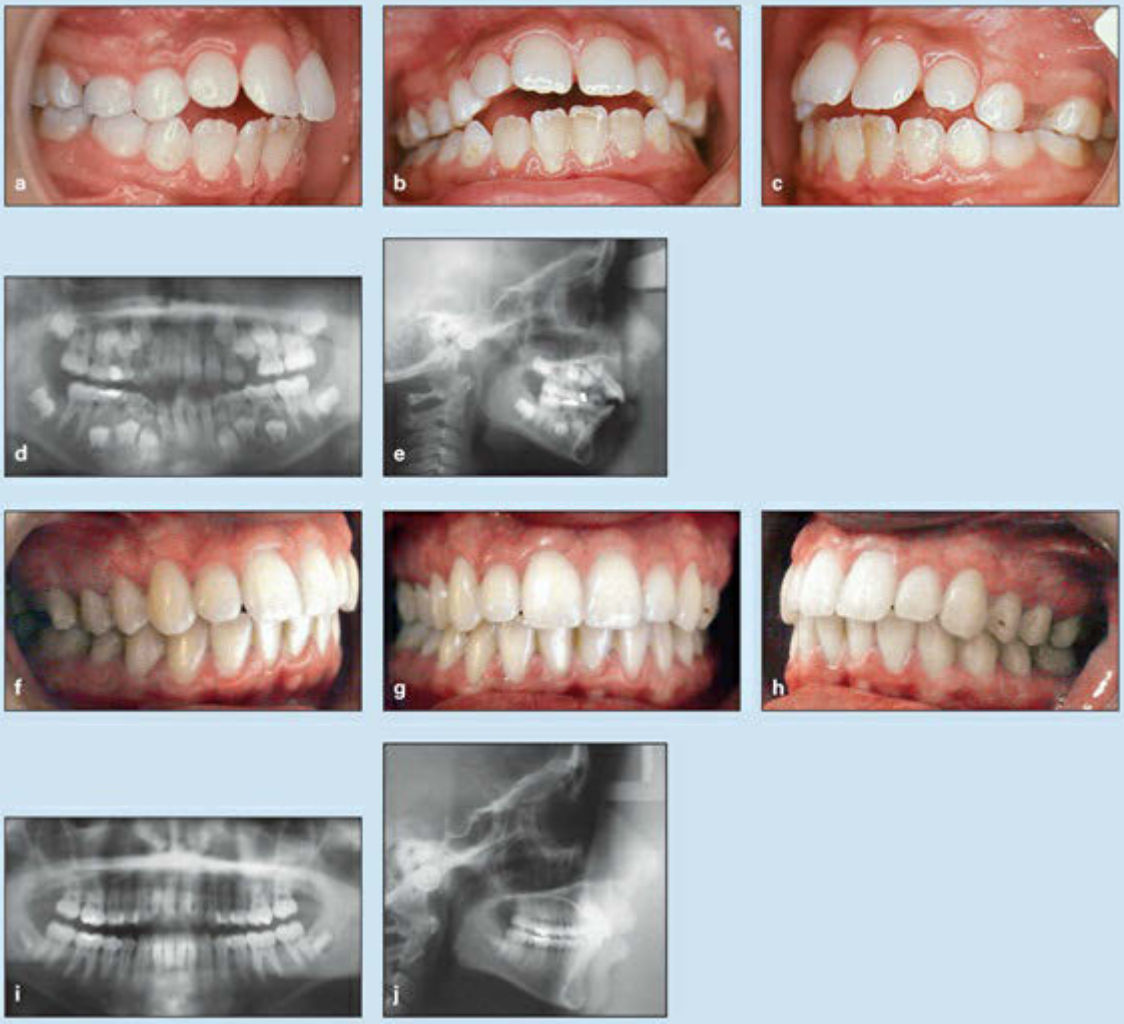


Fig 6-11 Treatment of a 7-year-old girl who is an active thumb sucker with a developing tongue thrust. She has a dentoskeletal Class I malocclusion, 5-mm overjet, 3-mm dental open bite, and mandibular dental protrusion. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e)* Pretreatment cephalometric radiograph. *(f to h)* Posttreatment occlusion after retention. *(i)* Posttreatment panoramic radiograph. *(j)* Posttreatment cephalometric radiograph.

Pacifier Habits

Sucking is a powerful reflex in infants in their early days of life. Babies who are breastfed benefit from both the nutrition and satisfaction of their sucking instinct. Therefore, if babies are not sufficiently breastfed, they may satisfy their sucking instinct by using pacifiers or other objects. Unfortunately, some parents believe that pacifier use or digit sucking in early childhood is a harmless habit, and use of a pacifier to satisfy children is common.

Several reports have shown that improper use of a pacifier can have disastrous effects on occlusion. Larsson[19] reported that anterior open bite is normally more

obvious and visible earlier in pacifier suckers than in digit suckers. As in digit suckers, the open bite is associated with tongue thrust during swallowing. He reported that the prevalence of posterior crossbite among pacifier-sucking girls in Falköping, Sweden, was 26%. Larsson[19] suggested that the transverse occlusal relationship should be evaluated in pacifier-sucking children between 2 and 3 years of age. He concluded that if inter-fering contacts of the primary canines exist, the parents should be instructed to reduce the "in the mouth time" of the pacifier.

In a review of the literature, Adair[20] concluded that dentists are generally well aware of the oral implications of non-nutritive sucking via digit or pacifier. He reported that some evidence indicates that pacifiers may be less harmful to the dentition than digit sucking, particularly because children often abandon pacifier habits spontaneously at 2 to 4 years of age. In contrast, children are more likely to persist with digit habits into their school years, and they may require appliance therapy to achieve discontinuation. He and other researchers also suggested that dentists should be aware of other harmful effects of pacifier use, such as failure of breastfeeding, dental deformities, recurrent acute otitis media and other infections, and safety issues. Castilho and Rocha[21] claimed that pacifiers prevent the establishment of breastfeeding and lead to weaning.

On the other hand, Li et al[22] and Cinar[23] concluded that use of a pacifier seems to reduce the risk of sudden infant death syndrome (SIDS) and possibly reduces the influence of known risk factors in the sleep environment. The American Academy of Pediatrics recommends that parents consider offering pacifiers to infants aged 1 month and older at the onset of sleep to reduce the risk of SIDS.[24] The American Academy of Family Physicians (AAFP) recommends weaning children from pacifiers between 6 months and 12 months of age to prevent otitis media.[25] According to the AAFP, pacifier use should not be actively discouraged and may be especially beneficial in the first 6 months of life.

In a study verifying the relationship between breastfeeding, bottle-feeding, and non-nutritive sucking habits, de Holanda et al[26] concluded that breastfeeding that continues for more than 6 months is a protective factor against the persistence of pacifier sucking, although the effects of the mother-child relationship require additional investigation. They added that pacifier habits appear to end earlier than digit habits. They reported that more than 90% of pacifier users ended the habit before 5 years of age and 100% stopped by age 8 years.

Some people believe that pacifier habits are easier to stop than digit habits and that the pacifier can be discontinued gradually or completely if the issue is explained

carefully to the child. Damage to occlusion created by pacifiers, depending on the type of pacifier and duration and intensity of sucking, is not much different from that resulting from thumb or finger sucking. If continued with high intensity and high duration, pacifier sucking can certainly have some deleterious effects. Bishara et al[27] proposed that the transverse occlusal relationship should be evaluated between 2 and 3 years of age, particularly in children who suck pacifiers.

Tongue Thrust

Clinically, *tongue thrust* is defined as a forward placement of the tip of tongue between the anterior teeth during swallowing. According to Graber,[28] humans swallow 1,200 to 2,000 times every 24 hours with about 4 lbs of pressure per swallow. The constant pressure of the tongue may force the teeth out of alignment. Graber[28] indicated that nervous thrusting also pushes the tongue against the teeth while it is at rest. This is an involuntary, subconscious habit that is difficult to correct.

This myofunctional disorder of the tongue in the oral cavity has been referred to as *deviate swallow*, *infantile swallow*, *abnormal swallow*, *reverse swallow*, and *immature swallow*. The terms most commonly used are *tongue thrust* and *tongue thrusting*.

To understand tongue thrust, it is necessary to learn about normal and abnormal tongue function.

Normal and abnormal deglutition

Moyers[29] described normal swallowing: The teeth are in occlusion, the lips are lightly closed, and the tip of the tongue is held lightly against the palate behind the anterior teeth, while the anterior middle dorsum of the tongue is elevated and in contact with the hard palate; there is no marked contraction of perioral musculature.

Tongue thrust can develop following finger sucking or because of other factors leading to tongue protrusion during swallowing. It can be present without any visible muscle contraction and can be self-corrected or reduced by age.

Proffit[15] evaluated tongue pressure in the transition and normal swallowing and indicated that individuals who place the tongue tip forward when they swallow usually do not have more tongue force against the teeth than those who keep the

tongue tip back; in fact, tongue pressure may be lower in the former individuals.

Kelly et al[13] produced epidemiologic data showing that the percentage of persons with infantile and transitional swallowing patterns is greater than the percentage of persons with open bite. They also reported that open bite is seen much more frequently in blacks than in whites. Therefore, it can be concluded that swallowing is not a learned behavior; rather it is a physiologically controlled function at the subconscious level. As Subtelny and Subtelny[30] stated after assessing cineradiographs and cinephotographs, tongue thrust must be considered as a concomitant, rather than a causative, factor in most cases of open bite.

Tongue thrust develops when an open bite is present between incisors because of previous abnormal sucking conditions and the tongue is placed more forward to maintain an anterior seal. Abnormal swallowing patterns play an important role in maintaining the open bite or worsening the problem and are related to development of posterior crossbite in early ages. Ovsenik[31] investigated the etiology of posterior crossbites to determine the correlation between posterior crossbite and orofacial function. She found that atypical swallowing was an important factor in the etiology for posterior crossbite development: Children with crossbite showed atypical swallowing patterns more often than children without crossbite. Ovsenik[31] concluded that for every child with sucking habits in the primary dentition, clinical examination should include assessment of the swallowing pattern and other orofacial functions.

Classification

Moyers[29] divided abnormal swallowing into three types: the simple tongue-thrust swallow, the complex tongue-thrust swallow, and retained infantile swallow.

Simple tongue thrust

Simple tongue thrust is usually associated with a history of digit sucking that has led to open bite. Even though the sucking habit may no longer be practiced, it is necessary for the tongue to thrust forward into the open bite area to maintain an anterior seal with the lips during swallowing. According to Moyers,[29] in simple tongue thrust the teeth are in occlusion during swallowing, some muscle contraction can be seen, and correction of malocclusion will correct the habit.

Complex tongue thrust

Complex tongue thrust is a more complicated type of swallowing pattern associated with chronic nasorespiratory issues such as mouth breathing, tonsillitis, or pharyngitis. When the tonsil is inflamed and enlarged, the root of the tongue exerts force on the tonsil and causes pain.

To avoid this force and resulting pain, the mandible will drop reflexively, separating the maxillary and mandibular teeth, enlarging the freeway space, and providing more room for the tongue to move forward. This will create a more comfortable position during swallowing and a more adequate airway.

The forward position of the tongue exerts continuous light force on the anterior teeth and alveoli, which will result in dental or dentoalveolar protrusion, interdental spacing, and open bite. Open bite might not be limited to anterior teeth. Treatment of this type of tongue thrust is more complicated; myofunctional therapy might also be required.

Retained infantile swallow

Infant gum pads are not brought together in function, because the mouth is designed for suckle feeding at this stage, and the space between the gum pads is occupied by the tongue. At this age, the tongue is advanced in development and is relatively larger than the surrounding jaws to facilitate suckling. The transition from the infantile swallowing pattern to an adult swallowing behavior occurs after 6 months, with tooth eruption.

Moyers[29] states that retained infantile swallow is an abnormal swallowing pattern in which the infantile swallow remains and the transition to an adult swallowing behavior has not occurred. Open bite is more severe in patients with this type of swallowing and may not be confined to the anterior segment.[32] Treatment is also more complicated and may include orthognathic surgery and myofunctional therapy.

Etiology

As already mentioned, simple tongue thrust usually develops to maintain anterior seal in the open bite area created by a finger- or thumb-sucking habit. Open bite caused by this type of tongue thrust is limited to undereruption of the anterior teeth or the dentoalveolar area. After control of abnormal tongue function, open bite is

corrected, and no relapse occurs (see case 6-4).

The specific causes of complex tongue thrust and retained infantile tongue thrust have not been determined. The following have been proposed as local or general causes for these types of abnormal swallowing:

- Hereditary factors, such as a large tongue
- Vertical skeletal problems such as a steep mandible or wide gonial angle
- Thumb or other finger sucking
- Short lingual frenum (tonguetie)
- Mouth breathing, which might be due to many factors that cause nasal obstruction, such as allergies, nasal congestion, deviated conchae, or large adenoid
- Certain artificial nipples used in bottle feeding
- Sore throat, enlarged tonsils, or adenoids that cause difficulty in swallowing
- Premature loss of primary teeth and abnormal tongue adaptation
- Muscular, neurologic, or other physiologic abnormalities, such as loss of muscle coordination

Examples of these conditions are shown in Figs 6-12 to 6-14.

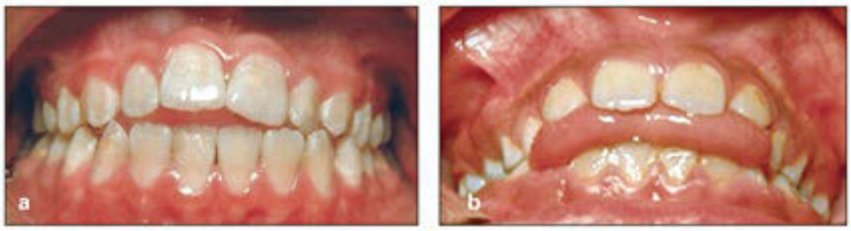


Fig 6-12 *(a and b)* Anterior tongue thrusts of different magnitudes can have different effects on the dentition.

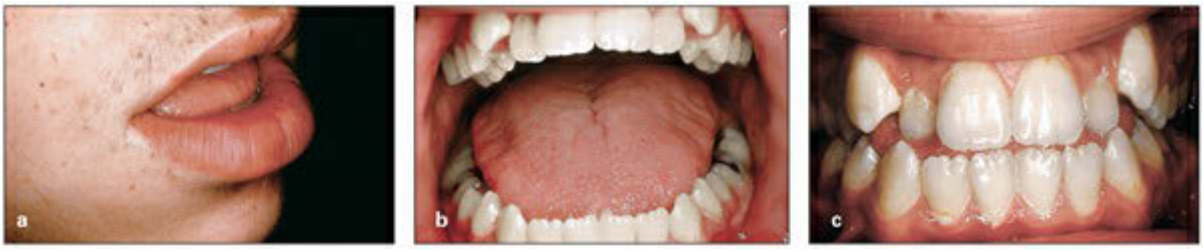


Fig 6-13 *(a to c)* Macroglossia causing open bite and tongue thrust. (Courtesy of J. Daniel Subtelny, Rochester, New York.)

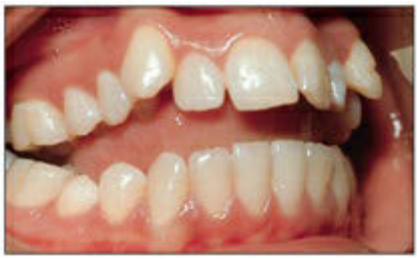


Fig 6-14 Skeletal open bite causing tongue thrust.

Different types of tongue thrust

Tongue thrust has many variations, depending on its etiologic factors and interaction with other environmental factors; the effects of these different patterns on occlusion can also vary.

Anterior tongue thrust

Anterior tongue thrust is one of the most common and typical types of tongue thrust. The resulting occlusal problem is anterior open bite. The extent of bite opening depends on the magnitude and position of tongue force (see Fig 6-12).

Sometimes anterior tongue thrust is accompanied by macroglossia. In such cases, the lips are apart, the tongue at rest protrudes beyond the lips, and anterior open bite is usually more extensive (see Fig 6-13).

Tongue thrust develops as an adaptive function to maintain anterior seal during swallowing when an open bite is present between incisors. The gap or open bite can be the result of previous abnormal sucking conditions or can be a skeletal open bite that has developed because of a vertical growth pattern and divergence between the maxilla and the mandible (see Fig 6-14).

Sometimes the bite opening is extensive and the only occlusion is in the molar area, and tongue dysfunction is secondary to this situation. An orthosurgical approach is usually the only treatment option.

Anterior tongue thrust combined with strong mentalis muscle dysfunction exaggerates maxillary incisor protrusion, mandibular incisor retroclination, and crowding and consequently causes severe overjet.

Lateral tongue thrust

Lateral tongue thrust is not as common as anterior tongue thrust and, depending on its

etiology, can cause unilateral or bilateral open bite. The anterior bite is usually closed; however, the posterior teeth may be open on one or both sides, from the first premolar to the distalmost molars. Correction of these anomalies is much more difficult.

Lateral tongue thrust usually develops following undereruption of primary molars due to ankylosis. This problem can be more severe where primary ankylosed molars are at a lower occlusal level. If the situation is neglected, patients will continue abnormal tongue function, which may strengthen the tongue, preventing even eruption of the permanent dentition. Early detection and extraction of ankylosed primary molars can prevent these abnormalities (Fig 6-15).

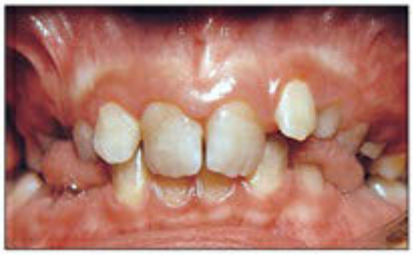


Fig 6-15 Posterior bilateral tongue thrust resulting from ankylosis of all primary molars.

Bilateral tongue thrust can develop following primary molar ankylosis on both sides or when macroglossia causes the tongue to rest over occlusal surfaces (see Fig 6-13). Proffit[15] stated that the failure of posterior teeth to erupt fully into occlusal contact is the cause of this kind of open bite.

Problems associated with tongue thrust

Abnormal pressure from the tongue during deglutition or speech can produce or maintain open bite, dental protrusion, or spacing. It is generally accepted that all infants show some tongue thrust during swallowing. At age 6 years, 50% of individuals show some degree of tongue thrust; at age 15 years, the proportion decreases to 25%. According to Profitt,[15] the maturation of the adult swallow pattern appears in some children as early as age 3 years but is not usual until about age 6 years and is never achieved in 10% to 15% of a typical population. Tongue thrust swallowing in older patients superficially resembles the infantile swallow. Some open bites might correct spontaneously as children pass through puberty.

Clinical observations also show that tongue thrust will persist as long as the open bite is present. In patients with narrow maxillary arches who have a persistent tongue thrust, maxillary expansion will accommodate the tongue and produce better

treatment results.

Orthodontic treatment will relapse if the force of the tongue is not controlled, whether the tongue thrust is a causative or contributing factor in the malocclusion.

Speech problems are also a consequence of tongue dysfunction and affected occlusion. The /*s*/ sounds are most affected, and lisping can result. Lateral lisping can occur in patients with lateral open bite caused by lateral tongue thrust during swallowing.

Clinical examination and differential diagnosis

The investigation of tongue function, tongue size, and tongue posture is an important part of oral diagnosis (see chapter 3). Many studies have demonstrated that tongue dysfunction plays an important role in the etiology of open bite as well as in the relapse of treated open bite patients.[25] Therefore, to determine the etiology of tongue dysfunction, it is important to identify patients with abnormal swallowing patterns.

Graber[28] described three types of swallowing pattern: *(1)* visceral, *(2)* somatic, and *(3)* inconstant. The visceral swallowing pattern, also termed *infantile swallowing*, is present at birth. It is characterized by a forward movement of the tip of the tongue during swallowing. Visceral swallowing facilitates suckling and is considered normal at this age because the tongue is relatively large compared with the mandible. Normally this type of swallowing pattern changes gradually into a so-called mature or somatic swallowing pattern. If the visceral swallowing pattern persists after the primary dentition, it is then considered abnormal or a dysfunction of the tongue, which can cause problems for occlusion.

The somatic swallowing pattern is a normal, mature type of swallowing in which the tip of the tongue is held lightly against the palate behind the anterior teeth. The anterior middle dorsum of the tongue is elevated and in contact with the hard palate and exerts pressure on the incisive papillae rather than on the teeth.

Graber et al[33] characterized inconstant swallowing as a transitory pattern of swallowing that normally occurs during the transitional period between the infantile and somatic swallowing patterns.

To diagnose and differentiate between infantile and mature swallowers, Peng et al[32] conducted an ultrasonography study in two groups of children. One group consisted of visceral swallowers, and the second group had the somatic swallowing pattern. The researchers found that the dorsal surface of the tongue, which was thought to be ideal for observing tongue function, was not suitable for differentiating

between visceral and somatic swallowing patterns. Instead, they found that the movements of the genioglossus muscle were identical within each group but significantly different ($P < .01$) between the two groups. They concluded that the genioglossus muscle appears to play an important role in visceral swallowing and can serve as a reliable means for differentiating between visceral and somatic swallowers.

Peng et al[32] also found that the activity and strength of the genioglossus muscle seems greater in individuals with a somatic swallowing pattern. They added that, because the movements of the tongue tip proved to be inconsistent in visceral swallowers, it is important that movements of the genioglossus muscle be used to distinguish between visceral and somatic swallowing even when the tongue tip moves atypically.

Examination of a child to determine the presence or absence of tongue dysfunction requires a thorough evaluation of both the occlusal conditions and all warning signs that can play a role in tongue dysfunction. For example, anterior open bite is one of the most common signs of anterior tongue thrust, but open bite is a multifactorial phenomenon, and no single factor can account for open bite. The following may indicate the presence of tongue dysfunction:

- Initial cause of open bite, such as thumb and finger sucking, prolonged use of pacifiers, and bottle feeding
- Mouth breathing resulting from a history of allergies or enlarged tonsils or adenoids
- Open mouth posture (lip incompetence)
- Tightening of facial and lip muscles (grimace or pursing of lips) during swallowing
- Speech difficulty during sounds such as /*s*/ and /*z*/
- Skeletal malformation of genetic origin

The clinician should also evaluate tongue size at rest (the position relative to the mandibular dentition, tongue scalloping) and tongue posture (the resting position of the tongue in the mouth). The clinician evaluates tongue function by keeping the child in an upright position, watching in the mirror, and instructing the patient to swallow saliva. During the patient's swallow, the evaluator should observe function of the perioral musculature and then watch the tongue activity by gently lowering and pulling the lip corners.

Treatment considerations

Some investigators, such as Subtelny and Subtelny,[30] believe that tongue thrust must be considered as a concomitant rather than a causative factor in most cases of open bite. Some reports indicate self-correction of or reduction in thrusting activity with increasing age.[34]

The author's clinical experience after years of observation of many patients is that, no matter what the cause of open bite, once space has been created between the maxillary and mandibular teeth, the tongue will continue to enter the created space and consequently enlarge the space or prevent self-correction. If the adapted tongue function persists beyond eruption of the incisors, self-correction may be impossible and the dentoalveolar deformity can worsen during jaw growth.

These deformities will be more destructive to the occlusion if the child's tongue thrust is combined with other hereditary or environmental factors, such as a vertical growth pattern and clockwise rotation of the mandible, mouth breathing, large tongue, or large tonsils.

If the problem is detected before permanent incisors erupt or during eruption and the original cause, such as pacifier or finger sucking, is stopped, the chance of self-correction is high. If mouth breathing is the result of some kind of airway obstruction, consultation with a specialist is necessary. If early detection and intervention are neglected, tongue thrust can persist, causing occlusal deformity and structural damage.

Treatment approaches

Two methods for treatment of tongue thrust have been proposed: *(1)* mechanotherapy with an orthodontic device and *(2)* oral habit training. Oral habit training is an exercise technique taught by a qualified speech therapist who tries to reeducate the muscles associated with swallowing by changing the swallowing pattern.

Mechanotherapy in tongue thrust patients can be accomplished with either a removable or a fixed appliance. The part of the appliance that controls the abnormal pressure from the tongue (tongue crib or tongue guard) must be designed in such a way that it covers the entire anterior tooth gap but does not interfere with the bite during chewing (Fig 6-16).

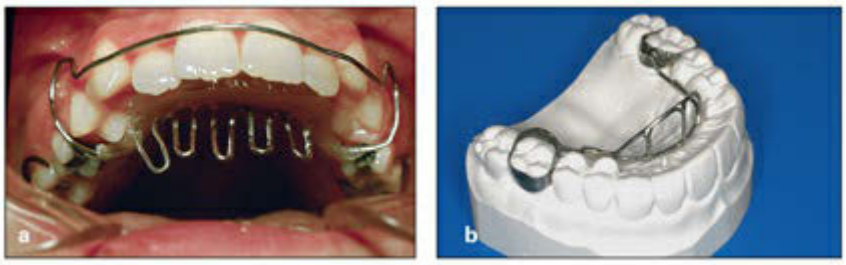


Fig 6-16 *(a)* Removable tongue guard. *(b)* Fixed tongue guard.

As with many other orthodontic procedures, the result is more successful with the sincere commitment and cooperation of the child and parents. If the patient is compliant, mechanotherapy is 100% successful in correcting simple tongue thrust, unless other complications such as airway problems, neuromuscular involvement, or vertical skeletal growth patterns are interacting with abnormal tongue function. This requires more comprehensive treatment in conjunction with tongue thrust mechanotherapy.

Correction of open bite with tongue guards alone may not be sufficient in every patient, and other treatment concomitant with a tongue guard might be necessary. For example, palatal expansion of the narrow palate and correction of mouth breathing would facilitate correction of tongue dysfunction.

In cases of severe tongue thrust, proper myofunctional therapy might be advised in addition to mechanotherapy. Good results can be achieved when the patient is instructed to complete daily exercises and use the fixed tongue guard. After the problem is clearly demonstrated to the child and parents on casts, photographs, and radiographs, the appliance function and the type of exercises must be explained.

The following is one useful exercise for tongue adaptation: The appliance is inserted, and the child is instructed to close the teeth, put the tip of tongue behind the tongue crib, and swallow while trying to hold the tongue tip so that it does not touch the crib. This exercise should be repeated several times during the day. Daily exercise with fixed tongue guard therapy facilitates tongue adaptation and is very effective in the treatment of tongue dysfunction.

To prevent relapse and impairment of orthodontic results, tongue dysfunction must be eliminated before completion of orthodontic treatment. Some practitioners suggest bonding lingual cleats over the palatal side of the central incisors to help the patient to avoid tongue thrust.

Case 6-3

A 14-year-old boy was concerned about a diastema in the anterior teeth (Figs 6-17a to 6-17d). The patient had a history of thumb sucking that was stopped by 9 years, but a severe tongue thrust habit continued.

Treatment:
The treatment plan involved application of a fixed tongue guard and tongue exercises. Active treatment was very effective and terminated after 9 months. Follow-up treatment continued with a Hawley retainer (Figs 6-17e to 6-17h).

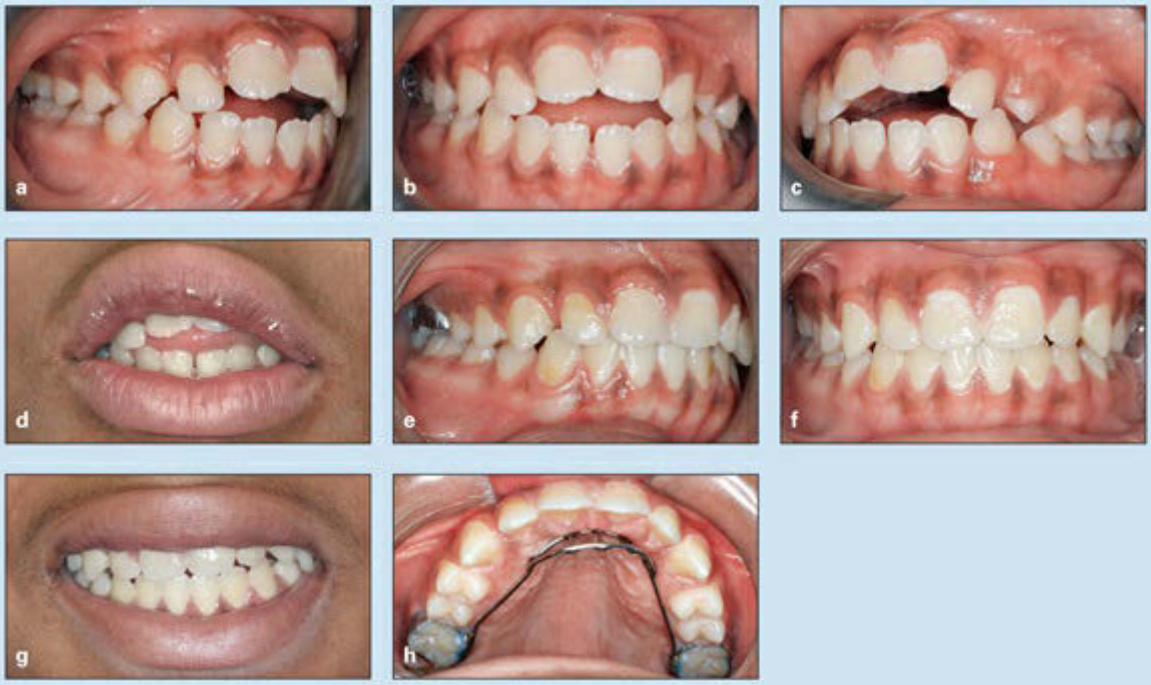


Fig 6-17 Treatment of a 14-year-old boy with severe tongue thrust who had a history of thumb sucking until the age of 9 years. *(a to d)* Pretreatment occlusion. *(e to h)* Posttreatment occlusion. The only appliance used was a tongue guard.

Case 6-4

A 12-year, 4-month-old girl with a normal skeletal pattern exhibited severe anterior open bite (3 to 5 mm) from the maxillary left to right primary second molars and simple tongue thrust (Figs 6-18a to 6-18c).

Treatment:
Because patient cooperation was excellent, the treatment plan included use of a removable Hawley appliance with anterior tongue crib and tongue exercises (Figs 6-18d and 6-18e). Figures 6-18f to 6-18i illustrate the final occlusion, and Fig 6-18j shows posttreatment dentoskeletal changes.

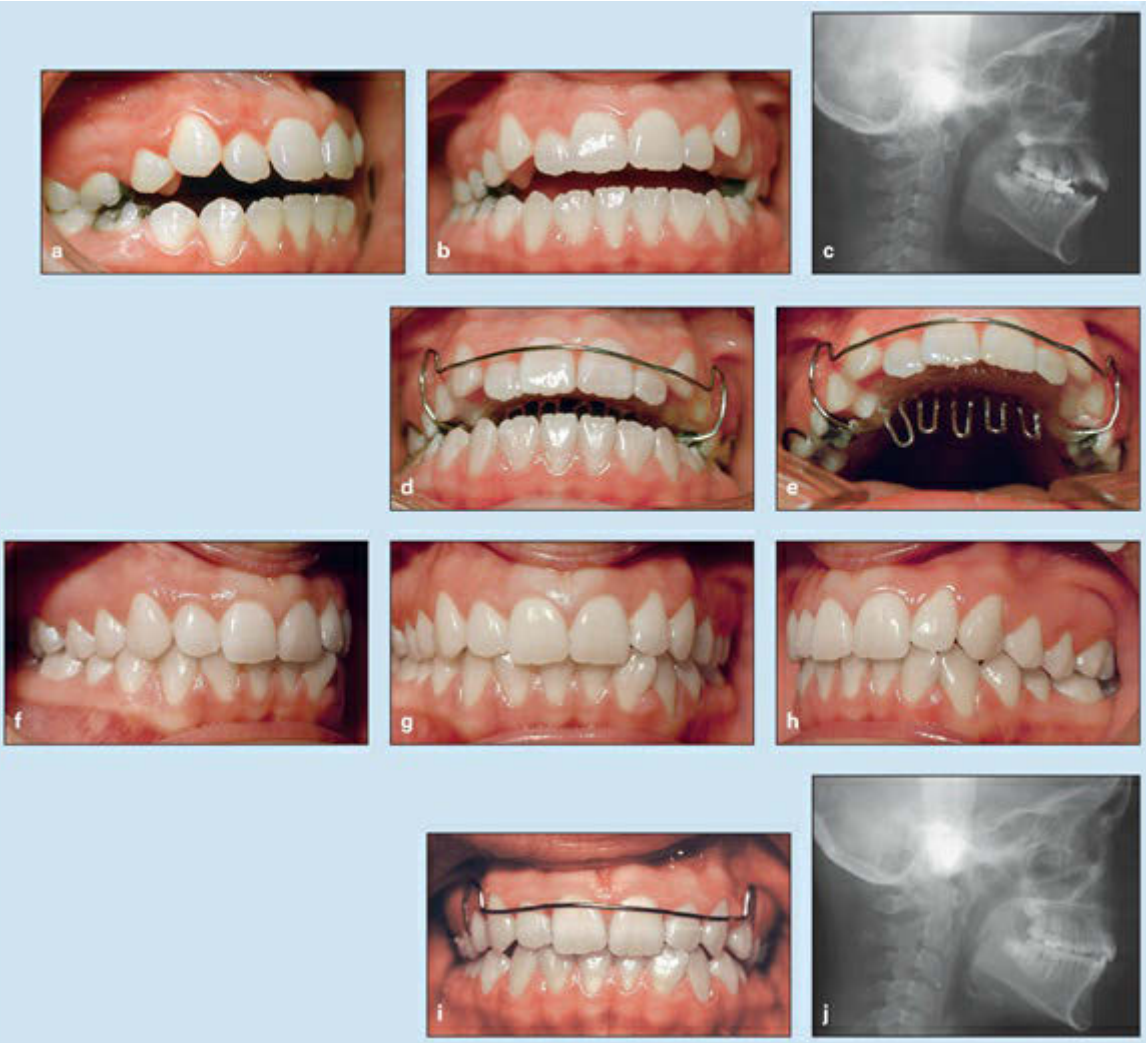


Fig 6-18 Treatment of a 12-year-old girl with severe anterior open bite and a simple tongue thrust habit. *(a and b)* Pretreatment occlusion. *(c)* Pretreatment cephalometric radiograph. *(d and e)* Removable Hawley appliance with tongue crib, the only appliance used. *(f to i)* Posttreatment occlusion before termination of the retainer. *(j)* Posttreatment cephalometric radiograph.

Mouth Breathing

Nasal respiration and its capsular matrix, the nasomaxillary complex, have an important role in and effect on normal maxillofacial morphology and growth and therefore on occlusion. Normal respiration requires adequate airway space through the nasal and nasopharyngeal areas. If the structures within this passage, such as the adenoids, tonsils, or nasal turbinate, are enlarged pathologically or anatomically, nasal respiration is precluded; the result can be an adaptation to oral respiration. Oral respiration and nasal obstruction are common findings among orthodontic patients.

The allegations that disturbed respiratory function can affect craniofacial growth and morphology and produce malocclusion have been a matter of controversy for more than 100 years. The *adenoid facial type* is characterized as a long, narrow face with anterior open bite, dental protrusion, incompetent lip, narrow maxillary arch, and deep palate; this facial morphology was considered to be caused by mouth breathing for many years.

There are many controversies regarding whether mouth breathing is the cause of malocclusion or whether craniofacial configurations have a genetic origin that would make airway problems a secondary cause.

Subtelny[35] hypothesized that mouth breathing can cause postural adaptations and affect the positional relationship of the jaws and the developing occlusion. He also stated that children with large adenoids exhibit a longer face, more retruded incisors, and lips that are parted to a greater degree than do children without large adenoids.

In 1960, Linder-Aronson[36] compared children selected for an adenoidectomy with a control group by cephalometric evaluation and found a retroclination of maxillary and mandibular incisors and a tendency toward an open bite in the children who required surgery.

Subtelny[37] also evaluated the changing dimension of adenoid tissue on serial cephalometric roentgenograms in 33 young patients with enlarged adenoids and compared them with a sample of children with minimal adenoid mass. He reported that adenoid tissue does exhibit a developmental cycle superimposed upon hard tissue, which may be hypertrophic reactions related to stress, nasorespiratory infections, and allergies.

Subtelny[37] stated that adenoid tissue becomes evident by 6 months to 1 year of age and occupies about one-half of the nasopharyngeal cavity by age 2 to 3 years. In his longitudinal cephalometric series, he found that peak mass was observed as early as 9 to 10 years and as late as 14 to 15 years of age, and thereafter the adenoid mass seemed to gradually diminish. He added that the growth of adenoid and tonsil tissue does not follow the classic growth curve described by Scammon[38] for lymphoid tissue.

Subtelny[37] also claimed that large adenoid masses often may be observed during the prepubertal stages of development, when the nasomaxillary complex is growing fast. The presence of large adenoid mass and protruding nasal airway during a growth spurt can have an adverse effect on the nasomaxillary complex and posture and consequently on occlusion.

Effects on occlusion and dentoskeletal structures

Many recent reports indicate that mouth breathing can have an adverse effect on the maxillary structure.[35,39–41] In addition, the growth of the nasomaxillary complex causes some degree of maxillary retrusion, maxillary deficiency, and counterclockwise rotation of the palate; deeper palatal height; retroclined mandibular incisors; increased total anterior facial height and lower facial height; a larger gonial angle; and greater sella-nasion plane, palatal plane, and occlusal plane to mandibular plane angles.

Chronic mouth breathing has negative effects on the maxillofacial complex and occlusion and can cause serious problems in the general health of patients at any age but especially growing children. Much human and animal research has focused on this complicated problem, which is of interest to many specialists, including speech scientists, otolaryngologists, allergists, pediatricians, and orthodontists (Fig 6-19).

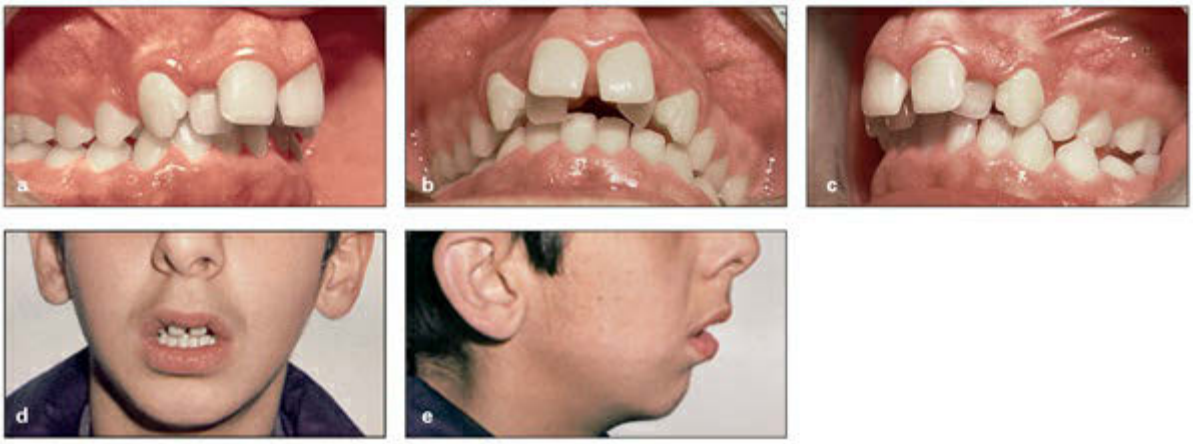


Fig 6-19 *(a to e)* Typical characteristics of patients with mouth breathing caused by nasal obstruction: narrow, dolichocephalic face; convex profile; tapering, narrow maxillary dentition; and anterior crowding.

In an experimental study in primates, Harvold et al[42] induced mouth breathing by obstructing the nasal passages of the animals with silicon nose plugs. They found that the monkeys adapted to nasal obstruction in different ways, and the experimental animals maintained an open mouth. Some increased the oral airway rhythmically, while others maintained the mandible in a lower position with or without protruding their tongue. All experimental animals gradually acquired a facial appearance and dental occlusion different from those of the control animals. Harvold et al[42] concluded that changes in neuromuscular activity in turn affect both muscle development and bone remodeling.

In a 5-year follow-up study, Kerr et al[43] treated 26 children for nasal obstruction

by adenoidectomy and used lateral skull radiographs to compare the patients with a control group of children with normal breathing. They used 36 digitized points to examine mandibular morphology and concluded that this method provided a valuable complement to isolated measurements in determining growth changes. Kerr et al[43] found that the group that underwent adenoidectomy had a more anterior direction of symphyseal growth after surgery and experienced some reversal of the initial tendency to a posterior rotation of the mandible.

Woodside et al[39] studied the amount of maxillary and mandibular growth and the direction of maxillary growth over 5 years following adenoidectomy in 38 children who had severe nasopharyngeal obstruction. They found that both the maxilla and mandible in children who had adenoidectomy grew significantly more than did the jaws of a control group without adenoidectomy; this increase was higher in boys than in girls. Woodside et al[39] detected no difference in the direction of maxillary growth between those who had undergone adenoidectomy and the control group.

Experiments with human subjects have shown that a change in posture does accompany nasal obstruction. For example, Tourne and Schweiger[44] artificially eliminated the respiratory patterns of 25 adults for a period of 1 hour. Cephalometric radiographs were taken before and after the experimental hour to determine the extent of the postural reflexive behavior of the cranium, mandible, hyoid bone, tongue, and lips. They found statistically significant parting of the lips, a drop in mandibular position, and downward movement of the hyoid bone. The change in cranial extension did not reach statistical significance. Tourne and Schweiger[44] added that if the same postural reactions are maintained over a long-term period, they may be instrumental in influencing the vertical craniofacial growth pattern.

As Gugino and Dus[45] stated, every malocclusion is associated with at least some degree of orofacial dysfunction that should be removed to maximize the chances of obtaining a stable treatment outcome. Prolonged, predominant oral respiration, particularly in growing children, can be associated with esthetically and functionally unacceptable facial features.

Etiology

Anything that blocks the nasal airway can reduce or prevent nasal breathing. To overcome this blockage, the mouth is forced to open so that the individual can breathe through the oral pathway.

One of the most common causes of mouth breathing is allergies. Allergies cause

swelling of the mucous membranes that line the nose. Even mild swelling can affect lung oxygenation by closing off the sinus openings; the greater the swelling of the mucosa, the greater percentage of blockage of nasal airway and therefore the more mouth breathing. Inflammation and enlargement of tonsils and adenoids are other factors that block the airway and promote mouth breathing.

Congenital malformations that cause structural deformities can also block nasal airways. These include deviated or enlarged conchae and deviated septum. Sometimes trauma such as a broken nose can distort the anatomy of the nose and cause blockage.

The low tongue posture observed in individuals with oral respiration can prevent normal exertion of lateral force of the tongue on the maxilla and impede the lateral expansion and anterior development of the maxilla.

In some children, mouth breathing is considered a habit. This habit is believed to be sustained abnormal breathing following thumb sucking and long pacifier habits in children who simply fail to breathe through the nose during infancy and childhood, while the face and upper airway are developing and growing fast. Góis et al[40] stated that early disturbed breathing can affect nasal pathway growth and mouth breathing and continue in later ages.

Clinical signs

Early detection of mouth breathing in young children is an important step in interceptive orthodontic treatment. Prolonged mouth breathing, especially during ages of rapid growth, can affect dental occlusion, skeletal morphology, and growth patterns, as well as the child's posture. To review this process and understand the mechanism of these changes, it is useful to first look at the characteristics of a child with these problems.

The child can be examined for signs of mouth breathing in two areas: *(1)* general body growth and posture of the child and *(2)* dentofacial characteristics such as orofacial morphology, soft tissue characteristics, and dental occlusion.

General body growth and posture

Children suffering from chronic mouth breathing usually have problems getting enough oxygen into their blood during sleep. Thus, the sleep cycle is disrupted, growth hormone production is interfered with, and general body growth (size and weight) and even school performance are affected.

Warren[46] studied the effect of airway obstruction on facial growth. He suggested that use of respirometric techniques offers a more quantitative assessment of respiratory behaviors and more objective definitions of airway impairment and mouth breathing. Warren[46] concluded that, in instances when the nasopharyngeal or oropharyngeal airway space is small, exaggerated postural responses in obligatory nasaloral breathers may be detrimental to growth.

Tourne and Schweiger,[44] in experiments with human subjects, showed that a change in posture does accompany nasal obstruction. When the nose is completely blocked, there is usually an immediate change of about 5 degrees in the craniovertebral angle. When the nasal obstruction is removed, the original posture immediately returns. This physiologic response occurs to the same extent, however, in individuals who already have some nasal obstruction, which indicates that it may not totally result from respiratory demands.

Sleep disorders of early childhood are most often the consequence of respiratory disorders due to airway insufficiency in the upper respiratory tract, which has three clinical symptoms: breathing with the mouth open, snoring, and sleep apnea.

Obstructive sleep apnea syndrome (OSAS) is a severe sleep disorder of early childhood, and any delay in diagnosis and treatment may cause prolonged morbidity; early treatment is simpler and more effective.

Many researchers relate SIDS to OSAS. Children with OSAS usually snore (snoring is a manifestation of a blocked airway, which in essence is a milder version of sleep apnea). Bedwetting, poor-quality sleep, and obesity are other effects on these children. These children are usually tired during the day, and their performance in school is poor; they often exhibit anger and frustration. Sometimes they can be misdiagnosed with attention deficit–hyperactivity disorder.

Many experiments in animals and humans have shown the effect of chronic mouth breathing and postural change.[39,42,46] Children who are chronic mouth breathers will inhale and exhale faster than normal breathers (about every 3 seconds instead of every 6 seconds). Sometimes it is noticeable that shoulders rise and fall during breathing, while in normal breathing there is no visible, outward sign of breathing.

Improvement in nasopharyngeal function may occur as a natural phenomenon of normal growth of the upper airway and specifically as a result of the involution of the lymphoid tissue in the respiratory passages. Orofacial dysfunctions are most successfully treated in young patients who still have to experience most of their somatic growth and their growth spurts.

Dentofacial characteristics

Recent studies support the hypothesis that prolonged oral respiration during critical growth periods has an adverse effect on the growth and development of the child's face and jaws. The adverse effects increase with the severity and duration of the problem; especially when chronic mouth breathing and abnormal tongue function are both present, the consequences would be more extensive.

Souki et al[47] reported epidemiologic data on the prevalence of malocclusion among 401 patients ranging from 2 to 12 years of age. All subjects of the study were evaluated by otorhinolaryngologists to confirm mouth breathing. The study found a higher prevalence of posterior crossbite, anterior open bite, and Class II malocclusion.

Trask et al[48] studied the effects of perennial allergic rhinitis on dentoskeletal structures and corroborated earlier reports that allergic children tend to have increased anterior face height and increased overjet/decreased overbite. In another study, Linder-Aronson[36] found that, on average, Swedish children with large adenoids, facial skeleton, and dentition had a significantly longer anterior face height than control children. These children also had a tendency toward maxillary constriction and more upright incisors. Linder-Aronson and Bäckström[49] also found that children who had undergone adenoidectomy tended to return toward the mean cephalometric norm of the control group.

Solow et al[50,51] and Solow and Tallgren[52,53] showed the relationship between facial proportions and head posture. Children who hold their head and chin up usually have vertical problems, a divergent face, greater anterior facial height, and open bite or open bite tendency. Those who exhibit a posture with their head and chin down are more associated with short anterior facial height and deeper bite.

Using lateral cephalograms, Zettergren-Wijk et al[54] compared children suffering from OSAS (mean age of 5.6 years) with a control group. They found that children with OSAS had a more retruded mandible, a more anteriorly inclined maxilla, greater lower anterior facial height, retroclined maxillary and mandibular incisors, reduced airway space, and a less pronounced nose. Over a 5-year period following adenoidectomy, tonsillectomy, or both procedures for treatment of OSAS, there were no statistically significant differences between the two groups. Zettergren-Wijk et al[49] concluded that early treatment of OSAS was successful and that dentofacial morphology was normalized after adenoidectomy/tonsillectomy. They contended that children with OSAS must be diagnosed early and evaluated medically and

dentofacially. This demands close cooperation among pediatricians, otolaryngologists, orthodontists, and pediatric dentists.

In another cephalometric study, Solow et al[51] assessed the anteroposterior diameters of the pharyngeal airway in a sample of 50 male OSAS patients and a reference sample of 103 male students to examine the relationship between these diameters and the posture of the head and the cervical column. They observed that the largest difference was behind the soft palate, where the diameter was 50% narrower in the OSAS sample than in the reference sample. They also concluded that differences in the awake erect posture in OSAS patients represented a compensatory physiologic postural mechanism to maintain airway adequacy.

Good lateral cephalometric radiographs can show the situation of adenoid tissue and the airway passage between the adenoid and soft palate in the sagittal view. Frontal or anteroposterior cephalometric radiographs also can be helpful in showing the width of nasal cavities and the size and shape of the nasal septum. These radiographs can also reveal the size and degree of hypertrophy of the bony nasal turbinates located on the lateral walls of the nasal cavities and the apparent amount of open airway space within the nasal cavity.

Problems associated with mouth breathing

There is an extensive amount of research and reports concerning the effect of chronic mouth breathing on general body health and especially on the dentoskeletal complex of children and adults.[35,39–41] The first consequence of nasal obstruction is the adaptation of the patient to mouth breathing. To facilitate these abnormal types of breathing, these children usually hold their head more upright to increase airway volume; their mandible is dropped down and the mouth is open; the tongue protrudes in the floor of the mouth and does not touch the maxillary dental arch; and suprahyoid muscles are stretched.

Holding the head upright and back produces a reflex forward head posture, putting a large load on the neck and upper back muscles, which, if sustained, will cause permanent postural changes such as abnormal curvature in the cervical and thoracic vertebrae and an altered shoulder posture. Keeping the mouth open can cause overeruption of posterior teeth, anterior open bite, more clockwise rotation of the mandible, and an increased lower facial height. In severe cases of head uprighting, the stretching of suprahyoid muscles through the anterior border of the mandible and downward can produce antegonial notching.

The downward position of the tongue and absence of tongue force on the maxillary arch disturbs the balance of the tongue and perioral muscles. Consequently, maxillary constriction, posterior crossbite, anterior crowding and protrusion, and lip incompetence will develop.

The consequences of these structural and functional problems are summarized in Box 6-2.

Box 6-2	Effects of chronic mouth breathing
Dental and soft tissue changes • Anterior crowding • Maxillary incisor proclination • Anterior open bite • Narrow anterior maxilla • Narrow maxillary arch and bilateral posterior crossbite • Overeruption of molars • Dry lips • Bad breath and periodontal disease, caused by the shift in the bacterial flora in the mouth • Dark circles under the eyes	***Skeletal changes*** • Clockwise rotation of the growing mandible • Increased anterior facial height • Increased lower anterior vertical face height • Long, narrow face • Retrognathia of the mandible • Antegonial notching • Increased mandibular steepness • Maxillary constriction, deep palate, and posterior crossbite • Downward rotation of the posterior palate • Maxillary deficiency • Narrowed nasal airway passage and decreased internasal capacity

Clinical examination

Early detection and diagnosis of mouth breathing is an important part of children's dental examination. All practitioners, and especially pediatric dentists and orthodontists, must pay careful attention in this regard.

Components of evaluation

Evaluation for mouth breathing consists of four steps:

1. The patient and parent should be questioned regarding the child's general health, the possibility of any allergy, the type of breathing during the day and while sleeping, the presence of daytime sleepiness, the presence of snoring, and the child's activities and school performance.
2. The child's general growth and posture, such as height and weight as compared with a normal growth chart, should be assessed. The child's posture should be

observed while he or she walks, sits, is weighed, and is measured for height. The child's shoulders should be watched for the presence of any asymmetry, and the natural head posture, which can be altered in mouth breathers, should be noted.

3. Dentoskeletal characteristics of mouth breathers can be checked by extraoral and intraoral examination. Incompetent lips, forward position of the tongue, increased overjet, maxillary anterior crowding and tapering arch, posterior crossbite or crossbite tendency, and deep and narrow palate are visual signs of mouth breathing.
4. Direct examination for evaluation of the child's breathing can be performed in the clinic while the patient is seated straight and relaxed on a dental chair:

   - First the patient's breathing is observed indirectly without informing the child.
   - Then the patient is asked to take a deep breath without closing the lips, while the examiner watches the patient's lip position and alar muscle reflex carefully.
   - The child is asked to close the lips and take a deep breath. Nose breathers dilate the external nares on inspiration by controlling alar muscles.
   - Other kinds of direct examination can be done, such as holding a cold dental mirror in front of the nose for a little while. Nose breathers cloud the mirror with nasal moisture during breathing. Some practitioners recommend briefly covering the mouth with the hand and watching the child's reaction.

Differential diagnosis for lip incompetence

Lip incompetence can be a sign of mouth breathing. Other factors can cause lip separation during the rest position; these must be differentiated during the patient's examination (see chapter 3):

- Short lips
- Hypotonic lips
- Increased lower facial height
- Severe dental protrusion

When examining the patient, the practitioner should also consider the possibility that any of the aforementioned factors might exist in conjunction with mouth breathing.

Adenoid tissue location

Airway space and adenoid tissue can be seen with a good lateral head radiograph. The status of the tonsils can be examined intraorally. The adenoid is located in the nasopharynx above the level of the soft palate and may extend as far forward as the posterior nasal conchae and downward, approaching the nasal surface of the soft palate, disrupting nasal breathing. Studies indicate that adenoid and tonsil tissues follow a specific cycle of growth. The classic growth curve described by Scammon[38] for lymphoid growth does not apply to adenoid and tonsillar tissues. Subtelny,[37] in a longitudinal cephalometric study, showed that the peak mass of adenoid tissue was observed as early as 9 to 10 and as late as 14 to 15 years.

An otolaryngologist must be consulted for an accurate evaluation, diagnosis, and treatment plan for the nasopharyngeal or oropharyngeal airway space and nasal obstructions.

Orthodontic management

Otolaryngologists have the ability to assess upper airway conditions and decide on any medical or surgical treatment of respiratory dysfunction. However, in patients with maxillary constriction, the orthodontic treatment technique of rapid palatal expansion results in significant changes in children's breathing patterns.[55,56] This type of treatment corrects transverse occlusal disharmony and functional problems and provides more space for teeth. It also increases nasal airway capacity as an immediate result of rapid skeletal expansion. Expansion also provides room for the tongue to rest and function normally in the palate.

In younger children, early detection of respiratory problems and proper orthodontic treatment can correct occlusal abnormalities and prevent adverse effects on dentoskeletal growth patterns. In addition, increasing the volume of the nasal passages will facilitate normal breathing in the child.

Even after medication, anatomical correction of problems, and orthodontic treatment, the child may continue the mouth breathing habit. Suggestions for breaking this habit include use of a piece of surgical tape to keep the mouth closed, use of an oral screen with a thin piece of rubber behind and between the lips to block airflow, or a device to control mandibular dropping during sleep.

In patients with short or hypotonic lips, in whom normal lip seal is distorted, exercises will strengthen the seal of the lips and obstruct the mouth to force

breathing through the nose. For example, the patient can be instructed to hold a sheet of paper between the lips and try to breath through the nose for a period of time a few times a day to improve lip seal. Another exercise involves use of a button with a piece of string tied to it. The patient should hold the button behind the lips and stretch the string while the lips are resisting; this exercise can improve lip seal and tonicity.

Speech and Malocclusion

Although it is widely accepted that teeth play an important role in speech production and although the incidence of malocclusion in persons with defective speech articulation has been reported,[57,58] the relationship between tooth position and speech remains controversial.[30,59]

The majority of sound is developed by the tongue and anterior teeth, and a causal relationship between speech defects and malocclusion has long been assumed to exist. One example is the case of lisping associated with malocclusion of the anterior teeth. Some other teeth irregularities that can be suspected to cause speech disorders are narrow, high palates; incisal irregularities; and spaced or absent teeth. Anteroposterior arch malrelationships (Angle Class II and Class III malocclusions) are also commonly listed as the malocclusions chiefly associated with defective speech.

This does not mean that malocclusions are always the cause of the speech defect or that malocclusions are always the result of a speech defect. Many etiologic factors are common to both problems. Speech defects can be found in persons with or without normal occlusion. In other words, if a speech defect is present, a strict cause-and-effect relationship with malocclusion cannot be assumed. The speech defect may be the result of an underlying emotional problem that is contributing to the malocclusion, or it can be due to a previous deleterious finger sucking habit which itself may be an expression of emotional disturbances.

Sometimes soft tissues, such as the lips and tongue, may adapt to tooth irregularities and compensate for difficulties of articulation and speech.

Subtelny and Subtelny[30] studied Class II division 1 patients with an excessive maxillary dental protrusion of at least 6-mm overjet. This condition has been reported to be associated with abnormal protrusive tongue habits during swallowing. The subjects were grouped into adaptive and maladaptive categories on the basis of speech adequacy (lispers and nonlispers). Speech samples were tape-

recorded, and cephalometric radiographs were obtained during the production of the /*s*/ sounds.

Subtelny and Subtelny[30] considered the /*s*/ sound to be the most commonly defective sound in American English; the teeth and supporting structures are directly involved in its production. They also found that most children between the ages of 10 and 16 years learn to adapt to this malocclusion without speech training. Adaptation can take the form of excessive lower lip function or tongue thrust, but generally these are not evident coincidentally.

The majority of children with speech defects protrude the tongue tip between the incisors during speech and swallowing. When the pattern of tongue thrust is consistent during both speech and swallowing, the pattern of tongue function may be related etiologically to the malocclusion.

Subtelny and Subtelny[30] also revealed, in a sample of orthodontic patients, that children with speech defects did not have a higher incidence of malocclusion than children with normal speech. It was only in the children with open bite malocclusion that speech defects presented constantly. In these cases, lisping was found to be significantly related to the open bite defect.

Speech and sound production mature by age 5 years. Some spontaneous correction of speech sound articulation occurs during eruption of some permanent teeth at the first phase of mixed dentition.

Johnson and Sandy[60] reviewed relevant studies and discussed the difficulties of scientific investigation in this area. They stated that although the adaptability of patients to compensate for abnormal tooth position during speech is recognizable, the mechanism for this adaptation remains incompletely understood. They concluded that certain tooth irregularities can cause some speech disorders, but there is no correlation with the severity of malocclusion and no indication that alteration of tooth position can improve articulation disorders.

Bruxism

Bruxism is defined as a grinding of teeth resulting from a nonfunctional rhythmic contraction of the masseter, temporalis, and internal pterygoid muscles. Bruxism usually occurs while a child is sleeping. Tooth grinding is a subconscious action that is characterized by an audible sound; it is silent during the daytime. Sometimes bruxism is associated with lip or nail biting. The natural reaction of these children is

to grind their teeth to calm themselves; often this practice later develops into a habit.

It seems that both children and adults grind their teeth when they are excited, scared, or as a response to facial pain. This is common in children suffering from ear pain or illness; hyperactive children seem to develop bruxism more often.[61–65]

Bruxism is a destructive habit that may result in structural damage, tooth wear, temporomandibular dysfunction (TMD), facial pain, muscle pain and tenderness, muscle spasms, headache, stiffness of the neck, and periodontal problems.

Incidence

According to Peterson and Schneider,[3] the prevalence of bruxism in children is very high, ranging from 5.1% to 96%. This wide range is due to differences in diagnostic criteria, definitions, the technique of sampling, and the population. In a study of 15- to 18-year-old Swedish adolescents, Nilner[66] reported a prevalence of 7% to 15.1%. Reding et al[67] reported a more frequent incidence among boys.

Etiology

The exact cause of bruxism is unknown; there is much controversy, and many different factors have been proposed. Most explanations center on local, systemic, and psychologic factors.

The local theory suggests that bruxism is a reaction to an occlusal interference of opposing teeth, high restoration, or some irritating dental condition during function or at rest.[68] Some systemic factors in relation to bruxism have also been proposed. These include intestinal parasites, nutritional deficiencies, allergies, or endocrine disorders.

Nervous disorders, psychologic dysfunction, emotional illness, anxiety, frustration, stress, allergy, and asthma are other proposed factors.[61–65] Children with cerebral palsy and the severely mentally retarded grind their teeth more commonly.[61–65]

Funch and Gale[69] state that bruxism is correlated with psychologic factors; they believed that the patient's lifestyle has great influence on severity, frequency, and duration of the habit. However, the exact contribution of psychologic factors remains debatable.

Attanasio[70] describes bruxism as a condition of multifactorial etiology,

determined by an association of psychologic, local, and systemic factors.

Antonio et al[71] evaluated two cases of severe bruxism. The patients were of similar age and dentition but had different life histories and socioeconomic backgrounds. One patient lived in a violent area and was apprehensive, while the other lived in a good area but suffered from night terrors. Antonio et al[71] concluded that the condition in both patients was triggered by psychologic disturbances, resulting from harrowing, although different experiences. They also concluded that bruxism is independent of socioeconomic status but closely associated with each patient's life events.

Reding et al[67] studied simultaneous recordings of brain waves, eye movements, and masticatory muscle potentials throughout the night and found a temporal relationship between episodes of bruxism and periods of rapid eye movements indicative of dreaming.

An association between mouth breathing and the occurrence of night bruxism has also been studied. Grechi et al[72] investigated the occurrence of bruxism in 60 children with nasal obstruction. The children were divided into two groups, one group with bruxism and one group without. The researchers concluded that bruxism and deleterious oral habits such as biting behavior (objects, lips, and nails) were significantly present, together with the absence of sucking habits, in children with nasal obstruction.

Effects on the dentition

Tooth wear, a common effect of bruxism, can range from mild to severe; it can be localized in some areas or can be seen extensively throughout the dentition. Attanasio[70] claimed that bruxism results in other traumas to the dentition and surrounding tissues. These include thermal hypersensitivity, tooth hypermobility, injury to the periodontal ligament and periodontium, hypercementosis, fractured cusps, pulpitis, and pulpal necrosis.

In a review of the literature from 1970 to 2007, Barbosa et al[73] concluded that the prevalence of TMD in children and adolescents varies widely. They also suggested that bruxism in children may be a consequence of immaturity of the masticatory neuromuscular system.

Some investigators report an association between bruxism with TMDs in the primary and mixed dentition. Pereira et al[74] studied the risk indicators for signs and symptoms of TMD in 106 children between 4 and 12 years of age and found that

12.26% of those studied showed at least one sign or symptom of TMD. Children with bruxing or clenching habits and those with posterior crossbite had a greater likelihood of developing signs and symptoms of TMD.

Treatment

Research indicates that the etiology of bruxism is not well understood and might be considered as a multifactorial problem. Before any treatment planning or management, the practitioner must look at all medical and dental data.

Therapeutic approaches to bruxism in children depend on the patient's health status and etiologic factors. Treatment can include occlusal adjustment of the dentition, use of interocclusal appliances, behavior modification, or pharmaceuticals. Consequently, application of a team approach with other specialties might be the best option.

A soft or hard occlusal bite plate is a useful orthodontic appliance with reasonable treatment results that also prevents the destructive effects of bruxism. Hachmann et al[75] evaluated two groups of children with bruxism aged 3 to 5 years, one group receiving no treatment and one using a nocturnal bite plate. Study casts of both groups were taken to compare the progression of wear facets during an 8-month evaluation period. The researchers found increased wear facets in the control group, while the treatment group showed no increase in wear facets, even after removal of the device.

Children with grinding and clenching habits usually have occlusal wear, deep bite, and in severe cases show shortened lower facial height. Vinyl bite guards are fabricated to cover the occlusal surfaces of all teeth and can be worn at night to prevent continuing abrasion.

The author recommends application of a simple Hawley appliance with anterior bite plate, which is not only easy for children to wear but also prevents occlusal wear. This appliance, by opening the posterior bite, promotes posterior tooth eruption and alveolar growth and consequently reduces the deep bite and enhances lower facial height during the early and middle mixed dentition.

Besides selective grinding and use of soft or hard interdental splints, other treatment approaches have been proposed, such as psychotherapy, hypnosis, physical therapy, and habit modification programs. Other factors to consider are the child's personality and home and social environments; other methods of management include increasing emotional support, removing threats of punishment, and

maintaining authority.

Summary

- Non-nutritive sucking is sucking on an object (digit or pacifier) that is not related to the ingestion of nutrients.
- Abnormal habits must be distinguished from normal habits that are a part of normal oropharyngeal function and that play an important role in craniofacial growth and occlusal physiology.
- Abnormal oropharyngeal functions such as finger sucking, mouth breathing, tongue thrust swallowing, low tongue position at rest, unilateral chewing, and abnormal postures of perioral musculature can cause malocclusion.
- There are two schools of thought regarding the cause of abnormal habits. The psychoanalytic school regards a habit as a symptom of some emotional disturbance. Few studies support the psychologic hypothesis. Behaviorists view a habit as a simple learned act, with no underlying neurosis; learning theory appears more relevant.
- Thumb sucking is one of the earliest examples of the neuromuscular learning process; it has been shown that the fetus sometimes sucks fingers in utero. Digit sucking has also been related to inadequate sucking (early weaning). In contrast, some investigators contend that prolonged nursing strengthens the oral drive and sucking habit. Practitioners must consider the psychologic background of the patient as well as the conditions under which children conduct the habit.
- The extent of damage of any abnormal force applied to the dentition, such as that from sucking on a finger or any other object, depends on the duration, frequency, and intensity of the habit. The duration seems to have the greatest effect, and research has that shown 5 to 6 hours of force per day is necessary to cause tooth movement.
- Finger sucking, depending on severity and type, can cause damage to the dentoskeletal structure, including open bite, overjet, dental protrusion and spacing, posterior crossbite, Class II malocclusion, and clockwise rotation of the mandible.
- Research indicates that two-thirds of finger suckers stop by age 5 years. Sucking habits during the primary dentition years usually have little, if any, long-term effect.

- Intervention is recommended after age 4 or 5 years, or earlier if it is a habit of high intensity and long duration that can cause clockwise rotation and antegonial notching of the mandible.
- Swallowing is not a learned behavior; it is a physiologically controlled function at the subconscious level. Tongue thrust must be considered as a concomitant, rather than causative, factor in most cases. Even after a thumb habit has ceased, a compensatory tongue thrust may be operating, which may offer considerable difficulty in management. • Three types of tongue thrust have been described: simple tongue-thrust swallow, complex tongue-thrust swallow, and retained infantile swallow. Factors that may prohibit someone from developing a mature or adult swallowing pattern and continue the tongue thrust habit are hereditary factors such as a large tongue and vertical skeletal problems such as a steep mandibular plane angle or wide gonial angle.
- Mouth breathing is an abnormal habit caused by nasal obstruction. Many factors cause nasal obstruction, including allergies, nasal congestion, deviated conchae, or large adenoids.
- Prolonged oral respiration, particularly in growing children, can cause dentoskeletal damage such as posterior cross-bite, anterior open bite, Class II malocclusion, and antegonial notching.
- Early intervention in and proper management of all deleterious oral habits can prevent or reduce dentoskeletal damage at later ages. Proper intervention requires careful diagnosis and thorough treatment planning.
- Before undertaking any orthodontic intervention and mechanotherapy for the treatment of oral habits, the practitioner should ensure that both the child and the family want to cooperate and that all parties are aware of the advantages and disadvantages of every available method of treatment.

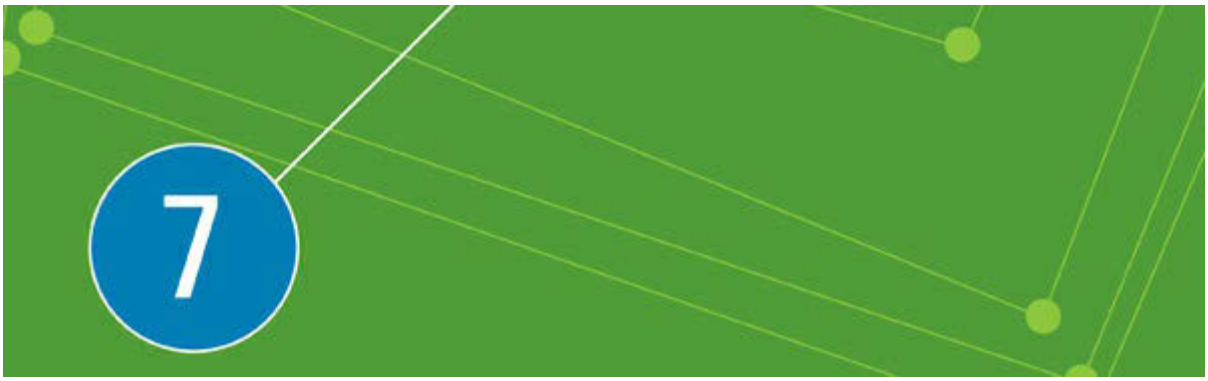
References

1. Bruun RA, Hertzberg JL, Tayer BH. Oral habits: Non-nutritive sucking and tongue thrusting. Am Assoc Orthod Orthod Dialogue 1991;4:2–3.
2. Bryant P, Gale E, Rugh J. Oral Motor Behavior Workshop, 16–17 May 1979 [Report NIH 79-1845]. Bethesda, MD: National Institutes of Health, 1979.
3. Peterson JE, Schneider PE. Oral habits: A behavioral approach. Pediatr Clin North Am 1991;38:1289–1307.
4. Freud S. Three Contributions to the Theory of Sex, ed 3. New York: Nervous and Mental Disease Publishing, 1919.
5. Haryett RD, Hansen FC, Davidson PO, Sandilands ML. Chronic thumb sucking: The psychologic effects and

the relative effectiveness of various methods of treatment. Am J Orthod 1967;53:569–585.
6. Levy SM, Slager SL, Warren JJ, Levy BT, Nowak AJ. Associations of pacifier use, digit sucking, and child care attendance with cessation of breast feeding. J Fam Pract 2002;51:465.
7. Moimaz SA, Zina LG, Saliba NA, Saliba O. Association between breast-feeding practices and sucking habits: A cross-sectional study of children in their first year of life. J Indian Soc Pedod Prev Dent 2008;26:102–106.
8. Sears R, Wise G. Relation of cup-feeding in infancy to thumb sucking and oral drive. Am J Orthopsychiatry 1950;20:123–138.
9. Benjamin L. Nonnutritive sucking and dental malocclusion in the deciduous and permanent teeth of the rhesus monkey. Child Dev 1962;33:29–35.
10. Hepper PG, Wells DL, Lynch C. Prenatal thumb sucking is related to postnatal handedness. Neuropsychologia 2005;43:313–315.
11. Helle A, Haavikko K. Prevalence of earlier sucking habits revealed by anamnestic data and their consequences for occlusion at the age of eleven. Proc Finn Dent Soc 1974;70:191–196.
12. Friman PC, McPherson KM, Warzak WJ, Evans J. Influence of thumb sucking on peer social acceptance in first-grade children. Pediatrics 1993;91:784–786.
13. Kelly JE, Sanchez M, Van Kirk LE. An Assessment of the Occlusion of Teeth of Children 6–11 Years, United States. National Center for Health Statistics, US Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. HRA 74-1612. Rockville, MD: National Center for Health Statistics, 1973.
14. Van Norman RA. Digit-sucking: A review of the literature, clinical observations and treatment. Int J Orofacial Myology 1997;23:14–34.
15. Proffit WR. Lingual pressure patterns in the transition from tongue thrust to adult swallowing. Arch Oral Biol 1972;17:555–563.
16. Romanou-Kouvelas K, Kouvelas N. Oral habits. Etiology and treatment. Hell Stomatol Chron 1988;32:285–291.
17. Slade PD, Owens RG. A dual process model of perfectionism based on reinforcement theory. Behav Modif 1998;22:372–390.
18. Haskell BS, Mink JR. An aid to stop thumb-sucking: The “Bluegrass” appliance. Pediatr Dent 1991;13:83–85.
19. Larsson E. Artificial sucking habits: Etiology, prevalence and effect on occlusion. Int J Orofacial Myology 1994;20:10–21.
20. Adair SM. Pacifier use in children: A review of recent literature. Pediatr Dent 2003;25:449–458.
21. Castilho SD, Rocha MA. Pacifier habit: History and multidisciplinary view. J Pediatr (Rio J) 2009;85:480–489.
22. Li DK, Willinger M, Petitti DB, Odouli R, Liu L, Hoffman HJ. Use of a dummy (pacifier) during sleep and risk of sudden infant death syndrome (SIDS): Population based case-control study. BMJ 2006;332(7532):18–22.
23. Cinar DN. The advantages and disadvantages of pacifier use. Contemp Nurse 2004;17:109–112.
24. Hitchcock S. Endorsing safe infant sleep: A call to action. Nurs Womens Health 2012;16:386–396.
25. Sexton S, Natale R. Risks and benefits of pacifiers. Am Fam Physician 2009;79:681–685.
26. de Holanda AL, dos Santos SA, Fernandes de Sena M, Ferreira MA. Relationship between breast- and bottle-feeding and non-nutritive sucking habits. Oral Health Prev Dent 2009;7:331–337.
27. Bishara SE, Nowak AJ, Kohout FJ, Heckert DA, Hogan MM. Influence of feeding and non-nutritive sucking methods on the development of the dental arches: Longitudinal study of the first 18 months of life. Pediatr Dent 1987;9:13–21.
28. Graber TM. The ‘three Ms’: Muscles, malformation, and malocclusion. Am J Orthod 1963;49:418–450.
29. Moyers RE. The infantile swallow. Rep Congr Eur Orthod Soc 1964;40:180–187.
30. Subtelny JD, Subtelny JD. Malocclusion, and speech, and deglutition. Am J Orthod 1962;48:685–697.
31. Ovsenik M. Incorrect orofacial functions until 5 years of age and their association with posterior crossbite. Am J Orthod Dentofacial Orthop 2009;136:375–381.
32. Peng CL, Jost-Brinkmann PG, Yoshida N, Miethke RR, Lin CT. Differential diagnosis between infantile and mature swallowing with ultrasonography. Eur J Orthod 2003;25:451–456.

33. Graber TM, Rakosi T, Petrovic AG. Dentofacial Orthopedics with Functional Appliances, ed 2. St Louis: Mosby, 1997.
34. Subtelny JD, Subtelny JD. Oral habits—Studies in form, function, and therapy. Angle Orthod 1973;43:349–383.
35. Subtelny JD. Oral respiration: Facial maldevelopment and corrective dentofacial orthopedics. Angle Orthod 1980;50:147–164.
36. Linder-Aronson S. Adenoids: Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. Acta Otolaryngol Scand Suppl 1970;265:1–132.
37. Subtelny JD. The significance of adenoid tissue in orthodontia. Angle Orthod 1954;24:59–69.
38. Scammon RE. The first seriatim study of human growth. Am J Phys Anthropol 1927;10:329–336.
39. Woodside DG, Linder-Aronson S, Lundstrom A, McWilliam J. Mandibular and maxillary growth after changed mode of breathing. Am J Orthod Dentofacial Orthop 1991;100:1–18.
40. Góis EG, Ribeiro-Júnior HC, Vale MP, et al. Influence of nonnutritive sucking habits, breathing pattern and adenoid size on the development of malocclusion. Angle Orthod 2008;78:647–654.
41. Gupta N, Gupta SD, Varshney S, Singh R, Bist SS, Barthwala J. Orthodontic treatment after adenoidectomy patients: Effect on jaw relations in saggital plane. Indian J Otolaryngol Head Neck Surg 2009;61:153–156.
42. Harvold EP, Tomer BS, Vargervik K, Chierici G. Primate experiments on oral respiration. Am J Orthod 1981;79: 359–372.
43. Kerr WJ, McWilliam JS, Linder-Aronson S. Mandibular form and position related to changed mode of breathing— A five-year longitudinal study. Angle Orthod 1989;59: 91–96.
44. Tourne LP, Schweiger J. Immediate postural responses to total nasal obstruction. Am J Orthod Dentofacial Orthop 1996;110:606–611.
45. Gugino CE, Dus I. Unlocking orthodontic malocclusions: An interplay between form and function. Semin Orthod 1998;4:246–255.
46. Warren DW. Effect of airway obstruction upon facial growth. Otolaryngol Clin North Am 1990;23:699–712.
47. Souki BQ, Pimenta GB, Souki MQ, Franco LP, Becker HM, Pinto JA. Prevalence of malocclusion among mouth breathing children: Do expectations meet reality? Int J Pediatr Otorhinolaryngol 2009;73:767–773.
48. Trask GM, Shapiro GG, Shapiro PS. The effects of perennial allergic rhinitis and dental and skeletal development: A comparison of sibling pairs. Am J Orthod Dentofacial Orthop 1987;92:286–293.
49. Linder-Aronson S, Bäckström A. A comparison between mouth and nose breathers with respect to occlusion and facial dimension. Odontol Revy 1960;11:343–376.
50. Solow B, Ovesen J, Nielsen PW, Wildschiødtz G, Tallgren A. Head posture in obstructive sleep apnoea. Eur J Orthod 1993;15:107–114.
51. Solow B, Skov S, Ovesen J, Norup PW, Wildschiødtz G. Airway dimensions and head posture in obstructive sleep apnoea. Eur J Orthod 1996;18:571–579.
52. Solow B, Tallgren A. Head posture and craniofacial morphology. Am J Phys Anthropol 1976;44:417–435.
53. Solow B, Tallgren A. Natural head position in standing subjects. Acta Odontol Scand 1971;29:591–607.
54. Zettergren-Wijk L, Forsberg CM, Linder-Aronson S. Changes in dentofacial morphology after adeno-/tonsillectomy in young children with obstructive sleep apnoea—A 5-year follow-up study. Eur J Orthod 2006;28:319–326.
55. Haas AJ. Palatal expansion: Just the beginning of dentofacial orthopedics. Am J Orthod 1970;57:219–255.
56. Matsumoto MA, Itikawa CE, Valera FC, Faria G, Anselmo-Lima WT. Long-term effects of rapid maxillary expansion on nasal area and nasal airway. Am J Rhinol Allergy 2010;24:161–165.
57. Weinberg B. A cephalometric study of normal and defective -s- articulation and variations in incisor dentition. J Speech Hear Res 1968;11:288–300.
58. Khinda V, Grewal N. Relationship of tongue-thrust swallowing and anterior open bite with articulation disorders: A clinical study. J Indian Soc Pedod Prev Dent 1999;17:33–39.
59. Doshi UH, Bhad-Patil WA. Speech defect and orthodontics: A contemporary review. Orthodontics (Chic) 2011;12:340–353.

60. Johnson NC, Sandy JR. Tooth position and speech—Is there a relationship? Angle Orthod 1999;69:306–310.
61. Vieira-Andrade RG, Martins-Júnior PA, Corrêa-Faria P, et al. Oral mucosal conditions in preschool children of low socioeconomic status: Prevalence and determinant factors [epub ahead of print 26 January 2013]. Eur J Pediatr.
62. Serra-Negra JM, Paiva SM, Auad SM, Ramos-Jorge ML, Pordeus IA. Signs, symptoms, parafunctions and associated factors of parent-reported sleep bruxism in children: A case-control study. Braz Dent J 2012;23:746–752.
63. Ghafournia M, Hajenourozali Tehrani M. Relationship between bruxism and malocclusion among preschool children in Isfahan. J Dent Res Dent Clin Dent Prospects 2012;6:138–142.
64. Renner AC, da Silva AA, Rodriguez JD, et al. Are mental health problems and depression associated with bruxism in children? Dent Oral Epidemiol 2012;40:277–287.
65. Ferreira-Bacci Ado V, Cardoso CL, Díaz-Serrano KV. Behavioral problems and emotional stress in children with bruxism. Braz Dent J 2012;23:246–251.
66. Nilner M. Prevalence of functional disturbances and diseases of the stomatognathic system in 15- to 18-year-olds. Swed Dent J 1981;5:189–197.
67. Reding GR, Rubright WC, Rechtschaffen A, Daniels RS. Sleep pattern of tooth-grinding: Its relationship to dreaming. Science 1964;145:725–726.
68. Lindqvist B. Occlusal interferences in children with bruxism. Odontol Revy 1973;24:141–148.
69. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. J Behav Med 1980;3:385–387.
70. Attanasio R. Nocturnal bruxism and its clinical management. Dent Clin North Am 1991;35:245–252.
71. Antonio AG, Pierro VS, Maia LC. Bruxism in children: A warning sign for psychological problems. J Can Dent Assoc 2006;72:155–160.
72. Grechi TH, Trawitzki LV, de Felício CM, Valera FC. Bruxism in children with nasal obstruction. Int J Pediatr Otorhinolaryngol 2008;72:391–396.
73. Barbosa Tde S, Miyakoda LS, Pocztaruk Rde L, Rocha CP, Gavião MB. Temporomandibular disorders and bruxism in childhood and adolescence: Review of the literature. Int J Pediatr Otorhinolaryngol 2008;72:299–314.
74. Pereira LJ, Costa RC, França JP, Pereira SM, Castelo PM. Risk indicators for signs and symptoms of temporomandibular dysfunction in children. J Clin Pediatr Dent 2009;34:81–86.
75. Hachmann A, Martins EA, Araujo FB, Nunes R. Efficacy of the nocturnal bite plate in the control of bruxism for 3- to 5-year-old children. J Clin Pediatr Dent 1999;24:9–15.



Orthodontic Management of Hypodontia

Tooth agenesis (hypodontia) is the most common craniofacial malformation; it can occur as an isolated condition (non-syndromic) or in association with other developmental anomalies (syndromic). Lack of one or more teeth is a congenital defect that occurs during the initiation and proliferation stages of tooth formation. The large number of genes involved in the odontogenic process means that there are numerous opportunities for mutations to disrupt this process. Recent advances in genetic techniques have begun to identify the complex process of reciprocal interactions in tooth development and the genetic mechanisms that underlie the odontogenic process.

Congenital absence of teeth has great potential to disrupt normal occlusal development; it can result in abnormal spacing, tipping of adjacent teeth, abnormal tooth relationships, and disturbed intercuspation and interdigitation. Inclined teeth can result in occlusal trauma to the affected teeth, areas of stagnation susceptible to caries, periodontal problems, bone loss, and mandibular shift. Missing anterior teeth can disturb physiologic functions such as speech and mastication and can seriously impair the patient's esthetics. These issues can profoundly affect the patient's self-esteem and confidence, resulting in psychologic problems.

Muller et al[1] reported an increased prevalence of hypodontia during the 20th century. Therefore, future identification and analysis of its genetic basis is essential for early detection and intervention to obtain acceptable alignment and occlusal

relationships. Timely management of hypodontia can be of great benefit to the oral health and occlusion of children who present with such anomalies. Early intervention reduces the complexity of the problems and, in some situations, may eliminate the need for a second phase of treatment.

Terminology

Hypodontia

Hypodontia is a general term used for the congenital absence of teeth. This anomaly can appear as absence of a single tooth, agenesis of several teeth, or even complete tooth agenesis in patients with ectodermal dysplasia syndrome.

Oligodontia

Oligodontia or *partial hypodontia* is a term that has been used to describe the absence of multiple teeth. Some investigators use this term to refer to the absence of three or more tooth buds, while others have defined this term as the congenital absence of at least four teeth. Oligodontia can be present concomitant with syndromic conditions or can arise in individuals without a syndromic condition or any general abnormalities.

Anodontia

Anodontia (*total hypodontia*), or absence of all teeth, is a very rare anomaly that has occurred in some patients with ectodermal dysplasia.

Prevalence

Many studies have been published on the subject of hypodontia, and they have reported a wide range of prevalence rates (Table 7-1). Excluding third molars, the incidence of other missing teeth has been reported to be between 1.6% and 9.6%. Almost all reports show a higher prevalence in girls than in boys. The teeth most frequently absent are the maxillary lateral incisors and mandibular second

premolars.

Table 7-1	Prevalence of hypodontia reported in various populations*		
Investigator	**Year**	**Country**	**Prevalence**
Brekhus et al[2]	1944	USA	1.60%
Rothenberg and Werther[3]	1939	USA	2.30%
Byrd[4]	1943	USA	2.80%
Dolder[5]	1937	Switzerland	3.40%
Shah and Boyd[6]	1978	Canada	3.60%
Buenviaje and Rapp[7]	1984	USA	3.70%
Brown[8]	1957	USA	4.30%
Rose[9]	1966	England	4.30%
Gimmes[10]	1964	Norway	4.50%
Eidelman et al[11]	1973	Israel	4.60%
Glenn[12]	1964	USA	5.10%
Hermel et al[13]	1971	Israel	5.30%
Grahnén[14]	1956	Sweden	6.10%
Lynham[15]	1990	Australia	6.30%
Thompson and Popovich[16]	1974	Canada	7.40%
Maklin et al[17]	1979	USA	7.50%
Locht[18]	1980	Denmark	7.70%
Magnússon[19]	1977	Iceland	7.90%
Haavikko[20]	1971	Finland	8.00%

Hunstadbraten[21]	1973	Norway	10.10%
Bahreman et al*	2007	USA	6.38%

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

Many of these studies were conducted with regard to specific populations, with some variations in the findings (see Table 7-1). For example, Brekhus et al[2] conducted a hypodontia survey in American children and found the prevalence to be 1.6%, whereas Hunstadbraten[21] conducted a survey of Norwegian children and found the prevalence to be 10.1%.

In an unpublished study performed at the Eastman Institute for Oral Health, 800 panoramic radiographs of children aged 6 to 17 years were randomly selected from both active and inactive chart pools with no regard to sex or ethnicity (Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007). Patients who were congenitally missing third molars and children who were syndromic or had clefts were excluded from the study. The aim of this investigation was to determine the prevalence of anomalies of tooth number (hypodontia and hyperdontia) of three racial/ethnic groups (whites, blacks, and Hispanics) in the greater Rochester, New York, area.

Prevalence data were calculated from the findings. Fisher exact test was performed to assess the statistical significance of the findings. Among the 800 panoramic radiographs evaluated, 51 children showed hypodontia (6.38%). The incidence was 6.57% in girls and 6.15% in boys (Table 7-2). The racial/ethnic distribution was as follows: 22 of 383 (5.74%) among black subjects, 23 of 292 (7.88%) among white subjects, and 5 of 103 (4.85%) among Hispanic subjects (Table 7-3). The ethnic distribution, when controlled by sex, showed that white females had the highest prevalence of hypodontia, 14 of 155 subjects (9.03%), while Hispanic males had the lowest rate, 2 of 47 (4.26%) (Table 7-4).

Table 7-2	Distribution of hypodontia by sex*			
Sex	Hypodontia		Total patients	Percentage†
	No	Yes		
Male	351	23	374	6.15%
Female	398	28	426	6.57%
Total	*749*	*51*	*800*	*6.38%*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.
†Fisher exact test (*P* value = .89) revealed no statistically significant difference.

Table 7-3	Distribution of hypodontia by ethnicity*			
Ethnic group	Hypodontia		Total patients	Percentage†
	No	Yes		
Black	361	22	383	5.47%
White	269	23	292	7.88%
Hispanic	98	5	103	4.85%
Other	21	1	22	4.54%
Total	*749*	*51*	*800*	*6.38%*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.
†Fisher exact test (*P* value = .64) revealed no statistically significant difference.

Table 7-4	Distribution of hypodontia by ethnicity and sex*			
Group	Hypodontia		Total patients	Percentage
	No	Yes		
Black males	167	12	179	6.70%
Black females	194	10	204	4.90%
Hispanic males	45	2	47	4.26%
Hispanic females	53	3	56	5.36%
White males	128	9	137	6.57%
White females	141	14	155	9.03%
Other males	11	0	11	0.00%
Other females	10	1	11	9.09%
Total	*749*	*51*	*800*	*6.38%*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

The most common congenitally missing tooth was the mandibular permanent second premolar, followed by the maxillary second premolar, followed by the maxillary lateral incisor (Table 7-5).

Table 7-5	Distribution of congenitally missing teeth (hypodontia) by location*		
Mandible		Maxilla	
Location	**No. of patients**	**Location**	**No. of patients**
Central incisor	2	Central incisor	0
Lateral incisor	7	Lateral incisor	12
Canine	0	Canine	4
First premolar	3	First premolar	2
Second premolar	32	Second premolar	13
First molar	2	First molar	0

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

There were 77 congenitally missing teeth distributed among the 51 panoramic radiographs exhibiting hypodontia. Of those, 36 radiographs revealed only one congenitally missing tooth, and 1 panoramic radiograph showed 7 congenitally missing teeth. The distribution of the number of congenitally missing teeth shown on each panoramic radiograph demonstrating hypodontia is shown in Table 7-6.

Table 7-6	Distribution of missing teeth by panoramic radiograph*
No. of missing teeth	No. of panoramic radiographs
1	36
2	12
3	0
4	1
5	0
6	1
7	1

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

A study by Bahreman and Shokoofan[22] examined the panoramic radiographs of 610 Iranian children between the ages of 9 and 14 years. The investigators found that, excluding third molars, the prevalence of hypodontia was 4.0% in boys and 6.5% in girls. The most frequently missing tooth was the mandibular second premolar.

According to Muller et al,[1] the prevalence of hypodontia in whites increased during the 20th century.

Grahnén and Granath[23] concluded that hypodontia is less frequent in the primary dentition but that there is a high correlation between primary tooth absence and permanent tooth absence.

Etiology

Different etiologic factors have been proposed for this anomaly:

- Genetic factors
- Environmental factors:
  - Allergy
  - Facial trauma
  - Maternal medications during pregnancy
  - Endocrine disturbances
  - Maternal health during pregnancy
  - Maternal rubella (German measles) during pregnancy
  - Evolutionary dental changes
  - Localized inflammation and infection during the initial stage of tooth formation
  - Systemic conditions (rickets, syphilis)
  - Dysplastic syndromes (ectodermal dysplasia) and abnormalities of the ectodermal structures (discussed later in this chapter)
  - Chemotherapy and irradiation

Genetic factors

Odontogenesis is a fascinating process consisting of a complex series of epithelial-mesenchymal interactions involving growth factors and other morphogenetic factors. A large number of genes are involved in this process, and the opportunity for mutations to disrupt the process of odontogenesis is very high.

Nonsyndromic agenesis of permanent teeth is the most common developmental dental anomaly. Excluding the third molars, the prevalence of other missing teeth has been reported with a wide range of between 1.6% and 9.6%; the prevalence for third molars is more than 20%. The prevalence of hypodontia in the primary dentition is less than 1%. Hypodontia usually occurs in the incisor region and is associated with missing succedaneous teeth.[14] Mandibular second premolars and maxillary lateral incisors are the most frequently missing teeth in the permanent dentition.

Genetically nonsyndromic tooth agenesis is a heterogenous condition, and it is believed that different phenotypic forms are caused by different mutated genes.[24] In a study of families, Burzynski and Escobar[24] established that lateral incisor and premolar hypodontia is inherited via an autosomal-dominant gene that demonstrates incomplete penetrance. This form of hypodontia, affecting one or a few teeth (maxillary lateral incisors and mandibular second premolars) is the most common.

Arte et al,[25] in a study of 214 Finnish family members in three generations of 11 probands, evaluated the characteristic of genetic linkage of incisor-premolar hypodontia. They confirmed the existence of an incisor-premolar hypodontia trait. They concluded that it is a genetic condition with autosomal-dominant transmission with reduced penetrance in other relatives. They found the prevalence of hypodontia and/or peg-shaped teeth to be more than 40% in first- and second-degree relatives and 18% in first cousins of the probands.

They also found that 4 out of 9 carriers of the hypodontia gene had some dental anomalies, including small maxillary lateral incisors, ectopic canines, taurodontism, and rotated premolars. These anomalies were also observed at higher than normal frequency in relatives affected with hypodontia.[25]

Advanced technology in molecular biology and genetics and the completion of the Human Genome Project have made the DNA sequence of all 24 human chromosomes available. Now, localization of all human genes and, ultimately, determination of their function are possible.

Homeoboxes, or *Hox* genes, are present in the genomes of many organisms, from fruit flies to humans. This DNA sequence is found within genes that are involved in

the regulation of patterns of development (morphogenesis) during embryonic life. A small mutation in a *Hox* gene could have profound effects on an organism.

One family of homeobox genes, called *MSX genes*, seems to play a crucial role in development. Recently, tooth development was shown to be inhibited in transgenic mice lacking a functional *Msx1* gene.[25] Initial discoveries indicate that the homeodomain proteins MSX1 and the paired-domain transcription factor PAX9 are causative genes in tooth morphogenesis in mice.[25]

Lidral and Reising[26] tested the hypothesis that *MSX1* mutations are a common cause of congenital tooth agenesis. They used single-strand conformation analysis to screen for mutations in 92 affected individuals representing 82 nuclear families. Two siblings from a large family with autosomal-dominant tooth agenesis exhibited a Met61Lys substitution. Complete concordance of the mutation with tooth agenesis was observed in the extended family. The siblings had a pattern of severe tooth agenesis similar to that described in other reports, suggesting that mutations in *MSX1* are responsible for a specific pattern of inherited tooth agenesis. No mutations were found in more common cases of incisor or premolar agenesis, supporting the theory that these have a different etiology.[26]

In a review of current literature about the molecular mechanisms responsible for selective tooth agenesis in humans, Mostowska et al[27] concluded that the only genes associated with the nonsyndromic form of tooth agenesis are *MSX1* and *PAX9*, which encode transcription factors that play a critical role during tooth development.

Seifi et al[28] conducted a study of 40 Iranian children; the subjects included 20 unaffected individuals and 20 affected individuals with at least one congenitally missing tooth. DNA was extracted from all 40 individuals, and a polymerase chain reaction test for *MSX1* was carried out. Results verified the presence of the mutation in all 20 affected individuals but not in the 20 unaffected individuals, indicating that mutations of the *MSX1* gene contribute to tooth agenesis in Iranian individuals.

Different results of genetic studies have drawn interest to phenotypic variations of this anomaly. Studies such as those by Nieminen et al[29] and Vastardis[30] have already shown that different phenotypic forms of tooth agenesis are caused by different mutated genes.

Genetic studies have begun to elucidate the relationship between specific gene and dentition anomalies and expression elsewhere in the body. Lammi et al[31] studied a Finnish family in which severe permanent tooth agenesis (oligodontia) and colorectal neoplasia segregated with dominant inheritance. Results indicated that Wnt signaling regulates embryonic pattern formation and morphogenesis of most

organs. (The Wnt signaling pathway is a network of signaling proteins; aberrations may lead to cancer.) Eleven members of the family lacked at least eight permanent teeth; two members developed only three permanent teeth each. Colorectal cancer or precancerous lesions of variable types were found in eight of the patients with oligodontia.

To identify the causative mutation, Lammi et al[31] used positional cloning and found that mutations in the *AXIN2* gene can cause agenesis of several permanent teeth and may be a predisposing factor in familial colon cancer. However, variable expression of the trait suggests that there is a polygenic mode of inheritance in which epistatic genes and environmental factors interact.

Environmental factors

Hypodontia has a significantly higher prevalence in the relatives of affected individuals than in the general population. Nevertheless, the differences found in hypodontia between monozygotic twins suggest that environmental factors can also influence odontogenesis.

To identify the differences in presence, severity, and location of hypodontia among family members, Parkin et al[32] examined 117 first-degree relatives of 41 index patients clinically and radiographically. The study concluded that the occurrence of hypodontia is not solely determined by genetic factors. Epigenetic and environmental factors are probably also important; the possibility that this condition has a multifactorial etiology must be considered.

Environmental factors can play a variety of roles in tooth agenesis and arrested tooth development. These factors can act independently or as an enforcing agent in positioning and physical development of the tooth buds. To evaluate the possible influence of environmental factors on tooth development and disturbances during odontogenesis, many investigators have assessed different environmental factors.

Allergy

Yamaguchi et al[33] investigated allergy as a predisposing factor for hypodontia in an examination of 3,683 patient files; they found 215 patients to have hypodontia (prevalence of 5.8%). They also found a high prevalence of systemic complications, including allergy, asthma, atopy, and enlarged adenoids, associated with hypodontia. However, only allergies showed a significant relationship with hypodontia ($P < .01$).

Facial trauma

Grahnén and Granath[23] indicated that trauma in the dental region, such as fractures, surgical procedures on the jaws, and extraction of the preceding primary tooth are local factors in hypodontia.

Medications

Thalidomide has been reported as a cause of congenitally missing teeth in children whose mothers took this drug during pregnancy.[34,35]

Endocrine disturbances

Cohen[36] also found that endocrine disturbances can be an environmental cause of hypodontia.

Maternal health during pregnancy

In their previously mentioned study of the severity and distribution of familial hypodontia, Parkin et al[32] also sought to determine if maternal health during pregnancy or the patient's birthweight had any influence. They reported that maternal health during pregnancy had no effect on the expression of hypodontia within a family.

Maternal rubella

Developmental anomalies may develop in the fetus of a pregnant woman infected with German measles during the first trimester. Maternal rubella or congenital rubella syndrome is transmitted to the baby through the placenta. This can cause miscarriage, stillbirth, or birth defects such as deafness, brain damage, heart defects, and cataracts.

Although rubella is now a rare condition, the effect of this syndrome on odontogenesis has also been investigated. In a prospective study of children aged 1 to 3 years with histories of maternal rubella, Lundstrom et al[37] found that 550 children born of women with rubella in the first 4 months of pregnancy had an average of 0.6 teeth fewer than 429 children with maternal rubella in the fifth and

following months and 639 matched control subjects.

Evolutionary dental changes

Recent findings of increased prevalence of hypodontia in humans and the findings by anthropologists in primates have influenced the assumption of the possibility of an evolutionary effect on hypodontia. In the evolution of primates, there has been a tendency toward reduction in jaw length and prognathism, mandibular canine size, and number of first molar cusps as well as an increase in the incidence of missing third molars. Anderson et al[38] compared these oral structures with cranial size, body height and weight, and finger length in 118 males and 102 females. They found a significant relationship between body weight and canine width, jaw length, and prognathism. These relationships were stronger in males than in females. They concluded that the evolutionary reduction in these dental dimensions may result from an evolutionary reduction in genetically determined body size.

In both sexes, agenesis of third molars and the length of the maxilla were related. In females, canine width was related to the number of cusps of the first molars, agenesis of third molars, and length of a finger. Females exhibited more frequent simultaneous reductions in dental structures.

Systemic diseases

Clinical reports of congenital syphilis,[39] familial hypophosphatemic rickets,[40] and other infectious diseases[41] have demonstrated their effects on the dentition: reduction in dimensions of the tooth and thinning of the enamel, abnormal tooth form such as Hutchinson incisors and Moon molars, and hypodontia.

Localized inflammation and infection of primary teeth

Another local factor that has been reported is prolonged neglect of localized inflammation and infection of primary teeth during the initial stage of tooth formation, which can disturb tooth formation during the stage of initiation.[42]

Chemotherapy and irradiation

Side effects of irradiation and chemotherapy in growing children have been investigated by several investigators, and their reports show irreversible effects on the dentition. Children who receive high-dose radiotherapy and chemotherapy during

the time of tooth formation are at risk of numerous dental abnormalities. Kaste et al[43] reviewed the clinical and radiographic records of 423 survivors of acute lymphoblastic leukemia. The researchers found root stunting in 24.4% of patients, microdontia in 18.9%, hypodontia in 8.5%, and taurodontia in 5.9%. (Taurodontism is characterized by enlargement of the crown relative to the root size and an elongated pulp chamber that extends deeply into the region of the roots.) They also observed overretention of primary teeth in 4% of patients.

They also found that patients who were younger than 8 years old or those who received cranial irradiation (in addition to chemotherapy) developed more dental abnormalities than did those who were older than 8 years at diagnosis and those who did not receive cranial irradiation.[43]

Other reports from different investigators in long-term survivors who had received different types of treatment for malignant diseases during childhood indicated different dental defects attributed to chemotherapy or irradiation. These defects include disturbances in enamel formation and arrested root development.[44–46]

All developing teeth are irreversibly affected by multiagent chemotherapy and radiation therapy. Irradiation seems to produce more severe effects than chemotherapeutic agents, especially in young patients.[44–46]

Association of Hypodontia with Other Syndromes

Hypodontia can also occur in association with other developmental anomalies. Cobourne[47] indicated more than 60 different syndromic conditions that include hypodontia as a part of their phenotypic spectrum of anomalies. The most common syndromes associated with hypodontia are ectodermal dysplasia, clefting, Down syndrome, and hemifacial microsomia.

Ectodermal dysplasia

Ectodermal dysplasia is a syndromic type of hypodontia in which several dental units are usually missing (oligodontia). The congenital absence of primary teeth is relatively rare. Patients who are missing a number of primary teeth usually have other ectodermal deficiencies, such as ectodermal dysplasia. Ectodermal dysplasia is not a single disorder; it is a group of conditions in which there is abnormal

development of the skin, hair, nails, teeth, or sweat glands.

Many gene defects can cause ectodermal dysplasia, and many different syndromes have been identified. Despite the fact that some syndromes have different genetic causes, the symptoms are sometimes very similar. The most common form of ectodermal dysplasia usually affects men. Other forms of the disease affect men and women equally. These conditions are usually X-linked or autosomal dominant.

Symptoms include dry skin, fine, sparse hair, frontal bossing, and congenital absence of multiple teeth. Underdevelopment of the maxilla and mandible in all three dimensions, but especially in the vertical dimension, is due to reduced growth of alveolar height. Hypodontia ranges from oligodontia to complete anodontia. This can affect both the primary and permanent dentitions.[48–50]

For children suffering complete or partial hypodontia in the primary dentition, partial dentures can be constructed at an early age and can be adjusted or remade at intervals to allow for the eruption of permanent teeth. This increases the ability to chew and also reduces the psychologic problem of the child feeling different from others.

Various types of clefts

In patients with cleft lip and palate, disruption of the dental lamina and abnormal induction or proliferation of the oral mesenchyme may disturb tooth formation, leading to hypodontia, hyperdontia, or both at the same time. In a review of tooth formation in children with cleft lip and palate, Ranta[51] reported that the maxillary lateral incisor is the tooth most susceptible to injury in the area of cleft in both primary and permanent dentitions. This tooth is affected in most instances, even in the cases of microforms of the cleft lip.

The prevalence of hypodontia increases strongly with the severity of cleft. Hypodontia is similarly prevalent in subjects with isolated cleft palate with and without a positive family history of clefts.

Shapira et al[52] studied the radiographs of 278 patients with cleft lip, cleft palate, or both and reported that the prevalence of hypodontia (excluding third molars) was 77% for the total cleft sample. The maxillary permanent lateral incisors were the teeth most frequently missing on the cleft side (259 teeth). The next most commonly absent were the maxillary (47 teeth) and mandibular (23 teeth) second premolars, in both boys and girls.

Down syndrome

Congenital absence of teeth is a common defect in patients with Down syndrome. Mestrović et al[53] examined a group of 112 subjects with Down syndrome, aged 12 to 36 years. After clinical examination and panoramic radiograph analysis, the researchers reported hypodontia in 38.6% of patients. The maxillary lateral incisors and mandibular second premolars were the most commonly missing teeth.

Suri et al[54] studied specific craniofacial features of 25 patients with Down syndrome (12 male and 13 female) and found hypodontia of one or more permanent teeth in 92% of the sample.

Kumasaka et al[55] investigated developmentally absent permanent teeth in 98 subjects with Down syndrome, aged 6 to 28 years. The prevalence of hypodontia was 63%, and 53% of subjects were missing two or more teeth. The most frequently absent teeth were the mandibular lateral incisors (23.3%), the maxillary second premolars (18.2%), the maxillary lateral incisors (16.5%), and the mandibular second premolars (15.3%).

Hemifacial microsomia

Hemifacial microsomia disorder, which affects the development of the lower half of the face, is the second most common congenital anomaly of the face, after cleft lip and palate. It is also referred to as *first and second brachial arch syndrome*, *oral-mandibular-auricular syndrome*, *lateral facial dysplasia*, and *otomandibular dysostosis*. Most commonly, the ear, the mouth, the mandible, and the tissues on one side of the face are underdeveloped. This anomaly can involve the skull as well as the face, and sometimes both sides of the face can be affected.

The etiology is not clear. Most agree that it results from a disturbance of the blood supply to the first and second branchial arches during the first 6 to 8 weeks of pregnancy. Hemifacial microsomia is the most frequently encountered form of isolated facial asymmetry, affecting approximately 1 in every 5,000 births. This anomaly involves underdevelopment of the temporomandibular joint, mandibular ramus, masticatory muscles, and the ear.

According to Silverman and Ackerman,[56] the incidence of hypodontia is five times greater in individuals with hemifacial microsomia than in the general population.

Maruko et al[57] found a 26.9% incidence of hypodontia among 125 patients with

hemifacial microsomia, based on assessment of records from the craniofacial center at Boston's Children's Hospital. They also indicated that normal odontogenesis requires the presence and interaction of neural crest ectoderm and mesenchymal cells and that disturbances in the odontogenic process can produce abnormal or incomplete dental development.

Association of Hypodontia with Dental Anomalies

An association of different dental anomalies concomitant with both syndromic and nonsyndromic hypodontia has been widely reported in the literature. The most common of these anomalies are microdontia, transposition of permanent teeth, ectopic eruption of the permanent canines, ankylosis of primary molars, and taurodontism. The prevalence of these dental anomalies in association with hypodontia can play a role in early detection; their presence is a sign of hypodontia that must be considered during examination.

Microdontia

The association between hypodontia and microdontia is well established; for example, the presence of small peg-shaped maxillary lateral incisors is frequently seen with unilateral missing contralateral incisors.

Lai and Seow[58] assessed the records of 1,032 patients to evaluate the association between other dental anomalies and hypodontia and reported an association with enamel hypoplasia (11.9%) and conical or peg lateral incisors (8.9%).

McKeown et al[59] measured the tooth dimensions of patients with hypodontia and compared the measurements with those of unaffected relatives and a control group. He reported that permanent tooth dimensions were significantly smaller in patients with severe cases of hypodontia (six or more congenitally missing teeth). Microdontia or smaller dimensions of other permanent teeth can be seen in some cases when multiple teeth are missing (Fig 7-1).

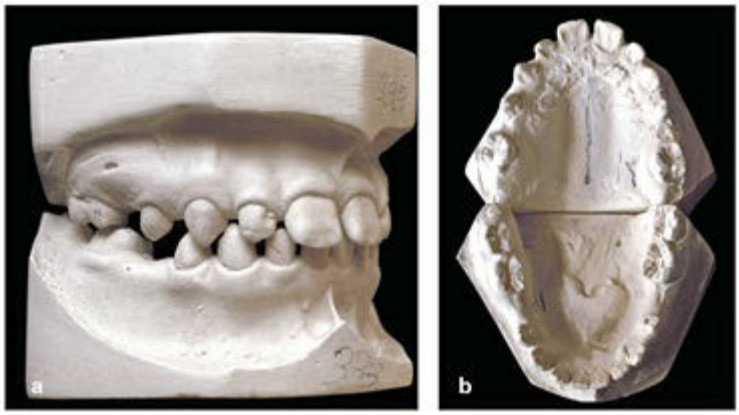


Fig 7-1 *(a and b)* Association of oligodontia (all four premolars missing) and microdontia of all teeth.

Transposition of permanent teeth

Tooth transposition is an abnormal eruption anomaly that can occur as a single anomaly or in association with congenitally missing teeth (see chapter 10). Cases of hypodontia seem to be more severe if they are accompanied by transposition.

In a sample of 43 subjects with maxillary canine–first premolar transposition, Peck et al[60] looked for evidence of other anomalies and found that tooth agenesis (excluding third molars) and/or peg-shaped maxillary lateral incisors accompanied canine–first premolar transposition in 49% (21) of the subjects, 4 to 10 times the normal rate of occurrence.

Ectopic eruption of permanent canines

The prevalence of hypodontia is increased not only in patients with transposition but also in patients with displaced canines. Peck et al[61] evaluated two selected sample groups with nonsyndromic hypodontia; one group included subjects with transposition of one or both maxillary canines–first premolars, and the second group included subjects with palatal displacement of the maxillary canine. They found a significant elevation of hypodontia in both samples.

Ankylosis of primary molars

The combination of missing premolar and retained primary molar, accompanied by primary tooth ankylosis, is often seen during the mixed dentition. Sometimes if the onset of fusion is early, there is a possibility of progressive infraoc-clusion, without

predictable exfoliation. This can have several other consequences, such as tipping of adjacent teeth and arch space loss, alveolar vertical growth deficiency, lateral open bite, and overeruption of opposing teeth (Fig 7-2). Early detection and removal of the ankylosed tooth and prophylactic treatment and restorative care are recommended in this situation.

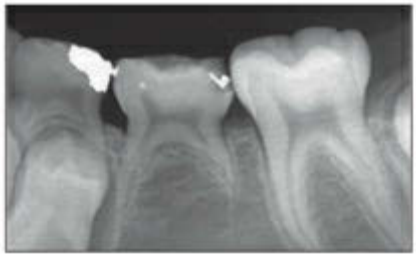


Fig 7-2 Ankylosis of the primary second molar associated with absence of the second premolar.

Via[62] suggested a genetic predisposition for ankylosis and reported that the incidence of submerged primary molars has familial tendencies, based on findings that the prevalence of ankylosis among siblings is 46% compared with a control group of 1.3% in American children.

Zengin et al[63] and Bianchi and Roccuzzo[64] reported that the incidence of primary molar impaction in the absence of the permanent successor is a very rare situation (1:10,000).

Figures 7-2 and 7-3 show a 14-year-old girl in whom all second premolars were missing and the four primary second molars were retained.

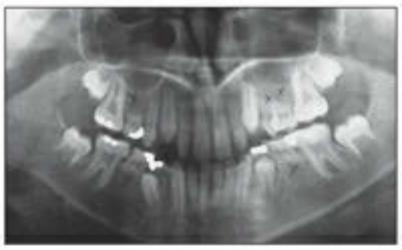


Fig 7-3 Absence of all permanent second premolars associated with overretained primary second molars.

Taurodontism

Taurodontism is a term derived from the Greek words *tauros*, meaning *bull*, and *dontia*, meaning *teeth*. This anomaly occurs mostly in molars. The body of the tooth and pulp chamber are enlarged vertically, and the floor of the pulp and furcation are positioned more apically than normal.

A higher prevalence of taurodontism in hypodontia patients has been reported. Lai and Seow[58] found taurodontism of the mandibular permanent first molar in 34.3% of subjects with hypodontia compared with 7.1% of subjects in the control groups.

Concomitant Hypodontia and Hyperdontia

The simultaneous occurrence of hypodontia and hyperdontia (supernumerary teeth) in the same individual is a rare anomaly, but it can be found occasionally in patients with syndromic or nonsyndromic conditions. This anomaly is also termed *concomitant hypo-hyperdontia*.

The etiopathogenesis of this simultaneous hypodontia-hyperdontia is obscure, but the suspected cause of this disturbance is migration, proliferation, and differentiation of the neural crest cells and interactions between the epithelial and mesenchymal cells during the initiation stage of tooth development.[65,66]

Very few cases of this condition have been reported in the literature, but most of those that have been reported occurred in the maxillary arches. Sharma[67] and Matsumoto et al[68] each reported a case of a missing maxillary lateral incisor and the presence of a midline supernumerary tooth. Ranta[57] reported a very rare nonsyndromic case of concomitant multiple supernumerary teeth in both arches and a missing maxillary permanent left canine in a 12-year-old girl.

Varela et al[69] investigated the frequency of concomitant hypodontia and hyperdontia in a large sample of nonsyndromic orthodontic patients. The records of 2,108 consecutive nonsyndromic orthodontic patients aged 7 to 16 years were examined retrospectively. They found single or multiple hypodontia in 137 patients (6.5%), 62 males and 75 females. One or more supernumerary teeth were found in 42 patients (2 %), 22 males and 20 females. Concomitant hypodontia-hyperdontia was diagnosed in only 7 subjects (0.33%), 4 males and 3 females.

In the aforementioned Eastman Institute study of 800 patients, only 3 (0.375%) exhibited concomitant hypodontia and hyperdontia (Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007).

Effects of Hypodontia on Occlusion, Soft Tissue, and Dentoskeletal Patterns

Depending on the number of missing teeth and the area where the teeth are missing, the following irregularities may result from hypodontia:

- Spacing
- Midline shifting
- Tipping of adjacent teeth
- Tooth displacement
- Overeruption of teeth in the opposing arch
- Canine impaction
- Premature contacts, occlusal trauma, and crossbite
- Mandibular shift
- Abnormal path of growth
- Soft tissue retrusion

The congenital absence of teeth, depending on the severity of the problem, can create serious problems that affect both the physical and emotional conditions of a young person. Even a single missing tooth can cause problems; for example, one missing maxillary lateral incisor can create an unwanted space that is esthetically disturbing and can also result in midline shift, abnormal path of eruption, or even impaction of the canine. One missing premolar can allow tipping of adjacent teeth, disturb interdigitation, cause overeruption of opposing teeth, and disrupt the posterior segment of occlusion.

Multiple hypodontia can create much more serious problems for children, including physical disabilities, emotional conditions, occlusal trauma, premature contacts, mandibular shift, temporomandibular joint dysfunction, adverse growth effects, difficulty in mastication and swallowing, and speech problems (Fig 7-4).

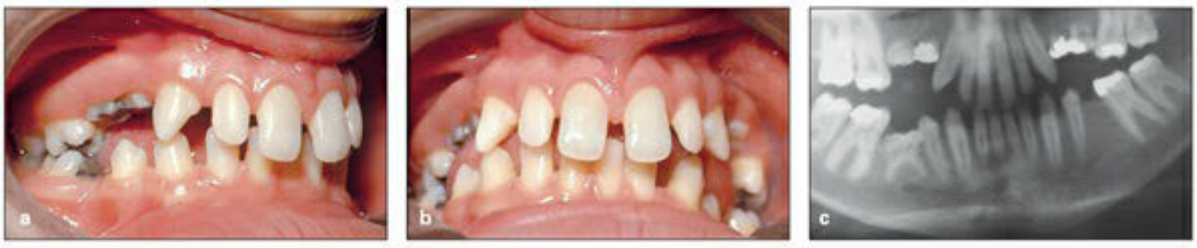


Fig 7-4 *(a to c)* Severely disturbed occlusion in a 16-year-old boy, resulting from oligodontia (10 teeth missing) and neglect. The remaining primary teeth are ankylosed. The situation is esthetically, functionally, and emotionally detrimental.

The dentoskeletal pattern of patients with multiple hypodontia can be unique, and

studies have shown various results. Ogaard and Krogstad[70] investigated the craniofacial structure and soft tissue profile changes in three groups of persons with mild, moderate, or severe hypodontia and compared their patterns with those of persons without hypodontia and with normal occlusion. The mean age of those studied was about 12 years. The researchers observed no consistent pattern when more than five teeth were missing, indicating that severe hypodontia has a different genetic mechanism than does mild hypodontia. A significant retroclination of the incisors and increased interincisal angle and lip retrusion were observed with increasing severity of hypodontia.

Increasing numbers of missing teeth also resulted in a decrease in the mandibular plane angle and a reduction in the anterior lower facial height. They did not find significant differences in the skeletal parameters and concluded that the typical dentofacial structure in persons with severe hypodontia may be the result of dental and functional compensation rather than a different growth pattern.[70]

Ben-Bassat and Brin[71] evaluated the dentoskeletal patterns of patients with multiple congenitally missing teeth. The authors compared lateral cephalograms of 115 subjects with at least three congenitally absent teeth (excluding third molars) with those of normal Israeli populations. The differences between lack of teeth in the anterior and posterior segments were also evaluated. In the subjects with hypodontia, the maxillomandibular relationship was normal but the maxillary and mandibular basal bones were more retruded and the profile was flatter than in the normal population. The dental pattern in those with hypodontia was characterized by upright incisors. In the vertical dimension, the study group exhibited a reduced Frankfort mandibular plane angle.[71]

Early Recognition and Clinical Signs of Hypodontia

Early referral of patients with hypodontia is helpful in the long-term planning of care. The pediatric dentist and general practitioner have an important role in this regard. Periodic longitudinal panoramic radiograph monitoring during the mixed dentition is very helpful in the early detection of many developmental and transitional problems occurring during this important period of dentition, including anomalies of tooth number (for more detail, see chapters 3 and 11).

If observed during the mixed dentition and transitional stages of occlusion, the following clinical signs can be helpful in early detection of hypodontia, which can be confirmed by radiograph investigation:

- Overretained primary teeth
- Incomplete eruption and ankylosis of primary teeth
- Abnormal spacing and tipping of adjacent teeth
- Midline shift
- Microdontia
- Asymmetric exfoliation of the primary dentition
- Evidence of hypodontia in other members of the family

Management

Recognition of hypodontia before complete development of the permanent dentition is not difficult, but after complete eruption the problem is more complicated and management is more difficult. Early detection of and proper intervention in hypodontia can reduce its severity or even prevent damage to occlusion at later ages. The best management of hypodontia is early recognition and intervention at the proper time to provide guidance of eruption.

Unfortunately, referral of patients with hypodontia is usually late, and the majority of oligodontia patients are managed when the patients seek orthodontic care because of their unesthetic malocclusion. As a long-time orthodontic practitioner, the author has treated many cases of hypodontia in the permanent dentition but very seldom had referrals of patients during the mixed dentition, which would have allowed for treatment with an interceptive or guidance strategy. Therefore, more attention must be paid by general practitioners and pediatric dentists during the transitional dentition to accurately detect this anomaly (see Fig 7-4).

Detection of this condition and intervention during the early stages of the dentition maximize the treatment options and therefore the potential to provide optimum clinical care. In addition, the result is achieved mostly through interceptive treatment and guidance, providing better function and esthetics.

Depending on the number and location of missing teeth, comprehensive management often requires a multidisciplinary approach. Interdisciplinary treatment, especially when several teeth are missing, involves a team of committed specialists; each member contributes his or her expertise to achieve an optimum outcome. The dental team has an important role in helping that young person during the important formative years.

Goals

The following are the main goals of comprehensive treatment of patients with congenitally missing teeth:

- Guidance of the existing dentition through necessary orthodontic tooth movement to achieve the best balanced position for occlusal restoration
- Maintenance of normal occlusion, preservation of esthetics, and improvement of the profile
- Maintenance of normal occlusal functions (mastication, deglutition, and speech)
- Preservation of the young patient's emotional and psychologic well-being

Treatment planning

Selection of the appropriate option and proper treatment planning for hypodontia, especially when multiple teeth are missing, requires a careful analysis and assessment of many factors:

- Design of future restorations (team consultation)
- Patient's chief complaints and preferred treatment
- Number and location of missing teeth
- Age of the patient and his or her developmental dental age
- Type of malocclusion
- Specific space requirements
- Conditions in the opposing arch
- Tooth size relationships (Bolton discrepancy)
- Oral and dentoalveolar health conditions
- Structure, form, color, and condition of adjacent teeth
- Patient's profile
- Incisor positions
- Status of primary teeth

Treatment options

Generally, there are four basic treatment options for management of missing teeth:

1. Moving the adjacent tooth to close the space created by the missing tooth, such as canine substitution for a lateral incisor
2. Opening and aligning space for a conventional prosthesis or an implant-supported replacement
3. Retaining the primary tooth (in special cases)
4. Autotransplantation

To determine the appropriate treatment option, the clinician must consider factors that can vary for each individual patient: *(1)* the type of malocclusion, *(2)* anterior tooth relationships, *(3)* the patient's profile, *(4)* specific space requirements, *(5)* condition of the adjacent teeth, and *(6)* the patient's desires.

Depending on the number and site of missing teeth, the type of occlusion, the patient's age, the patient's desires, and the patient's oral health, other variations of treatment plan might be suitable for each individual patient. The ideal treatment option is the most conservative one that will maintain existing dentition, satisfy individual esthetic desires, and fulfill functional requirements.

Management of Missing Lateral Incisors

Failure to provide early intervention when a maxillary lateral incisor is missing, especially unilaterally, can cause several occlusal problems, including midline shift, diastema, palatal tipping of the central incisor, dental crossbite, space deficiency for the canine, and even impaction. Approaches to early orthodontic intervention, space distribution, and tooth alignment vary depending on the type of damage.

When selecting the best treatment option, the clinician should consider the type of malocclusion, tooth size relationships relative to adjacent teeth, specific space requirements, the size and shape of the canine, and the importance of interdisciplinary treatment planning.

After space redistribution and tooth alignment are completed, the available space can be restored in different ways, depending on the age of the patient and the kind of occlusion. Restorations can take the form of a single implant, a cantilevered fixed partial denture, a conventional complete-coverage fixed partial denture, or a Maryland bridge or other resin-bonded fixed partial denture. In the early dentition, a provisional prosthesis (flipper) can also be used to maintain the space for a future

prosthesis or implant.

Orthodontic uprighting of tipped teeth is important to make a more favorable abutment for a prosthesis or implant.

Implant placement usually has to be postponed until the completion of skeletal growth. An exception would be in patients with ectodermal dysplasia; where there are large edentulous spaces and no expectation of growth of the alveolar processes, implants can be placed at an earlier age.

Therefore, early management of missing incisors, depending on the patient's type of occlusion and dental age, usually has two or occasionally three phases of treatment. During the interim phase, the available space has to be preserved by a provisional removable or fixed device. At the same time, the device can play an important role in esthetics and function for the young child.

Absence of the maxillary lateral incisor is a common type of hypodontia. It can occur unilaterally or bilaterally; occasionally a small, peg-shaped maxillary lateral incisor is found concomitant with absence of the contralateral incisor. As previously mentioned, Lai and Seow[58] assessed 1,032 patient records to determine the association of other dental anomalies with hypodontia and found an association with conical incisors or peg lateral incisors in 8.9% of those patients. The presence of a peg-shaped lateral incisor, with or without concomitant hypodontia, is not only disturbing esthetically but also has a considerable impact on the final occlusion because of its tooth size discrepancy. Reshaping of the teeth and a composite resin buildup or veneers can easily solve the problem.

Space closure by canine substitution

The absence of lateral incisors often raises difficult treatment planning issues; achievement of the best esthetic, functional, and stable results depends on accurate initial treatment planning.

One orthodontic option for closing the space left by a missing lateral incisor is to move the canine to the lateral incisor space, a procedure termed *canine substitution*.

Indications

Space closure and canine substitution can be an excellent treatment option for replacement of missing lateral incisors; it should result in a lasting, natural, healthy dentition. However, if it is used in the wrong situation, the final result will not be

ideal. The following are the best conditions for canine substitution when a maxillary lateral incisor is missing:

- Class I crowded mandibular dentition requiring mandibular extraction
- Class II division 1 malocclusion with severe overjet and good mandibular dentition
- Canine with proper form, size, and color
- Normal or moderate overbite
- Concomitant missing maxillary lateral incisor and missing mandibular incisor

Evaluations

To establish a normal overbite and overjet relationship, the clinician must first evaluate the patient's profile, lip line, anterior tooth size relationships, and the canine shape, color, and size.

Patient's profile. The ideal profile for canine substitution is a balanced and relatively straight one; however, patients with mild convexity (especially if the convexity is the result of maxillary dental protrusion), increased overjet, and good mandibular dentition are suitable cases for canine substitution.

Patients with a convex profile, mandibular retrusion, and a retrognathic chin are not appropriate candidates for canine substitution. These patients can end with good results if treated early with a combination of orthopedic treatment and enhancement of mandibular growth.

Lip line. Patients who show excessive gingiva on smiling because of an excess of vertical alveolar size or short upper lip are not good candidates for canine substitution.

If the gingival margin of the canine is at a higher level than that of the central incisors, the gingival margin of the canine can be positioned slightly incisal to the gingival margin of the central incisors. Kokich and Kinzer[72] suggested gingivectomy in this situation if this is a concern to the patient.

Brackets for canine extrusion must be positioned according to gingival margin height rather than incisal edge; the incisal edge of the canine can be reduced later.

Canine shape and color. The size, shape, and color of the canines are important factors to consider before replacement. Canines, with their wide and more convex

labial surface, are naturally larger than lateral incisors, but recon-touring of the canine through some reduction of coronal structure and buildup with composite resin or porcelain veneer can achieve a normal occlusion and acceptable esthetic and functional results. As Zachrisson[73] has shown, contouring and reshaping the canine crown may lead to dentin exposure, which requires restorative intervention. Depending on the shape of the incisal edge, it may be necessary to restore the mesioincisal and distoincisal edges with composite resin to recreate normal lateral incisor contours. Some palatal reduction of the cingulum area also might be necessary in order to place the canine in the normal vertical position of a lateral incisor (Fig 7-5).

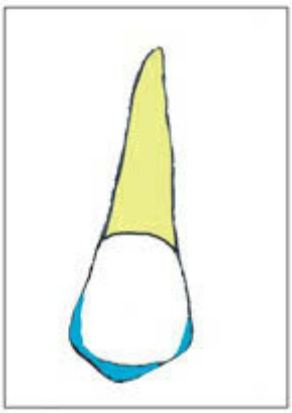


Fig 7-5 Reshaping the canine (areas of reduction shown in blue).

The canine might be more saturated and slightly darker than the central incisors. The easiest option is canine bleaching. If this is not sufficient, application of a veneer may be indicated.

Zachrisson and Mjör[74] showed that extensive grinding using diamond instruments with abundant water spray cooling can be performed on young teeth without long-term changes in tooth sensitivity. They noted that some patients experienced short-term increases in tooth sensitivity for 2 to 3 days.

Anterior tooth size relationships. In treatment planning for canine substitution, the anterior Bolton ratio also must be considered to evaluate the final relationship between the maxillary and mandibular anterior teeth.

Space creation and alignment for a prosthesis

Another option for management of hypodontia is alignment of adjacent teeth in the region and preparation of the occlusion for prosthetic replacement or implants.

Maxillary lateral incisors and mandibular second premolars are the most commonly missing teeth. Application of this option is the most conservative and ideal alternative that satisfies individual esthetic and functional requirements. This technique can be applied to a missing lateral incisor or a missing premolar, with some modifications between the two. In both situations, all other available options must be closely examined.

In hypodontia cases, especially if proper early intervention has been performed, the second phase requires only some orthodontic space redistribution to facilitate prosthetic or implant replacement.

One consequence of a single missing lateral incisor or second premolar is tipping of adjacent teeth, which necessitates reopening of space and uprighting and alignment of the misplaced teeth.

Indications

Management of a missing maxillary lateral incisor by replacement with an implant or a prosthesis has certain indications and cannot be applied in every situation. For example, in patients with missing maxillary lateral incisors and severe mandibular incisor crowding that requires extraction, alignment of the remaining maxillary incisors and subsequent prosthetic replacement of the lateral incisor is definitely an incorrect procedure.

The following are the best situations for management of a missing maxillary lateral incisor with an implant or prosthetic replacement:

- Class I malocclusion, minimum overjet, and good mandibular dentition
- Maxillary deficiency with normal mandibular dentition
- Severe deep bite

Treatment approaches

The restorative treatment approach can be divided into two categories: single-tooth implant and tooth-supported restoration. Different types of tooth-supported restorations can be used in the replacement of missing lateral incisors: cantilevered fixed partial denture, resin-bonded fixed partial denture, and conventional complete-coverage fixed partial denture. The choice of the implant-supported or the tooth-supported option depends on the skill of the practitioner and the quality of the prosthesis as well as the general health and oral hygiene of the patient. An

interdisciplinary approach must be considered.

As previously mentioned, a neglected missing maxillary lateral incisor can cause several occlusal problems, including midline shift, diastema, palatal tipping of the central incisor, dental crossbite, space deficiency for the canine, and impaction. Therefore, after comprehensive evaluation of the patient, depending on the type of damage, the first step is interdisciplinary approaches and consultation for missing tooth restoration after orthodontic treatment; this is followed by orthodontic intervention, which consists of space redistribution and tooth alignment.

Orthodontic intervention

Early orthodontic intervention and tooth movement of existing incisors depends on the type of problem. Treatment usually consists of closure of diastemata, correction of midline shifts, elimination of any crowding or rotation, and correction of crossbite or any other irregularities present.

Orthodontists play an important role in determining and establishing the space required for missing tooth replacement. To determine the appropriate amount of space needed for the missing lateral incisor, Kokich[75] suggested three methods:

1. Application of the "golden proportion," which is a 1:0.618 ratio for the width of the central incisor to the width of the lateral incisor. For example, with an 8-mm-wide central incisor, the appropriate space for a lateral incisor is 5 mm.
2. Use of the width of the contralateral incisor, provided that it has a normal width, to judge the space for the missing incisor. He did not recommend this method for adults or for patients with peg lateral incisors.
3. Application of the Bolton analysis.[76] The proper space for an edentulous area can be mathematically calculated by using the anterior ratio.

Fabrication of a diagnostic setup cast is very useful for determining ideal spacing.

Prosthetic replacement

Depending on the patient's occlusion, amount of overbite, and the size, form, and condition of adjacent teeth, many options for replacement of missing lateral incisors are available.

Fiber-reinforced composite resin fixed partial denture. Adolescent patients with

congenitally missing lateral incisors require an interim space maintainer after orthodontic treatment to restore function and esthetics before craniofacial growth is completed and an endosseous implant can be placed. Direct and indirect fiber-reinforced composite resin fixed partial dentures are a new way to rapidly produce a metal-free tooth replacement that is minimally invasive, esthetic, and cost-effective.

For young patients, because of the size of the immature pulp, a resin-bonded prosthesis is preferable to conventional fixed partial dentures. This technique can also be used as an interim prosthesis before placement of an implant. In addition to allowing chairside tooth replacement, it is also economically feasible for patients who cannot afford more traditional treatment regimens.

This technique is also useful for medically compromised patients who are unable to withstand the physical stress associated with fabrication of traditional fixed prostheses.

According to Botelho et al,[77] two-unit cantilevered resin-bonded prostheses have a mean service life of 4 years, 4 months (standard deviation, 20 months).

Removable partial denture. In young children, after anterior tooth alignment and preparation of the region for prosthetic replacement of the missing tooth, overdentures and removable partial dentures can be used as a provisional retainer and as an interim prosthesis.

This type of partial denture can be constructed from a simple Hawley retainer with an acrylic resin tooth placed in the area of the missing tooth. This can serve as a space maintainer before definitive restorative work. It can also be used as a bite plane to increase the vertical dimension in cases of overclosure.

This appliance not only is an economically feasible option but also restores the patient's appearance and function, especially in cases of severe oligodontia. Therefore, the patient's compliance is ensured.

Disadvantages include the possibility of appliance loss and the fact that prolonged use may increase the risk of caries and periodontal disease. Maintenance of excellent oral hygiene is required.

Single-tooth implant. Osseointegrated implants have been used successfully in dentistry for more than 43 years. Their direct alveolar anchorage provides a predictable and successful means of supporting crowns, fixed partial dentures, overdentures, and other maxillofacial prosthetics to replace missing teeth. Placement of a single-tooth implant has become a popular method of replacing missing teeth

(Fig 7-6). Several longitudinal studies of conventional implant treatment have shown success rates of 91% to 99% in the mandible and 84% to 92% in the maxilla.[78–82]

Achievement of optimal results for single implants requires an interdisciplinary approach, because there are several options to provide the best treatment outcome. Orthodontic treatment must be based on the final restorative treatment plan, in order to position the adjacent teeth in a way that facilitates the definitive restoration and provides the best possible esthetics and function.

One of the main advantages of a single-tooth implant is the ability to leave the adjacent teeth untouched, while most restorative treatments require varying amounts of tooth structure removal. This is a particularly important advantage when single implants are used in young patients with an unrestored dentition; however, the implant cannot be placed unless facial growth is completed.

Achieving root parallelism and maintaining enough space for implants are other important parts of the orthodontist's responsibility before any dental replacement (see Fig 7-6). Periodic examination of periapical radiographs of the edentulous area prior to removal of orthodontic appliances is highly recommended to confirm that treatment has created ideal root position and adequate space for the implant.

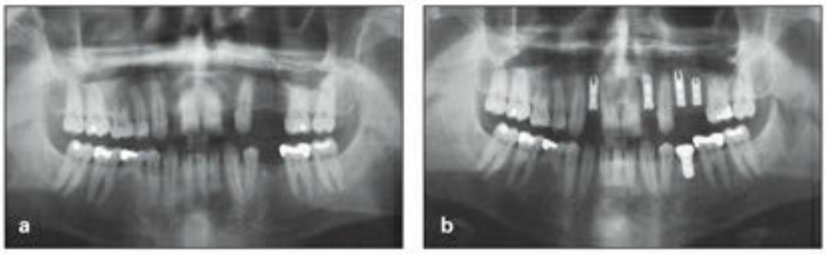


Fig 7-6 *(a)* Pretreatment panoramic radiograph of a 23-year-old man, revealing missing teeth, diastemata, and tooth displacement. *(b)* Panoramic radiograph after alignment, space distribution, and placement of several single-tooth implants.

The buccolingual thickness of the alveolar bone must also be adequate to allow proper implant placement. Because of the absence of the lateral incisor, the ridge of alveolar bone in the area is usually narrow and underdeveloped. If the osseous ridge has not fully developed, it may be necessary to place a bone graft either prior to or in conjunction with implant placement to achieve optimal alveolar ridge thickness.

Another means of developing sufficient bone support in the edentulous area is orthodontic tooth movement. Kinzer and Kokich[83] suggested that early extraction of the primary lateral incisor encourages the permanent canine to erupt adjacent to the central incisor and that its large buccolingual width will influence the thickness of the edentulous ridge. After complete eruption of the permanent canine, it is moved

distally, leaving behind an adequate buccolingual width for implant placement. Research indicates that the buccolingual width of the alveolus remains stable if the implant site is developed by orthodontic tooth movement.[75,84]

Placement of implants is contraindicated during growth changes, because residual facial growth causes infraocclusion of the implant. The implant becomes ankylosed to the alveolar bone, while the adjacent teeth erupt and alveolar bone growth continues.[85]

Autotransplantation

Autotransplantation is a surgical repositioning of a tooth from one site to another within the same individual. The new location may be a fresh extraction socket or an artificially prepared socket in an edentulous alveolar ridge.

Autotransplantation has been performed for different reasons, including management of impacted teeth, carious and nonrestorable teeth, and hypodontia. Autotransplantation has been carried out for many years, but with varying success rates. Reports of successful autotransplantation first appeared in the 1950s, when carious first molars were replaced with transplanted immature third molars.[86,87] Evidence for the successful transplantation of premolars and molars to the incisor region has also been presented.[88,89]

The preservation and regeneration of the periodontal ligament is the key to success of this treatment strategy. Jonsson and Sigurdsson[90] carried out a long-term study of 370 autotransplanted premolar teeth to determine a standardized surgical procedure that would optimize pulpal and periodontal healing. They concluded that careful surgical technique without trauma can maximally preserve an intact periodontal ligament. They added that if Hertwig's root sheath is traumatized, future root growth will be limited or inhibited, depending on the severity of the trauma.

Tooth transplantation is considered a viable treatment method because it allows dentofacial development and maintains alveolar bone volume, especially in young patients with tooth agenesis. This procedure is obviously more cost-effective than conventional prosthetic restorations (implants or fixed partial dentures). Another major advantage of transplantation over implantation is the applicability of transplantation for the management of patients before puberty, while implants are contraindicated during growth changes. In addition, transplanted teeth are biologic material and are able to erupt in harmony with adjacent teeth and growing jaws.

A disadvantage of transplantation is the need for an available donor tooth of

suitable size and morphology. In addition, implants have higher success rates than transplanted teeth. The surgical success of autotransplantation depends on good oral hygiene of the patient and a medical history that does not contraindicate transplantation (eg, cardiac defect).

Management of Missing Mandibular Second Premolars

The second premolar is one of the most common congenitally absent teeth. Its absence can cause serious problems for occlusion and represents a complicated treatment situation. Therefore, absence of the second premolar requires early detection and intervention.

There are two general strategies for management of congenitally missing second premolars: *(1)* space opening for future restorations and *(2)* space closure so that natural teeth touch each other. However, the formulation of a comprehensive treatment plan depends on the basic orthodontic diagnosis and evaluation of special criteria:

- Existing malocclusion (dental and skeletal relationships)
- Age of the patient
- Dental age of the patient
- Arch length deficiency
- Facial profile
- Condition of the primary molar
- Willingness of the patient to cooperate with extensive treatment
- Financial considerations

This anomaly is often associated with retained and infraoccluded primary molars, which require early intervention. Delayed treatment or failure to provide treatment can cause the following sequelae:

- Reduction of alveolar height
- Supraeruption of opposing teeth
- Tipping of adjacent teeth with space loss
- Deep curve of Spee
- Impaction of the first premolar

This clinical situation is a challenge to pediatric dentists, prosthodontists, and orthodontists and requires early detection and intervention (see chapter 10).

Replacement of congenitally missing premolars generally involves one of four basic treatment options:

1. Retaining the primary molar
2. Closing the space left by the missing tooth
3. Placing a tooth-supported restoration or a single-tooth implant
4. Autotransplantation

Retention of the primary tooth

Primary teeth with agenesis of their succedaneous tooth usually have a very long stage of root resorption and exfoliation, as the literature reports.[91] Depending on occlusion and their relationship to adjacent and opposing teeth, they may stay intact for a long period of time.

There have been reports in the literature of preserving primary teeth without permanent successors as an orthodontic option for management of missing permanent teeth.[91]

These kinds of overretained primary teeth can be kept if the permanent successor is missing, provided that the condition of the primary tooth roots and the occlusion indicate a good prognosis.

Before deciding to retain the primary molars in patients who are missing their permanent successors, the orthodontist must consider the age of the patient, the occlusal status, the condition of the infraoccluded tooth, including the degree of infraocclusion and root resorption, and adjacent alveolar bone levels. If the primary tooth is in normal condition and in normal relationship to the adjacent and opposing teeth (excluding the aforementioned abnormal condition) but the succedaneous tooth is missing, it can be preserved as long as it remains naturally. The retained primary tooth has an advantage as a space maintainer and serves an esthetic and functional purpose. In addition, alveolar bone can also stay intact for future implant or prosthetic replacement.

In cases of infraocclusion of primary molars, building the occlusal surface with composite is recommended. However, Kurol and Olson[92] assessed 143 permanent molars adjacent to 119 infraoccluded primary molars and 24 normal contralateral primary molars in 68 individuals. The subjects were reexamined both clinically and

radiographically about 8 years after the exfoliation or extraction of the infraoccluded primary second molars. The researchers found that all but two permanent first molars had a normal alveolar bone level mesially.

When the primary molar is nonmobile, functioning, and meets a patient's esthetic standards, its retention can be another management option in patients with missing premolars. Other advantages include the psychologic benefits of a person keeping his or her own teeth and the ability for that tooth to maintain the surrounding bone and soft tissue intact.

Sletten et al[93] evaluated the long-term retention of primary molars without the permanent successor in 20 patients who had at least one retained primary mandibular second molar without a permanent successor. The mean age at the initial examination was 36.1 years, and the mean age at the final examination was 48.5 years. Of the 28 retained primary molars initially identified, 24 (86%) continued to function; the other 4 mandibular primary molars were lost because of caries or periodontal breakdown.

Furthermore, they found that the average shortening of all primary root lengths was negligible (0.16 mm). Based on this study in adults and earlier studies in adolescents, they concluded that retention of mandibular primary second molars is a viable treatment alternative.[93]

Although restoration of infraoccluded teeth to the occlusal plane may provide a good immediate result, progressive infraocclusion, which can happen during facial growth, or continued root resorption, and later exfoliation, is often not a long-term solution.

Management of a missing premolar by retaining the primary molar is not the best option in all cases (eg, premolar hypodontia concomitant with severe crowding in another area of dentition that may need extraction). Depending on the age of the patient and the type of occlusion, the treatment of choice in these conditions can be extraction of the primary molar and space closure, as explained in the following section.

Space closure

Space closure to manage congenital absence of permanent teeth is definitely a more attractive solution in adolescent patients than retaining primary teeth. Space that has resulted from mandibular second premolar hypodontia can be closed in two ways: *(1)* as an intervention during the early mixed dentition or *(2)* as an option during the

permanent dentition. There are two different orthodontic treatment approaches to closing the premolar space:

1. Gradual slicing of the distal surface of the primary second molar to allow the permanent first molars to drift slowly, thus facilitating future orthodontic treatment
2. Extraction of the primary second molar to allow the permanent first molar to drift mesially followed by immediate orthodontic uprighting and closure of residual space

Early gradual slicing

Early intervention for a missing mandibular premolar is the ideal and easy way to accomplish space closure, if started at the proper time. This technique can be started before complete eruption of the permanent first molar and can be continued during the early and middle mixed dentition with gradual disking of the distal surfaces of the primary second molars.

In a study of 34 patients with 52 missing premolars diagnosed between 8 and 11 years of age, Valencia et al[94] found 42 premolars missing in the mandible (81%) and 10 missing in the maxilla (19%). The patients were divided into different groups to allow comparison of slicing versus extraction of primary molars. These groups were subdivided into two age groups: 8 to 9 years and 10 years or older.

Sequential slicing followed by hemisection had a greater success rate than did extraction. Permanent molars showed an 80% bodily space closure within 1 year, without mesial rotation, midline loss, or any inflammation, leading to better final orthodontic results. A 90% success rate was achieved when the technique was applied at an early age (8 to 9 years), and the success rate tended to decrease as the age at start of treatment increased. Extraction of the primary second molars, without the controlled slicing technique, showed an average to poor result in 75% of the patients, with no different clinical response in any age group.

Extraction and space closure

Another option in the orthodontic management of congenitally missing second premolars is the early extraction of primary molars during transitional development and closure of the extraction space. Lindqvist[95] investigated the effectiveness of this procedure in 101 children aged 5 to 12 years. A planned extraction of the primary second molar was carried out after cast and profile registration. Extraction was followed annually by cast registration and oblique lateral radiography. The presence

or absence of mandibular dysfunction was recorded after the second molars had come into occlusion. She reported that residual space was an average of 2 mm in the mandible and less than 1 mm in the maxilla 4 years after extraction.

She also stated that extraction after root development of the adjacent teeth led to closure of the extraction space by tipping of these teeth. Correct timing of the extraction of the primary second molars was advocated to facilitate bodily movement of the permanent first molars. Lindqvist[96] concluded that early extraction of the corresponding primary molar should be seriously considered as a treatment alternative in cases of hypodontia of the second premolar.

Mamopoulou et al[96] investigated space closure and occlusal changes in 11 subjects (mean age of 11.8 years). Treatment began when the first premolars came into occlusion. Subjects were followed for 4 years. Dental casts were prepared at the start of treatment and after 1, 2, and 4 years. Lateral cephalograms were taken at the start of treatment and after 2 and 4 years.

The researchers reported that most of the extraction space closed during the first year (55% in the maxilla, 46% in the mandible). At the end of the follow-up period, 89% of the extraction space had closed in the maxilla and 80% in the mandible, leaving mean residual extraction spaces of 0.9 and 2.0 mm, respectively. They also indicated that unilateral extraction had no influence on the maxillary midline, while it caused a statistically significant mandibular dental shift to the extraction side. Extraction therapy had no impact on the overjet, overbite, or incisor inclination.[96]

Tooth-supported or implant-supported restoration

Alignment of adjacent teeth in the region of missing premolars and preparation of the occlusion for prosthetic replacement or implants is another option that can be applied in patients with missing premolars. This option is usually applied in the permanent dentition when adjacent teeth have tipped toward the missing space and tooth irregularity and occlusal interference are problematic for the proper application of a restoration. This situation is usually the consequence of a lack of interceptive treatment at an earlier age. However, even after proper intervention and interceptive treatment during the mixed dentition, such as retention of primary molars or gradual space closure, some minor orthodontic tooth movement, such as uprighting or final space closure, may be necessary.

Preparation of the occlusion for prosthetics or implants in the premolar region can be simple or complicated, depending on the changes to occlusion after primary tooth

loss. Goals may include uprighting of teeth, root paralleling, correction of rotations, leveling, and, in cases of overeruption, intrusion of the opposing teeth. The orthodontic needs of premolar hypodontia that have been treated at the proper time by simple interceptive treatments can be managed much easier at the later age.

Depending on the patient's age, type of occlusion, and desires, different types of restorations can be applied. These include single-tooth implants, cantilevered fixed partial dentures, conventional complete-coverage fixed partial dentures, or different types of resin-bonded fixed partial dentures.

Autotransplantation

One kind of autotransplantation that has been reported widely in the literature is transplantation of an extracted premolar to the site of a missing premolar in the same patient. Slagsvold and Bjercke[97] postoperatively observed 34 premolars transplanted before completion of root formation. Follow-up periods ranged from 3 to 13 years. All 34 teeth in question were found to still be in place. Root formation continued, supporting tissues developed, and teeth erupted. In most instances, the teeth functioned normally without giving rise to any problems. The authors concluded that most of the teeth functioned normally and predicted that they would for many years to come, perhaps for a lifetime. They also added that substitution by autotransplantation cannot be applied unless a suitable transplant is available, such as a premolar that has been extracted for orthodontic reasons.[97]

Kvint et al[98] performed a follow-up study of 215 patients, aged 9.1 to 56.4 years, who had undergone transplantation of a total of 269 teeth. The researchers evaluated the success rate and analyzed factors affecting the outcome. The transplants were recorded as unsuccessful if the tooth had been extracted or as surviving but with root resorption or ankylosis. The interval between transplantation and final follow-up was a median of 4.8 years. Kvint et al[98] recorded 175 (81%) of the transplantations as surviving and 40 (19%) as unsuccessful.

Jonsson and Sigurdsson[90] investigated 32 orthodontic patients with missing premolars who had 40 premolars transplanted into contralateral or opposing jaw quadrants. The long-term outcome, pulp survival rate, periodontal condition, and root development of autotransplanted premolars were evaluated. The teeth were examined systematically with clinical and radiographic measures, and the observation time varied from 2 years, 5 months to 22 years, 3 months (mean of 10 years, 4 months).

At the last examination, 37 of 40 transplanted teeth and their supporting structures were healthy, a 92.5% success rate. Transplanted teeth with closed apices received end-odontic therapy; however, among those with open or half-open apices, a 66% pulp survival rate was observed. No teeth in the sample had signs of replacement resorption or developed periodontal attachment loss.[90]

It seems that autotransplantation is an ideal treatment option for child or adolescent patients who suffer from hypodontia and who need premolar extraction as a part of orthodontic treatment. This treatment strategy is especially suitable for premolar-to-premolar transplantation. As previously mentioned, the preservation and regeneration of the periodontal ligament is the key to success in this type of treatment.

Case Reports

As already discussed, management of hypodontia depends on the number of teeth missing, the location in the arch, the patient's type of occlusion, the condition of adjacent teeth, and the patient's desires. The following case reports present different treatment options with an emphasis on early treatment.

Case 7-1

A 15-year, 3-month-old girl with a Class I malocclusion, normal skeletal pattern, and straight profile was congenitally missing a maxillary right lateral incisor and mandibular left second premolar (Figs 7-7a to 7-7d). Their absence had caused diastemata and midline shift. The mandibular left molar and first premolar had tipped toward the missing space.

Treatment:
Because she had a good profile, no crowding, and good interdigitation, the treatment plan was limited to tooth uprighting, diastemata closure, alignment of the anterior segment, and placement of a resin-bonded fixed partial denture to replace the right lateral incisor. The mandibular premolar space was closed orthodontically (Figs 7-7e to 7-7g).

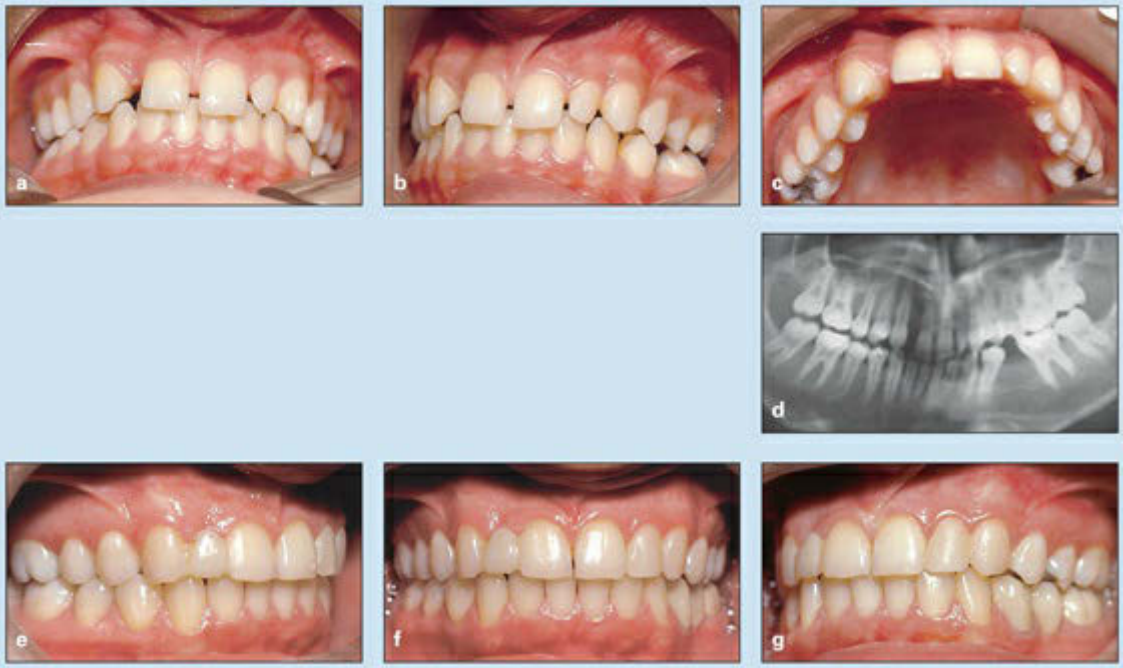


Fig 7-7 Treatment of a 15-year, 3-month-old girl who is missing her maxillary right lateral incisor and mandibular left second premolar. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e to g)* Posttreatment occlusion, after orthodontic treatment and placement of a resin-bonded prosthesis.

Case 7-2

A 12-year, 5-month-old girl with a Class I malocclusion exhibited severe overjet (14-mm), 2-mm open bite, and dental protrusion (Figs 7-8a to 7-8f). Her tongue was very active during swallowing, and she had a steep mandibular plane, retrognathic mandible, and very convex profile. Both maxillary lateral incisors were missing, but the primary canines were present. She had mandibular incisor protrusion and minor crowding in the mandibular posterior segment.

Treatment:
The treatment plan included extraction of two mandibular premolars and the maxillary primary canines. Mechanotherapy was performed in two phases:

In the first phase, a mandibular lingual arch and a maxillary tongue crib–transpalatal arch (TC-TPA) (see chapter 6) were inserted to control tongue activity. Maxillary and mandibular molar intrusion was carried out in an attempt to achieve mandibular counterclockwise rotation. After tongue control was achieved and the retained maxillary canines were extracted, maxillary dental proclination and overjet were significantly decreased.

The second phase of mechanotherapy was removal of the TC-TPA, insertion of Nance anchorage, and full bonding for retraction of the anterior and then retention. Figures 7-8g to 7-8m illustrate the posttreatment changes.

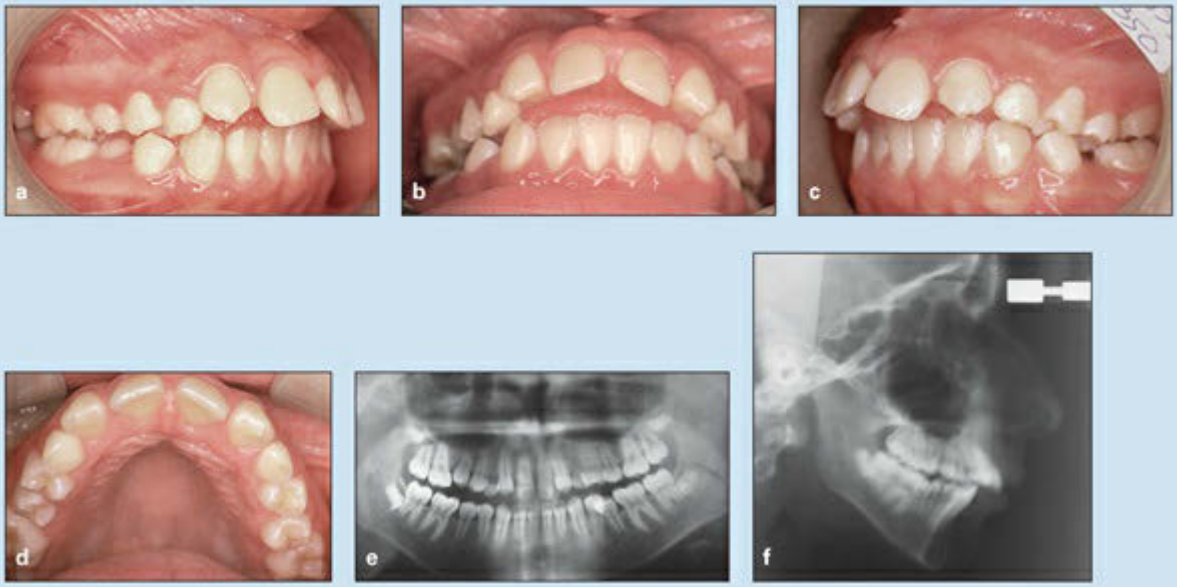


Fig 7-8 A 12-year, 5-month-old girl who is missing both maxillary lateral incisors. *(a to d)* Pretreatment occlusion. Both maxillary primary canines are present. *(e)* Pretreatment panoramic radiograph. *(f)* Pretreatment cephalometric radiograph.

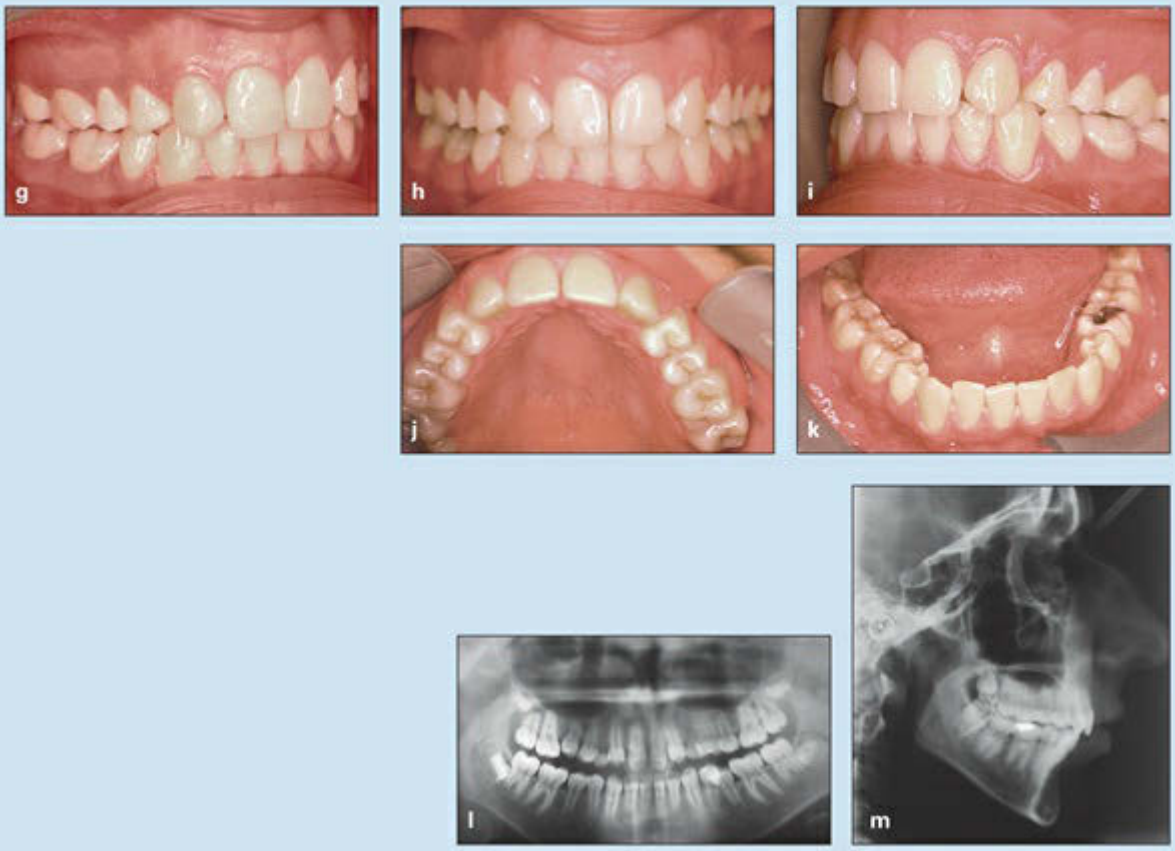


Fig 7-8 *(cont) (g to k)* Posttreatment occlusion, after extraction of both mandibular first premolars and the maxillary left and right primary canines. *(l)* Posttreatment panoramic radiograph. *(m)* Posttreatment cephalometric radiograph.

Case 7-3

An 11-year, 8-month-old girl with a Class I malocclusion, 6-mm overjet, moderate overbite, and upper lip protrusion presented with a missing maxillary left lateral incisor (Figs 7-9a to 7-9d). The mandibular right second premolar was impacted. The maxillary right primary canine was still present. The maxillary midline had shifted laterally to the side of the missing tooth. The mandibular dentition was good.

Treatment:
Because the position and inclination of the mandibular incisors were normal, the maxillary right lateral incisor and primary canine were removed to correct the maxillary midline, allow retraction of the maxillary anterior segment, and allow the canines to shift to the position of the lateral incisors. The impacted mandibular second premolar was extracted, adjacent teeth were uprighted, and the space was closed. Figures 7-9e to 7-9h illustrate the posttreatment occlusion.

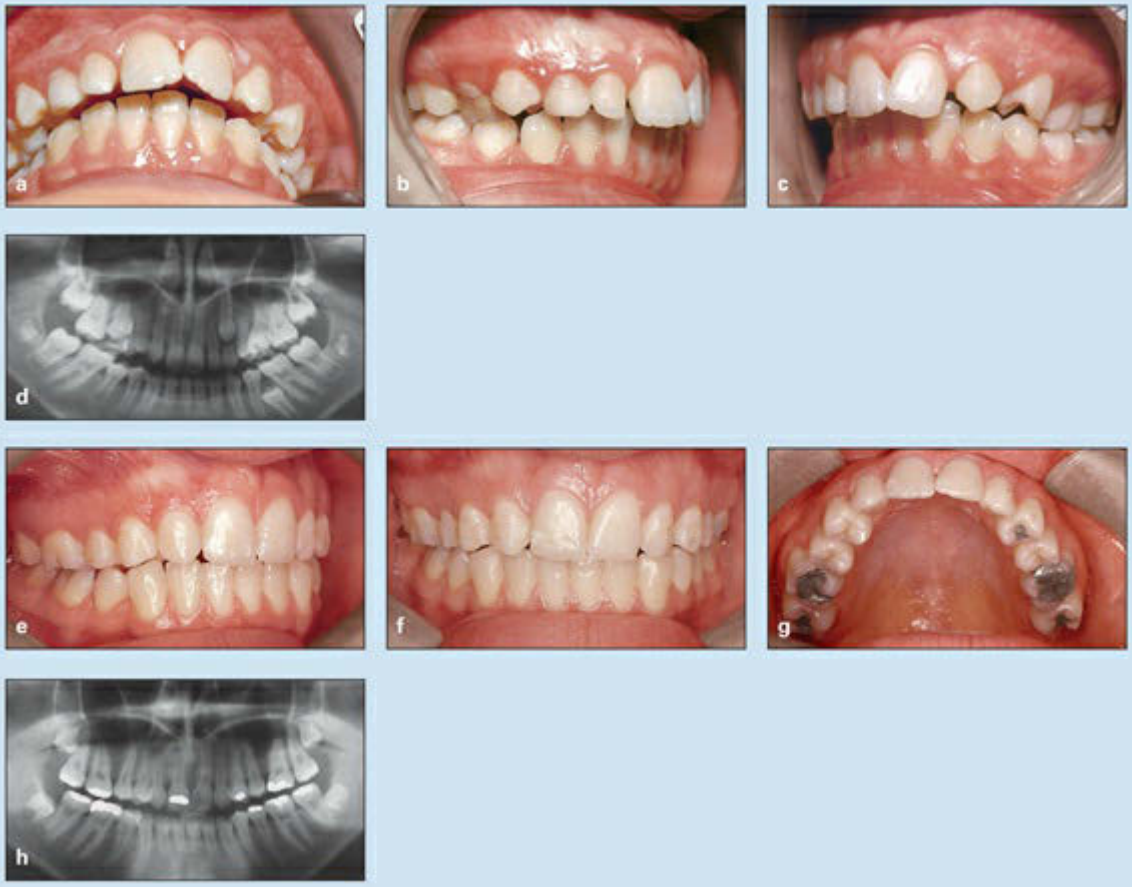


Fig 7-9 Treatment of an 11-year, 8-month-old girl who is missing her maxillary left lateral incisor and has an impacted mandibular right second premolar. The maxillary right primary canine is still present. *(a to c)* Pretreatment occlusion. The maxillary midline has shifted to the side of the missing lateral incisor. *(d)* Pretreatment panoramic radiograph. *(e to g)* Posttreatment occlusion, after extraction of the maxillary permanent right lateral incisor and mandibular second premolar to correct the midline and upright the teeth. *(h)* Posttreatment panoramic radiograph.

Case 7-4

A 9-year-old girl in the middle mixed dentition presented with a diastema and locked traumatized incisor crossbite (Figs 7-10a to 7-10c). Radiographic evaluation indicated that both maxillary lateral incisors were missing (Fig 7-10d).

Treatment:
The treatment plan was designed in two phases. The first phase involved correction of crossbite, incisor alignment, and closure of the diastema to prevent occlusal trauma. The maxillary primary lateral incisor and canines were extracted to guide permanent canine eruption and prevent impaction. The mandibular primary canine, and later the mandibular primary first molar, were extracted to accelerate mandibular canine eruption.

Phase 2 treatment was started after all canines had erupted, including extraction of both mandibular first premolars (Figs 7-10e to 7-10h).

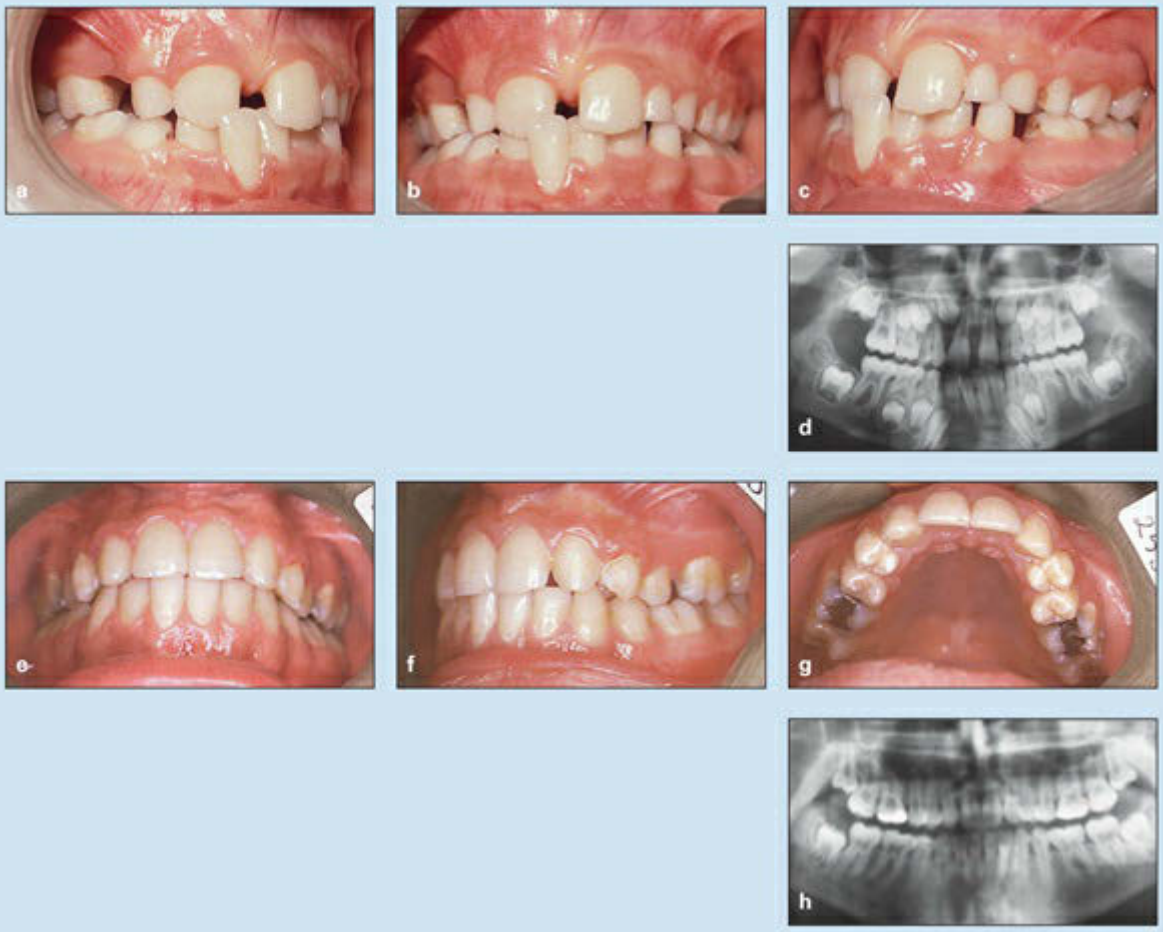


Fig 7-10 Treatment of a 9-year-old girl with open diastemata and central incisor crossbite. Both maxillary lateral incisors are missing. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e to g)* Posttreatment occlusion. *(h)* Posttreatment panoramic radiograph.

Case 7-5

A 10-year-old boy presented for orthodontic treatment. His chief complaints were his inability to close his lips and the resulting esthetic issue. There were no remarkable features in his medical history, although a mouth breathing habit, which perhaps was related to the severe lip incompetence, was

reported by the parents. He presented with a Class I malocclusion and severe overjet of around 16 mm (Figs 7-11a to 7-11i). Both mandibular central incisors were congenitally missing. Severe lip dysfunction was aggravating the amount of overjet.

The original factors creating overjet seemed to be mandibular clockwise rotation and retrognathism, congenitally missing mandibular central incisors, and retroclination of the mandibular anterior segment.

Treatment:

The treatment objective was to achieve normal overjet, normal lip closure, and close the gap caused by the missing incisors without aggravating retroclination of the mandibular anterior segment. Both maxillary first premolars and remaining mandibular primary central incisors were extracted.

A lower holding arch was placed to prevent any incisor retroclination and move the lateral incisors around the arch mesially to close the gap. Then the permanent mandibular canines were mesialized around the arch without reducing arch circumferences.

A maxillary Nance holding appliance was used during maxillary canine retraction. After maxillary canine retraction and consolidation of the mandibular anterior segment (mandibular lateral incisor and canine), retraction of the maxillary four central incisors with good, controlled root torque was started.

At the same time, movement of one mandibular premolar on each side was started with chain and heavy class 2 elastics. After consolidation of all mandibular premolars with the anterior segment around the lingual arch, the mandibular lingual arch was removed, and the mandibular first permanent molars were mesialized with continuation of heavy class 2 mechanics combined with maxillary anterior retraction and control of lingual root torque.

Figures 7-11j to 7-11p show posttreatment views of the patient's occlusion, lip relationships, and dentoskeletal relationships.

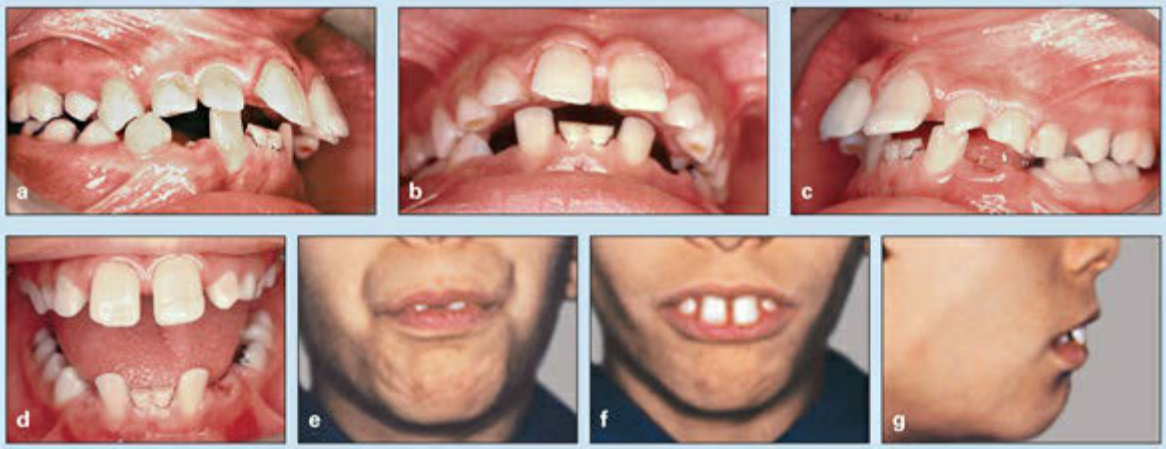


Fig 7-11 Treatment of a 10-year-old boy who exhibited lip dysfunction and was missing both mandibular central incisors. *(a to d)* Pretreatment occlusion. *(e to g)* Severe lip strain on closing, as a result of the severe overjet.

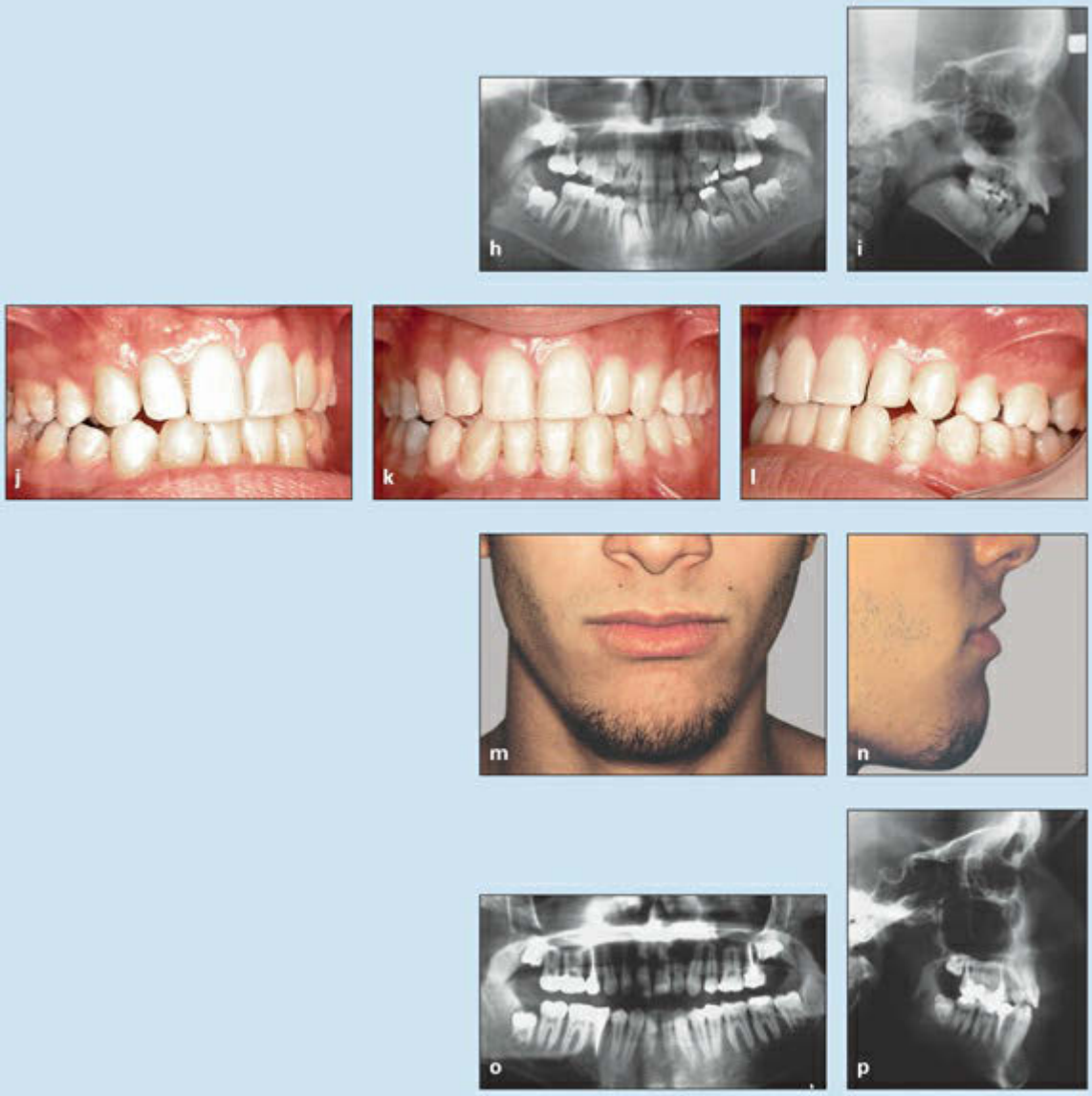


Fig 7-11 *(cont) (h)* Pretreatment panoramic radiograph. *(i)* Pretreatment cephalometric radiograph. *(j to l)* Posttreatment occlusion, after maxillary first premolar extraction and mandibular space closure. *(m and n)* Posttreatment lip closure. *(o)* Posttreatment panoramic radiograph. *(p)* Posttreatment cephalometric radiograph.

Case 7-6

A 14-year-old girl presented with oligodontia; all second premolars and third molars were missing. The patient also had microdontia and significant interdental spacing (Figs 7-12a to 7-12f). The parents and the patient desired space closure and no prosthetic replacement. The initial panoramic radiograph (see Fig 7-12a) was taken by the dentist 3 years before the orthodontic treatment. Unfortunately, nothing was done at that time. Early detection and intervention, such as removal of the primary molars, might have reduced the severity of the problems.

Treatment:

Despite all the problems present, one helpful condition was the presence of maxillary and mandibular incisor proclination and lip protrusion that would be corrected automatically by space closure. The treatment mechanics were similar to those applied in case 7-4, but in both arches. That is, maxillary and mandibular holding arches were inserted to preserve arch circumferences and start space closure from the anterior segment. This was followed by anterior consolidation and tooth-by-tooth mesial movement of the buccal segment and consolidation for posterior teeth. The only restoration was composite resin buildup of the maxillary lateral incisors. Figures 7-12g to 7-12k show the final occlusion and improvement of the profile.

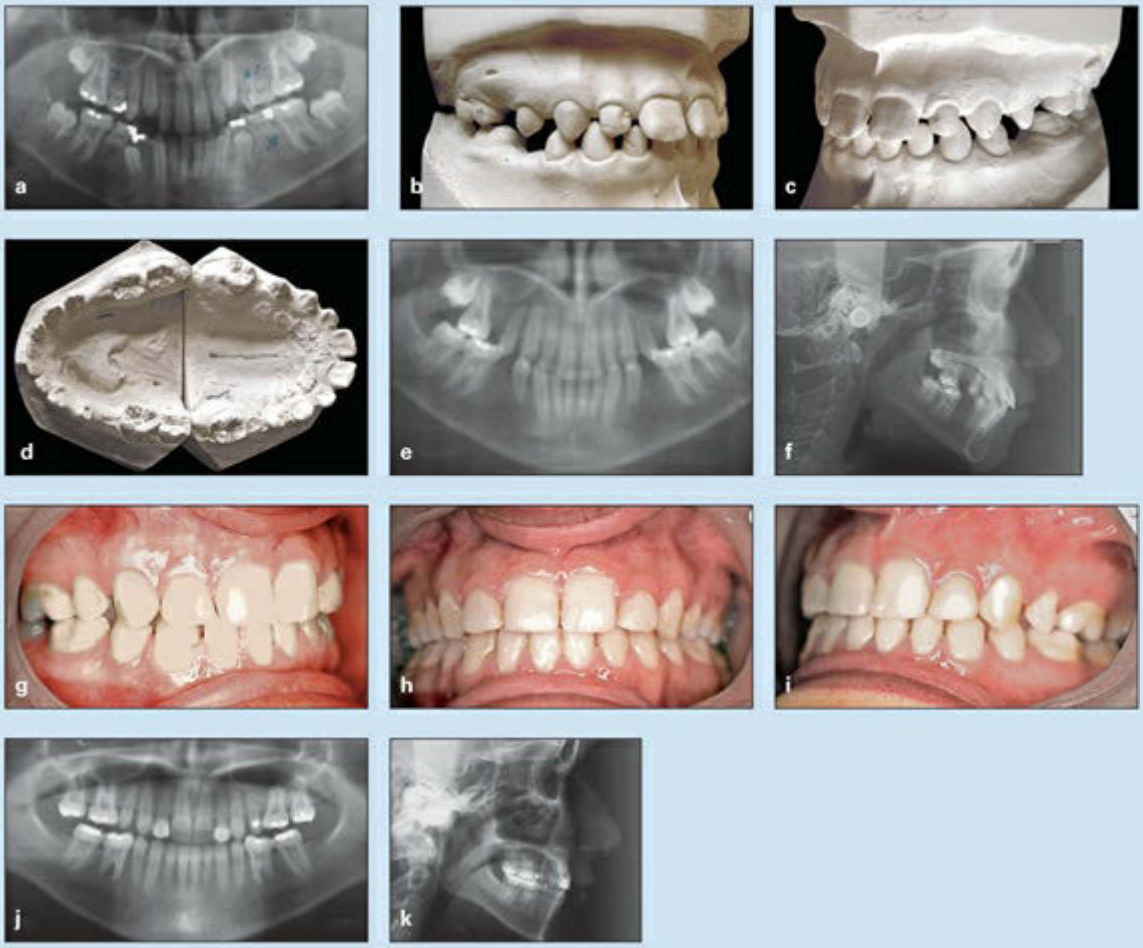


Fig 7-12 Treatment of a 14-year-old girl with oligodontia. All second premolars and third molars are missing. She also exhibits microdontia and interdental spacing. *(a)* Pretreatment panoramic radiograph taken 3 years before orthodontic referral. *(b to d)* Pretreatment casts of the occlusion. *(e)* Pretreatment panoramic radiograph, taken at the time of orthodontic referral. *(f)* Pretreatment cephalometric radiograph. *(g to i)* Posttreatment occlusion. All spaces are closed, and the soft tissue profile is improved. *(j)* Posttreatment panoramic radiograph. *(k)* Posttreatment cephalometric radiograph.

Case 7-7

Cases 7-7 through 7-9 present patients who have several missing teeth (oligodontia), requiring combined orthodontic-prosthodontic treatment.

In this case, a 22-year-old man with 12 missing teeth (including third molars) had a partial denture

that had been constructed to work with the tipped and shifted teeth and space availability (Figs 7-13a to 7-13f). The patient's complaints were diastemata, unequal lateral incisors, and a shifted dental midline.

Treatment:
Based on the patient's desires, orthodontic treatment was limited to bonding in the maxillary arch. After leveling was completed, the midline diastema was shifted to the normal place and closed, the lateral incisor spaces were equalized, and the canine and central incisors were aligned and uprighted to leave the necessary spacing for implants (Figs 7-13g to 7-13j). Figures 7-13k and 7-13l show the final results of treatment.

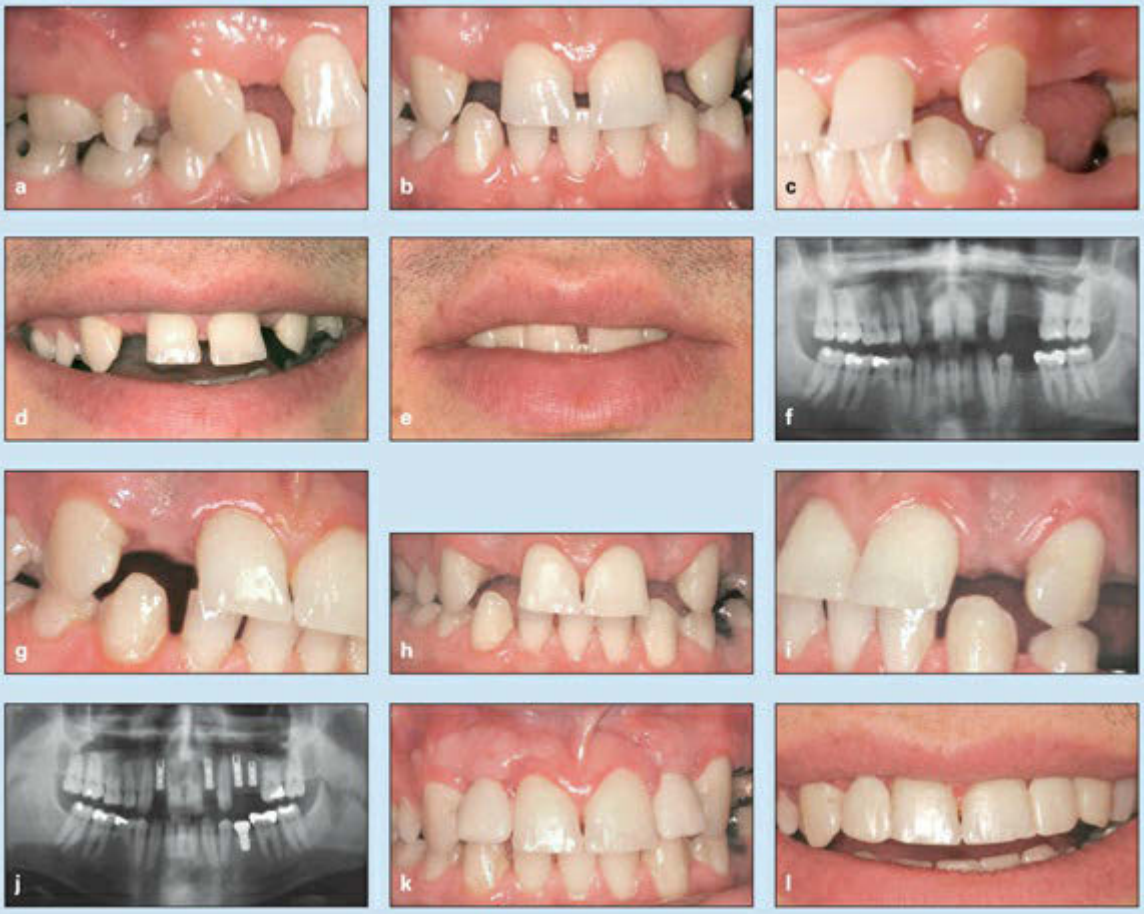


Fig 7-13 Treatment of a 22-year-old man with oligodontia. He is missing 12 teeth, including the third molars. *(a to c)* Pretreatment occlusion. *(d and e)* Pretreatment smile, revealing that the midline has shifted and maxillary lateral incisor spacing is unequal. *(f)* Pretreatment panoramic radiograph showing that other teeth are missing. *(g to i)* Occlusion after orthodontic treatment, including midline shift and closure, root parallelism, and opening of equal space for the lateral incisors. *(j)* Panoramic radiograph taken after implant insertion. *(k and l)* Occlusion and smile after prosthetic treatment. (Orthodontic treatment performed at the Eastman Dental Institute for Oral Health, University of Rochester, New York. Prosthetic treatment by Dr Guglielmi Marcello.)

Case 7-8

In selected cases, one option for management of hypodontia is retention of the primary tooth. A 13-year-old girl with a Class I malocclusion was missing both maxillary permanent lateral incisors. The

maxillary right primary lateral incisor had exfoliated, but the left lateral incisor remained intact.

Early detection and proper intervention could have prevented the problems that occurred after exfoliation of the primary right canine. The lack of treatment resulted in diastemata opening, midline shift, and displacement, rotation, and an edge-to-edge relationship of the maxillary right canine to mandibular canine (Figs 7-14a to 7-14d).

Treatment:

Because the occlusion of the mandibular arch was acceptable and the condition of the maxillary left primary canine was good, orthodontic treatment was confined to the maxillary arch. The orthodontic procedures included closure of the diastemata, correction of the midline, correction of the maxillary permanent canine rotation and displacement, and preparation of adequate space for replacement of the maxillary right lateral incisor. After completion of orthodontic treatment, the primary left lateral incisor was built up to the shape of a permanent lateral incisor. A resin-bonded prosthesis was used to replace the missing right lateral incisor (Figs 7-14e to 7-14h).

A postretention panoramic radiograph, taken 8 years after treatment, revealed that the primary lateral incisor remained in good condition (Fig 7-14i).

Fig 7-14 Treatment of a 13-year-old girl who is missing both maxillary lateral incisors. The left primary lateral incisor is present. *(a to d)* Pretreatment casts of the occlusion. *(e to g)* Posttreatment occlusion, after orthodontic treatment and placement of a resin-bonded prosthesis. *(h)* Posttreatment panoramic radiograph. *(i)* Postretention panoramic radiograph, revealing the good condition of the retained primary lateral incisor 8 years after treatment.

Case 7-9

A 12-year, 8-month-old girl presented with a Class 1 occlusion, a good profile, and moderate maxillary and mandibular incisor crowding. Her chief complaint was the unattractive appearance of her incisors due to crowding and deformed maxillary lateral incisors (peg lateral) and chipped mandibular lateral incisors (Figs 7-15a to 7-15d) . Figures 7-15e and 7-15f show the pretreatment panoramic and

cephalometric radiographs.

Treatment:
Because of the perfect interdigitation in the posterior segments and the patient's good, straight profile, orthodontic treatment was confined to extraction of the four lateral incisors. Figures 7-15g to 7-15l show the post-treatment photographs, panoramic radiograph, and cephalometric radiograph.

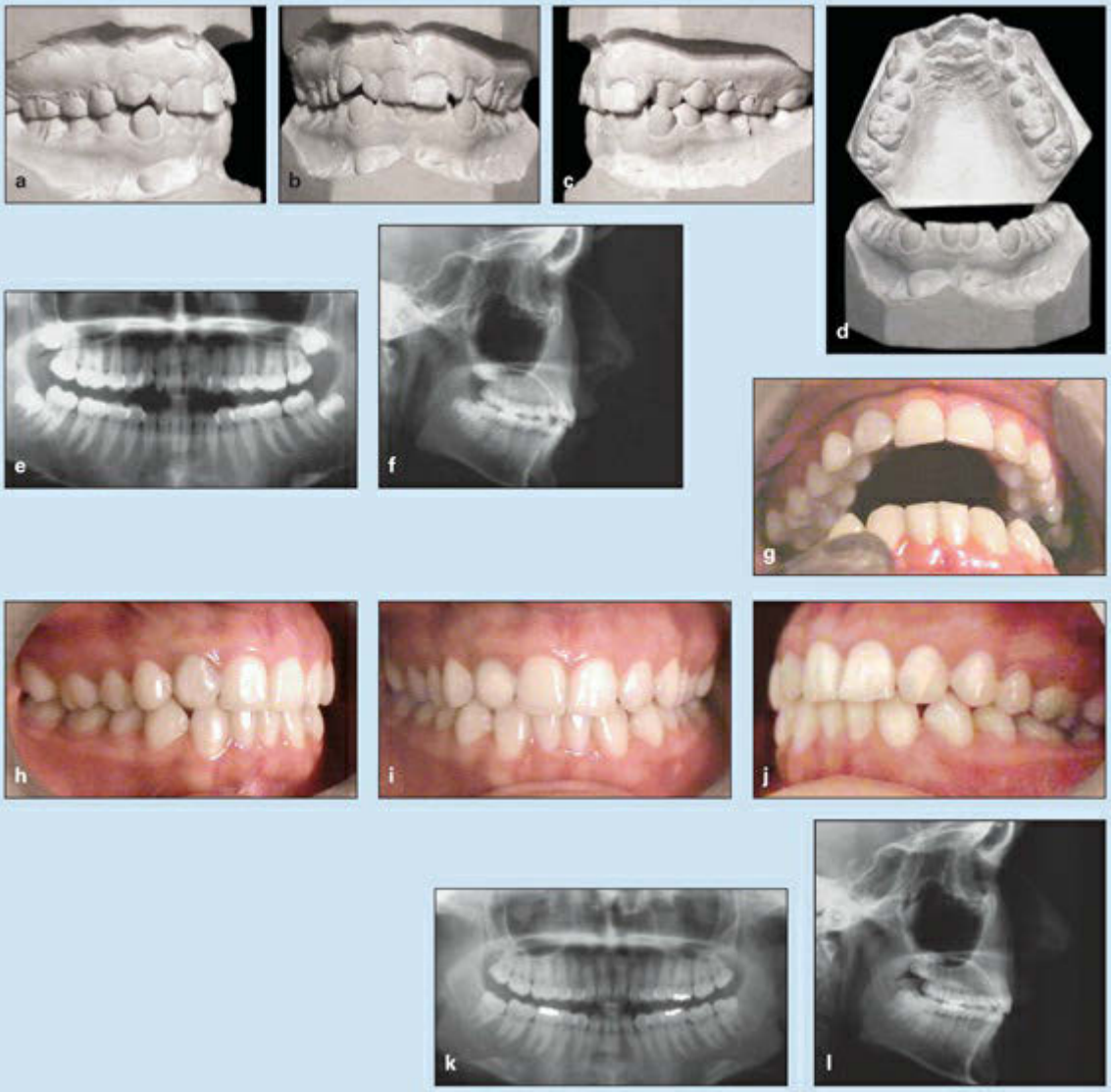


Fig 7-15 *(a to d)* Pretreatment casts of the occlusion. *(e)* Pretreatment panoramic radiograph. *(f)* Pretreatment cephalometric radiograph. *(g to j)* Posttreatment results. *(k)* Posttreatment panoramic radiograph. *(l)* Posttreatment cephalometric radiograph.

Summary

- Hypodontia is the most common craniofacial malformation. It can be isolated (nonsyndromic) or can occur in association with other

developmental anomalies (syndromic).

- *Hypodontia* is a general name for congenital absence of teeth. The term *oligodontia* has been applied to patients who are missing at least four teeth. *Anodontia* is used when patients are missing all teeth, a rare condition that has been found in some patients with ectodermal dysplasia.
- An increase in the prevalence of hypodontia in white individuals during the 20th century has been reported.
- Hypodontia is less frequent in the primary dentition, but there is a high correlation between primary tooth absence and permanent tooth absence.
- Hypodontia is an anomaly that arises during the initiation stage of tooth formation.
- The odontogenic process consists of a complex series of epithelial-mesenchymal interactions involving growth factors and other morphogenetic factors. A large number of genes are involved, and the opportunity for disruptions caused by mutations is very high.
- Initial discoveries indicate that *MSX1* and *PAX9* play critical roles in tooth morphogenesis and that mutations can lead to agenesis.
- The occurrence of hypodontia is not solely determined by genetic factors; epigenetic and environmental factors are probably also important. The possibility that this condition has a multifactorial etiology must be considered.
- Many environmental factors have also been associated with hypodontia, including allergy, facial trauma, maternal medications during pregnancy, endocrine disturbances, maternal health during pregnancy, maternal rubella (German measles) during pregnancy, evolutionary changes, radiation or chemotherapy during tooth development, dysplastic syndromes (ectodermal dysplasia), localized inflammation and infection during the initial stage of tooth formation, and systemic conditions (rickets, syphilis).
- Association of different dental anomalies concomitant with hypodontia has also been reported. These conditions include microdontia, tooth transposition, ectopic permanent canine, taurodontism, and infraocclusion of primary molars.
- Concomitant hypodontia and hyperdontia has also been reported. In the Rochester study, the prevalence was 0.375%.
- Depending on the number of teeth missing and the location in the dental arch, hypodontia can significantly damage occlusion. Hypodontia can result in spacing, midline shift, tipping of adjacent teeth, tooth displacement, overeruption of the opposing teeth, canine impaction, premature contacts,

occlusal trauma, crossbite, mandibular shift, abnormal path of growth, shorter lower facial height, and soft tissue retrusion.

- There are some clinical signs during the transitional stage of occlusion that can be helpful in early detection of hypodontia. These signs, which can be confirmed by radiographic investigation, include overretained primary teeth, incomplete eruption and ankylosis of primary teeth, abnormal spacing and tipping of adjacent teeth, midline shift, microdontia, asymmetric exfoliation of the primary dentition, and evidence of missing teeth in other members of the family.
- Management of hypodontia usually requires a multidisciplinary team approach to ensure proper design of future restorations.
- The practitioner must assess all the conditions carefully, including the patient's complaints and desire for different treatment options; the number and area of missing teeth; the age of the patient and his or her developmental dental age; the type of malocclusion; specific space requirements; the situation of the opposing arch; tooth size relationships (Bolton discrepancy); oral and dentoalveolar health conditions; the structure, form, color, and condition of adjacent teeth; the patient's profile; the incisor position; and the status of the primary teeth.
- Treatment decisions depend on the basic orthodontic diagnosis, evaluation of the aforementioned criteria, the patient's desire, and financial considerations.
- Treatment options include moving the adjacent tooth to the area of the missing tooth for space closure, such as canine substitution for a lateral incisor; opening and aligning space for a traditional prosthesis or an implant-supported replacement; retaining the primary tooth; and autotransplantation.

References

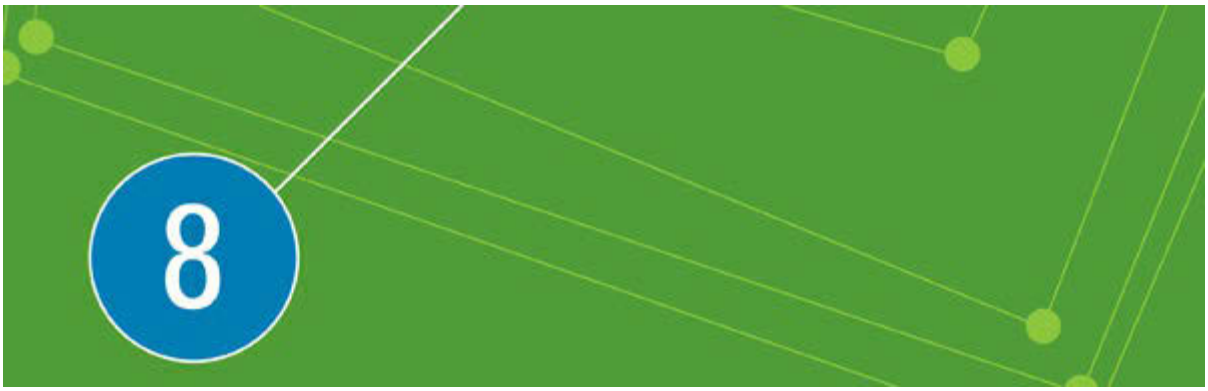
1. Muller TP, Hill IN, Peterson AC, Blayney JR. A survey of congenitally missing permanent teeth. J Am Dent Assoc 1970;81:101–107.
2. Brekhus P, Oliver C, Montelius G. A study of the pattern and combinations of congenitally missing teeth in man. J Dent Res 1944;23:117–131.
3. Rothenberg F, Werther A. Anodontia. Am J Orthod 1939;25:61–81.
4. Byrd ED. Incidence of supernumerary and congenitally missing teeth. J Dent Child 1943;10:84–86.
5. Dolder E. Deficient dentition. Dent Rec 1937;57:142–143.
6. Shah R, Boyd M. Studies of permanent tooth anomalies in 1,886 Canadian individuals. J Can Dent Assoc

1978;6:265– 268.
7. Buenviaje TM, Rapp R. Dental anomalies in children: A clinical and radiographic survey. J Dent Child 1984;51:42– 46.
8. Brown RV. The pattern and frequency of congenital absence of teeth. Iowa State Dent J 1957;43:60–61.
9. Rose JS. A survey of congenitally missing teeth, excluding third molars in 6000 orthodontic patients. Dent Pract Dent Rec 1966;17:107–114.
10. Gimmes H. Congenital absence of teeth in Oslo school children. Dent Abstr 1964;9:236–237.
11. Eidelman E, Chosack I, Rosenzweig KA. Hypodontia: Prevalence amongst Jewish populations of different origin. Am J Phys Anthropol 1973;39:129–133.
12. Glenn FB. A consecutive 6 year study of the prevalence of congenitally missing teeth in private paedodontic practice of 2 geographically separate areas. J Dent Child 1964;31:264–270.
13. Hermel J, Hermel G, Ulmansky M. Dental abnormalities. A study based on 2123 roentgenograms. Refuat Hapeh Vehashinayim 1971;20:1–4.
14. Grahnén H. Hypodontia in the permanent dentition. A clinical and genetical investigation. Odontol Revy 1956;7(sup-pl 3):1–100.
15. Lynham A. Panoramic radiographic survey of hypodontia in Australian Defence Force recruits. Aust Dent J 1990;35:19–22.
16. Thompson GW, Popovich F. Probability of congenitally missing teeth: Results in 1,191 children in the Burlington Growth centre in Toronto. Community Dent Oral Epidemiol 1974;2:26–32.
17. Maklin M, Dummett CO, Weinberg R. A study of oligodontia in a sample of New Orleans children. J Dent Child 1979;46:478–482.
18. Locht S. Panoramic radiographic examination of 704 Danish children aged 9–10 years. Community Dent Oral Epidemiol 1980;8:375–380.
19. Magnússon TE. Prevalence of hypodontia and malformation of permanent teeth in Iceland. Community Dent Oral Epidemiol 1977;5:173–178.
20. Haavikko K. Hypodontia of permanent teeth. An orthopan-tomographic study. Suom Hammaslaak Toim 1971;67:219– 225.
21. Hunstadbraten K. Hypodontia in the permanent dentition. J Dent Child 1973;40:115–117.
22. Bahreman AA, Shokoofan K. Prevalence of hypodontia in Iranian children. Iranian Dent Assoc J 1991;15:2–11.
23. Grahnén H, Granath L. Numerical variations in primary dentition and their correlations with the permanent dentition. Odontol Revy 1961;12:348–357.
24. Burzynski N, Escobar V. Classification genetics of numeric anomalies of the dentition. Birth Defects 1983;13:95–106.
25. Arte S, Nieminen P, Apajalahti S, Haavikko K, Thesleff I, Pirinen S. Characteristics of incisor-premolar hypodontia in families. J Dent Res 2001;80:1445–1450.
26. Lidral AC, Reising BC. The role of MSX1 in human tooth agenesis. J Dent Res 2002;81:274–278.
27. Mostowska A, Kobielak A, Trzeciak WH. Molecular basis of non-syndromic tooth agenesis: Mutations of MSX1 and PAX9 reflect their role in patterning human dentition. Eur J Oral Sci 2003;111:365–370.
28. Seifi M, Kazemi B, Golkar P. The role of MSX1 in tooth agenesis in Iranians. Int J Paediatr Dent 2007;17:254–258.
29. Nieminen P, Arte S, Pirinen S, Peltonen L, Thesleff I . Gene defect in hypodontia: Exclusion of MSX1 and MSX2 as candidate genes. Hum Genet 1995;96:305–308.
30. Vastardis H. The genetics of human tooth agenesis: New discoveries for understanding dental anomalies. Am J Orthod Dentofacial Orthop 2000;117:650–656.
31. Lammi L, Arte S, Somer M, et al. Mutations in AXIN2 cause familial tooth agenesis and predispose to colorectal cancer. Am J Hum Genet 2004;74:1043–1050.
32. Parkin N, Elcock C, Smith RN, Griffin RC, Brook AH. The aetiology of hypodontia: The prevalence, severity and location of hypodontia within families. Arch Oral Biol 2009;54(suppl 1):S52–S56.
33. Yamaguchi T, Tomoyasu Y, Nakadate T, Oguchi K, Maki K. Allergy as a possible predisposing factor for

hypodontia. Eur J Orthod 2008;30:641–644.
34. Axrup K, D'Avignon M, Hellgren K, et al. Children with thalidomide embryopathy: Odontological observations and aspects. Acta Odontol Scand 1966;24:3–21.
35. Schübel F, Partsch CJ. Thalidomide-embryopathies and their effect on dentition [in German]. Dtsch Zahnarztl Z 1965;20:1278–1283.
36. Cohen MM. Congenital, genetic, and endocrinologic influences on dental occlusion. Dent Clin North Am 1975;19:499–514.
37. Lundstrom R, Lysell L, Berghagen N. Dental development in children following maternal rubella. Acta Paediatr 1962;51:155–160.
38. Anderson BL, Thompson GW, Popovich F. Evolutionary dental changes. Am J Phys Anthropol 1975;43:95–102.
39. Jacobi KP, Cook DC, Corruccini RS, Handler JS. Congenital syphilis in the past: Slaves at Newton Plantation, Barbados, West Indies. Am J Phys Anthropol 1992;89:145–158.
40. Sattur A, Naikmasur VG, Shrivastava R, Babshet M. Familial hypophosphatemic rickets. J Indian Soc Pedod Prev Dent 2010;28:302–306.
41. Lambert PM. Infectious disease among enslaved African Americans at Eaton's Estate, Warren County, North Carolina, ca. 1830–1850. Mem Inst Oswaldo Cruz 2006;101(suppl 2):107–117.
42. Mcdonnell ST, Liversidge H, Kinirons M. Temporary arrest of root development in a premolar of a child with hypodontia and extensive caries. Int J Pediatr Dent 2004;14:455–460.
43. Kaste SC, Hopkins KP, Jones D, Crom D, Greenwald CA, Santana VM. Dental abnormalities in children treated for acute lymphoblastic leukemia. Leukemia 1997;11:792–796.
44. Näsman M, Björk O, Söderhäll S, Ringdén, Dahllöf G. Disturbances in the oral cavity in pediatric long-term survivors after different forms of antineoplastic therapy. Pediatr Dent 1994;16:217–223.
45. Pajari D, Lahtela P, Lanning M, Larmas M. Effect of antineoplastic therapy on dental maturity and tooth development. J Pedod 1988;12:266–274.
46. Sonis AL, Tarbell N, Valachovic RW, Gelber R, Schwenn M, Sallan S. Dentofacial development in long-term survivors of acute lymphoblastic leukemia. Cancer 1990;66:2645– 2652.
47. Cobourne MT. Familial human hypodontia—Is it all in the genes? Br Dent J 2007;203:203–208.
48. Bansal M, Manchanda K, Pandey SS. Hypohidrotic ectodermal dysplasia. Int J Trichology 2012;4:167–168.
49. Agarwal S, Gupta S. Hypohidrotic ectodermal dysplasia. Indian Dermatol Online J 2012;3:125–127.
50. Koyuncuoglu CZ, Metin S, Minoglu-Saylan I, Calisir K, Tuncer O, Alpdogan K. Full-mouth rehabilitation of a patient with ectodermal dysplasia patient with dental implants [epub ahead of print 19 November 2012]. J Oral Implantol.
51. Ranta R. A review of tooth formation in children with cleft lip/palate. Am J Orthod Dentofacial Orthop 1986;90:11–18.
52. Shapira Y, Lubit E, Kuftinec MM. Hypodontia in children with various types of clefts. Angle Orthod 2000;70:16–21.
53. Mestrović JS. Hypodontia in patients with Down's syndrome. Coll Antropol 1998;22(suppl): 69–72.
54. Suri S, Tompson BD, Cornfoot L. Cranial base, maxillary and mandibular morphology in Down syndrome. Angle Orthod 2010;80:861–869.
55. Kumasaka S, Miyagi A, Sakai N, Shindo J, Kashima I. Oligodontia: A radiographic comparison of subjects with Down syndrome and normal subjects. Spec Care Dentist 1997;17:137–141.
56. Silverman NE, Ackerman JL. Oligodontia: A study of its prevalence and variation in 4032 children. ASDC J Dent Child 1979;46:470–477.
57. Maruko E, Hayes C, Evans CA, Padwa B, Mulliken JB. Hypodontia in hemifacial microsomia. Cleft Palate Craniofac J 2001;38:15–19.
58. Lai PY, Seow WK. A controlled study of the association of various dental anomalies with hypodontia of permanent teeth. Pediatr Dent 1989;11:291–296.
59. McKeown HF, Robinson DL, Elcock C, al-Sharood M, Brook AH. Tooth dimensions in hypodontia patients, their unaffected relatives and a control group measured by a new image analysis system. Eur J Orthod

2002;24:131–141.
60. Peck L, Peck S, Attia Y. Maxillary canine-first premolar transposition, associated dental anomalies and genetic basis. Angle Orthod 1993;63:99–109.
61. Peck S, Peck L, Kataja M. Site-specificity of tooth agenesis in subjects with maxillary canine malpositions. Angle Orthod 1996;66:473–476.
62. Via WF Jr. Submerged deciduous molars: Familial tendencies. J Am Dent Assoc 1964;69:127–129.
63. Zengin AZ, Sumer AP, Karaarslan E. Impacted primary tooth and tooth agenesis: A case report of monozygotic twins. Eur J Dent 2008;2:299–302.
64. Bianchi SD, Roccuzzo M. Primary impaction of primary teeth: A review and report of three cases. J Clin Pediatr Dent 1991;15:165–168.
65. Low T. Hypodontia and supernumerary tooth: Report of a case and its management. Br J Orthod 1977;4:187–190.
66. Sharma A. A rare non-syndrome case of concomitant multiple supernumerary teeth and partial anodontia. J Clin Pediatr Dent 2001;25:167–169.
67. Sharma A. A rare case of concomitant hypo-hyperdontia in identical twins. J Indian Soc Pedod Prev Dent 2008;26(suppl 2):S79–S81.
68. Matsumoto M, Nakagawa Y, Sobue S, Ooshima T. Simultaneous presence of a congenitally missing premolar and supernumerary incisor in the same jaw: Report of case. ASDC J Dent Child 2001;68:63–66.
69. Varela M, Arrieta P, Ventureira C. Non-syndromic concomitant hypodontia and supernumerary teeth in an orthodontic population. Eur J Orthod 2009;31:632–637.
70. Ogaard B, Krogstad O. Craniofacial structure and soft tissue profile in patients with severe hypodontia. Am J Orthod Dentofacial Orthop 1995;108:472–477.
71. Ben-Bassat Y, Brin I. Skeletodental patterns in patients with multiple congenitally missing teeth. Am J Orthod Dentofacial Orthop 2003;124:521–525.
72. Kokich VO Jr, Kinzer GA. Managing congenitally missing lateral incisors. 1. Canine substitution. J Esthet Restor Dent 2005;17:5–10.
73. Zachrisson BU. Improving orthodontic results in cases with maxillary incisors missing. Am J Orthod 1978;73:274– 289.
74. Zachrisson BU, Mjör IA. Remodeling of teeth by grinding. Am J Orthod 1975;68:545–553.
75. Kokich VG. Maxillary lateral incisor implants: Planning with the aid of orthodontics. Int J Oral Maxillofac Surg 2004;62:48–56.
76. Bolton WA. The clinical application of a tooth-size analysis. Am J Orthod 1962;48:504–529.
77. Botelho MG, Leung KC, Ng H, Chan K. A retrospective clinical evaluation of two-unit cantilevered resin-bonded fixed partial dentures. J Am Dent Assoc 2006;137:783–788.
78. Adell R, Eriksson B, Lekholm U, Brånemark PI, Jemt T. Long-term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws. Int J Oral Maxillofac Implants 1990;5:347–359.
79. Mayer TM, Hawley CE, Gunsolley JC, Feldman S. The single-tooth implant: A viable alternative for single-tooth replacement. J Periodontol 2002;73:687–693.
80. Naert I, Koutsikakis G, Duyck J, Quirynen M, Jacobs R, van Steenberghe D. Biologic outcome of single-implant restorations as tooth replacements: A long-term follow-up study. Clin Implant Dent Relat Res 2000;2:209–218.
81. Noack N, Willer J, Hoffmann J. Long-term results after placement of dental implants: Longitudinal study of 1,964 over 16 years. Int J Oral Maxillofac Implants 1999;14:748– 755.
82. Zarb GA, Schmitt A. The longitudinal clinical effectiveness of osseointegrated dental implants: The Toronto study. 1. Surgical results. J Prosthet Dent 1990;63:451–457.
83. Kinzer GA, Kokich VO Jr. Managing congenitally missing lateral incisors. 3. Single-tooth implants. J Esthet Restor Dent 2005;17:202–210.
84. Ostler MS, Kokich VG. Alveolar ridge changes in patients congenitally missing mandibular second premolars. J Prosthet Dent 1994;71:144–149.
85. Thilander B, Odman J, Grondhi K, Friberg B. Osseointegrated implants in adolescents. An alternative in

replacing missing teeth. Eur J Orthod 1994;16:84–95.
86. Apfel H. Preliminary work in transplanting the third molar to the first molar position. J Am Dent Assoc 1954;48:143– 150.
87. Miller H. Transplantation and reimplantation of teeth. Oral Surg Oral Med Oral Pathol 1956;9:84–95.
88. Natiella JR, Armitage JE, Greene GW. The replantation and transplantation of teeth. A review. Oral Surg Oral Med Oral Pathol 1970;29:397–419.
89. Kristerson L, Lagerström I. Autotransplantation of teeth in cases with agenesis or traumatic loss of maxillary incisors. Eur J Orthod 1991;13:486–492.
90. Jonsson T, Sigurdsson TJ. Autotransplantation of premolars to premolar sites. A long-term follow-up study of 40 consecutive patients. Am J Orthod Dentofacial Orthop 2004;125:668–675.
91. Sabri R. Management of over-retained mandibular deciduous second molars with and without permanent successors. World J Orthod 2008;9:209–220.
92. Kurol J, Olson L. Ankylosis of primary molars—A future periodontal threat to the first permanent molars? Eur J Orthod 1991;13:404–409.
93. Sletten DW, Smith BM, Southard KA, Casko JS, Southard TE. Retained deciduous mandibular molars in adults: A radiographic study of long-term changes. Am J Orthod Dentofacial Orthop 2003;124:625–630.
94. Valencia R, Saadia M, Grinberg G. Controlled slicing in the management of congenitally missing second premolars. Am J Orthod Dentofacial Orthop 2004;125:537–543.
95. Lindqvist B. Extraction of the deciduous second molar in hypodontia. Eur J Orthod 1980;2:173–81.
96. Mamopoulou A, Hägg U, Schröder U, Hansen K. Agenesis of mandibular second premolars. Spontaneous space closure after extraction therapy: A 4-year follow-up. Eur J Orthod 1996;18:589–600.
97. Slagsvold O, Bjercke B. Indications for autotransplantation in cases of missing premolars. Am J Orthod 1978;74: 241–257.
98. Kvint S, Lindsten R, Magnusson A, Nilsson P, Bjerklin K. Autotransplantation of teeth in 215 patients: A follow-up study. Angle Orthod 2010;80:446–451.



Orthodontic Management of Supernumerary Teeth

Supernumerary teeth are defined as teeth formed in excess of the normal dental complement in a normal primary or permanent dentition. This phenomenon, also known as *hyperdontia*, is a disorder of odontogenesis that develops at the stage of initiation during teeth formation. This abnormality develops because of continued bud formation of the enamel organ or excessive proliferation of cells. Excessive cell proliferation or continued budding can be responsible for a variety of irregularities in the primary and transitional dentition.

The presence of an extra tooth in the dental arch has great potential to disrupt normal occlusal development. Early detection and intervention to remove the supernumerary tooth, or monitoring until the proper time for extraction, is a type of interceptive or guidance treatment that can be performed at early ages.

Prevalence

Supernumerary teeth are relatively common in the general population. They can affect both the primary and the permanent dentitions. The association of multiple hyperdontia with different syndromes, such as Gardner syndrome, cleidocranial syndrome, or facial fissures, has also been reported widely in the literature[1–4]; however, supernumerary teeth can also appear in nonsyndromic situations.

Supernumerary teeth may occur singly or in multiples, unilaterally or bilaterally, and in one or both arches. The occurrence of one or two supernumerary teeth is most common in the anterior region of the maxilla, followed by the mandibular premolar region. When multiple (more than three) supernumerary teeth are present, the most common site affected is the mandibular premolar region.

The incidence of one or two supernumerary teeth occurred most frequently in the maxillary anterior region (46.9% of the patients); this was followed by supernumerary premolars (24.1%), then supernumerary molars (18%), and, finally, supernumerary paramolars in 5.6% of cases.[5] Yusof[5] reported that hyperdontia from different ethnic groups varies widely, from 0.1% to 3.8%. This range could be due to the methodology for detection or the population studied in different reports.

Table 8-1 is a comparison of reports of hyperdontia in various populations. In a study performed in Rochester, NY, panoramic radiographs of children distributed among three racial/ethnic groups (whites, blacks, and Hispanics) were randomly selected from the records of 800 children aged 6 to 17 years (Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007). The radiographs were evaluated for the presence of hyperdontia and hypodontia. Hyperdontia was found in 2.25% of the total group.

Table 8-1	Prevalence of supernumerary teeth reported in various populations*		
Investigator	Year	Country	Prevalence
Shah[6]	1978	Canada	0.25%
Boyne[7]	1954	USA	0.3%
Buenviaje and Rapp[8]	1984	USA	0.5%
Wallfeldt[9]	1961	Sweden	0.5%
Frome et al[10]	1977	USA	1.0%
Schulze[11]	1960	Germany	1.0%
Morris et al[12]	1969	USA	1.4%
Billberg and Lind[13]	1965	Sweden	1.4%
McKibben and Brearley[14]	1971	USA	1.5%

Jarvinen[15]	1976	Finland	1.7%
Locht[16]	1980	Denmark	1.7%
Clayton[17]	1956	USA	1.9%
Luten[18]	1967	USA	2.0%
Parry and Iyer[19]	1961	India	2.5%
Lacoste et al[20]	1962	France	2.8%
Salcido-García et al[21]	2004	Mexico	3.2%
Bäckman and Wahlin[22]	2001	Sweden	1.9%
Bahreman et al*	2007	USA	2.25%

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

Multiple supernumerary teeth are not a common occurrence, although a single supernumerary tooth or a few such teeth in a single patient have been widely reported in the literature. The occurrence of supernumerary teeth is less frequent than the congenital absence of teeth, and supernumerary teeth occur twice as often in men as in women. Hyperdontia occurs more often in the maxilla than in the mandible, particularly in the maxillary anterior region. The condition is very rare in the primary dentition.

The most common site for supernumerary teeth is the maxillary incisor area. The occurrence of supernumerary teeth in several members of the same family has been also observed, which indicates a familial pattern.

All reports indicate that hyperdontia is more prevalent in males than in females. Table 8-2 shows the sex distribution of hyperdontia in the subjects of the author's investigation. Among the three racial/ethnic populations, the distribution of supernumerary teeth was highest in the white group, followed by the black group; the prevalence was lowest in the Hispanic group (Table 8-3). A comparison of the distribution of hyperdontia by race/ethnicity and sex is found in Table 8-4. The highest prevalence rates were found among white males and black males.

Table 8-2	Distribution of supernumerary teeth by sex*		
Sex	Supernumerary teeth	Total patients	Percentage†

	No	Yes		
Male	362	12	374	3.21%
Female	420	6	426	1.41%
Total	*782*	*18*	*800*	*2.25%*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.
†Fisher exact test (*P* value = .10) revealed no statistically significant difference.

Table 8-3	Distribution of supernumerary teeth by ethnicity*			
Ethnic group	Supernumerary teeth		Total patients	Percentage†
	No	Yes		
Black	376	7	383	1.83%
White	286	6	292	2.05%
Hispanic	101	2	103	1.94%
Other	19	3	22	13.6%
Total	*782*	*18*	*800*	*2.25%*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.
†Fisher exact test (*P* = .05) revealed no statistically significant difference.

Table 8-4	Distribution of supernumerary teeth by ethnicity and sex*			
Group	Supernumerary teeth		Total patients	Percentage
	No	Yes		
Black males	174	5	179	2.79%
Black females	202	2	204	0.98%
Hispanic males	46	1	47	2.13%
Hispanic females	55	1	56	1.79%
White males	132	5	137	3.65%
White females	154	1	155	0.65%
Other males	10	1	11	9.09%

Other females	9	2	11	18.2%
Total	*782*	*18*	*800*	*2.25%*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

Almost all the literature about the distribution of supernumeraries reveals a higher prevalence in the maxilla than in the mandible and the highest occurrence in the maxillary anterior area. The author's Rochester study confirmed earlier findings. Of the 24 supernumerary teeth found, 6 were in the mandible and 18 were in the maxilla. The most prevalent location was the anterior maxilla, and the next was the mandibular premolar area (Table 8-5).

Table 8-5 Distribution of supernumerary teeth by location*

Mandible		Maxilla	
Location	**No. of teeth**	**Location**	**No. of teeth**
Incisor	1	Incisor	15
Canine	0	Canine	0
Premolar	3	Premolar	1
Molar	2	Molar	2
Total	*6*	*Total*	*18*

*Bahreman AA, Jensen MO, Lothyan JD, unpublished data, 2007.

Etiology

The etiology of supernumerary teeth is not fully understood. Some hypothesize that they have a genetic background; a similarity to primates has also been proposed. Because this anomaly is more commonly found in relatives of affected individuals than the general population, genetics may play a part. However, Yusof[5] reported that the inheritance pattern does not follow Mendelian principles. Therefore, environmental factors must also be considered in the etiology of supernumerary teeth.

Neville et al[23] reported evidence of supernumerary teeth in some genetic

syndromes such as cleidocranial dysostosis, Gardner syndrome, cleft lip and palate, Apert syndrome, Crouzon syndrome, and ectodermal dysplasia.

Three general etiologic theories—dichotomy, hyperactivity, and atavism—for the development of supernumerary teeth have been proposed.

Dichotomy theory

Dichotomy theory suggests that the supernumerary tooth is created as a result of a dichotomy of the tooth bud. Advocates of the dichotomy theory contend that the tooth bud splits into two equal- or different-sized parts, resulting in two teeth of equal size or one normal and one dimorphic tooth, respectively. This hypothesis is supported by animal experiments in which split germs have been cultivated in vitro.[24,25]

Hyperactivity theory

Another theory, which is more supported in the literature, is the hyperactivity theory, which suggests that supernumeraries are formed as a result of local, independent conditions by hyperactivity of the dental lamina.[26,27] This abnormality develops due to continued bud formation of the enamel organ or excessive proliferation of cells during the stage of initiation. Sharma[28] explained that disturbance in migration, proliferation, and differentiation of the neural crest cells and interaction between the epithelial and mesenchymal cells during the initiation stage of tooth development have been suspected as the possible cause.

Depending on the stage of differentiation, this anomaly may terminate to a cyst, an odontoma, or a supernumerary tooth.

Atavism theory

Shapira and Kuftinec[29] accepted the theories of hyperactivity and dichotomy and also suggest the phylogenetic process of atavism syndromes as a factor for hyperdontia.

Atavism is a reappearance of an earlier characteristic that had disappeared generations ago. It occurs because genes for previously existing phenotypic features are often preserved in DNA, even though the genes are not expressed in some or most of the organisms possessing them. The late development of supernumerary teeth or a "postpermanent dentition" may also be an example of this condition.

Becker et al[30] reported a case of a child in whom a maxillary midline supernumerary tooth was removed at age 8 years. When the child was 12 years old, multiple anterior and posterior maxillary and mandibular supernumerary teeth were detected. This may be an example of development of postpermanent dentition.

Paramolars and parapremolars would also seem to fit a model of postpermanent dentition development consistent with continued dental lamina activity.

Concomitant Hypodontia and Hyperdontia

Concomitant hypodontia-hyperdontia is a rare anomaly of mixed numeric condition of the human dentition; very few cases have been reported in the literature.[28,31,32] The etiopathogenesis of this simultaneous hypodontia-hyperdontia is obscure, but according to Low[26] and Sharma,[28] the suspected possible cause of this disturbance is migration, proliferation, and differentiation of the neural crest cells and interaction between the epithelial and mesenchymal cells during the initiation stage of tooth development (see chapter 7).

Odontoma

Hyperactivity of the dental lamina during the stage of initiation can terminate to extra tooth formation (supernumerary tooth) or a dysmorphic tooth tissue called *odontoma*. Odontoma is the most common benign odontogenic tumor of epithelial and mesenchymal origin. The World Health Organization[33] has defined odontomas as either complex odontoma or compound odontoma.

Complex odontoma is a malformation in which all dental tissues are present but arranged in a more or less disorderly pattern. In compound odontoma, all of the dental tissues are represented in a pattern that is more orderly than that of the complex type. Enamel, dentin, cementum, and pulp are arranged as they would be in the normal tooth.

In another classification, this anomaly has been described as simple, complex, and compound odontoma. The simple kind is classified as a supernumerary tooth that has all the structures of a tooth, with separate dentin, enamel, and pulpal structures.[33]

Eruption of odontoma into the oral cavity is extremely uncommon. There are reports of the exceptional spontaneous eruption of this tumor (both compound and

complex odontoma).[34–36] A literature review by Litonjua et al[37] recorded only 14 cases in the English literature from 1980 to 2003. In 2007, Vengal et al[38] reported a case in Canada; this case was particularly interesting because of a possible relationship of the malformation to a traditional tooth extraction that was performed on the patient as a young child.

The average age of people found with odontoma is 14 years, and the condition is frequently associated with unerupted adjacent teeth. Odontomas are mostly associated with permanent teeth, and they are rarely associated with primary teeth.

Formation of an odontoma, depending on the location and size, can cause severe damage to the dental arch and dentition. If intervention is late or completely neglected, the odontoma can require major surgery and result in loss of adjacent teeth. Periodic radiographic evaluation and monitoring of the dental transition can be very helpful in early detection of and intervention in this kind of problem.

Figure 8-1 presents a case of neglected odontoma. Retrospective evaluation of the radiographs clearly show the extent of damage and final undesirable results. This case highlights the importance of longitudinal monitoring of panoramic radiographs and the need for early intervention to prevent adverse consequences.

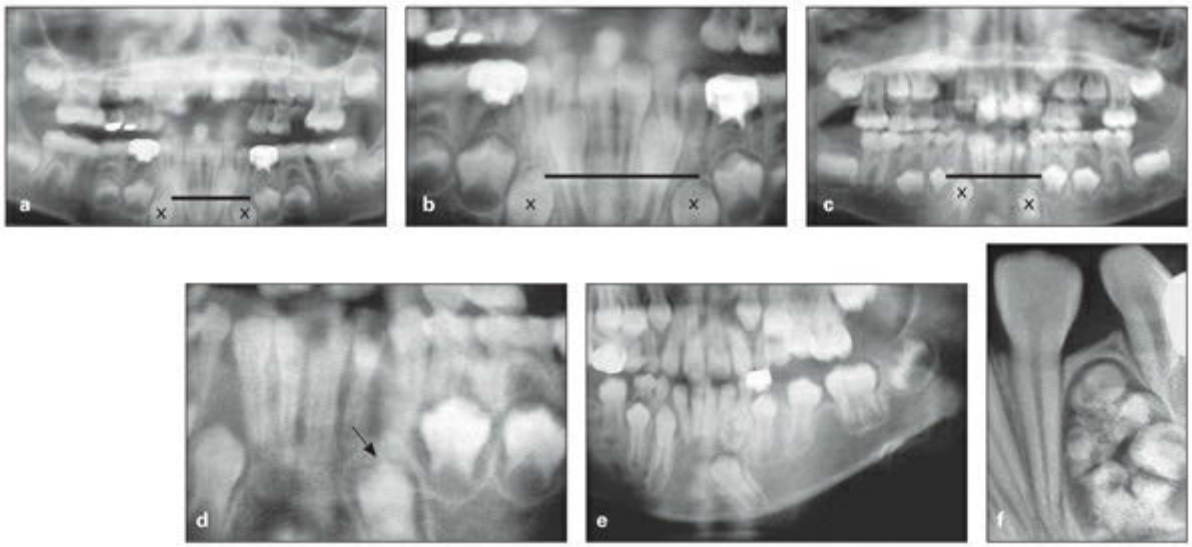


Fig 8-1 Neglected odontoma, resulting in extensive damage. *(a and b)* Panoramic radiograph of a 9-year-old boy. The eruption pattern seems normal, but there is a slight difference of canine heights *(horizontal lines)*. *(c and d)* Mandibular canines, 15 months later, with a significant difference of canine heights. A radiopacity has developed around the left canine crown *(arrow in d)*. *(e)* Panoramic radiograph taken 2 years later, showing significant changes in the position of the left canine due to growth of the odontoma. *(f)* Periapical radiograph taken on the same day, showing the huge odontoma.

Dentigerous Cyst

A *dentigerous cyst* or *follicular cyst* is an odontogenic cyst that forms around the crown of an unerupted or partially erupted tooth and appears on the radiograph as a pericoronal radiolucency.

The cyst cavity is lined with epithelial cells derived from the reduced enamel epithelium of the tooth-forming organ.

Its pathogenesis is thought to be pressure exerted by an erupting tooth on the follicle, which may obstruct venous flow and induce accumulation of exudates between the reduced enamel epithelium and the tooth crown.[39]

Most patients with this type of cyst are young or middle-aged adults. The teeth most commonly affected are third molars and maxillary canines, but any other unerupted tooth can be involved (Figs 8-2 and 8-3). Formation of dentigerous cysts during the primary dentition is very rare.

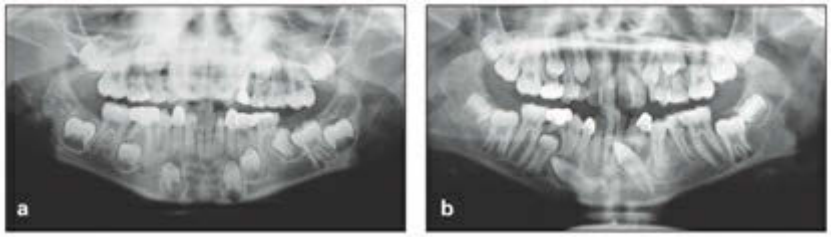


Fig 8-2 *(a and b)* The primary canines in this case crowned without previous evaluation of the pulp vitality. This led to the formation of cysts, which were not checked properly and consequently caused deflection of both permanent canines.

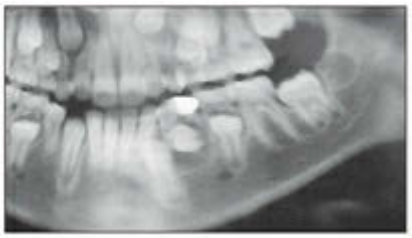


Fig 8-3 Cyst formation displacing the permanent first premolar and preventing eruption.

Shen et al[40] evaluated and monitored 100 embedded supernumeraries and found that 35% showed cystic changes by biopsies.

Classification of Supernumerary Teeth

Supernumerary teeth have been classified into two categories according to their shape and their position (Box 8-1).

Box 8-1	Classification of supernumerary teeth
Classification on the basis of location in the arch	***Classification on the basis of form***
• Mesiodentes	• Conical types
• Parapremolars	• Tuberculate types
• Paramolars	• Supplemental types
• Distomolars	• Odontomas

Supplemental types are those that are closely similar to the adjacent tooth (Fig 8-4).

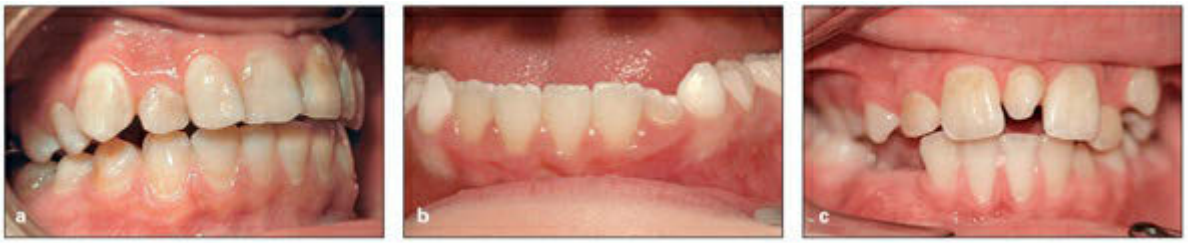


Fig 8-4 *(a)* Supplemental maxillary right lateral incisor. *(b)* Supplemental mandibular left lateral incisor. *(c)* Conical maxillary supernumerary tooth.

A *mesiodens* is a type of supernumerary tooth that is located between the central incisors. Kaler[41] reported that the incidence of mesiodentes varied from 0.15% to 3.0%, as reported among a large population of different ethnic origins.

Low[26] found that the conical types of supernumerary teeth, which are located near the midline, often erupt in childhood and have root formation chronologically matched with teeth in the region.

Ectopic Eruption of Supernumerary Teeth

Ectopic eruption of the teeth in various areas of the dentition has been widely reported by different investigators, but ectopic eruption of a tooth in other sites of the jaws, especially for supernumeraries, is rare. Ectopic teeth may be permanent, primary, or supernumerary. Erkmen et al[42] reported the case of an ectopic supernumerary tooth in the left maxillary sinus.

Many theories have been proposed for the etiology of this type of anomaly, including developmental disturbances such as cleft palate, traumatic tooth displacement, cysts, genetic factors, or crowding and dense bone.[43–45]

Most ectopic supernumeraries are asymptomatic and are usually found during

routine clinical or radiologic investigations. Periodic use of panoramic radiographs during the transitional dentition is a useful technique for early detection of these types of anomalies. The definitive treatment is surgical removal of the tooth.

Late Development of Supernumerary Teeth

Late development of supernumerary teeth is a situation that demands careful attention and follow-up monitoring even after completion of orthodontic treatment.

Gibson[46] reported late development of a supernumerary mandibular premolar in a patient between the ages of 11 and 20 years. Other cases of late development of parapremolar supernumerary teeth, or "recurring supernumerary mandibular premolar," have also been reported by Rubenstein et al,[27] Chadwick and Kilpatrick,[47] and Poyton et al.[48]

Rubenstein et al[27] reported that 8% to 10% of recurring supernumeraries are found in the premolar area, occurring in 0.29% to 0.64% of the population. Males are affected twice as often as females. Most late supernumerary teeth are seen in the premolar area of the mandible. Evidence of late development of the supernumerary tooth comes from consecutive panoramic radiographs even after treatment (see case 8-4).

Effect of Hyperdontia on Occlusion

The presence of supernumerary teeth in the dental arch is almost always harmful to adjacent teeth and occlusion. Depending on the number, location, size, and type of supernumeraries, different kinds of irregularity of occlusion may result:

- Delayed eruption
- Impaction
- Ectopic eruption
- Displacement of adjacent teeth
- Damage to tooth structure, including root resorption, malformation, dilaceration, and loss of vitality of adjacent teeth
- Crowding
- Spacing

- Premature contact and occlusal interference
- Cystic formation

As a rule, the damage to adjacent teeth varies depending on the number, type, and position of supernumeraries. The more supernumerary teeth, the greater the damage to occlusion; management is more complicated. Supernumerary teeth in the maxillary anterior region can prevent eruption and cause crowding, tooth rotation, abnormal diastemata, ectopic eruption, and impaction. Figure 8-5 shows different kinds of supernumeraries causing many occlusal problems in different patients. Mandibular supplemental supernumerary teeth can cause crowding, midline shift, and arch asymmetry (Fig 8-6).

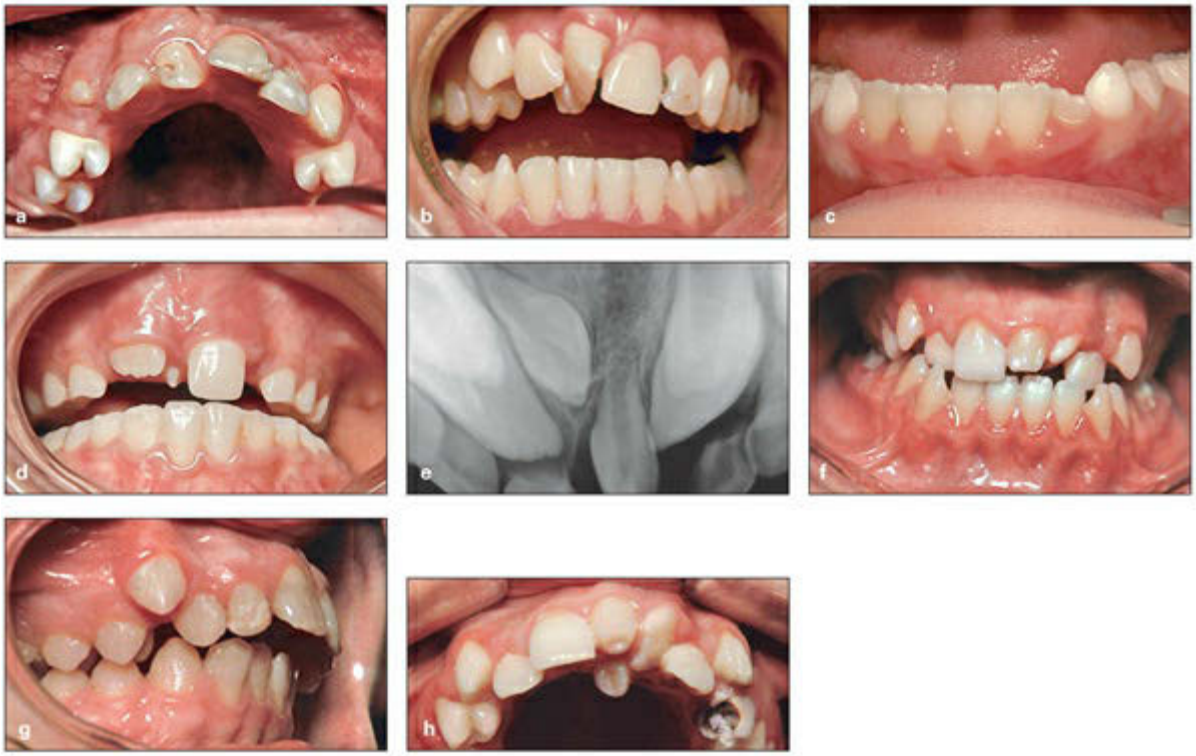


Fig 8-5 *(a to h)* Various supernumerary teeth, affecting occlusion in many different ways.

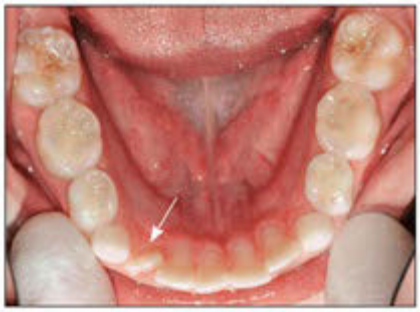


Fig 8-6 Supplemental mandibular supernumerary tooth *(arrow)* causing crowding, midline shift, and arch asymmetry.

Hyperdontia in the buccal segments of the arch can also cause many problems for adjacent teeth and the occlusion. These supernumeraries can prevent eruption and cause crowding, impaction, and posterior open bite. Figure 8-7a shows four parapremolar supernumeraries that have prevented eruption of all mandibular premolars. Figure 8-7b shows two paramolar supernumerary teeth that are damaging the roots of the permanent first molars.

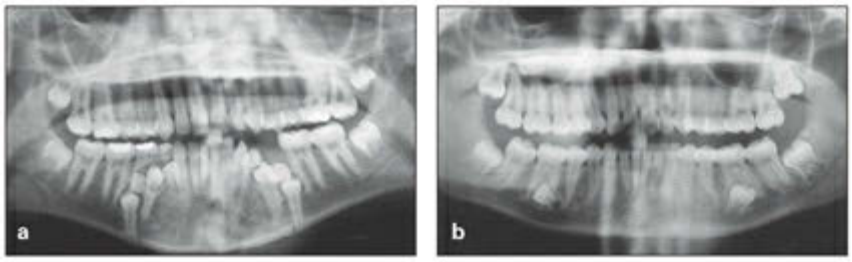


Fig 8-7 *(a)* Parapremolar supernumerary teeth preventing eruption of mandibular premolars. *(b)* Paramolar supernumerary teeth damaging the permanent first molar roots.

Most of these conditions develop because of an irregularity during the developing occlusion and especially during the transitional dentition. Careful examination and monitoring can detect the problem easily. With proper intervention, development of irregularities can be prevented, or the severity of the problem can be reduced. Unfortunately, most cases are referred after the occlusion is damaged. Longitudinal monitoring of panoramic radiographs is an easy technique for the general practitioner and pediatric dentist, who see children at an early age. Radiographs should be compared at 6, 8, and 10 years of age (see chapter 3).

Early Recognition and Clinical Signs of Hyperdontia

Development of supernumerary teeth can occur any time during the primary dentition, mixed dentition, and the permanent dentition. They are almost always harmful to adjacent teeth and to the occlusion. Most cases of supernumerary teeth are asymptomatic and are usually found during routine clinical or radiologic investigations. Therefore, early recognition of and treatment planning for supernumerary teeth are important components of the preliminary assessment of a child's occlusal status and oral health, which is based on careful clinical and paraclinical examinations.

Clinical examination

Clinical examination of children during the primary or mixed dentition is discussed in detail in chapter 3. When assessing supernumerary teeth in the developing occlusion of a child, the clinician must consider the number, size, and form of teeth, the eruption time, the sequence of eruption, the position of each tooth, and local and general factors that can affect occlusion during transitional changes. The following are clinical signs of the presence of supernumerary teeth:

- Abnormal pattern and abnormal sequence of eruption
- Delayed eruption
- Absence of eruption
- Ectopic eruption
- Asymmetric eruption between the left and right sides of more than 6 months' duration
- Local spacing or crowding
- Abnormal tooth bulge

Radiographic examination

If presence of a supernumerary tooth is suspected, special radiographic techniques such as posteroanterior radiographs, orthopantomograms, occlusal radiographs, and computed tomograms are helpful to detect and localize the supernumerary tooth. *Computed axial tomography* (CAT or CT) is the process of using a computer to generate a three-dimensional image from flat, two-dimensional radiographs, one slice at a time. CT scans of internal organs, bone, teeth, and soft tissue provide greater clarity and reveal more details than regular radiographs. Figure 8-8 reveals the presence of multiple supernumerary teeth three dimensionally.

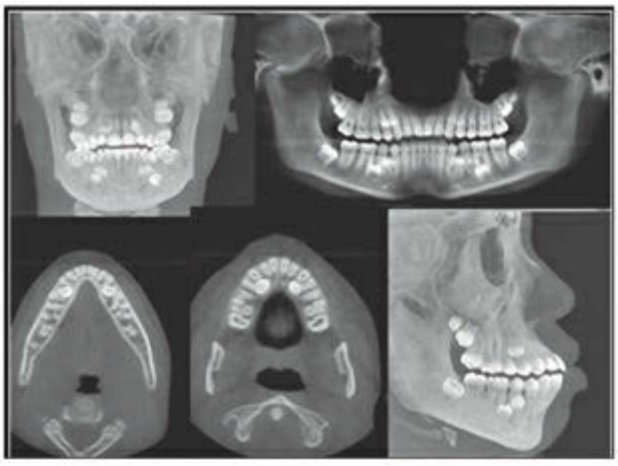


Fig 8-8 Three-dimensional views of a patient with multiple supernumerary teeth (oligodontia).

Saini et al[49] presented some cases where supernumerary teeth were recognized as incidental findings during routine radiographic examination in patients with an age range of 5 to 39 years. They showed that the initiation and development of supernumerary premolars are often delayed by a magnitude of years. Therefore, periodic examination is recommended to assess for late occurrence of this type of anomaly (see case 8-4).

In the presence of multiple supernumeraries, clinicians must assess for a possible association with other syndromes, such as Gardner syndrome and cleidocranial dysostosis.

Management

The majority of, if not all, supernumerary teeth are harmful to the occlusion and must be removed at the proper time. Management of supernumerary teeth depends on their type, number, and position; their effect or potential effect on adjacent teeth; the root development of adjacent teeth; the dental age of the patient; and the patient's occlusion.

Treatment options

Early detection and removal before any damage

The best option and the best type of management is to detect the supernumerary tooth early and remove it at the proper time, before any damage has happened. This includes cases in which the supernumerary tooth is located within unerupted dentition and has the potential to disturb normal occlusal development. Cozza et al[50] reported a case of a supplemental tooth with morphology similar to that of a lateral incisor, found in the canine region of the mandible of a 6-year-old boy. Treatment was extraction of the mandibular primary canine to facilitate spontaneous eruption of the supplemental tooth. Extraction was followed by spontaneous permanent canine eruption.

Another possibility is when supernumerary teeth erupt before or at the same time as the adjacent teeth. Detection is easy, and after careful clinical and radiographic evaluation, orthodontic management usually consists of extraction of the

supernumerary tooth followed by orthodontic therapy.

Early detection and observation for proper time of removal

In some situations, the practitioner may detect the presence of a supernumerary tooth early but decide to postpone surgery and continue observation. This course is prudent if the tooth is located high and close to the apex of developing roots and is not harmful to the dentition or if surgical removal might harm normal root development of the adjacent teeth.

Late detection and management of damaged situation

The third type of management arises when supernumeraries have not erupted and either have not been detected early or have been neglected. These are supernumeraries that prevent eruption of adjacent teeth, displace them, and damage tooth structure and occlusion. In this situation, careful clinical and radiographic evaluation and localization of the supernumerary is the first step in treatment planning.

Depending on the location and number of supernumerary teeth, the patient's occlusion, and the developmental stage of the adjacent teeth, a decision can be made to intervene surgically and follow up with the proper comprehensive orthodontic treatment. Several factors can be helpful to consider when determining whether to extract immediately or wait and monitor the condition.

The indications for immediate removal and orthodontic treatment include the following:

- When the supernumerary tooth is erupted and causing space problems for the adjacent teeth
- When the presence of the supernumerary tooth is preventing or delaying eruption or damaging adjacent teeth
- When the supernumerary tooth is displacing adjacent teeth
- When a pathologic lesion such as a cyst is evident
- When the supernumerary tooth is an obstacle to active orthodontic tooth movement

The indications for postponed extraction and continued monitoring include the following:

- When the supernumerary tooth is located extremely high in the apex area of permanent teeth and surgical removal can damage root development
- When the supernumerary tooth is very high and does not interfere with urgent interceptive tooth movement (the extraction can be postponed until after the initial phase of treatment)

Orthodontic considerations following supernumerary tooth removal

The following are important points to be considered before, during, or after removal of supernumerary teeth:

- If the supernumerary has not erupted and delayed eruption of permanent teeth is predicted, the space for unerupted teeth must be maintained.
- Long-delayed eruption and the forces of mastication might have caused formation of fibrous tissue or a bone barrier that will have to be removed to facilitate eruption.
- In many cases of neglected supernumeraries and delayed eruption of permanent teeth, the adjacent teeth often shift, creating a space deficiency for unerupted teeth. In these situations, besides removal of the supernumerary, space regaining is also recommended.
- If the permanent tooth or teeth are located high in the arch and/or displaced, it is necessary to bond an attachment for orthodontic traction at the time of extraction of the supernumerary tooth.

Case 8-1

A 15-year-old girl had a supplemental supernumerary lateral incisor on the maxillary right segment, resulting in crowding, disturbed occlusion in the right segment, overjet, and midline shift (Figs 8-9a and 8-9b). This type of problem can be considered an easy type of supernumerary tooth to manage; nevertheless, early recognition of an erupted supernumerary tooth and early treatment could prevent damage and even prevent the need for orthodontic treatment.

Treatment:
The treatment plan involved extraction of the supernumerary lateral incisor and only 2 × 6 bonding of the maxillary and mandibular arches to correct anterior crowding and the maxillary midline (Figs 8-9c and 8-9d).

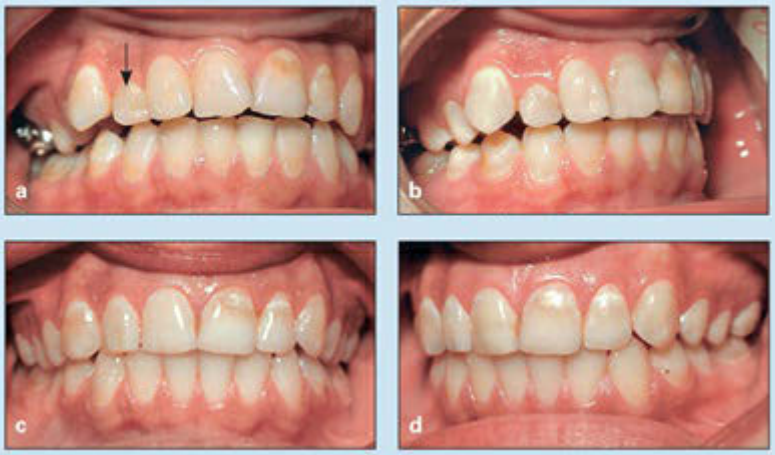


Fig 8-9 Treatment of a 15-year-old girl with a supplemental lateral incisor causing malocclusion, crowding, and a midline shift. *(a and b)* Maxillary right supplemental supernumerary tooth *(arrow). (c and d)* Posttreatment result after extraction of the supernumerary tooth and 2 × 6 bonding of maxillary and mandibular arches to eliminate crowding and correct the maxillary midline.

Case 8-2

A 14-year, 6-month-old girl complained that her maxillary permanent central incisors had not erupted. Her orthodontic problems included high-positioned, impacted permanent central incisors. The impaction was the result of negligence, as the primary incisors and two mesiodentes were allowed to remain for a long period of time. The other consequence was inadequate space for eruption of the permanent central incisors because of mesial tipping of lateral incisors after canine eruption.

Two panoramic radiographs were taken by her dentist before referral for orthodontic treatment. The initial radiograph (Fig 8-10a) shows the presence of two mesiodentes and both primary central incisors. The second panoramic radiograph (Fig 8-10b) was taken 1 year after extraction of both mesiodentes and the primary central incisors at the time of referral.

When supernumerary teeth are detected, extraction and space maintenance of the adjacent teeth are recommended in the majority of situations, except in rare cases where they are positioned close to developing roots and observation is an option.

The patient also had severe maxillary and mandibular dental protrusion and moderate mandibular crowding (Figs 8-10c to 8-10e). The patient's lateral cephalometric radiograph revealed severe maxillomandibuar dental protrusion and a steep mandibular plane (Fig 8-10f).

Treatment:

Because of the space loss and severe maxillary and mandibular anterior dental protrusion and crowding, the treatment plan called for extraction of four first premolars. After canine retraction and space opening for the central incisors, both maxillary incisors received a chain attachment for orthodontic traction (Figs 8-10g).

Total treatment time in this case was more than 3 years (Figs 8-10h to 8-10l). Early extraction of the primary incisors and space maintenance might have prevented the damage and drastically shortened the treatment time.

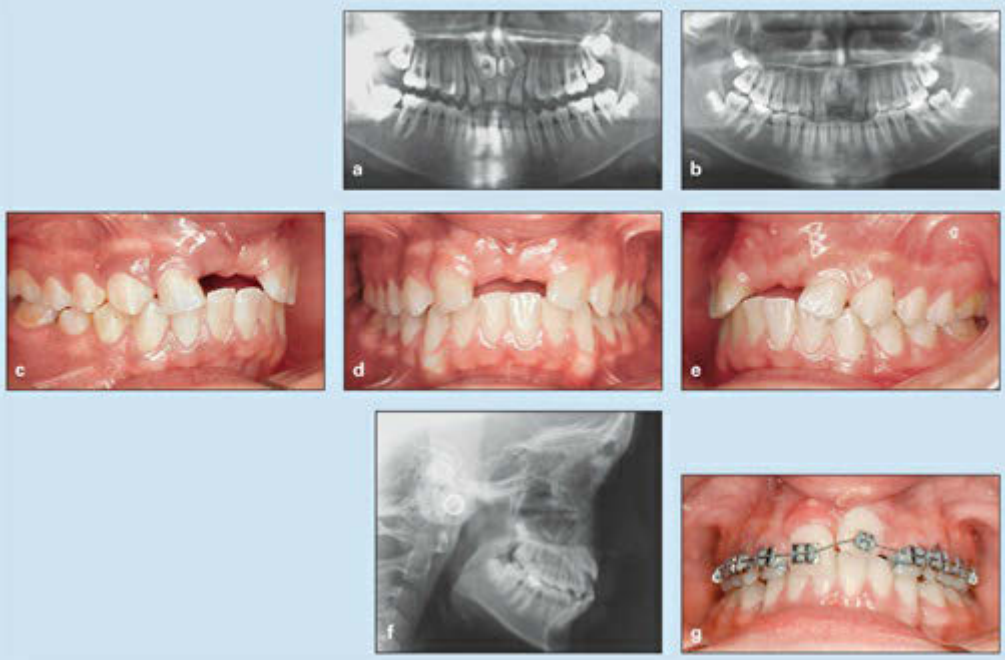


Fig 8-10 Treatment of a 14-year, 6-month-old girl with unerupted maxillary central incisors. *(a)* Panoramic radiograph taken by a dentist 2 years before orthodontic referral. *(b)* Panoramic radiograph taken after extraction of both mesiodentes and before orthodontic treatment. *(c to e)* Pretreatment occlusion. *(f)* Pretreatment lateral cephalometric radiograph revealing anterior crowding and severe dental protrusion. *(g)* Orthodontic traction of the central incisors.

Fig 8-10 *(cont) (h to j)* Posttreatment occlusion. *(k)* Posttreatment panoramic radiograph. *(l)* Posttreatment cephalometric radiograph.

Case 8-3

A 13-year, 7-month-old girl exhibited Class I minor anterior crowding and a buccally tipped maxillary right lateral incisor crown. Asymmetric eruption is evident. The permanent left canine and both premolars are completely erupted, while the maxillary primary right canine and first molar are still present. No canine bulge is present. The maxillary right canine and first premolar are deeply impacted

because of a complex odontoma (Figs 8-11a to 8-11d). Radiographs revealed the odontoma and damage to the occlusion (Fig 8-11e to 8-11h).

The position of the impacted canine over the apex of the maxillary right central and lateral incisors caused resorption of the central and lateral incisor roots.

Treatment:

The treatment plan included removal of the odontoma and chain attachment to both the impacted canine and premolar (see chapter 10 for more details). Because of the size and complexity of the odontoma, this patient had to undergo two surgeries. Figure 8-11i shows the chain attachments before traction. Figures 8-11j to 8-11m show the posttreatment occlusion.

Careful observation and periodic radiograph monitoring during the transitional dentition of this patient could have reduced the severity of her problems and the damage to the maxillary incisor roots.

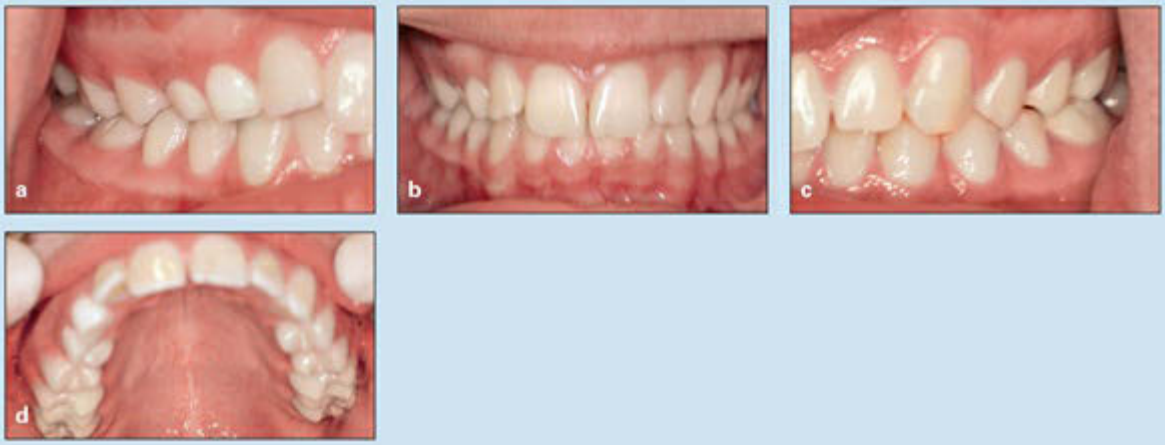


Fig 8-11 *(a to d)* Treatment of a 13-year, 7-month-old girl with asymmetric eruption.

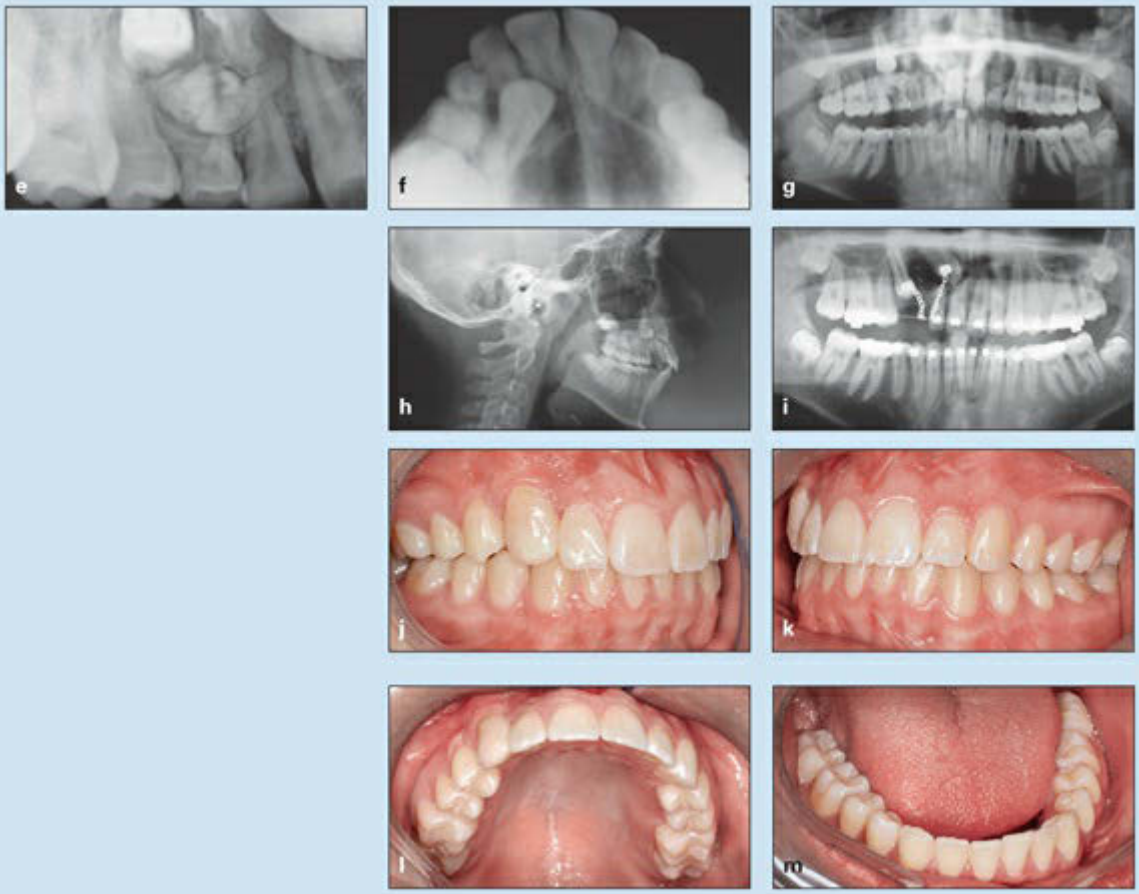


Fig 8-11 *(cont) (e)* Pretreatment periapical radiograph showing the odontoma and impacted maxillary right canine and first premolar. *(f)* Pretreatment occlusal radiograph showing the damage to the occlusion. *(g)* Pretreatment panoramic radiograph. *(h)* Pretreatment cephalometric radiograph. *(i)* Panoramic radiograph showing chain attachment before traction. Note the damage to the maxillary right central and lateral incisors from the odontoma and canine displacement. *(j to m)* Posttreatment occlusion.

Late development of new supernumerary teeth

Another important point to consider in the treatment of patients with hyperdontia, especially in cases with multiple supernumerary teeth, is the phenomenon of supernumerary reoccurrence, or late development of new supernumerary teeth after removal of the initial supernumerary teeth and even after orthodontic treatment. Careful monitoring is therefore required even after the completion of treatment. The following case is an example of this condition.

Case 8-4

A 10-year, 6-month-old boy with a Class II division 1 malocclusion and impinging deep bite had two

mesiodentes preventing eruption of both maxillary central incisors (Figs 8-12a to 8-12d).

Treatment:
Because of maxillary anterior dental crowding, maxillary protrusion, and severe space deficiency for the maxillary central incisors, both maxillary first premolars and both supernumeraries were extracted (Figs 8-12e to 8-12g).

Figure 8-12h shows a panoramic radiograph taken 3 years after treatment, which revealed late development of five new supernumeraries. This panoramic radiograph clearly demonstrates the potential damage that a newly developed supernumerary can inflict on the permanent dentition.

Late development of new supernumeraries is possible after removal of the initial supernumerary tooth or teeth and even after orthodontic treatment. This is especially true in patients with multiple supernumerary teeth. This possibility means that patients with hyperdontia require careful radiographic monitoring, even after the completion of treatment.

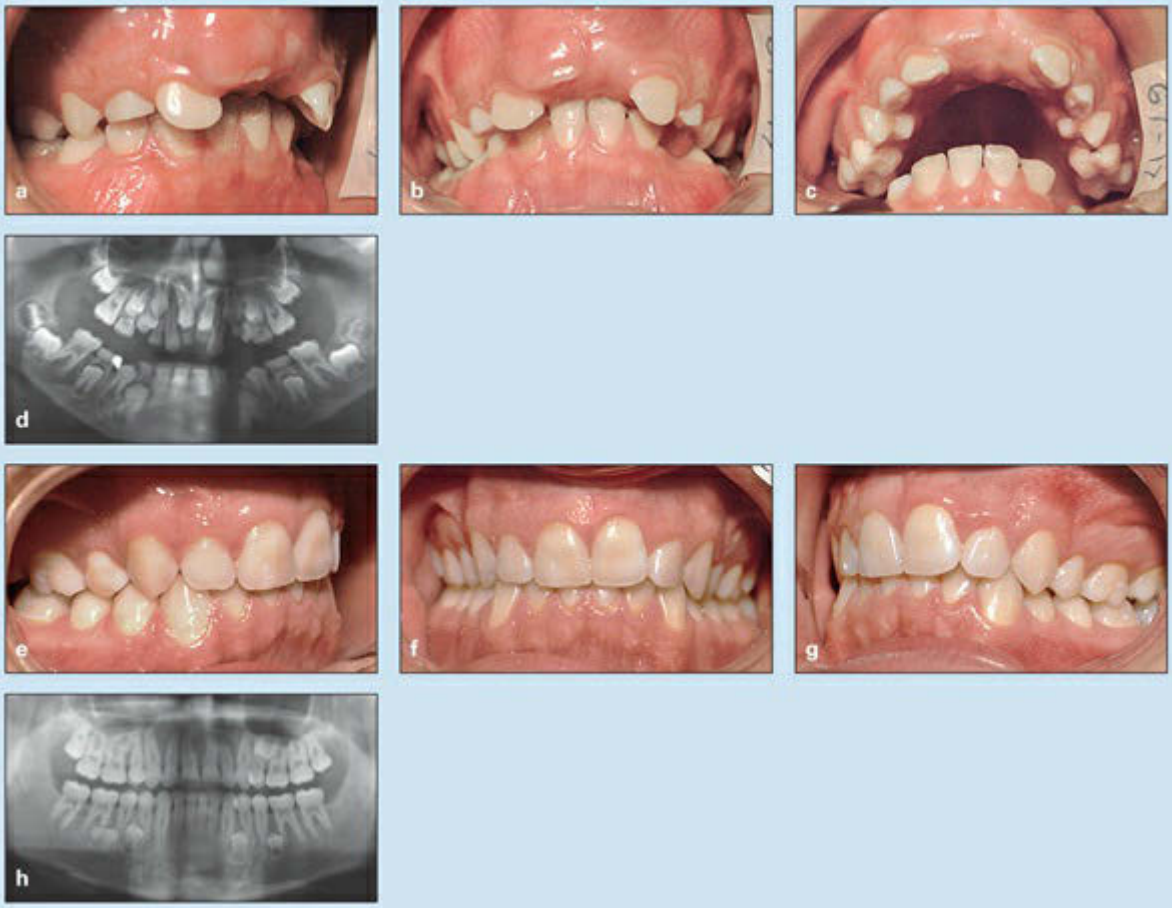


Fig 8-12 Treatment of a 10-year, 6-month old boy with a Class II division 1 impinging deep bite and recurring supernumerary tooth development. *(a to c)* Pretreatment occlusion. Two mesiodentes are preventing eruption of the central incisors. *(d)* Pretreatment panoramic radiograph. *(e to g)* Posttreatment occlusion. *(h)* Post-treatment panoramic radiograph demonstrating development of five new supernumerary parapremolars.

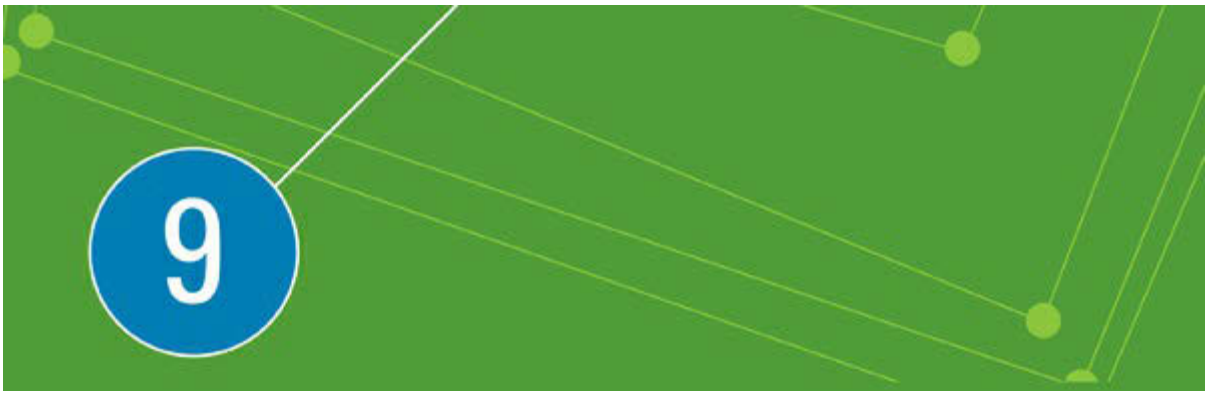
Summary

- Supernumerary teeth (hyperdontia) are teeth formed in excess of the normal dental complement.
- Prevalence reports from different ethnic groups vary widely, from 0.1% to 3.8%.
- The presence of supernumerary teeth is less common than congenital absence of teeth (hypodontia). Supernumerary teeth are seen twice as often in males as in females. Supernumerary teeth can also be found in the primary dentition but with less frequency.
- Depending on the shape and position of the teeth in the dental arch, several kinds of supernumeraries have been distinguished. The most common is found in the maxillary incisor area (mesiodens).
- Supernumeraries can occur in multiple numbers; the site most commonly affected by multiple supernumeraries is the mandibular premolar region.
- Three theories for morphogenesis of this anomaly have been proposed: dichotomy theory, hyperactivity theory, and atavism theory.
- The suspected possible cause of this disturbance is the migration, proliferation, and differentiation of the neural crest cells and interaction between the epithelial and mesenchymal cells during the initiation stage of tooth development.
- Depending on the nature of the disturbance during initiation, the result may be hypodontia, hyperdontia, or formation of an odontoma or cyst.
- The presence of supernumerary teeth is always problematic for occlusal development. Depending on their number, location, and vicinity to adjacent teeth, supernumerary teeth can cause crowding, spacing, delayed or lack of eruption of adjacent teeth, impaction, and structural damage to roots, pulp, and supporting bone.
- Management of supernumerary teeth depends on their location and number and the stage of dental development. Almost all supernumeraries are harmful and must be removed. The best management is early detection and intervention to prevent further damage to the dentition and occlusion.
- The timing of extraction of a supernumerary tooth depends on its position and the probable damage it will cause. If it is erupted, preventing eruption of other teeth, or damaging adjacent teeth, it should be removed. If it is located high, close to the apex, extraction can be postponed until after root completion.
- New supernumeraries can occur even after completion of orthodontic treatment; therefore, follow-up radiographic evaluation is recommended.

References

1. Vadiati Saberi B, Shakoorpour A. Apert syndrome: Report of a case with emphasis on oral manifestations. J Dent (Tehran) 2011;8:90–95.
2. Bhat MA, Laway BA, Mantoo S, Choudry K, Kotwal S, Mir SA. Cleidocranial dysplasia: A rare cause of disproportionate severe short stature. Oman Med J 2012;27:408–410.
3. D'Alessandro G, Tagariello T, Piana G. Cleidocranial dysplasia: Etiology and stomatognathic and craniofacial abnormalities. Minerva Stomatol 2010;59:117–127.
4. Panjwani S, Bagewadi A, Keluskar V, Arora S. Gardner's syndrome. J Clin Imaging Sci 2011;1:65.
5. Yusof WZ. Non-syndromal multiple supernumerary teeth: Literature review. J Can Dent Assoc 1990;56:147–149.
6. Shah R, Boyd M. Studies of permanent tooth anomalies in 1,886 Canadian individuals. J Can Dent Assoc 1978;6:265– 268.
7. Boyne PJ. Supernumerary maxillary incisors. Oral Surg 1954;7:901–905.
8. Buenviaje TM, Rapp R. Dental anomalies in children: A clinical and radiographic survey. J Dent Child 1984;51:42– 46.
9. Wallfeldt A. Overtaliga tander: Overkakens incisivregeion. Odont Revy 1961;12:67–74.
10. Frome K, Dickert P, Silko K, Miller AS. Panoramic survey. Dent Hyg (Chic) 1977;51:208–210.
11. Schulze C. Incidence of supernumerary teeth. Stoma 1960;13:128–140.
12. Morris CR, Marano PD, Swimley DC, Runco JG. Abnormalities noted on panoramic radiographs. Oral Surg 1969;28:772–782.
13. Billberg B, Lind V. Medfodda antalsvariationer I permanenta dentitionen. Odont Revy 1965;16:259–272.
14. McKibben DR, Brearley IJ. Radiographic determination of the prevalence of selected dental anomalies in children. J Dent Child 1971;28:390–398.
15. Jarvinen S. Supernumerary and congenitally missing permanent upper anterior teeth in 7-year-old Finnish children. A radiographic study. Proc Finn Dent Soc 1976;72:99–102.
16. Locht S. Panoramic radiographic examination of 704 Danish children aged 9–10 years. Community Dent Oral Epidemiol 1980;8:375–380.
17. Clayton JM. Congenital dental anomalies occurring in 3,557 children. J Dent Child 1956;23:206–208.
18. Luten JR. The prevalence of supernumerary teeth in primary and mixed dentitions. J Dent Child 1967;34:246–353.
19. Parry RR, Iyer VS. Supernumerary teeth amongst orthodontic patients in India. Br Dent J 1961;111:257–258.
20. Lacoste L, Hirsch C, Frank R. Les inclusions dentaires surnumeraires chezl'enfant. Revue Fr Odonto-Stomat 1962;9:967–983.
21. Salcido-García JF, Ledesma-Montes C, Hernández-Flores F, Pérez D, Garcés-Ortíz M. Frequency of supernumerary teeth in Mexican population. Med Oral Patol Oral Cir Bucal 2004;9:403–409.
22. Bäckman B, Wahlin YB. Variations in number and morphology of permanent teeth in 7-year-old Swedish children. Int J Paediatr Dent 2001;11:11–17.
23. Neville BW, Damm D, Allen C, Bouquot J. Oral & Maxillofacial Pathology, ed 3. St Louis: Saunders, 2008.
24. Liu JF. Characteristics of premaxillary supernumerary teeth: A survey of 112 cases. ASDC J Dent Child 1995;62: 262–265.
25. Taylor GS. Characteristics of supernumerary teeth in the primary and permanent dentition. Dent Pract Dent Rec 1972;22:203–208.
26. Low T. Hypodontia and supernumerary tooth: Report of a case and its management. Br J Orthod 1977;4:187–190.
27. Rubenstein LK, Lindauer SJ, Isaacson RJ, Germane N. Development of supernumerary premolars in an orthodontic population. Oral Surg Oral Med Oral Pathol 1991;71:392– 395.

28. Sharma A. A rare non-syndrome case of concomitant multiple supernumerary teeth and partial anodontia. J Clin Pediatr Dent 2001;25:167–169.
29. Shapira Y, Kuftinec MM. Multiple supernumerary teeth: Report of two cases. Am J Dent 1989;2:28–30.
30. Becker A, Bimstein E, Shteyer A. Interdisciplinary treatment of multiple unerupted supernumerary teeth. Report of a case. Am J Orthod 1982;81:417–422.
31. Matsumoto M, Nakagawa Y, Sobue S, Ooshima T. Simultaneous presence of a congenitally missing premolar and supernumerary incisor in the same jaw: Report of case. ASDC J Dent Child 2001;68:63–66.
32. Ratna R. Numeric anomalies of teeth in concomitant hypodontia and hyperdontia. J Craniofac Genet Develop Biol 1988;8:245–251.
33. Satish V, Parabhadevi MC, Sharma R. Odontome: A brief overview. Int J Clin Dent 2011;4:177–185.
34. Serra-Serra G, Berini-Aytés L, Gay-Escoda C. Erupted odontomas: A report of three cases and review of the literature. Med Oral Patol Oral Cir Bucal 2009;14:E299–E303.
35. Tejasvi MLA, Babu BB. Erupted compound odontomas: A case report. J Dent Res Dent Clin Dent Prospects 2011;5:33–36.
36. Amado Cuesta S, Gargallo Albiol J, Berini Aytés L, Gay-Escoda C. Review of 61 cases of odontoma. Presentation of an erupted complex odontoma. Med Oral 2003;8:366–373.
37. Litonjua LA, Suresh L, Valderrama LS, Neiders ME. Erupted complex odontoma: A case report and literature review. Gen Dent 2004;52:248–251.
38. Vengal M, Arora H, Ghosh S, Pai KM. Large erupting complex odontoma: A case report. J Can Dent Assoc 2007;73:169–173.
39. Main DM. Epithelial jaw cysts: A clinico-pathological reappraisal. Br J Oral Surg 1970;8:114–125.
40. Shen WJ, Shen YM, Zha Z, et al. The clinical and pathologic study of embedded supernumerary teeth [in Chinese]. Shanghai Kou Qiang Yi Xue 2001;10:108–109,118.
41. Kaler LC. Prevalence of mesiodens in a pediatric Hispanic population. ASDC J Dent Child 1988;55:137–138.
42. Erkmen N, Ölmez S, Önerci M. Supernumerary tooth in the maxillary sinus: Case report. Aust Dent J 1998;43:385– 386.
43. Carver DD, Peterson S, Owens T. Intranasal teeth. Oral Surg Oral Med Oral Pathol 1990;70:804–805.
44. Spencer MG, Couldery MG. Nasal tooth. J Laryngol Otol 1985;99:1147–1150.
45. Gadalla GH. Mandibular incisor and canine ectopia. A case of two teeth erupted in the chin. Br Dent J 1987;163:236.
46. Gibson N. A late developing mandibular premolar supernumerary tooth. Aust Dent J 2001;46:51–52.
47. Chadwick SM, Kilpatrick NM. Late development of supernumerary teeth: A report of two cases. Int J Pediatr Dent 1993;3:205–210.
48. Poyton GH, Morgan GA, Crouch SA. Recurring supernumerary mandibular premolars. Report of a case of post-mature development. Oral Surg Oral Med Oral Pathol 1960;13:964–966.
49. Saini T, Keene JJ Jr, Whetten J. Radiographic diagnosis of supernumerary premolars: Case reviews. ASDC J Dent Child 2002;69:184–190.
50. Cozza P, Laganà G, Mucedero M. Early diagnosis and treatment of supplemental mandibular tooth: Report of a case. ASDC J Dent Child 2002;69:180–183.



Diagnosis and Management of Abnormal Frenum Attachments

An abnormal labial frenum attachment can cause many problems for the dentition, such as an abnormal midline diastema. A *midline diastema* is a gap between the maxillary central incisors; it can be a normal occurrence during transitory stages of the dentition (during the primary, early, and middle mixed dentitions). However, a diastema can also be considered as an abnormal separation of incisors, that is, as a malocclusion.

Clinicians must be able to differentiate between these two types of diastema and know when and how to manage the abnormal diastema. Figure 9-1 shows two diastemata, developed from two different etiologic factors: an abnormal diastema developed because of impinging deep bite and an abnormal diastema caused by abnormal frenum attachment. Many factors other than an abnormal frenum attachment can cause an abnormal diastema; ways to differentiate between these causes are presented in this chapter.

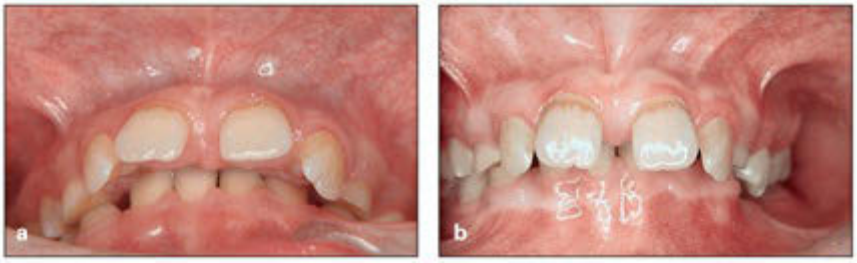


Fig 9-1 *(a)* Diastema caused by impinging deep bite. *(b)* Diastema caused by abnormal frenum attachment.

As Baum[1] stated, the midline diastema is usually a part of normal dental development during the transitional stage of the eruption of maxillary lateral incisors. It gradually disappears as the canines erupt, and the central incisors come into complete contact with each other.

In 1907, Angle[2] described the midline diastema as a common form of malocclusion distinguished by a space of varying degrees (1 to 5 mm in width) between the maxillary incisors. He suggested that it always presented an unpleasing appearance and noted that it was rarely seen between mandibular central incisors. He also suggested that the frenum was a cause of a midline diastema and outlined a method for its removal.

Broadbent[3] described the "ugly duckling stage" as a normal developmental phenomenon seen in most children. The permanent maxillary central incisors are flared laterally at the beginning of eruption because the unerupted lateral incisors constrain the roots of the central incisors. During this process, the medial erupting path of the maxillary lateral incisors, and later the maxillary canines, results in normal closure of the midline diastema. However, in some individuals, the diastema may not close spontaneously; an abnormal space remains between the incisors and causes other problems for adjacent teeth and for occlusion.

The presence of a diastema between the maxillary central incisors is considered an unacceptable esthetic and malocclusion problem by many adults. However, other individuals, including some actors, singers, and other celebrities, have a distinct diastema and do not mind.

In contrast to the maxillary diastema, which can be a part of the normal transitional stages of occlusal development, the mandibular diastema is not a normal growth characteristic. Because of the relatively larger size and the position of mandibular permanent incisors compared with the primary incisors, the mandibular permanent incisors often erupt with some amount of crowding.

Prevalence

A review of older literature indicates that the midline diastema was of concern to patients and was considered an unacceptable malocclusion, and clinicians were very interested in the etiology and treatment of this problem. In 1882, Farrer[4] described a method for the correction of diastema.

According to many epidemiologic investigations, the prevalence of midline diastema is high during the early mixed dentition and decreases between the ages of 9 and 12 years. In normal conditions, it closes after complete eruption of the permanent canines.[5–7]

In general, the midline diastema is more prevalent in females than in males. Richardson et al[8] studied the sex prevalence of diastema in 5,307 children between 6 and 14 years of age. They found that, at age 6 years, females showed a higher prevalence than males, but, at age 14 years, males had a higher prevalence.

Racial differences also exist; Lavelle[9] investigated the distribution of midline diastema in different human populations and reported that the prevalence is greater in Africans (West Africa) than in British whites or Chinese from Hong Kong and Malaysia.

Horowitz[10] evaluated the racial differences of midline diastema in a group of over 700 children, 397 black children and 321 white children, between the ages of 10 and 12 years. He found that 19% of the total sample of black children had a midline maxillary diastema of 2 mm or more compared with 8% of the while children.

Taylor[5] examined 516 children between 5.5 and 11.0 years of age and found that 66 of 68 children in the 5.5- to 6-year age group (97%) showed spacing between incisors. In the 6- to 7-year group, the prevalence of diastema was 87.7%, and in the 10- to 11-year group, 48.7% showed midline diastema. In another study of children aged 12 to 18 years, Weyman[11] reported a 7% occurrence.

Brunelle et al,[12] analyzing a national health and nutrition survey carried out in the United States, reported that a maxillary diastema of 2 mm or more was present in 19% of 8- to 11-year-olds, 6% of 12- to 17-year-olds, and 5% of adults aged 18 to 50 years.

Etiology of Midline Diastema

Initiation of any treatment for a midline diastema must be based on proper evaluation

of the extent, etiology, and pathogenesis of the diastema. The possible influence of the superior labial frenum in relation to the midline diastema has been of great interest to clinicians for many years. The presence of persistent diastema has often been blamed on an enlarged labial frenum. Sometimes, without a proper evaluation of the etiology of a particular diastema, frenectomy has been performed unnecessarily, although many other factors can be involved in this abnormality. Therefore, the cause of a diastema must be evaluated in regard to both the anatomical structure of frenum tissue and other causes of abnormal diastema.

Morphogenesis and structure of the frenum

The frenum can be the original cause of a diastema in only a small proportion of midline diastemata. According to Huang and Creath,[13] the superior labial frenum begins to form in the 10th week of gestation, and by the 3rd month in utero the morphology of the fetal frenum is similar to that of the abnormal frenum of postnatal life. It extends as a continuous band of tissue from the tuberculum on the inner side of the lip, over and across the alveolar ridge, to be inserted in the palatine papilla. Before birth, the lateral halves of the alveolar ridge unite, and the continuous band of tissue becomes totally enclosed by bone; it is divided into a palatal portion (palatine papilla) and a labial portion (superior labial frenum) by this closure.

The frenum of a newborn at the gum pad stage and before eruption of the dentition is a large, fleshy attachment with a wide origin. The tissue gradually narrows, attaching in the midline to the connective tissue of the internal maxillary suture and alveolar process. With the eruption of teeth and development of the alveolar ridge, the position of the frenum changes. As growth progresses, the frenum may atrophy and may assume a higher position or maintain its attachment to the papilla.

It has been suggested that a slight clefting of intercrestal bone can hold the teeth apart and cause an abnormal diastema.[14]

In a study of dogs, Picton and Moss[15] hemisectioned the mandibular first molar and disked the mesiodistal contact of other teeth in one side of the arch and produced artificial interdental spacing between the halves of the first molar on one side. After a few weeks, they found an enlarged space between the two parts of the sectioned tooth. One half moved mesially, and the other half moved distally because the transseptal fibers were not present between the roots of the divided molar, while other created spaces were spontaneously closed. They concluded that the transseptal fibers are one of the principle factors that bring about and maintain the interstitial

mesiodistal contacts of the teeth in each arch.

Stubley[16] compared the transseptal fibers to an orthodontic chain used for interdental space closure. He stated that transseptal fibers do not contain elastic tissue; they are made of collagen, and the motive power that they exert derives from tiny coils into which they contract as they mature; the force generated varies from individual to individual.

Stubley[16] described persistent midline diastema where interdental fibers are characterized by their strength and a wide space develops in the maxillary midline. In the absence of any pathologic condition, the underlying cause is congenital and is due to the presence of a persistent midline suture that interferes with arrangement of the transseptal fibers. Instead of passing directly across the interdental space from one tooth to the other, the fibers turn upward at a right angle from the mesial aspect of each central incisor to enter the suture. Such an arrangement is like a break in the transseptal fiber chain, and the distal transseptal fibers of developing central incisors can move them apart. This fiber pattern could account for difficulty in the spontaneous diastema closures.

Martin and Jones[17] examined 17 consecutive individuals with various forms of holoprosencephaly at autopsy or during clinical evaluation and found that 88% of the patients were missing the superior labial frenum, regardless of the severity of holoprosencephaly or other associated craniofacial defects. *Holoprosencephaly* is a cephalic disorder in which the prosencephalon (the forebrain of the embryo) fails to develop into two hemispheres. *Hox* genes, which guide placement of embryonic structures, fail to activate along the midline of the head, allowing structures that are normally paired on the left and right to merge. Martin and Jones[17] stated that absence of the frenum in individuals with holoprosencephaly provides evidence that the frenum has its embryonic origin in the medial nasal process.

Dean et al[18] stated that the maxillary labial frenum is composed of two layers of epithelium enclosing a loose vascular connective tissue. In some instances, the frenum may include muscle fibers that originate from the orbicularis oris muscle of the upper lip. The origin from the lip is often wide, but the tissue of the frenum itself narrows and is inserted in the midline into the outer layer of the periosteum and into connective tissue of the internal maxillary suture and alveolar process. The frenum may attach at variable locations in the attached gingival tissue some millimeters above the crest of the ridge, or on the ridge, or the fibers may pass between the central incisors and attach to the palatine papilla. Figure 9-2 shows an abnormal diastema caused by heavy and low attachment of an abnormal frenum.

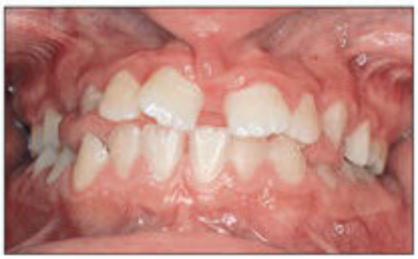


Fig 9-2 Abnormal diastema caused by heavy, low frenum attachment.

The frenum may be completely absent, or its existence may be a part of normal transition stage of dentition. With the completion of incisor and canine eruption, the gap spontaneously closes.

Other causes of midline diastema

Besides the anatomical structure of the frenum (size, position, and type of insertion), other causes of midline diastema must be considered before treatment planning and frenectomy are initiated. Possible therapeutic approaches include orthodontics, restorative dentistry, surgery, or various combinations of these. The ideal treatment should seek to close the diastema in question but also address the cause behind it.

During incisor transition, because permanent incisors are larger than primary incisors (incisor liability), some mechanisms interact to compensate for these width differences and enable normal development of occlusion. Mandibular incisors generally erupt with some degree of crowding, while maxillary incisors, because of their position and inclination, erupt with some degree of spacing. Depending on their size, this can be considered a normal transitional condition or can be a sign of an abnormal situation that deserves early detection and intervention before complete development.

In the normal transitional stage of maxillary incisors, a midline gap of up to 3 mm is normal. The mesial inclination of maxillary lateral incisors and their mesial force during eruption reduce or completely close this gap; eruption of the maxillary permanent canines with the same mechanism will completely close the remaining gap between central incisors. However, persistent midline diastema is also an ethnic norm for some races that have large dentoalveolar arches, such as African and Mediterranean groups.

To differentiate between normal transitional changes in the dentition that result in spacing in the maxillary permanent incisors from other factors that cause abnormal spacing, clinicians must carefully examine the patient's occlusion intraorally and

extraorally through clinical and radiographic evaluation. Incisor separations can result from one or more of the following factors:

- Lateral incisor hypodontia
- Microdontia of maxillary lateral incisors (peg-shaped lateral incisors)
- Presence of mesiodens or odontoma
- Deleterious oral habits
- Muscle imbalance (lip dysfunction)
- Impinging deep bite
- Anterior Bolton discrepancy
- Pathologic tooth migration

Lateral incisor hypodontia

One common cause of midline diastema is congenital absence of one or both maxillary lateral incisors (Fig 9-3). Sustained diastema after complete eruption of the central incisors is a diagnostic clue for early detection of this problem, which must be evaluated by radiograph. A remaining diastema with no midline shift is usually accompanied by absence of both lateral incisors, and a remaining diastema with a shift of midline is usually the consequence of one absent lateral incisor.

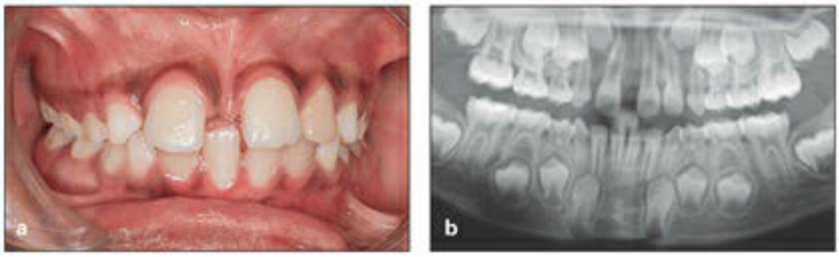


Fig 9-3 *(a and b)* Severe diastema caused by a congenitally missing maxillary right lateral incisor.

Depending on the type of occlusion and other conditions, treatment options are closing the diastema and using canine substitution or closing the diastema, aligning the anterior teeth, and replacing the missing teeth with conventional prostheses or implants (see chapter 7).

Microdontia of maxillary lateral incisors

Abnormal maxillary midline diastema can be the result of small incisors, that is, peg lateral incisors, which can be unilateral or bilateral (Fig 9-4).

Fig 9-4 Diastemata caused by missing teeth and microdontia of the central incisors.

Depending on the type of occlusion, the age of the patient, and the patient's desires, treatment options can be building up the small lateral incisor with composite resin, veneers, or crowns or extraction and space closure (see chapter 7).

Presence of mesiodens or odontoma

Another common cause of midline diastema is the presence of supernumerary teeth. The mesiodens is the most common type of supernumerary tooth (Fig 9-5). Early detection and diagnosis are not difficult if the incisor transition is carefully monitored. The presence of supernumerary teeth in the incisor area has some clinical signs that can help in early detection, including asymmetric or retarded tooth eruption, retained diastema after complete eruption of the lateral incisors, and rotation of the central incisors.

Fig 9-5 Diastema caused by mesiodens.

The treatment of choice depends on the patient's occlusion, dental age, and root formation of the incisors. Some treatment options include early detection and extraction of the supernumerary tooth and guidance of eruption and incisor alignment by segmental bonding (see chapter 8).

Deleterious oral habits

Another common cause of midline diastema or interdental spacing of incisors is abnormal habits, such as finger sucking, tongue thrust, and lip biting or lip dysfunction (Figs 9-6 and 9-7).

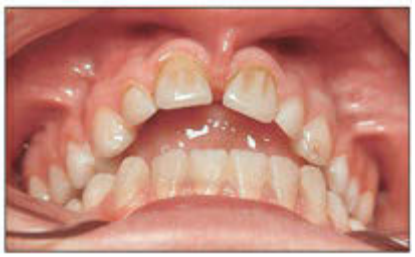


Fig 9-6 Diastema caused by thumb sucking.

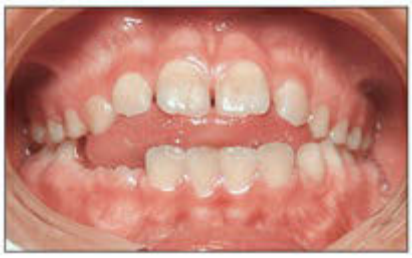


Fig 9-7 Diastema caused by tongue dysfunction.

The treatment of choice in this situation is early detection, habit control, and space closure (see chapter 6).

Muscle imbalance (lip dysfunction)

One of the most important factors influencing incisor relationships and maintaining normal occlusion is inner and outer muscle balance around the dental arches. Midline diastema can be caused by orofacial muscular imbalances, such as macroglossia, improper tongue posture and position, and flaccid lip muscles (Fig 9-8).

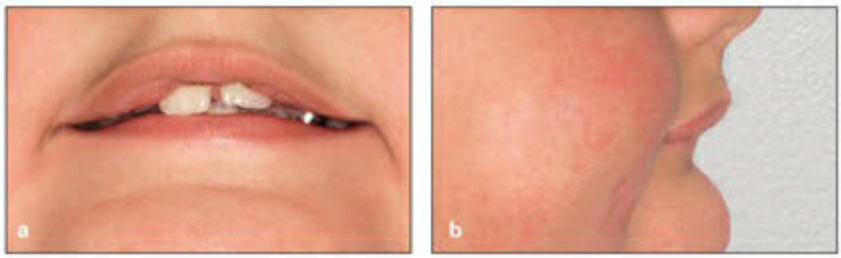


Fig 9-8 *(a and b)* Diastema caused by lip dysfunction.

Proper treatment of these conditions involves elimination of causative factors and space closure. Long-term stability depends on proper long-term retention.

Impinging deep bite

Deep bite malocclusions, especially Class II division 1 malocclusions with impinging bite caused by a severe curve of Spee and overeruption of mandibular incisors, is a common type of malocclusion that can cause incisor flaring and midline diastema.

The treatment of choice for this type of diastema is early intervention and correction of the curve of Spee, correction of deep bite, and then space closure and incisor retraction (see Fig 9-1a).

Anterior Bolton discrepancy

Another cause of midline diastema is an abnormal anterior ratio, which can be determined through the Bolton analysis.[19] This discrepancy results from the presence of relatively large mandibular incisors or small maxillary incisors in comparison to each other.

Space closure must be accompanied by a reduction in the size discrepancy between maxillary and mandibular incisors, either by enamel stripping or extraction of the bigger incisors or buildup of the smaller incisors (see chapter 5 on correction of incisor crowding).

Pathologic tooth migration

Another cause of incisor spacing and flaring, especially in the adult dentition, is development of a pathologic condition, such as cystic formation, bone loss due to periodontal problems, or some systemic condition that causes bone loss and tooth migration. Increased areas of inflammation cause bone loss, loss of attachment, and consequently migration of teeth from the pocket (Fig 9-9).

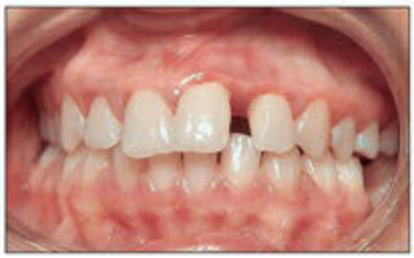


Fig 9-9 Diastema caused by pathologic tooth migration.

Management of this condition, especially in advanced situations, is very challenging and requires a multidisciplinary approach. Any orthodontic tooth movement and treatment must wait until a thorough evaluation has been completed and the cause has been eliminated.

Effects of Abnormal Frenum Attachment on Occlusion

An abnormal labial frenum, in addition to causing a maxillary midline diastema, can produce other undesirable clinical conditions:

- Abnormal frenum attachment can cause an abnormal space between all incisors as well as tooth displacement and rotation.
- An abnormal frenum attachment and a sustained diastema between central incisors can complicate the eruption of lateral incisors and canines, resulting in delayed eruption or complete absence of eruption, displacement, crossbite of lateral incisors, and/or canine impaction.
- Depending on their size and position, the abnormal frenum attachment may restrict movements of the lip or tongue and may interfere with speech and swallowing.
- Frenum tissue with a heavy band and low attachment can interfere with proper toothbrushing.
- Tight fiber attachment of frenum tissue, especially the mandibular labial frenum, may cause stretching of the labial and gingival tissues and tissue recession from the necks of the teeth.

Differential Diagnosis of Abnormal Frenum

Midline diastema is a common occurrence that can be seen in all races, sexes, and ages.

Because of its potential to have multiple etiologies, differential diagnosis must be based on a thorough medical and dental history and a complete clinical and paraclinical evaluation (radiograph and cast analysis if tooth size or arch size discrepancy is suspected). Several points should be investigated in evaluations of suspicious frenum attachment: hereditary background, age of the patient, local factors, and the size, position, and strain of the frenum tissue.

Hereditary background

Among the patients with abnormal frenum attachment whom the author has examined over the past 40 years of practice, almost all had some type of familial pattern of involvement, whether in parents, siblings, or other close relatives. For differential

diagnosis of sustained diastema, the author strongly recommends that the clinician look for any familial history of diastema.

In a follow-up evaluation of 96 consecutive cases, Shashua and Artun[20] assessed the relapse after orthodontic correction of maxillary midline diastema (ranging from 0.50 to 5.62 mm) 4 to 9 years after completion of active treatment. The only significant risk factors for relapse were pretreatment diastema size and presence of a family member with a similar condition.

Age of the patient

The presence of a midline diastema can be a normal transitional change of the dentition during the early and middle mixed dentitions. Such diastemata are self-corrected after complete eruption of all incisors and canines, provided that all other environmental conditions are normal.

Local factors

Any local obstacle that can interfere with normal diastema closure must be thoroughly assessed clinically and paraclinically. These obstacles include missing teeth, supernumerary teeth, microdontia, deleterious habits, muscle dysfunction, impinging deep bite, and pathologic conditions (periodontal involvement or cyst formation).

Clinical and radiographic signs of true abnormal frenum

The size, position, and strain of the frenum tissue are another important area of examination. Certain signs related to these aspects can help in the detection and confirmation of true abnormal frenum. These signs are the size, position, and location of tissue attachment; the shape of the midline gap; blanching of the papilla; and the presence of a bony cleft on radiographs.

Size, shape, and location of tissue attachment

An abnormal frenum is usually large, and its location is low, close to the ridge, between the central incisors, or even extended to the palatine papilla. In severe

conditions, tied frenum, especially tongue-tie of the lingual frenum, may negatively affect the patient's speech.

Shape of midline gap

A common shape of midline gap or diastema is a reverse V-shaped space between the central incisors, wider toward the incisal edge. This is sometimes accompanied by mesial rotation of the central incisors (Fig 9-10).

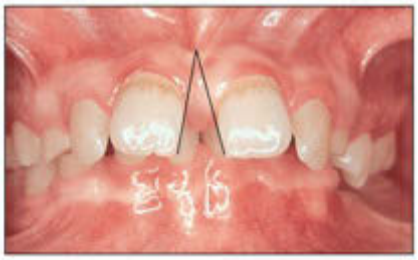


Fig 9-10 Reverse V-shaped separation between central incisors, a common sign of abnormal frenum attachment.

Blanching test

A simple diagnostic test for an abnormal frenum involves stretching the upper lip up and forward intermittently. If a heavy band of tissue is attached to the palatine papilla, the force exerted produces blanching of the papilla. This is called the *blanching test* (Fig 9-11).

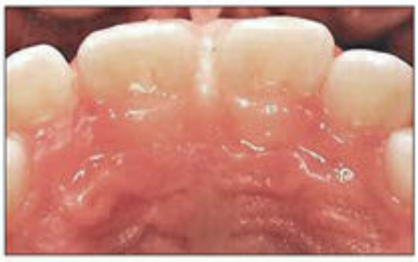


Fig 9-11 Local anemia or blanching of the papilla in response to stretching of the lip, another sign of abnormal frenum attachment.

Periapical radiograph

Evaluation of the midline and the condition of the alveolar suture is a helpful means of diagnosis. Midline bony clefts have been proposed as an etiologic factor for abnormal diastemata,[14] which often exhibit a V-shaped midline bony cleft and open suture (Fig 9-12).

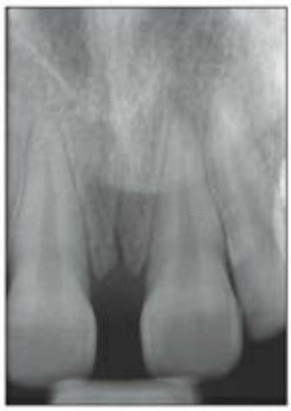


Fig 9-12 Sutural opening in the alveolar crest, a sign of frenum tissue insertion.

Management of Abnormal Frenum

Depending on the etiologic factor or factors, abnormal midline diastemata can be treated with various options that have been discussed in previous chapters (such as abnormal habits [see chapter 6], hypodontia [see chapter 7], and supernumerary teeth [see chapter 8]). This section focuses on the management of abnormal diastema caused by abnormal frenum attachment. Management varies depending on the stage of dentition.

Management in adults

Management of diastema in adults varies, depending on the type of malocclusion, etiologic factors, the health of the periodontium, and the patient's desires. There are four basic choices for treating diastemata in adults:

1. If the patient has an acceptable occlusion and does not want to undergo comprehensive orthodontic treatment, he or she may opt to forego treatment. Some people are fine with diastemata, while others find them unappealing and wish to have them changed or closed.
2. If the patient has good occlusion and a mild diastema but is not willing to undergo orthodontic treatment, the second choice is to close the space through porcelain veneers or composite resin buildup.
3. Another choice is placement of a conventional crown and fixed partial denture prosthesis. Depending on the size of the incisors and the width of the diastema, space closure can be accomplished by crown placement only on the central

incisors or by distribution of space closure among all incisors.

4. The most complicated treatment plan includes frenectomy and proper orthodontic treatment, which can be accomplished with comprehensive orthodontic treatment or 2 × 6 bonding. Retention is critical after most frenum corrections, especially in adults, and requires extended or permanent retention.

Management during the mixed dentition

Intervention for an abnormal frenum is easier during the early mixed dentition, and the results are more stable. Also, young patients are more willing to accept orthodontic options, and often parents find a diastema to be unacceptable in their children and do not like the appearance.

Early interception not only can solve the current problems for the child but also can prevent many negative consequences for the occlusion. Management options for an abnormal diastema during the early and middle mixed dentitions, whether it is due to abnormal frenum attachment or other etiologic factors, vary greatly. The timing of intervention is important for achieving satisfactory results. Timing depends on the initial cause, the patient's age, and the stage of the dentition.

Generally there is controversy among practitioners about the timing of frenum removal; some advocate that treatment be delayed until full eruption of all maxillary permanent anterior teeth, including the canines. This rule may be considered correct, provided that the frenum is not disturbing occlusal transition and the patient does not mind abnormal diastema and needs comprehensive orthodontic treatment.

However, there are many situations when the presence of abnormal frenum tissue during early stages of the dentition disturbs the normal development of occlusion. It may prevent normal eruption of adjacent teeth; disturb the normal sequence of transition; cause crowding, rotation, or displacement of adjacent teeth; and even cause malocclusion such as crossbite or canine impaction.

The foundations of permanent occlusion are the eruption of the permanent first molars in the posterior region and the incisors in the anterior region. Any disturbances anywhere in this structure can interfere with the normal development of the whole structure. Angle[2] based his idea of normal occlusion on the position of the maxillary permanent first molar as "the key of occlusion."

The question facing clinicians is whether they should neglect the conditions that they observe in the anterior region of the occlusion that are causing a sustained diastema, claiming that they can correct it at the later age. The answer is "No";

instead, they should eliminate the cause and maintain the integrity of the anterior segment of the arch at a natural time, to enhance the normal development of the rest of the arch.

Removal of any etiologic factors other than the frenum that are causing diastema and disturbing the eruption of adjacent teeth usually can be initiated on diagnosis and after sufficient development of the central incisors; however, tooth movement is usually deferred until eruption of the permanent lateral incisors or canines. There are different management options to choose from, depending on the severity of the abnormal diastema and its effects (eg, interference in eruption or interruption of normal transitional changes).

Early treatment procedures

The design of the treatment plan and the timing of intervention depend on the severity of the frenum problems, including size, type of insertion, the potential for affecting adjacent teeth, and patient occlusion.

Delaying treatment until complete eruption of all maxillary incisors. If the presence of an abnormal frenum is confirmed but there is no evidence of severe, disturbing effects on the occlusion and adjacent teeth, the treatment should be postponed until eruption of all maxillary incisors is completed. Treatment procedures are initiated with 2 × 4 bonding for leveling and correction of rotation and alignment of all incisors. After this phase, the patient is referred for frenectomy before diastema closure; closure of the diastema must begin the same day as frenectomy to prevent regeneration of tissue and collagen fibers in the suture areas.

Urgent intervention, frenectomy, and diastema closure. Gap closure is urgent when the presence of the diastema interferes with normal eruption or the transitional stage of dentition. For example, if the presence of heavy frenum tissue interferes with normal eruption of the maxillary lateral incisors, the intervention and diastema closure can be started even before eruption of the lateral incisors. This is accomplished with a removable appliance or by attaching two brackets on central incisors with a sectional arch to facilitate lateral incisor eruption. In this situation, if the patient's molar relationships, overjet, overbite, and midline relationship are normal and no other treatment is needed, all maxillary incisors must be retained until eruption of the canines.

Figure 9-13 shows the normal pattern of maxillary incisor transition (see chapter 2). During this process, any factor that interferes with normal, gradual closure of the

midline gap can affect normal eruption and transition of adjacent teeth, causing problems such as lateral crowding, rotation, lack of eruption, or crossbite. Eruption of canines also can be disturbed if the midline diastema remains open.

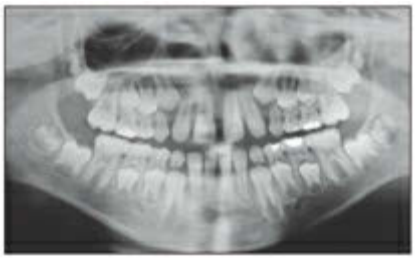


Fig 9-13 Normal pattern of maxillary incisor and canine eruption.

Figure 9-14 illustrates an abnormal frenum attachment that caused a diastema that remained mostly unchanged for 15 months, before and after eruption of the lateral incisors. The best treatment option in this case would be early intervention: frenectomy and diastema closure.

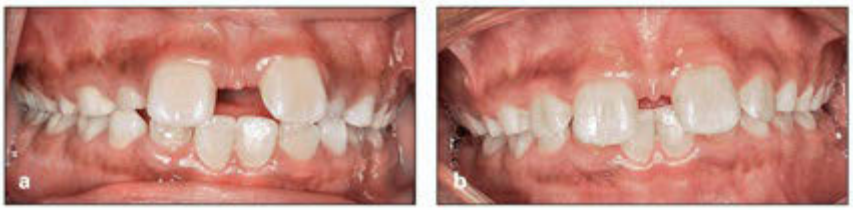


Fig 9-14 Wide diastema that has persisted even after eruption of both lateral incisors because of abnormal frenum attachment. (a) Pre-eruption. (b) Posteruption.

Figure 9-15 shows an abnormal frenum that caused problems for the maxillary canines and had the potential to result in lateral incisor root resorption and canine impaction. The patient was treated with frenectomy, diastema closure, and guidance of canine eruption.

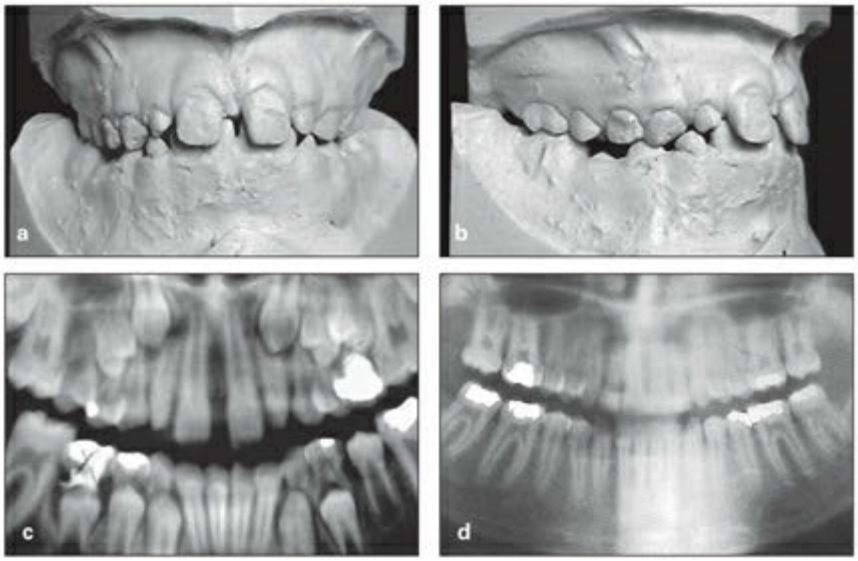


Fig 9-15 *(a to c)* Frenum that is preventing diastema closure and with the potential to cause canine impaction and lateral root resorption. *(d)* Panoramic radiograph after fre-nectomy and diastema closure.

Two-phase management. When other problems such as abnormal habits, overjet, and overbite are associated with a midline diastema and abnormal frenum and further treatment will be needed in the permanent dentition, the procedure can be managed in two phases. Phase 1 includes correction of the diastema and incisor alignment. After an interim phase of continued monitoring, the second phase of treatment is begun. However, not all diastemata can be treated the same way, in terms of modality or timing; careful pretreatment evaluation, proper orthodontic-surgical planning, and proper intervention are needed to achieve an ideal solution.

Sometimes local factors other than the frenum, such as abnormal habits, absence of the lateral incisors, or a mesiodens, are causing an abnormal diastema; however, if the frenum attachment is low in the ridge area, the appearance can be misleading because it looks like an abnormal frenum. Frenectomy is not recommended in these situations; elimination of the cause and orthodontic space closure are usually followed by spontaneous atrophy of the frenum tissue (see case 9-5).

Management during the primary dentition

Presence of an abnormal frenum is not as common in the primary dentition as in the mixed dentition, but there are patients with abnormal frenum attachment even during the primary dentition who may show severe diastema and incisor displacement. These are usually patients with severe frenum problems, but frenectomy during the primary dentition is not recommended, and the treatment must be postponed to the

mixed dentition period.

The author's clinical observation is that all cases of abnormal frenum attachment during the primary dentition have a hereditary background; children with this trait will show abnormal frenum attachment during the permanent dentition too. Therefore, frenectomy during the primary stage has no benefits for these children. Recurrence of the frenum in the permanent dentition is inevitable and will necessitate another phase of treatment (see case 9-3).

Management in infants

Kotlow[21] reported the presence of labial dental caries on the maxillary anterior teeth in breastfeeding infants due to abnormal attachment of the maxillary frenum. He added that abnormal frenum attachment may interfere with proper latching of an infant to a mother's breast and impede lip flanging, thereby preventing nursing. He recommended diode laser surgery for these infants to prevent this type of caries.

In contrast, Dean et al[18] stated that the effectiveness of surgical reduction of the labial frenum in infants cannot be substantiated.

Frenectomy

Despite advances in diagnostic methods, orthodontic treatment, and surgical techniques, some cases of relapse after diastema treatment have been reported. A careful pretreatment assessment is crucial, and the interaction of orthodontic treatment with the anticipated surgical procedure must be analyzed in treatment planning. For optimum results and stability, besides the frenum attachment and diastema, other aspects of the dentoskeletal malocclusion and perioral neuromuscular balance and function must also be considered; these include deleterious habits, tongue function, lip tonicity, and lip competency.

For the surgical technique, Dean et al[18] suggested removal of a wedge-shaped section of the tissue between the central incisors on either side of the frenum, extending the incision palatally to the nasal palatine papilla and to the depth of the underlying bone. The free marginal tissue on the mesial side of the central incisors should not be disturbed. The wedge of tissue can be picked up with tissue forceps and excised with tissue shears at an area close enough to the origin of the frenum to provide a desirable cosmetic effect. Sutures are placed inside the lip to approximate

the free margins of the tissue. It is generally not necessary to suture or pack the tissue between the incisors.

Recently, application of lasers in general dentistry and especially in soft tissue surgery has been advocated by many practitioners. Haytac and Ozcelik[22] compared the degree of postoperative pain, including discomfort and functional complications (eating and speech), in 40 patients requiring frenectomy. Patients were assigned randomly to a conventional surgical technique or surgery with a carbon dioxide laser. They concluded that carbon dioxide laser treatment is safe for frenectomy and that patients treated with the laser experience less postoperative pain and better function than do those treated with the scalpel technique.

Olivi et al[23] clinically evaluated the efficacy of an erbium, chromium-doped yttrium-scandium-gallium-garnet laser in removing the labial frenum in 143 adolescent and prepubescent patients. Patients returned for recall, and surgical areas were checked for adverse events, recurrence of frenum, functional complications, and patient acceptance. They reported that only two adolescent patients exhibited recurrence of the frenum. None of the three prepubescent patients needed additional intervention at follow-up, 3 years after treatment.

Kotlow[24] stated that traditional methods of oral surgery by scalpels or electrosurgery may place pediatric patients at risk during the use of a general anesthetic for an elective procedure and may produce significant postoperative discomfort; these procedures also require sutures and prolonged healing. Laser surgery provides a simple and safe in-office alternative for children, reducing the chances of infection, swelling, discomfort, and scarring.

In cases of deep attachment of frenum tissue in the midline suture, complete removal of the tissue in the suture and immediate space closure are necessary to prevent any tissue regeneration (Fig 9-16).

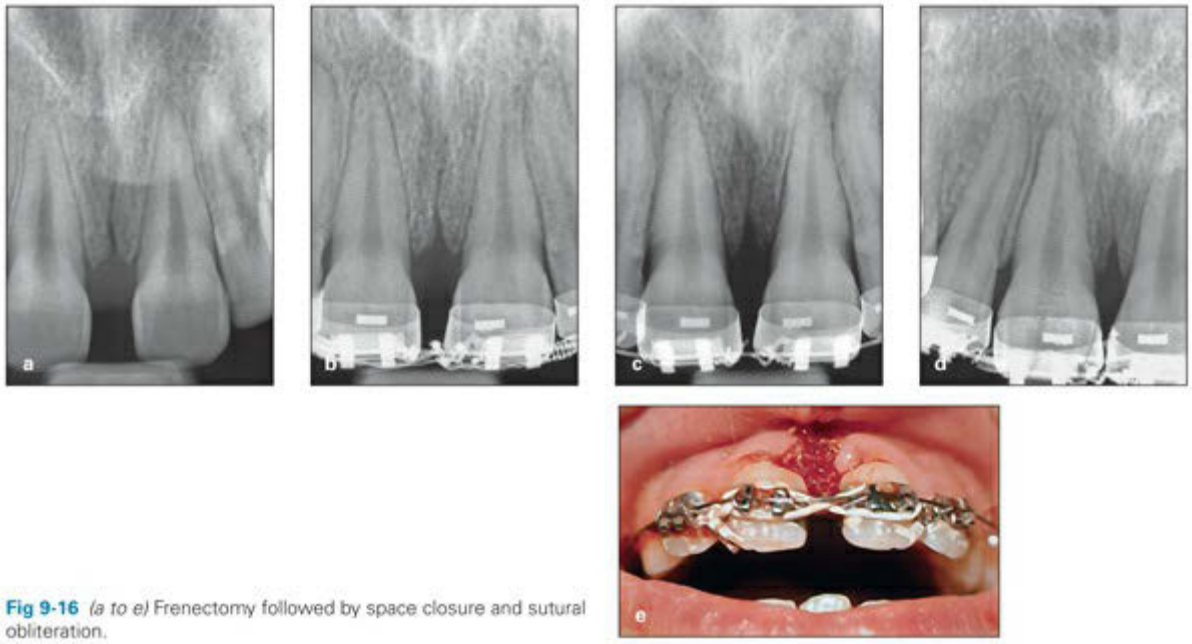


Fig 9-16 *(a to e)* Frenectomy followed by space closure and sutural obliteration.

Fig 9-16 *(a to e)* Frenectomy followed by space closure and sutural obliteration.

Ankyloglossia (Tongue-Tie)

The lingual frenum located under the tongue is a normal part of the oral cavity. It is a thin, vertical fold of tissue attached to the undersurface of the tongue and to the floor of the mouth. Ankyloglossia (tongue-tie), in which shortness and consolidation of the lingual frenum tissue reduce tongue mobility, is an uncommon oral anomaly that can cause some difficulty for tongue function.

In many children, ankyloglossia might be asymptomatic; in some the condition may resolve spontaneously, or affected children may learn to compensate adequately for their decreased lingual mobility. There are also children who may benefit from surgical intervention for tongue-tie.

Signs of ankyloglossia

A person with tongue-tie usually cannot protrude the tongue beyond the edges of the mandibular incisors or touch the palatine papilla of the maxillary incisors. This limitation has the potential to disturb speech, normal deglutition, and suckling and can have an effect on the dentition, such as increasing mandibular interincisal spacing (Fig 9-17).

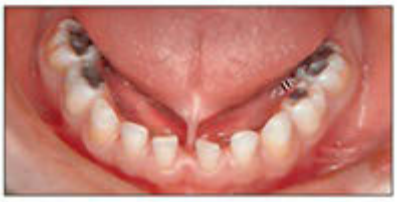


Fig 9-17 Ankyloglossia, or tongue-tie, a rare lingual frenum attachment. The condition can affect the dentition and tongue movement.

Management of ankyloglossia

The literature includes several reports, some exaggeration of functional complications in patients having this anomaly, and many different proposals for surgical therapy.[25,26] Management of lingual frenum has been mostly based on the results of clinical examination; because there is no distinct border between normal and tied mandibular frenums, the dental practitioner is often unsure of an appropriate course of action for a patient with suspected ankyloglossia.

Wright[27] retrospectively reviewed the records of 158 patients who had undergone a frenectomy and concluded that the operation requires general anesthesia, except in older children, for whom local anesthesia is appropriate. He added that speech difficulties related to tongue-tie are overrated and mechanical problems are underestimated.

Indications for lingual frenotomy include articulation difficulties confirmed by a speech pathologist and mechanical limitations such as inability to lick the lips or perform internal oral toilet. Limitations in the tongue's ability to sweep the teeth and spread saliva can cause dental caries and periodontal health problems. There may be rare instances in infants where problems with feeding and suction can be helped by frenectomy.

Fiorotti et al[28] indicated that a short lingual frenum can result in incorrect speech sounds, dentofacial alterations, and acquired functional adaptations. They assessed 15 patients who underwent frenectomy utilizing the carbon dioxide laser. The researchers indicated that the technique is safe, effective, and perfect for use in young children and can be performed in an outpatient unit. They also contended that, when frenectomy is indicated, it should be performed as early as possible to prevent functional alterations.

Suter and Bornstein[29] searched the literature and found 64 articles regarding the classifications of tongue-tie and many other proposed criteria. They concluded that lack of an accepted definition and classification of ankyloglossia makes

comparisons between studies almost impossible. No conclusive suggestions regarding the method of choice can be made; therefore, it remains controversial which tongue-ties should be surgically removed and which can be left to observation. Many children with the tied frenum compensate well and have normal-sounding speech.

Each individual with ankyloglossia requires a thorough individual assessment and evaluation to ensure that the most appropriate management strategies are selected. Parents should be educated about the possible long-term effects of tongue-tie.

Case 9-1

A 10-year-old boy presented with a hereditary abnormal frenum attachment, a Class I malocclusion, and a wide diastema that persisted even after eruption of the lateral incisors (Figs 9-18a to 9-18d). He exhibited deep bite and maxillary incisor flaring but good mandibular dentition. Figures 9-18e and 9-18f show the diastemata of his mother and his aunt.

Treatment:
Treatment was designed to include one phase of active treatment followed by a period of observation. First 2 × 4 bonding was used in conjunction with an anterior bite plate to align the incisors, and then a utility arch and bite plate were used to reduce overbite and flaring (Fig 9-18g). After these goals were accomplished, the second step was frenectomy (Fig 9-18h). The same day, space closure by elastic ligature was applied (Fig 9-18i). The final active part of treatment was incisor retraction and overbite control.

The final step was placement of a Hawley retainer to retain the anterior changes and control overbite until completion of permanent tooth eruption. Figures 9-18j to 9-18m show the posttreatment occlusion after 2 years of observation and eruption of all permanent teeth. This case represented interceptive treatment, using only 2 × 4 maxillary bonding.

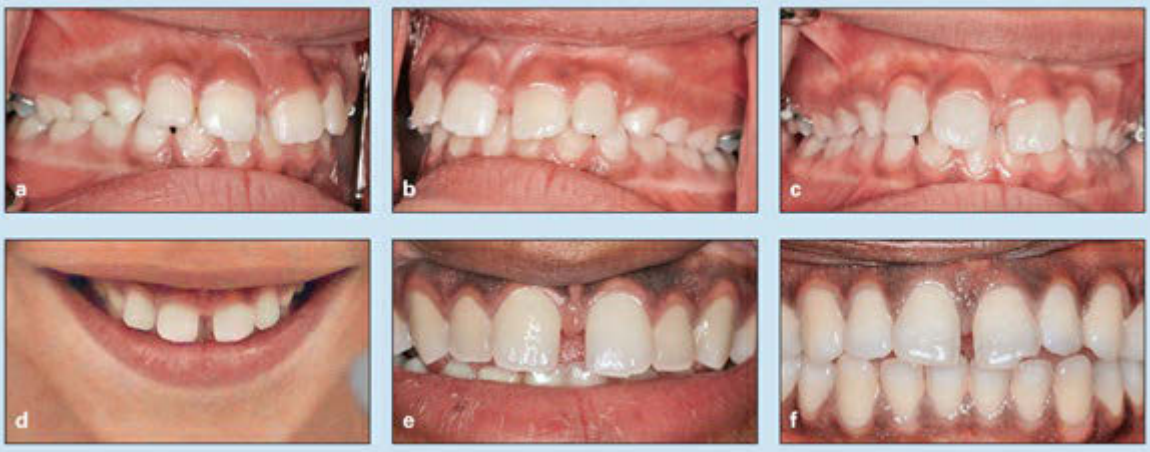


Fig 9-18 Treatment of a 10-year-old boy with a hereditary abnormal frenum attachment resulting in a wide, persistent diastema, deep bite, and flaring incisors. *(a to d)* Pretreatment occlusion. *(e and f)* Diastemata exhibited by the patient's mother and aunt.

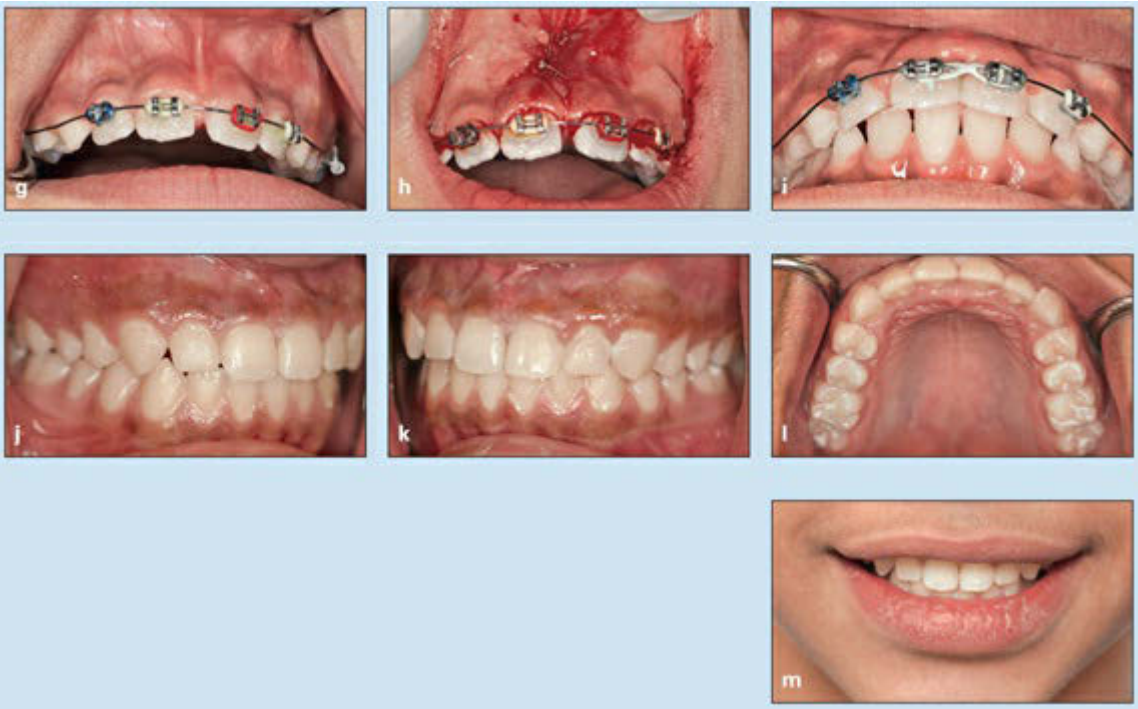


Fig 9-18 *(cont) (g)* After incisor alignment and overbite reduction but before frenectomy. *(h)* Frenectomy and initiation of space closure. *(i)* Space closure after 1 week. *(j to m)* Posttreatment occlusion.

Case 9-2

A 10-year, 8-month-old girl exhibited a Class II division 1 malocclusion and maxillary and mandibular incisor protrusion. In addition, an invasive frenum attachment caused severe maxillary incisor crowding, displacement, and cystic formation (Figs 9-19a to 9-19e).

Treatment:
The treatment plan included removal of the frenum, the cyst, and all abnormal soft tissue attachment and extraction of the four first premolars, carried out as a serial step-by-step extraction.

After the surgical procedure and tissue healing, a removable maxillary Hawley appliance was inserted to achieve slow, minor incisor alignment, and use of a lower holding arch for about 1 year was followed by step 1 of the extraction series: removal of the maxillary primary canines, both maxillary primary first molars, and both mandibular primary first molars. Figure 9-19f shows alignment of the maxillary incisors and the canine bulges before serial extraction.

Step 2 was extraction of all four first premolars. Maxillary anchorage was prepared with a Nance appliance, and the lower holding arch was removed as reciprocal anchorage.

Step 3 of the extraction sequence was removal of the remaining primary second molars. This was followed by maxillary and mandibular bonding to start maxillary canine retraction. Then mandibular and later anterior retraction and space closure were accomplished. Some mesial movement of the mandibular molars was allowed, in order to achieve a Class I molar relationship (Figs 9-19g to 9-19k).

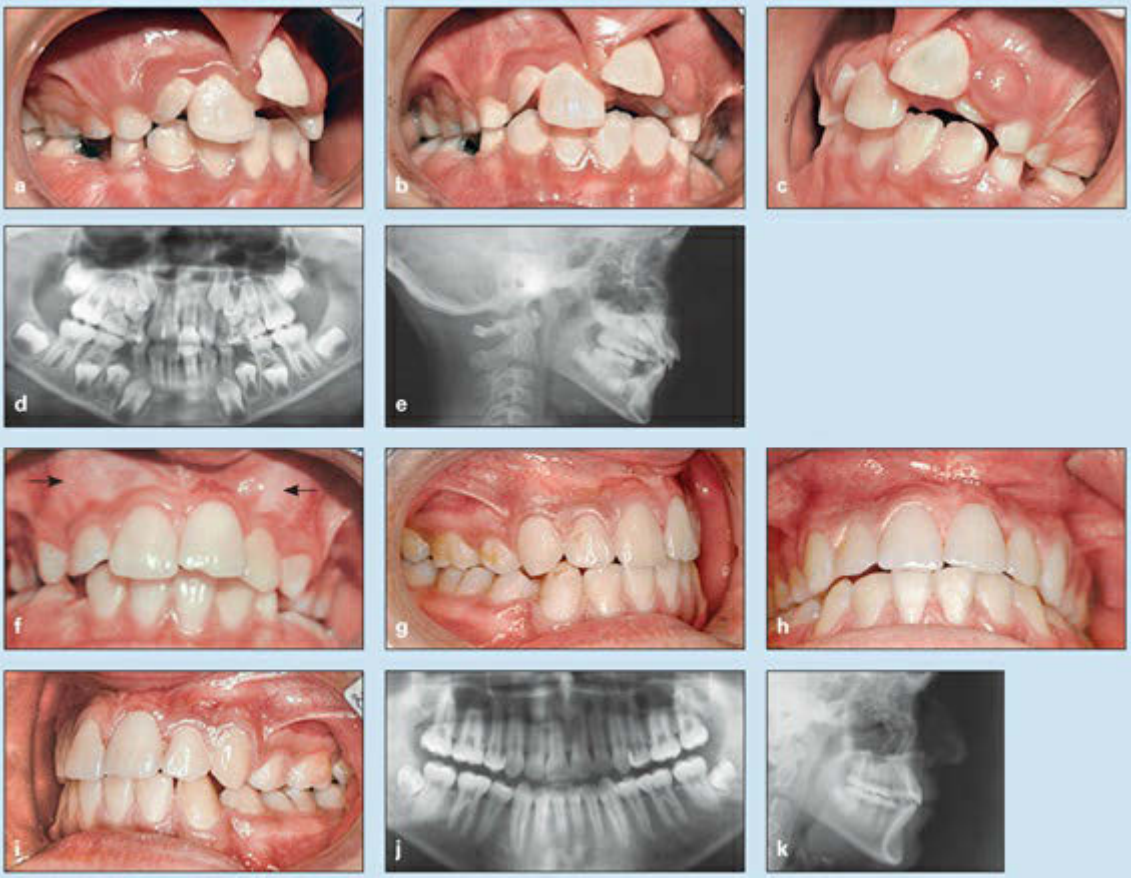


Fig 9-19 Treatment of a 10-year, 8-month-old girl with a Class II division 1 malocclusion and maxillary and mandibular protrusion. An invasive frenum attachment has caused tooth displacement, maxillary incisor crowding, and formation of a cyst. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e)* Pretreatment cephalometric radiograph. *(f)* Tissue healing and some incisor alignment. The *arrows* show canine bulge. *(g to i)* Posttreatment occlusion. *(j)* Posttreatment panoramic radiograph. *(k)* Posttreatment cephalometric radiograph.

Case 9-3

A 5-year-old girl presented with a mesial step and good interdigitation in the posterior segments but a severe hereditary diastema, resulting from a strong and low abnormal frenum attachment, and displaced primary incisors (Figs 9-20a to 9-20d). Because of the patient's age and the hereditary nature of the diastema (her mother also had a severe diastema and abnormal attachment), no treatment was suggested at this age. The parents were advised to bring back the child when all incisors had erupted.

The patient returned during the late mixed dentition with the same problem and a more intensive effect on the permanent incisors, including a severe, V-shaped diastema, incisor rotation, and incisor displacement (Figs 9-20e to 9-20h).

Treatment:
Orthodontic problems consisted of severe frenum attachment; a wide, V-shaped diastema; rotated and displaced incisors; anterior open bite; crowding; severe maxillary and mandibular dental protrusion; and steep mandibular plane. The treatment plan included deep frenectomy and extraction of the four first premolars.

The treatment followed this order: *(1)* placement of a maxillary Nance appliance and a lower holding arch as well as extraction of four first premolars; *(2)* 2 × 6 bonding, incisor alignment, frenectomy, and diastema closure; *(3)* canine retraction, anterior retraction, and completion of active treatment; *(4)* retention. Figures 9-20i to 9-20l show the posttreatment results.

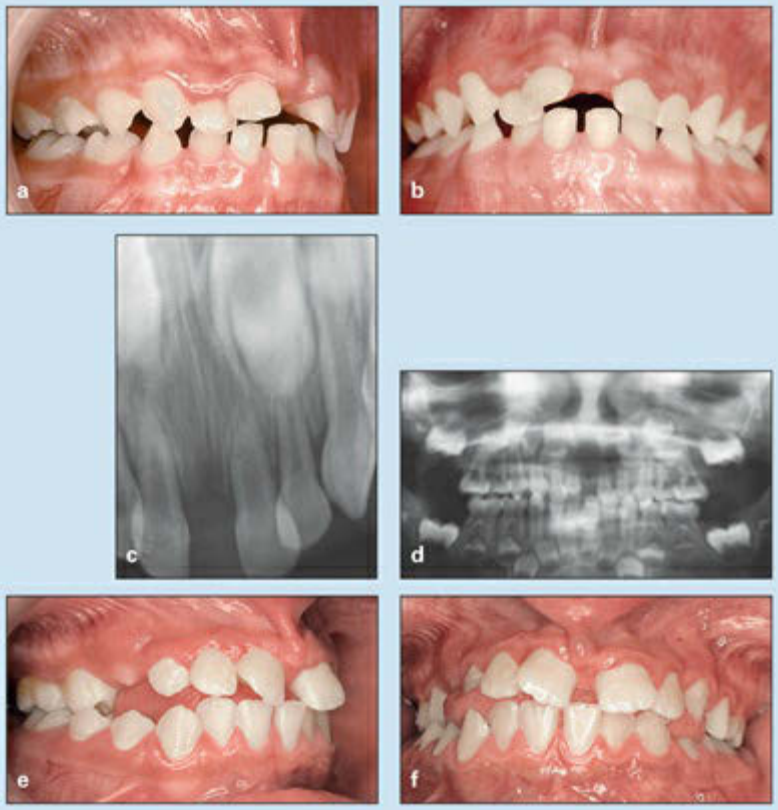


Fig 9-20 Treatment of a girl with a severe hereditary diastema, resulting from a strong and low abnormal frenum attachment in the primary dentition, and displaced incisors. *(a to c)* Occlusion at 5 years. Her parents were advised to return for evaluation when all the child's incisors had erupted. *(d)* Panoramic radiograph of the primary dentition. *(e and f)* Occlusion in the late mixed dentition, showing significant frenum attachment, a severe V-shaped diastema, and incisor displacement.

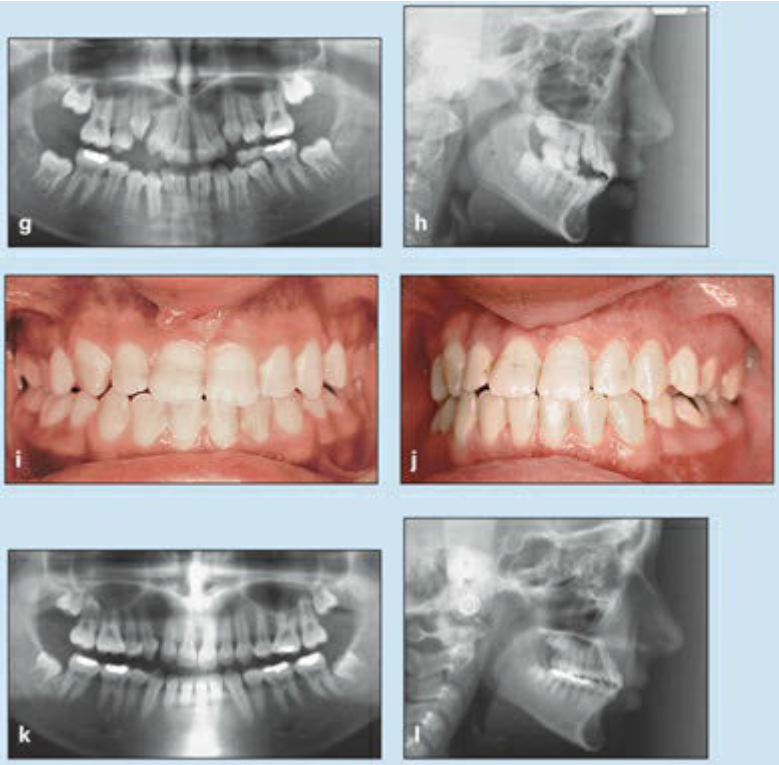


Fig 9-20 *(cont) (g)* Pretreatment panoramic radiograph during the late mixed dentition. *(h)* Pretreatment cephalometric radiograph during the late mixed dentition. *(i and j)* Posttreatment occlusion. *(k)* Posttreatment panoramic radiograph. *(l)* Post-treatment cephalometric radiograph.

Case 9-4

Many factors besides abnormal frenum attachment can cause abnormal diastema, and it is very important to differentiate and verify the cause before frenectomy. Cases 9-4 and 9-5 are examples of conditions in which a diastema was not caused by abnormal frenum attachment.

An 8-year, 5-month-old girl presented for correction of her diastema. Her orthodontic problems were a Class III malocclusion tendency in the molars and canines, maxillary deficiency, zero overjet, and minor mandibular incisor crowding. Cephalometric evaluation indicated a negative value for the point A–nasion–point B angle and maxillary incisor protrusion. Therefore, the cause of the diastema was diagnosed to be the point A–point B discrepancy, the absence of overjet, and the force of mandibular growth (Figs 9-21a to 9-21e).

Treatment:
The treatment plan included face mask therapy for maxillary protraction in order to increase the overjet, labial root torque, and then midline closure. No frenectomy was planned. Figures 9-21f to 9-21j show the posttreatment occlusal and dentoskeletal changes.

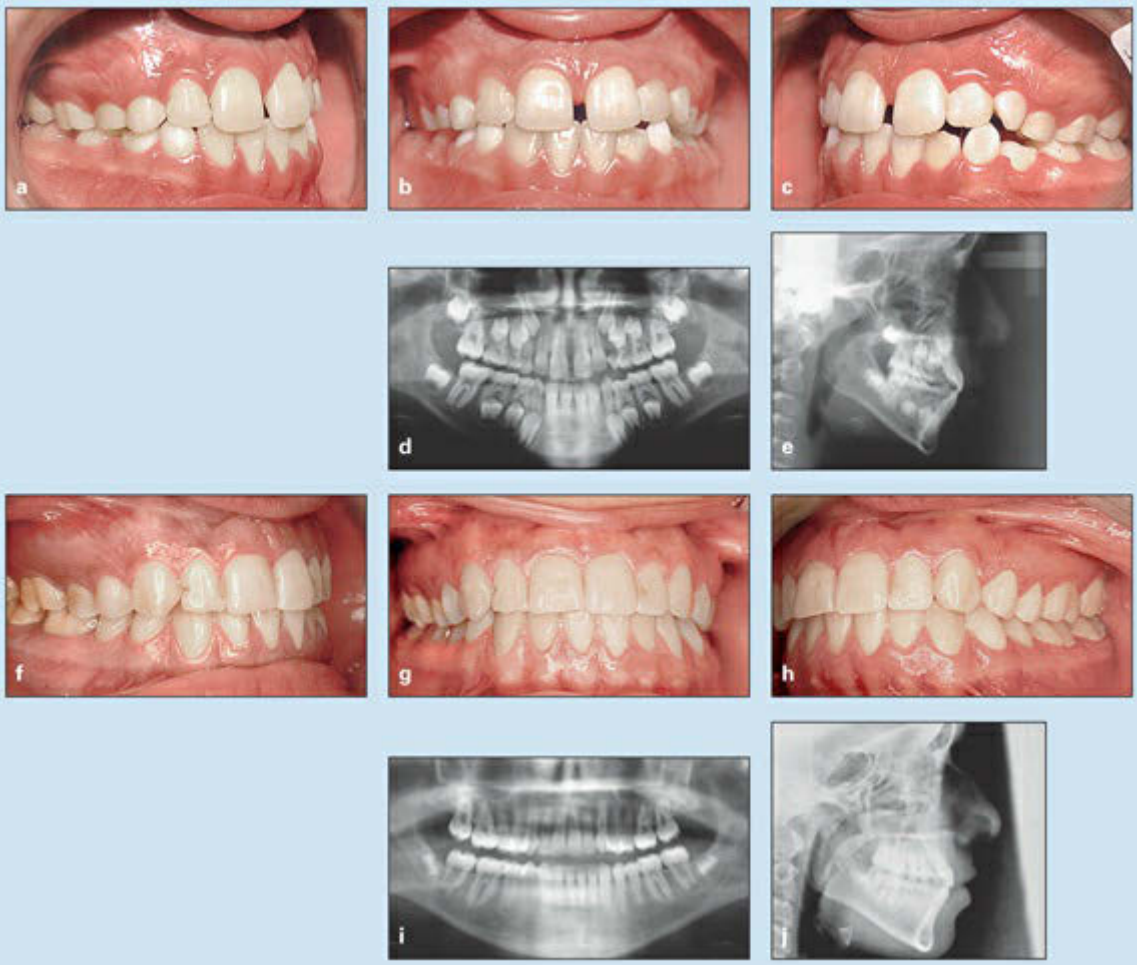


Fig 9-21 Treatment of an 8-year, 4-month-old girl with a Class III tendency, maxillary deficiency, and zero overjet, resulting in diastema and space deficiency in the maxillary anterior segment. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e)* Pretreatment cephalometric radiograph. *(f to h)* Posttreatment occlusion after maxillary protraction. *(i)* Post-treatment panoramic radiograph. *(j)* Posttreatment cephalometric radiograph.

Case 9-5

A 12-year-old girl presented with a midline diastema and severe incisor protrusion. Orthodontic problems included a Class II division 1 malocclusion, a 12-mm overjet, interdental spacing, an impinging deep bite, and lip dysfunction because of a two-step mandibular occlusion, the latter two of which caused an abnormal diastema. The frenum attachment was low but not the cause of the diastema (Figs 9-22a to 9-22d).

Treatment:
First, cervical headgear was used to correct the Class II molar relationship and reduce overbite. Then canine retraction was accomplished, while the headgear was kept as anchorage. Figure 9-22e shows the situation after a Class I molar relationship was achieved, during canine retraction with sectional retractor springs and anchorage with the cervical headgear; no anterior banding was applied as yet. This step was followed by mandibular complete bonding and correction of the two-step occlusion. Finally, treatment was concluded with anterior bonding and incisor retraction and diastema closure (Figs

9-22f to 9-22h).

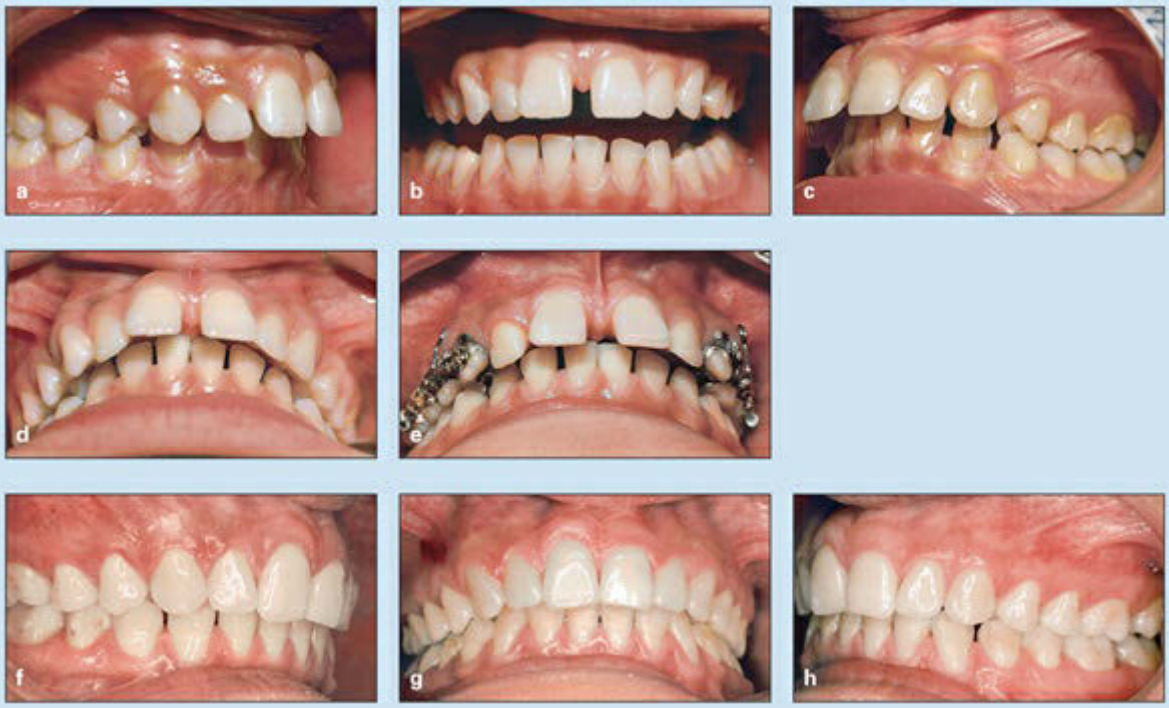


Fig 9-22 Treatment of a 12-year-old girl with a Class II division 1 malocclusion, impinging deep bite, and two-step mandibular occlusion that are causing a diastema. The frenum is low but is not the cause of the diastema. *(a to d)* Pretreatment occlusion. *(e)* Occlusion after achievement of a Class I molar relationship, during canine retraction but before anterior retraction and diastema closure. *(f to h)* Posttreatment occlusion. The frenum has atrophied and been eliminated without a frenectomy.

Summary

- Labial abnormal frenum attachment can cause many problems for the dentition, including an abnormal midline diastema that is often considered to be a malocclusion and esthetically unpleasing.
- A midline diastema is a gap between the maxillary central incisors that is part of the normal transitional stage of dentition; it usually closes automatically after incisor and canine eruption is completed.
- The prevalence of diastema is high in the early mixed dentition and decreases between the ages of 9 and 12 years.
- Other factors can cause abnormal diastema or prevent automatic closure of the normal developmental diastema; these diastemata must be differentiated from diastemata caused by abnormal frenum attachment.
- These other causative factors include lateral incisor hypodontia,

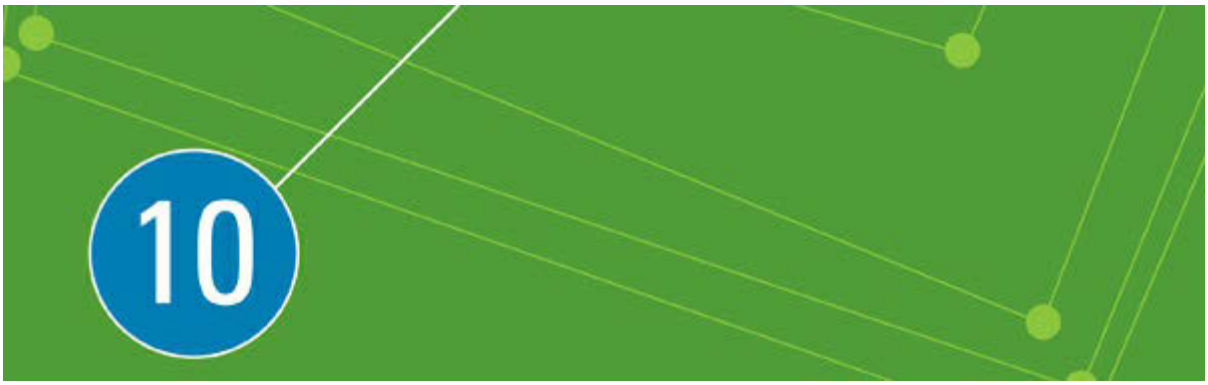
microdontia of maxillary lateral incisors (peg lateral incisors), presence of mesiodens or odontoma, deleterious oral habits, muscle imbalance, impinging deep bite, anterior Bolton discrepancy, and pathologic tooth migration.

- Abnormal diastema is more prevalent in females than in males; racial differences also exist.
- Abnormal frenum attachment can cause several problems in the dentition, including midline diastema with a malocclusion, rotation and displacement of central incisors, displacement of lateral incisors, lateral incisor crossbite, and a disrupted sequence of eruption.
- Treatment planning for the midline diastema must be based on proper evaluation of the extent, etiology, and pathogenesis of the diastema.
- Abnormal attachment of the frenum has special characteristics that aid recognition, such as the size, shape, and location of the tissue attachment and the shape of the midline gap. The blanching test can also be used, and open midline interseptal sutures can be diagnosed by periapical radiograph.
- Management of the frenum depends on the age of the patient and stage of dentition and on local factors such as the size, position, and strain of the frenum tissue.
- All diagnostic evaluation and assessment of all other possible causes must be completed before frenectomy is selected as a treatment.
- If frenectomy is performed, after involved tissue is removed, orthodontic closure must begin the same day to prevent fibrous tissue regeneration.

References

1. Baum AT. The midline diastema. J Oral Med 1966;21:30–39.
2. Angle EH. Treatment of Malocclusion of the Teeth, ed 7. Philadelphia: SS White, 1907.
3. Broadbent BH. Ontogenetic development of occlusion. Angle Orthod 1941;11:223–241.
4. Farrer JN. Regulation of teeth made easy by the positive system. Dent Cosmos 1882;24:186–193.
5. Taylor JE. Clinical observation relating to the normal and abnormal frenum labii superioris. Am J Orthod Oral Surg 1939;25:646–660.
6. Gardiner JH. Midline spaces. Dent Pract 1987;17:287–298.
7. Weyman J. The incidence of median diastema during the eruption of the permanent teeth. Dent Pract 1987;17: 276–298.
8. Richardson ER, Malhotra SK, Henry M, Coleman HT. Biracial study of the maxillary midline diastema. Angle Orthod 1973;43:438–443.
9. Lavelle CL. The distribution of diastemas in different human population samples. Scand J Dent Res 1970;78: 530–534.

10. Horowitz HS. A study of occlusal relations in 10- to 12-year old Caucasian and Negro children: Summary report. Int Dent J 1970;20:593–605.
11. Weyman J. The incidence of median diastema during the eruption of the permanent teeth. Dent Pract Dent Rec 1967;17:276–298.
12. Brunelle JA, Bhat M, Lipton JA. Prevalence and distribution of selected occlusal characteristics in the US population, 1988–1991. J Dent Res 1996;75:706–713.
13. Huang WJ, Creath CJ. The midline diastema: A review of its etiology and treatment. Pediatr Dent 1995;17:171–179.
14. Higley LB. Maxillary labial frenum and midline diastema. ASDC J Dent Child 1969;36:413–414.
15. Picton DC, Moss JP. The effect on approximal drift of altering the horizontal component of biting force in adult monkeys (Macaca irus). Arch Oral Biol 1980;25:45–48.
16. Stubley R. The influence of transseptal fibers on incisor position and diastema formation. Am J Orthod 1976;70:645–662.
17. Martin RA, Jones KL. Absence of the superior labial frenu-lum in holoprosencephaly: A new diagnostic sign. J Pediatr 1998;133:151–153.
18. Dean JA, Avery DR, McDonald RE. Dentistry for the Child and Adolescent, ed 9. St Louis: Mosby, 2010.
19. Bolton WA. The clinical application of a tooth-size analysis. Am J Orthod 1962;48:504–529.
20. Shashua D, Artun J. Relapse after orthodontic correction of maxillary median diastema: A follow-up evaluation of consecutive cases. Angle Orthod 1999;69:257–363.
21. Kotlow LA. The influence of the maxillary frenum on the development and pattern of dental caries on anterior teeth in breastfeeding infants: Prevention, diagnosis, and treatment. J Hum Lact 2010;26:304–308.
22. Haytac MC, Ozcelik O. Evaluation of patient perceptions after frenectomy operations: A comparison of carbon dioxide laser and scalpel techniques. J Periodontol 2006;77:1815–1819.
23. Olivi G, Chaumanet G, Genovese MD, Beneduce C, Andreana S. Er, Cr: YSGG laser labial frenectomy: A clinical retrospective evaluation of 156 consecutive cases. Gen Dent 2010;58:e126–e133.
24. Kotlow LA. Lasers and soft tissue treatments for the pediatric dental patient. Alpha Omegan 2008;101:140–151.
25. Edmunds J, Miles SC, Fulbrook P. Tonguetie and breastfeeding: A review of the literature. Breastfeed Rev 2011;19:19–26.
26. Berg KL. Tonguetie (ankyloglossia) and breast feeding: A review. J Hum Lact 1990;6:109–112.
27. Wright JE. Tonguetie. J Paediatr Child Health 1995;31: 276–278.
28. Fiorotti RC, Bertolini MM, Nicola JH, Nicola EM. Early lingual frenectomy assisted by CO_2 laser helps prevention and treatment of functional alterations caused by ankyloglossia. Int J Orofacial Myology 2004;30:64–71.
29. Suter VG, Bornstein MM. Ankyloglossia: Facts and myths in diagnosis and treatment. J Periodontol 2009;80:1204–1219.



Early Detection and Treatment of Eruption Problems

Tooth eruption is an occlusal physiologic movement of the tooth within the jaw from its developmental position to its functional position in the occlusal plane. Active eruption starts when crown formation is completed and the root begins to form. The eruption process moves the tooth toward its functioning position. Massler et al[1] defined *tooth eruption* as the "movement of a tooth from its site of development within the alveolar process to its functional position in the oral cavity."

An understanding of the mechanisms of eruptive tooth movement is essential in all areas of dentistry. In general, tooth eruption is a complex process that involves a regulated interrelationship of root genesis and cementogenesis. Bosshardt and Schroeder[2] stated that prefunctional development of the roots of permanent teeth is a protracted phenomenon in humans and may continue for 5 to 7 years prior to emergence of the tooth in the oral cavity. Tooth eruption, which is a continuous process to maintain the vertical dimension of every individual, continues throughout life. Movement can be observed following occlusal wear or as overeruption when a tooth is unopposed.

Early orthodontic treatment affords clinicians the opportunity to use this phenomenon in young patients. With proper mechanics, the eruption process can be used to increase or control vertical dimension during tooth eruption in patients with an overbite or underbite. There are also situations in space management when the space and mesiodistal position of a tooth must be controlled and overeruption of

opposing teeth must be prevented (if a tooth is congenitally missing or has been extracted in one arch).

Tooth buds and the alveolar process develop simultaneously, and when a crown is completed it is enclosed in a crypt within alveolar bone. When eruption and movement of the tooth start, localized bone resorption begins to produce an eruption pathway for the tooth. At the same time, bone starts to form to fill the space left behind by the moving crown and growing root. During this complex interaction of tooth movement and jaw growth, the teeth move in three-dimensional space to maintain their position in the growing jaw and to compensate for masticatory function.

Phases of Tooth Eruption

For convenient categorization and for descriptive purposes, physiologic tooth movement is described in three phases:

1. Pre-eruptive phase
2. Eruptive phase
3. Posteruptive phase

Pre-eruptive phase

The pre-eruptive phase starts with follicular growth when the crown is being formed within its bone crypt. Growing teeth move in various directions to maintain their position in the expanding jaws. These movements in permanent teeth are mainly the result of primary tooth eruption and alveolar growth.

Pre-eruptive movement within the crypt wall occurs during bone remodeling. When there is bone resorption on one side and bone apposition on the opposite side, the tooth germ moves bodily as a filling-in process. However, when eccentric growth takes place and only bone resorption occurs on one side, the shape of the crypt changes, and the tooth germ moves according to this shape.

Primary tooth germs are very small when they develop, have plenty of space in the developing jaw, and grow very rapidly. However, they become crowded; further jaw growth in all directions accommodates all tooth germs.

Successional tooth germs develop on the lingual aspect of their primary

predecessors, in the same bony crypt, and as the jaw develops they shift from their positions. For example, the permanent incisors and canines eventually occupy a position lingual to the roots of their primary predecessors, and the premolar tooth germs are positioned between the divergent roots of the primary molars.

Permanent molar tooth germs, which have no predecessors, develop from a backward extension of dental lamina. Maxillary molar tooth germs develop first, with their occlusal surfaces facing distally, and after sufficient jaw growth they swing into normal inclination. Mandibular permanent molars develop with their axes showing a mesial inclination, which becomes vertical when sufficient jaw growth has occurred.

All movements in this phase take place within the crypts of the developing and growing crowns before root formation.

Eruptive phase

The eruptive, or prefunctional, phase begins with the initiation of root formation and ends when the teeth reach occlusal contact. The mechanisms of eruption for primary and permanent teeth are similar, and many histologic changes occur around the tooth structure to accommodate tooth eruption. These changes include the following:

- Root formation
- Development of the periodontal ligament, which develops only after root formation has been initiated (the periodontal ligament must be remodeled to accommodate continued eruptive tooth movement)
- Resorption of primary roots
- Resorption of overlying bone

Any disturbances during these histologic processes can disturb tooth eruption.

This stage of tooth eruption is a relatively rapid phase that lasts until the teeth reach the occlusal level. In a radiographic study, Shumaker[3] observed that each tooth starts to move toward occlusion at approximately the time of crown completion. The interval from crown completion and the beginning of eruption until the tooth is in full occlusion is approximately 5 years for permanent teeth.

Posteruptive phase

The posteruptive, or functional, phase begins when the teeth reach occlusion. During this period the teeth continue to move occlusally, alveolar processes increase in height, roots continue to grow to accommodate jaw growth, alveolar bone density increases, and principal fibers of the periodontal ligament establish themselves. Posteruptive changes have been divided into three categories:

1. Juvenile occlusal equilibrium: This is a phase of very slow eruption that parallels the rate of vertical growth of jaws.
2. Circumpubertal occlusal eruptive spurt: This is the second phase of active eruption, coinciding with the skeletal growth spurt and compensatory alveolar growth to maintain the freeway space and increase lower facial height. This phase has a faster rate of eruption, which slows between the ages of 11 to 16 years as facial growth reaches maturity.
3. Adult occlusal equilibrium: This is an extremely slow rate of eruption that occurs throughout life, in order to maintain facial height in response to situations such as occlusal tooth wear or tooth loss in one arch and overeruption of the tooth in the opposing arch.

Mechanisms of Eruptive Tooth Movement

The mechanism of tooth eruption still is not fully understood, but a review of this subject leads to the conclusion that eruption is a multifactorial process. Several theories to explain tooth eruption have been proposed.

Root formation

Because tooth eruption coincides with root formation, some investigators, such as Massler et al,[1] considered that root elongation is a force responsible for tooth eruption. Shumaker,[3] in a radiographic study, indicated that eruption starts at approximately the time of crown completion. In a study of dogs, in which the tooth germs were removed and replaced with dead crown shells and synthetic substitutes, Marks and Cahill[4] showed that the follicular changes and the path of eruption were no different from those seen in normally erupting teeth. They concluded that the tooth itself played no part in the eruptive process. Marks and Cahill[4] described the movement of the teeth during eruption as a three-dimensional movement and concluded that root elongation cannot move the tooth in three dimensions; it may,

however, accelerate eruption speed.

Through animal histologic experiments and root dissection, Berkovitz and Thomas[5] also showed that eruption of rootless teeth continues in the same manner as eruption of teeth with normal roots. The normal eruption of teeth that had been subjected to irradiation and consequently had shortened root formation is another example.[6]

Considering the high position of the maxillary canine before eruption and the long distance it moves during eruption, this tooth would have a long root formation if the root were responsible for the eruption process, which is not the case.

Hydrostatic pressure

Some studies have demonstrated that a differential hydrostatic pressure exists between the tissues investing the crown and its apical extent (ie, higher tissue pressure or accumulation of tissue fluid in the apex during apex development pushes the tooth germ toward occlusion).

Van Hassel and McMinn,[7] in a study of dogs, found that the tissue pressure apical to the erupting tooth was greater than that found occlusally and thus advocated the theory that tissue pressure generates an eruptive force. However, they did not demonstrate any association between the magnitude of the force and the rate of eruption. Surgical excision of the growing root and associated tissues and elimination of the periapical vasculature did not stop eruption.

In root-resected incisors of rats, Chiba and Ohshima[8] investigated the effect of a single injection of colchicine and daily injections of hydrocortisone on eruption rate. The study found that the agents had opposite effects on tooth eruption; colchicine and hydrocortisone caused a marked reduction and a marked increase, respectively, in unimpeded eruption rates. Root-resected and normal, nonresected incisors responded identically to these two drugs, supporting the view that the eruptive process following root resection is physiologic. No direct evidence was found to confirm or refute the hydrostatic pressure or periodontal fibroblast hypothesis of tooth eruption.

Dental follicle

The soft tissue of the dental follicle, which is located between the bony crypt and unerupted crown, exhibits osteo-clastic activity and occlusal bone resorption and

creates an eruption tunnel to facilitate eruption. Simultaneously, new bone forms to fill the space left behind the crown and growing root.

Marks[9] studied crypt surfaces during eruption and confirmed the polarization of alveolar bone metabolism around a tooth with respect to both bone cells and mineralized surface topography. In experimental studies of tooth eruption, he showed that the dental follicle, the dense connective tissue investment of the tooth, is necessary for eruption and that neither bone resorption nor bone formation occurs without the adjacent part of the dental follicle.

Wise et al[10] used scanning electron microscopy to study bone formation in the base of the alveolar crypt of the first molar in the rat as a potential motive force of tooth eruption. Results indicated that the motive force of tooth eruption is likely bone formation at the base of the alveolar crypt and that this osteogenesis may relate to production of bone morphogenetic protein 2 in the dental follicle.

Gubernaculum dentis

At an early stage, the primary tooth and its successor share one bony crypt, but eruption and alveolar growth cause the permanent bud to be located in an inferior crypt. The gubernaculum dentis attachment acts to maintain the relative position of the follicle within the alveolar process and to prevent the tooth from submerging; in addition, it acts as a guide for the path of eruption.

Cahill and Marks,[11] through radiographic and histologic evaluation in dogs, studied the roles of the gubernaculum dentis, root formation, the tooth crown, and the dental follicle in prefunctional eruption of a mandibular premolar. They concluded that the dental follicle was the only one of these structures required for the coordinated enlargement of the eruption pathway and formation of bone in the base of the bony crypt.

Periodontal ligament

Two mechanisms have been suggested to support the theory that the periodontal ligament influences tooth eruption. One involves the maturation of fibers, and the other suggests that the periodontal ligament may function as myofibroblasts to induce tension, moving the tooth toward occlusion:

- As the collagen fibers of the intermediate plexus mature, they contract and, as they

are vertically inclined, draw the tooth vertically.

- Recent studies report a contractile protein in some fibroblasts of periodontal ligament that act as muscle cells.

Ten Cate[12] supported the theory that the periodontal ligament has a major role in determining tooth eruption. He stated that the periodontal ligament, which is derived from the dental follicle, provides the force required for eruption and that the cells responsible are the fibroblasts within the periodontal ligament, which have contractile potential.

In a discussion of the role of fibroblasts in the remodeling of periodontal ligament, Ten Cate et al[13] explained the role of fibroblasts in phagocytosis during remodeling. According to these investigators, increased fibroblastic activity during eruption plays a key role in physiologic tooth movement. However Marks[9] stated that a limited period of growth and the presence of a periodontal ligament do not assure tooth eruption.

Berkovitz[14] favored Ten Cate et al's theory[13] of fibroblast contraction, although he recognized the limitations of in vitro tissue studies. He indicated that formation and renewal of the periodontal ligament are associated with the continuous eruption of permanent incisors in rodents.

Bone remodeling

Bone remodeling in the pre-eruptive phase is theorized to move teeth by selective deposition and resorption in the immediate neighborhood of the tooth. Brash[15–18] proposed that the formation of bone apical to developing teeth is one mechanism for tooth eruption.

Marks and Cahill's experiment[4] in dogs showed that the dental follicle must be present for tooth eruption and that bone remodeling occurs around the erupting follicle, regardless of the presence or absence of a tooth crown. The investigators suggested that the remodeling process may be under the control of the dental follicle because the periodontal ligament, cementum, and alveolar bone proper are derivatives in part of the follicle, so that later events could be controlled by these and other related tissues.

Alveolar bone growth, tooth development, and eruption are interdependent phenomena, and bone formation itself is not sufficient for tooth eruption. Cleidocranial dysostosis (also known as *cleidocranial dysplasia*) is a good

example of a disorder in which eruption is delayed, absent, or ectopic, despite the fact that bone formation is normal.

Cellular and Molecular Bases of Tooth Eruption

Review of the mechanisms of tooth eruption and exploration of the different proposals and hypotheses related to tooth eruption suggest that tooth eruption requires the presence of three important components: the dental follicle, alveolar bone resorption for creation of an eruption pathway, and alveolar bone formation at the base of the bony crypt. In order for a tooth to erupt, resorption of alveolar bone must take place to remove the overlying bone covering the tooth bud and open a pathway; then the tooth bud must erupt through the formed pathway through a biologic process, and alveolar bone must form at the base of the bony crypt.

Marks and Cahill[4] stated that tooth eruption is a series of metabolic events in alveolar bone characterized by bone resorption and formation on opposite sides of the dental follicle; the tooth itself does not contribute to this process. The question is, what biologic process regulates both the osteoclastic activity and the osteogenesis needed for tooth eruption?

In a comparison between human premolars and rodent molars, Bosshardt and Schroeder[2] used light and electron microscopy and various measurements to assess the development and repair of cementum during and after root formation and tooth eruption. They indicated that cementum is a highly responsive mineralized tissue, and its biologic activity is necessary for root integrity and for bringing and maintaining the tooth in its proper position. They added that cementum formation and periodontal fiber attachment in premolars are characterized by a long-lasting phase of prefunctional development and that initial cementogenesis and root development last an average of 5 to 7 years in human premolars.

Larson et al[19] removed the external layer of the enamel organ (dental follicle) before eruption in 13 mandibular premolars in dogs and followed eruption clinically, radiographically, and histologically to determine the role of the enamel organ in eruption. They found that none of the teeth without dental follicles erupted, but three teeth from which the follicle was separated and then replaced did erupt. They concluded that the enamel organ cannot support tooth eruption without the dental follicle.

As mentioned earlier, Wise et al[10] examined bone formation in the base of bony crypts as a potential motive force of tooth eruption. They used scanning electron

microscopy to study the rat mandibular molar from postnatal days 3 to 18 and concluded that the motive force of tooth eruption is bone formation at the base of the alveolar crypt. They suggested that this osteogenesis may relate to production of bone morphogenetic protein 2 in the dental follicle.

In another study aimed at finding what mechanism regulates both osteoclastogenesis and osteogenesis, which molecules are needed to initiate this regulation, and which cells and tissues are involved in this process, Wise[20] reported the existence of a major burst of osteoclastogenesis in the rat mandibular first molar at day 3 and a minor burst at day 10 postnatally. Therefore, the osteoclastogenesis and osteogenesis needed for eruption are regulated by differential gene expression in the dental follicle both chronologically and spatially.

Stages of Permanent Tooth Eruption

The process of tooth eruption is an organized sequence of events that starts when crown formation is completed and the root begins to form. During this process, the tooth moves from its crypt through bone and soft tissue to its functional position. *Tooth emergence* is a part of the eruption process that starts with the appearance of any part of the cusp or crown through the gingiva.

Philbrick et al[21] demonstrated that the time interval from crown completion and the beginning of eruption to full occlusion of permanent teeth is approximately 5 years.

After a review of the literature, Suri et al[22] described the process of eruption as a dynamic process encompassing completion of root development, establishment of the periodontium, and maintenance of a functional occlusion.

During this complicated process, jaw growth is also interacting with numerous skeletal growth changes, such as increase in size, change in shape, and change in position. Normal bone remodeling under the control of genetic and environmental factors is another process necessary for progression of tooth eruption, which is occurring within the jaws.

The exfoliation of primary teeth and eruption of permanent successors result from a series of complex, reciprocal interactions between the root and surrounding structures. These interactions are controlled by a series of biochemical and biologic events that occur under the influence of several genetic signals. All of these events are consecutive steps; every step is a prerequisite for the next to occur and

facilitates normal permanent tooth eruption after primary tooth exfoliation.

The following are the consecutive steps for replacement of a primary tooth by a permanent successor that can be seen at a site of normal dental transition. These steps can be a useful guide in monitoring tooth eruption to detect problems and design a treatment plan accordingly:

1. Resorption of the primary tooth root
2. Lengthening of the permanent tooth root
3. Exfoliation of the primary crown
4. Resorption of overlying bone to clear the path of eruption
5. Bone apposition under the moving tooth germ to fill the space left behind
6. Alveolar vertical growth and increasing alveolar height according to vertical tooth eruption
7. Movement of the permanent tooth through bone
8. Piercing of the alveolar crest at two-thirds' root formation
9. Piercing of the gingival margin at three-quarters' root formation
10. Root completion after several months in occlusion

Early detection of developmental problems at their initial stage of development is the most important step in early intervention in orthodontics. Many dental anomalies arise during tooth formation and eruption, and recognition of these problems requires a thorough understanding of these developmental changes. Longitudinal panoramic radiograph monitoring of these changes can be very helpful in early detection of and intervention in these abnormalities (see chapter 3).

Factors That Can Disturb Tooth Eruption

Numerous in vivo animal experiments and radiographic studies in humans have been performed to better understand the process of tooth eruption. As previously mentioned, Cahill and Marks[11] stated that tooth eruption is a series of metabolic events in alveolar bone, characterized by bone resorption and formation on opposite sides of the dental follicle, and that the tooth itself does not contribute to this process. In a molecular study, Wise and King[23] revealed more precisely that eruption is a tightly coordinated process regulated by a series of signaling events between the dental follicle and the osteoblasts and osteoclasts found in the alveolar

bone.

Any disruption in this process can affect normal tooth eruption; outcomes range from delayed eruption to a complete failure of eruption. These abnormalities can occur as part of a syndrome or as a nonsyndromic disorder (isolated or familial).

Defects in tooth formation and eruption have been attributed to three general factors, categorized as systemic factors, genetic factors, and local factors. These factors can disturb matrix formation or the calcification process of tooth formation, resulting in structural defects such as decalcification and demineralization of tooth structure or abnormal tooth form. These factors can also arrest tooth development (hypodontia) or cause extra bud formation (hyperdontia). Likewise, if the apposition-resorption mechanism in alveolar bone is defective for any reason, the eruption process can be affected. Depending on the stage of eruption, syndromic and nonsyndromic problems can range from delayed eruption to a complete failure of eruption, including tooth ankylosis, primary failure of eruption (PFE), secondary failure of eruption, transposition, ectopic eruption, and impaction.

There is variation in the normal chronologic range of eruption of primary and permanent teeth because ethnic, racial, sex, and individual variations can influence this phenomenon. However, attentiveness to severe deviations of eruption time, sequence, and other disturbances in the tooth eruption process is critical to the timing and selection of different orthodontic treatment modalities. The purpose of this discussion is to provide a review of etiologic factors affecting normal eruption processes and the pathogenesis and differential radiographic interpretation of impaired tooth eruption.

Systemic factors

Disturbances of endocrine glands have a profound effect on the entire body, including tooth eruption. Baume and Becks[24] assessed the response of the incisors of rats to growth hormone and thyroxine and the combination of both. The growth hormone resulted in an increase in size without hastening the eruption rate. Thyroxine treatment increased tooth dimensions and accelerated the eruption rate 36% in the young group and 46% in the older group.

To elucidate the effects of parathyroid hormone–related protein (PTHrP) on osteoclast regulation, Philbrick et al[21] established a primary culture of epithelial stellate reticulum cells and mesenchymal dental follicle cells surrounding the teeth. The addition of osteoclastogenesis inhibitory factor resulted in a decrease in the

number of osteoclasts formed in the co-cultures, suggesting that osteoclast formation is mediated by osteoclast differentiation factor. They concluded that PTHrP seems to regulate osteoclast formation via mediation of the dental follicle, in a manner analogous to the osteoblast-mediated process in the peripheral skeleton.

Tse Mdo et al[25] used hemidecortication to investigate the effect of thyroid hormone in rats. This procedure, which consists of removing one cerebral hemisphere and leaving the thalamus and hypothalamus intact, produced hypothyroidism. The investigators also found a significant decrease in the weekly eruption rate of the maxillary incisors and partial recovery after the administration of thyrotropin-releasing hormone in rats.

Congenital hypothyroidism

Congenital hypothyroidism, also known as *cretinism*, is the result of the absence or underdevelopment of the thyroid gland and insufficient levels of thyroid hormone at birth. Today hypothyroidism is routinely diagnosed and corrected at birth. If undetected and untreated, hypothyroidism causes mental deficiency and disproportionate dwarfism. In addition, development of the dentition is delayed in all stages, including primary tooth eruption, exfoliation, and eruption of permanent teeth. The teeth are usually normal in size but crowded.

Juvenile hypothyroidism (acquired hypothyroidism)

Juvenile hypothyroidism is the result of a malfunction of the thyroid gland that is usually seen around the age of 10 to 12 years. Because most of the growth has been completed by this age, the body disproportion is not present, as it is in congenital hypothyroidism, but some degree of obesity is found. Development of the dentition is slow, and delayed exfoliation and delayed permanent eruption are common.

Hypopituitarism

Pituitary dwarfism is the result of an early hypofunction of the pituitary gland. Delayed eruption of the dentition is characteristic in patients with hypopituitarism; the primary teeth may be retained throughout the life of the person.

Extraction of the primary teeth is not indicated, because eruption of the permanent teeth cannot be ensured. The dental arch has been reported to be smaller than normal; thus, it cannot accommodate all the teeth, and crowding develops. The roots

of the teeth are shorter than normal in conditions of dwarfism, and the supporting structures are retarded in growth.

Achondrodysplasia (achondroplastic dwarfism)

Achondrodysplasia is an autosomal-dominant disorder that causes disproportionate dwarfism. In this abnormality, growth of cartilage, or endochondral ossification, is disturbed; thus, the secondary displacement of the nasomaxillary complex is insufficient, and consequently the upper face is retruded, and the bridge of the nose is depressed. The maxilla is deficient, with resultant crowding of the teeth and a tendency for a Class III sagittal relationship and open bite. The development of the dentition is slightly delayed.

Defects in development of cartilage, especially of long bones, result in arrested growth and dwarfism that are usually diagnosed in babies or children. The head is disproportionately large, although the trunk is normal in size. The fingers may be almost equal in length.

In a radiographic study of the jaws and teeth in a group of 48 individuals with pituitary dwarfism, Kosowicz and Rzymski[26] found an absence of root resorption and delayed exfoliation of primary teeth, marked delay in eruption of the permanent teeth, deep retention of permanent teeth in the maxillary and mandibular sockets, small jaw size, and crowding.

Barbería Leache et al[27] studied 50 children with growth deficit regarding their endocrine and dentition status. They planned to investigate the relationship between the chronology of tooth eruption and delayed growth, regardless of whether the delayed growth is associated with low genetic height or caused by hormones. They concluded that retardation in bone age does not necessarily imply retardation in the dentition.

Genetic factors

Eruption defects have been found to be a feature in many genetic disorders and syndromes, including cleidocranial dysostosis and Down syndrome. Both the sequence and timing of eruption seem to be largely genetically determined. The recent finding that the parathyroid hormone receptor 1 (*PTH1R*) gene can be a causative factor for familial cases of PFE[28] suggests that other disturbances in tooth eruption may have a genetic etiology.

Cleidocranial dysostosis

Cleidocranial dysostosis is a condition that primarily affects the development of the bones and teeth. Depending on the severity of the problem, signs and symptoms of cleidocranial dysostosis can vary widely, even within the same members of the family. Cleidocranial dysostosis occurs in approximately one individual per million worldwide.

Individuals with this syndrome usually have underdeveloped or absent collarbones (clavicles). As a result, their shoulders are narrow and sloping, and they can bring their shoulders unusually close together in front of the body. Delayed closure of cranial sutures and fontanels is another characteristic of this condition. Fontanels that usually close in early childhood may remain open into adulthood in people with this disorder.

Researchers believe that the RUNX2 protein acts as a master switch, regulating a number of other genes involved in the development of cells that build osteoblasts for bone formation. This protein is essential for osteoblastic differentiation and skeletal morphogenesis. It is believed that the *RUNX2* gene provides instructions for making a protein that is involved in bone and cartilage development and maintenance.[29]

In individuals with cleidocranial dysostosis, the development of the dentition is delayed, resulting from delayed resorption of the primary teeth, and delayed eruption of the permanent teeth is common. Retarded eruption, failure of eruption, impaction of multiple teeth, the presence of supernumerary teeth, and the absence of other teeth are other dental problems that complicate occlusion.

Down syndrome

Down syndrome, or *trisomy 21 syndrome*, is a congenital anomaly. The cause of this syndrome is the presence of three copies of chromosome 21 rather than the normal two (diploid).

In patients with Down syndrome, delayed eruption of the teeth is common. Sometimes the first primary teeth may not appear until the individual is 2 years old, and the primary dentition may be completed at 5 years of age. The eruption often follows an abnormal sequence, and some of the primary teeth may be retained until age 15 years.

Because of the special characteristics of facial patterns, diagnosis is not difficult. The bridge of the nose is more depressed than normal, the orbits are small, and the eyes slope upward.

The tongue tends to be larger than normal and is usually protruded because the mandible is smaller.

Another characteristic of Down syndrome in most children is mental disability in the mild-to-moderate range.

Local factors

Many local factors can cause defects of tooth eruption. These defects can be classified as nonsyndromic types of tooth eruption disorders. The first step in early intervention in and treatment of these types of eruption problems is differential diagnosis and understanding of the initial cause of the problem. For example, lack of eruption can result from an obvious cause, such as inadequate arch length, or it can represent a PFE with many possible causes.

Examples of local factors that can interfere with normal eruption processes include supernumerary teeth, odontomas, cysts, other pathologic conditions, primary tooth ankylosis, remaining primary tooth roots, fibrotic soft tissue, bone barriers, lack of space due to early primary tooth loss, overretained primary teeth, primary periapical lesions that may hasten permanent eruption, or habits and muscle dysfunction. In addition, extraction of primary teeth has some effect on the eruption of their permanent successors.

In a study of 874 children, Gron[30] observed that tooth emergence is associated more closely with the stage of root formation than with the chronologic or skeletal age of the child. By the time of clinical emergence, approximately three-quarters of root formation has occurred. Teeth reach occlusion before root development is complete.

Posen[31] reviewed records of children with unilateral extraction of primary molars. He reported that eruption of premolars is delayed when primary molars are extracted by age 4 or 5 years. Delayed eruption is decreased when primary molars are lost after age 5 years. He also found that a significant acceleration of premolar eruption occurs when primary molars are lost at 8, 9, and 10 years of age.

Types of Eruption Disturbance

Before any treatment is planned for eruption disorders, the type of problem and the etiologic factors disrupting normal eruption must be determined. Eruption

disturbances can be broadly classified into two general types:

1. Disturbances related to the time of eruption, such as delayed or premature eruption or failure of eruption
2. Disturbances related to the position of the affected tooth, such as ectopic eruption, tooth transpositions, and impactions

Box 10-1 lists different eruptive disturbances that can result from different systemic or local factors.

Box 10-1	Classification of eruptive disturbances
Disruptions in timing • Delayed tooth eruption • Early exfoliation of primary teeth and premature eruption of permanent teeth • Failure of eruption (PFE and secondary failure of eruption) • Retarded tooth development • Abnormal sequence of eruption	***Disruptions in position*** • Ectopic eruption • Transposition • Impaction • Ankylosis

Delayed Tooth Eruption

Emergence that is outside the normal chronologic range of eruption includes delayed or early types of tooth eruption. According to numerous population studies conducted over the past century, there are variations in dental chronology based on race, ethnicity, and sex as well as environmental factors. Therefore, eruption that is early or delayed by a few months is not harmful unless there are local or systemic problems preventing normal eruption.

Delayed tooth eruption (DTE) is a common clinical finding that can occur in both the primary and permanent dentitions as a localized condition caused by local factors or as a sign of a generalized condition caused by systemic or genetic factors. Eruption of primary teeth usually occurs around 6 to 8 months of age, but, because of genetic or constitutional factors, the dentition sometimes may be delayed up to 1 year. A 1-year-old child with no primary teeth cannot be ignored and must be assessed for the possibility of some systemic disorder or syndromic conditions.

In such children, if there is no evidence of any ectopic tooth position, physical obstruction, or structural defect of tooth and the eruption status is within normal

limits, periodic observation is the best treatment option to follow. However, any clinically detected instance of early or late eruption that is beyond the chronologic norms of emergence must be evaluated and monitored radiographically, especially if it is asymmetric. This condition must not be ignored, especially if eruption is delayed more than 10 months beyond the normal chronologic standard of emergence.

Timely screening and follow-up by the practitioner can prevent or minimize consequences that can develop at a later age and affect the patient's dentition and health. Longitudinal panoramic radiograph monitoring of different stages of the dentition can be very helpful in early detection of and intervention in these abnormalities.

Etiology

There is considerable controversy regarding the terminology and pathogenesis of DTE. Suri et al[22] published a table listing the terminology used for DTE. These terms include *primary retention*, *embedded teeth*, *late eruption*, *retarded eruption*, *arrested eruption*, *impaired eruption*, and others. Box 10-2 lists causes of delayed eruption in both the primary and permanent dentitions.

Box 10-2	Causes of delayed eruption
Primary teeth • Constitutional delay • Vitamin D deficiency in rickets (affects calcium metabolism, causing delayed eruption of teeth and bone abnormalities) • Hypothyroidism (low synthesis of thyroid hormone causes delayed development, including delayed eruption of teeth) • Hypopituitarism (results in developmental delay and DTE) • Cleidocranial dysostosis • Gardner syndrome • Apert syndrome • Down syndrome • Cerebral palsy • Protein-energy malnutrition, or protein-calorie malnutrition (a form of malnutrition caused by inadequate protein intake that occurs most frequently in infants and young children and is the leading cause of death in children in some developing countries)	***Permanent teeth*** • All of the factors that cause delayed emergence of primary teeth can also cause delayed emergence of permanent teeth • Crowding of the jaw and space deficiency • Supernumerary teeth and odontomas • Odontogenic cysts or other pathologic lesions • Overretained primary teeth or remaining roots • Tooth deformity and defective tooth development • Sclerotic gingiva or bone barrier covering the tooth

Diagnostic procedures

Diagnostic procedures for DTE consist of careful intraoral and extraoral examinations, assessment of medical and dental histories, and radiographic evaluation. As mentioned earlier, longitudinal panoramic radiograph monitoring can help in early detection and differential diagnosis of disrupted tooth eruption before its complete development.

The patient's medical and family histories and information related to eruption problems of the patient and close relatives are very important parts of the examination. A wide variety of disorders have been reported in association with DTE.

Clinical examination

Clinical examination must begin with the overall physical evaluation of the patient. For example, the presence of syndromes is usually obvious, but in patients with mild forms only a careful examination can reveal abnormalities and alert the clinician to perform further investigation if needed.

Specific oral examination

Specific oral examinations should include inspection, palpation, percussion, and necessary radiographic examinations to assess tooth eruption problems. Inspection includes evaluation of the number and morphology of the teeth present by assessing tooth counts, eruption timing, and eruption sequence.

The examination must also include clinical inspection of the alveolar ridge both buccally and lingually. The size and shape of the alveolar ridges usually show the characteristic bulge of a tooth in the process of eruption and can help to reveal DTE. A thin alveolar ridge indicates the absence of teeth in the area close to the ridge, and a wide and full alveolar ridge indicates the presence of a tooth. Palpation of the ridge can indicate the condition of an unerupted tooth, whether it is in the bone or is close to piercing the soft tissue. Careful observation and palpation of the alveolar ridges and soft tissue can also reveal the presence of swelling, scars, and fibrous or dense tissue.

Another important component of intraoral evaluation is assessment of the general tooth eruption pattern in the mandible and maxilla and especially comparison of the left and right segments of each arch. Any asymmetric pattern of eruption, including

overretained primary teeth or asymmetric eruption of the permanent dentition on the left and right sides of the arch (more than 6 months' difference), can be an important sign of eruption problems (Figs 10-1 and 10-2).

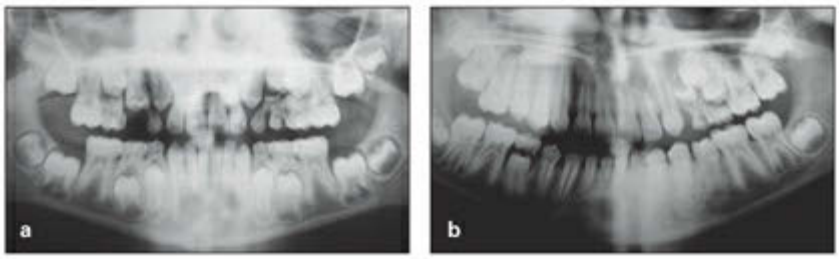


Fig 10-1 *(a)* Developing, detectable eruption problem in the maxillary left segment, which received no intervention. *(b)* Result of asymmetric eruption in the maxillary left segment.

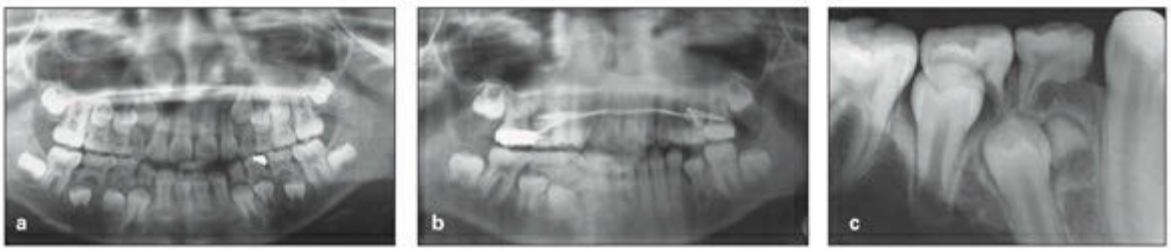


Fig 10-2 *(a)* Original, normal eruption pattern. *(b)* Developing asymmetric eruption. *(c)* Resulting problem.

Diagnostic procedures for eruption problems cannot be completed without careful radiographic evaluation. Several kinds of radiographic techniques are available for this purpose (see chapter 3).

Systemic factors also must be considered in clinical examination. Further evaluation and consultation with other medical professionals might be required. Delayed eruptions caused by systemic factors are usually associated with a generalized delayed timing in all tooth emergence and show no change in the sequence of eruption. In contrast, when delayed eruption has a local cause, the number of affected teeth are few (in a short segment), and the order of tooth eruption is also altered, such as an asymmetric type of eruption on the left and right sides of the arch.

Radiographic evaluation

Gron[30] reported that the basis for distinguishing a delayed eruption is the root length at the time of eruption. Under normal circumstances, tooth emergence begins when three-quarters of the final root length is established. The lengths of roots at the time of eruption are not exactly the same for different teeth, however. For example, the

canines and permanent second molars erupt after three-quarters' root length has been completed, and mandibular incisors and permanent first molars erupt before completion of three-quarters' root length.

DTE resulting from local factors or abnormalities in tooth or root development, such as ankylosis, space loss, or the presence of any obstacle, such as a supernumerary tooth, odontoma, or cyst, can be diagnosed by careful radiographic evaluation. DTE might be the result of defective tooth formation, and the first step in examination should be to assess whether the defect is localized or generalized. Retarded formation and eruption of mandibular second premolars is a common problem that must be carefully evaluated in all kinds of treatment planning and especially before serial extraction and space management (Fig 10-3).

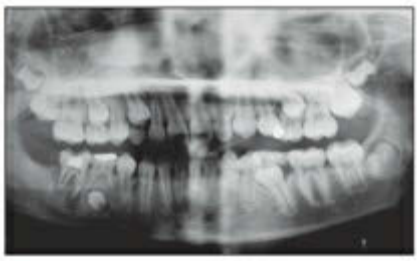


Fig 10-3 Retarded development of the mandibular right premolar. The left premolar shows complete development.

As mentioned earlier, several local and general factors can delay eruption of the permanent dentition, including ankylosis, cyst formation, tooth displacement, supernumerary teeth, and space deficiencies (Figs 10-4 to 10-7).

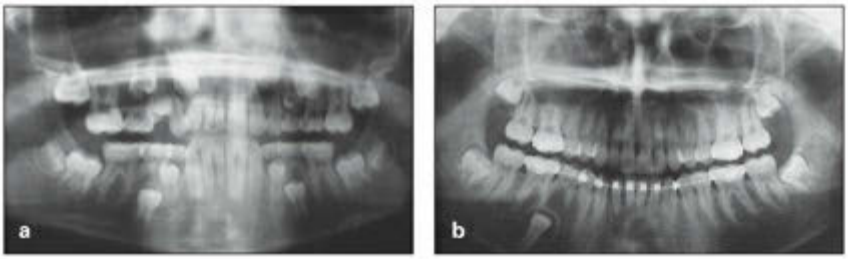


Fig 10-4 Mandibular right premolar, unerupted because of ankylosis. *(a)* Panoramic radiograph taken in the late mixed dentition. *(b)* Panoramic radiograph taken 3 years later, showing changes to the right premolar, which should have been extracted earlier.

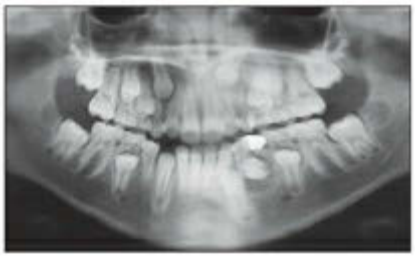


Fig 10-5 Cyst formation, preventing eruption of the mandibular left canine and premolar.

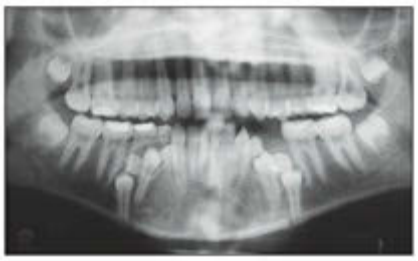


Fig 10-6 Four supernumerary teeth, preventing eruption of all mandibular premolars. The supernumerary teeth should have been extracted long ago.

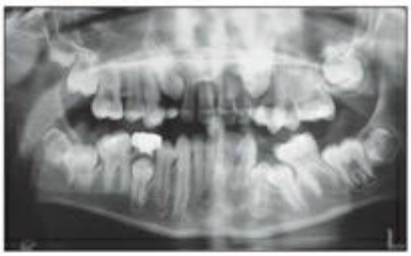


Fig 10-7 Space loss, preventing eruption of both mandibular left premolars.

Therapeutic considerations

DTE presents a challenge for orthodontic treatment if it is not diagnosed and treated at the initial stages of development. The best management of DTE, when the teeth do not erupt at the expected age, is early detection through longitudinal monitoring of panoramic radiographs and timely intervention in the problem.

If the problem has already developed, a careful evaluation should be performed first to establish the etiology of the problem and the stage of the problem; then the treatment plan can be designed accordingly. After careful examination, determination of the problem and the etiologic factors, and establishment of the type and stage of DTE, a number of options are available for management of this abnormality:

- If all diagnostic data indicate a poor prognosis and hopeless situation for the affected tooth, the management is extraction of the affected tooth or teeth and subsequent space closure or prosthodontic or implant replacement. These types of teeth include unerupted and deeply ankylosed permanent teeth and unerupted teeth with severely deformed tooth structure (see Fig 10-4).
- If DTE has affected a primary tooth, it must first be kept under close observation. An unerupted primary tooth with serious defects should be extracted, and,

depending on the condition of the succedaneous tooth and stage of eruption, space must be maintained until permanent tooth eruption.

- If the affected tooth is severely malformed, permanent teeth usually must be extracted, but the extraction can be postponed until after close observation of the growth period and appropriate development and preservation of the surrounding alveolar ridge. Occasionally, defective teeth can be saved after eruption with a crown or to serve as abutments for restorative options.
- If an obstacle is preventing normal eruption, the best option is surgical elimination of the obstacle to facilitate eruption. This obstacle can be fibrotic tissue, a bone barrier, an ankylosed primary molar, a remaining root, a cyst, a supernumerary tooth, or an odontoma. If the DTE results from a lack of space and crowding, the best option is space regaining or space creation, space maintenance, and guidance of eruption. Depending on etiologic factors and the type of problem, different options might be needed after surgical removal of the obstacle:
  - Some patients may need only a period of observation or space maintenance for the unerupted tooth and then observation.
  - Some patients may need space regaining after removal of the obstructing factor if the space has been lost and then observation.
  - Some patients, even after surgical removal of the obstacle, may also need orthodontic traction of the delayed or unerupted tooth, which can be achieved by bonding an attachment to the affected tooth and then initiating tooth traction.
- If clinical and radiographic evaluation of DTE indicates no obvious developmental defect, such as root deformity, ectopic tooth position, or physical obstruction, periodic observation is recommended.
- If observation indicates that the tooth is lagging in its eruption status and the root has reached two-thirds its length, active orthodontic treatment and traction must be implemented.
- If DTE has a systemic or syndromic cause, a team of specialists might be required for proper testing and treatment procedures.

Early Exfoliation of Primary Teeth and Premature Eruption of Permanent Teeth

In the same way that the timing of eruption of primary and permanent dentition may vary, the timing of exfoliation may differ among individual children. There are also

certain occasions when early exfoliation of primary teeth and early eruption of permanent successors might need attention and management.

Etiology

Local factors

Untreated and severe caries affecting primary teeth and mouth injuries suffered during sports or accidents are common factors in early tooth loss. The most frequent cause of premature loss of teeth is accidents, especially in children. The teeth that are lost with the greatest frequency are the maxillary central incisors, especially in children with severe overjet due to severe incisor protrusion.

Local factors that can cause early exfoliation of the primary teeth are longstanding periapical abscess and aggressive periodontitis, which cause early shedding of primary teeth and early resorption of the covering bone of permanent successors and early permanent tooth eruption. Figure 10-8 shows panoramic radiographs of two patients who lost their right primary molars due to local infection and abscess, resulting in early eruption of the first premolars, which have short roots, are mobile, and will need to be maintained. In contrast, on the left side the primary molars are present, and the permanent successor is far from eruption.

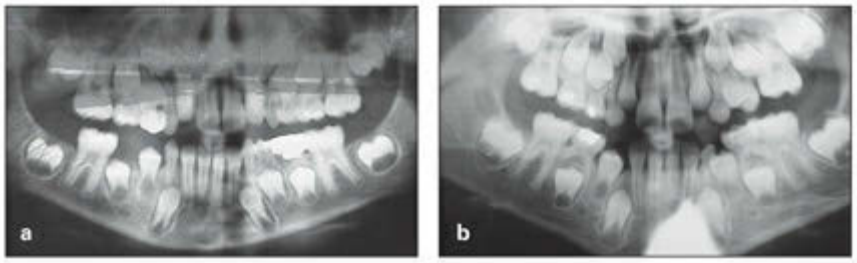


Fig 10-8 *(a and b)* Early eruption of the mandibular right premolar, which has very short roots and is very mobile.

Pathologic and systemic factors

Although premature loss of the primary teeth in conjunction with early eruption may be of no clinical significance, early exfoliation in children younger than 5 years of age without any history of trauma should not be overlooked by the clinician. These situations need special attention because they can be related to pathologic conditions of local or systemic origin. The following are some examples of systemic conditions that cause early exfoliation.

Hypophosphatasia. Hartsfield[32] stated that premature loss of teeth associated with systemic disease results from some change in the immune system or connective tissue. The most common of these conditions appear to be hypophosphatasia and early onset of periodontitis. Hypophosphatasia is a rare inherited metabolic disease that affects bone mineralization due to mutations in the *ALPL* gene. The *ALPL* gene plays an important role in signaling the production of an enzyme called *alkaline phosphatase*, which plays an essential role in mineralization of the skeleton and teeth. The loss of function that results from mutation of the *ALPL* gene disrupts the process of mineralization; deposition of minerals such as calcium and phosphorus in developing bones and teeth is affected.

Hypophosphatasia can appear anywhere from before birth to adulthood. The most severe forms of this disorder occur before birth and in early infancy. Because of mineral deficiency, bones are weak and soft, causing skeletal abnormalities similar to those found with rickets. Childhood forms are less severe, and early loss of anterior primary teeth associated with deficient cementum is one of the first signs of the condition in children. Tooth loss may be spontaneous or may result from slight trauma to the anterior teeth.

Agranulocytosis. Agranulocytosis, also known as *agranulosis* or *granulopenia*, is an acute condition in which there is a severe decrease in the production of granulocytes, most commonly neutrophils, so that a pronounced neutropenia affects the body, leaving it defenseless against bacterial invasion. Agranulocytosis is associated with gingival diseases such as gingival bleeding, hypersalivation, halitosis, osteoporosis, destruction of the periodontal ligament, and consequently early exfoliation of teeth.

Severe oral infection. Early exfoliation and tooth loss can also be seen in patients with severe oral infections caused by other disorders, such as Wiskott-Aldrich syndrome, diabetes mellitus, or herpes zoster.

Wiskott-Aldrich syndrome was described first by Wiskott in 1937 and later by Aldrich in 1954. It is a rare, X-linked recessive immunodeficiency disease characterized by eczema, thrombocytopenia (low platelet count), immune deficiency, and bloody diarrhea (secondary to thrombocytopenia).

Periodontitis. Another cause of early tooth loss is periodontitis. The term *periodontitis* is used to describe a group of multifactorial diseases that can result in the progressive destruction of the periodontal structures, including the periodontal ligament, cementum, and alveolar bone, and ultimately lead to tooth loss.

Acrodynia. Acrodynia, also known as *mercury poisoning* or *pink disease*, is a disease caused by exposure of young children to mercury or its compounds. Clinical manifestations of acrodynia include fever, anorexia, desquamation of the soles and palms (causing them to be pink), sweating, tachycardia, gastrointestinal disturbance, and hypotonia.[33] The oral findings include inflammation and ulceration of the mucous membranes, excessive salivation, loss of alveolar bone, and premature exfoliation of teeth.

Radiation therapy. Radiation therapy for malignancies of the orofacial region causes xerostomia resulting from salivary gland destruction; the hyposalivation is responsible for the development of cervical caries. Another complication is osteonecrosis, which in many instances is initiated by severe periodontal involvement. Loss of teeth is the end result of these secondary radiation effects.

Failure of Eruption

Another kind of eruption problem is complete failure of eruption of the tooth. PFE is a rare and poorly understood condition usually characterized as a nonsyndromic failure of eruption of permanent teeth when no mechanical obstruction is present.

PFE affects posterior quadrants and causes a dramatic posterior open bite that requires a complex management strategy. Proffit and Vig[34] stated that failure of posterior teeth to erupt fully into occlusal contact is the cause of this kind of open bite. According to Proffit and Vig,[34] this problem cannot be attributed to a mechanical interference with the eruptive process (eg, ankylosis or soft tissue interference). PFE affects all teeth distal to the most mesial involved tooth, while mechanical failure of eruption affects only the involved tooth or teeth and is primarily the result of ankylosis or soft tissue interference with eruption. Early distinction between PFE and mechanical failure of eruption is clinically important because it determines whether all posterior teeth, or only an individual affected tooth, will not respond to orthodontic force (Fig 10-9).

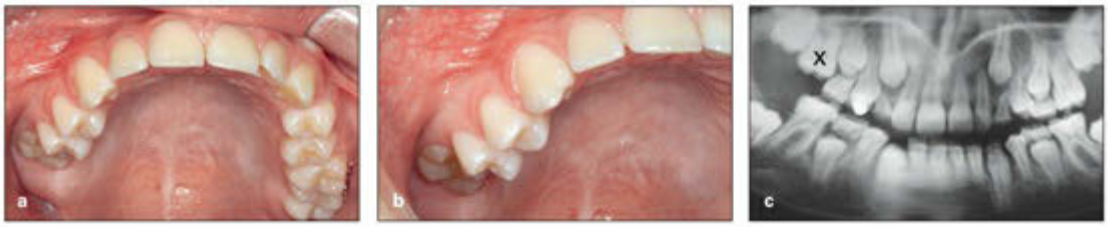


Fig 10-9 *(a to c)* Primary failure of eruption of the maxillary right first molar.

Periodic radiographic assessment is recommended to monitor the pattern of eruption of the teeth distal to the most mesial affected tooth. PFE usually manifests as a unilateral involvement of the maxillary and mandibular quadrants, but it can be bilateral.

Etiology

Rasmussen and Kotsaki[35] explained that unerupted primary teeth are rare and that most cases of failure involve the primary second molars. They classified instances of this anomaly as those in which the impaction is primary, meaning that the teeth have never erupted, or secondary, meaning that after partial eruption the teeth stop erupting and become reimpacted. They concluded that inheritance was the etiology of PFE in the patients they examined and explained that the mode of transmission was autosomal dominant.

Tooth eruption requires the presence of a dental follicle; as Wise[20] indicated, the dental follicle regulates the osteoclastogenesis and osteogenesis needed for alveolar bone resorption to create an eruption pathway, alveolar bone formation at the base of the bony crypt, and tooth eruption. Philbrick et al[21] concluded that PTHrP seems to regulate osteoclast formation via mediation of the dental follicle, in a manner analogous to the osteoblast-mediated process in the peripheral skeleton.

Recent studies show that PFE is inherited. Mutations in *PTH1R* genes explain several familial cases of PFE. Therefore, genetic analysis can be combined with clinical diagnostic information to ensure improved orthodontic management of PFE. Frazier-Bowers et al[36] investigated the etiology of eruption disorders by using a network pathway analysis in a rat model and found that *PTH1R* acts in a pathway with genes such as *PTHrP* that have been shown to be important in bone remodeling and eruption.

In one of the largest and most detailed analyses of teeth affected by PFE, Ahmad et al[37] conducted a systematic review of 40 cases in the literature. The review found a family history of eruption failure in almost 50% of the sample. Ahmad et al[37] reported that 60% of the affected patients were female, and first and second molars were the teeth most commonly affected. Incisors, canines, and premolars were also involved but with less frequency. There were no significant differences in incidence between the maxilla and mandible or between the left and right sides.

In a more recent study, Proffit and Frazier-Bowers[38] reviewed pre-emergent and postemergent eruption, with particular emphasis on distinguishing between isolated

molar ankylosis, PFE, and genetic considerations in eruption problems. The study revealed that, in conditions of PFE, all teeth distal to the most mesial affected tooth will fail to erupt and cannot be moved orthodontically. If the problem is isolated ankylosis of the first molar, however, the second and third molars are likely to be normal and will erupt on their own and can be moved orthodontically into the first molar area when the ankylosed tooth is removed.

The study concluded that PFE is an often overlooked cause of posterior open bite and that the diagnostic distinction between isolated ankylosis and PFE is critically important in planning treatment. Proffit and Frazier-Bowers[38] also added that genetics is an important consideration in the mechanism and control of tooth eruption and becomes a factor in differential diagnosis of eruption failure.

Stellzig-Eisenhauer et al[28] conducted a clinical and molecular genetics study in four families where at least two members were affected by nonsyndromic PFE. The study indicated that nonsyndromic PFE has an autosomal-dominant mode of inheritance. Molecular genetic analysis of the *PTHR1* gene revealed three distinct heterozygous mutations, while unaffected persons exhibited no mutations. The researchers concluded that the genetic causes of non-syndromic PFE can be used for the differential diagnosis of eruption failure.

Therapeutic considerations

Primary failure of tooth eruption can cause severe posterior open bite with a poor treatment prognosis that sometimes requires a complex management strategy. Depending on the age of the patient, the number and position of affected teeth, the developmental stage of the roots, and the severity of infraocclusion, various treatment approaches have been proposed, some with successful results, and some with failure.[39,40] Simple procedures include extraction of ankylosed primary molars, space control, and monitoring of the permanent teeth for eruption. In more complicated cases, surgical luxation followed by elevation and orthodontic traction of the affected teeth and stabilization of the lux-ated teeth in young patients with incomplete root formation have also been proposed.

When primary molars fail to erupt, orthodontic guidance of eruption is rarely indicated if problems can be detected early and managed properly. Early detection and removal of affected primary molars at the proper time, space maintenance, and monitoring of canine and premolar eruption seem to encompass the first preventive stage for managing this problem; this sequence usually facilitates permanent tooth

eruption. In some cases, orthodontic traction may be needed; however, in rare cases, traction and tooth eruption may result in failure and extraction of permanent teeth. Control of space availability and removal of bone barriers and overlying soft tissue can also facilitate premolar eruption.

Mc Cafferty et al[39] reported on an 8-year-old boy with severe posterior open bite due to PFE; both right permanent first molars failed to erupt. No significant medical history was present, and the siblings had no similar dental abnormalities. The infraoccluded right primary first and second molars were extracted, and the right permanent first molars were surgically exposed. Eruption of the premolars and molars was monitored over the next 2 years. The premolars showed signs of eruption and continued root development. Exfoliation of the remaining primary teeth proceeded as usual. The right permanent first molars did not erupt and were subsequently extracted. When the patient was 13 years old, orthodontic extrusion was used to further reduce the interdental distance between the canine and premolars.

Lygidakis et al[40] reported on a 7.5-year-old boy who presented with localized secondary eruption failure of the mandibular right permanent first molar with infraocclusion. There was no history of eruption failure or ankylosis in any other family member. Treatment was surgical luxation of the tooth, followed by elevation to the occlusal plane and immobilization to the adjacent primary molar. The splint was removed after 4 weeks. Three years' follow-up revealed successful results and no clinically or radiologically evident pathosis of the area. Root development of the affected tooth was arrested; there was no sign of pulpal necrosis.

Retarded Tooth Development

Retarded eruption of the second premolars is occasionally encountered during the transitional dentition and requires careful attention before treatment planning. This type of eruption disturbance is different from eruption problems commonly observed after space loss and following premature loss of the primary molars and subsequent tilting of the permanent molar that can cause eruption difficulties for second premolars. Retarded eruption of the second premolars is a rare anomaly that is associated with late development of tooth germs. In these cases, eruption of the premolars is excessively late, and it is difficult to predict the time of their emergence (see Fig 10-3). Because of the developmental delays, radiographic evaluation can even lead retarded tooth formation to be misdiagnosed as congenital

absence of teeth (Fig 10-10).

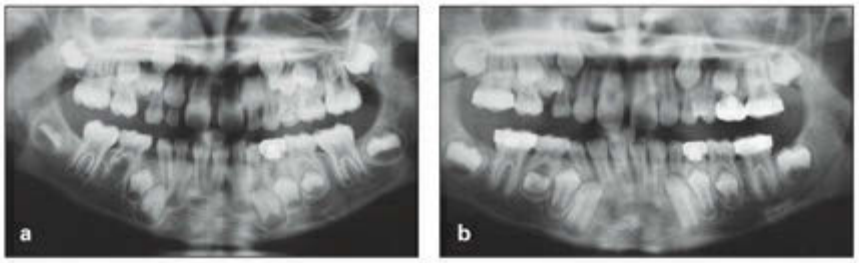


Fig 10-10 *(a)* Initial panoramic radiograph, considered to reveal congenital absence of the mandibular right premolar. *(b)* Panoramic radiograph taken 3 years later, revealing a premolar with retarded formation.

According to Massler et al,[1] calcification of the second premolar usually begins at the age of 2 to 3 years, and crown formation is usually completed by 6 to 7 years. However, there is always a wide variation in individual timing of premolar calcification and crown formation. In individuals with retarded premolar calcification, the appearance of the tooth germ may not be recognized even at 8 to 12 years.

This eruption disturbance is usually seen unilaterally but can occur bilaterally. In cases of unilateral occurrence, the degree of tooth formation on the contralateral side may help to predict the approximate age of emergence of the affected side.

In an evaluation of five unusual cases of anomalous eruption of the maxillary second premolars that related to late development of the germs, Taguchi et al[41] reported that the affected premolars emerged between the ages of 12 years, 3 months and 14 years, 6 months. The study also found that, in some cases, this anomaly is associated with microdontia. Peterka et al[42] reported a higher prevalence of retarded tooth development in the affected side of patients with unilateral cleft palate.

In a study assessing the timing of exchange of the maxillary primary and permanent teeth in boys with three types of orofacial clefts (unilateral, bilateral, and isolated cleft), Peterka and colleagues[43] concluded that the developmental disturbances of the maxilla and maxillary teeth in patients with orofacial clefts are also associated with alteration of timing of dental exchange.

If a defect or retardation of tooth formation is present, the first step should be to assess whether the defect is localized or generalized. Local delayed eruption can be due to retardation of tooth development. Mandibular and maxillary second premolars are the most common teeth to show retardation in development that is of unknown etiology and disharmonious with the development of adjacent teeth. Therefore, the eruptive development of these teeth must be watched more carefully

during the transitional dentition. Treatment planning in the absence of a clear-cut understanding of the condition of these teeth can complicate proper treatment.

Abnormal Sequence of Eruption

A normal sequence of eruption is an important aspect of occlusal development. Variations in the sequence of tooth eruption can have direct effects on the development of the dentition and can be more problematic than tooth eruptions that are a few months early or late. Individual variations in sequence of eruption can play an important role in orthodontic treatment planning and can have a direct clinical application in occlusal guidance and early orthodontic treatment.

According to Moorrees et al, "exactly how the leeway space is utilized depends on the sequence of shedding and eruption of the maxillary and mandibular posterior teeth and the molar occlusion."[44] The normal sequence of exfoliation of the primary dentition plays an important role in the normal sequence of eruption of permanent canines and premolars and in preservation of leeway space for the permanent dentition. Any disruption during the transitional stage, such as premature loss or overretention of primary teeth, may interfere with normal tooth transition and development of occlusion. (The mechanism of the transitional dentition is discussed in more detail in chapters 2 and 5.)

The sequence of eruption of the primary and permanent dentition has been studied extensively over the past several decades. In 1953, Lo and Moyers[45] studied the sequence of eruption in 236 Canadian schoolchildren by intraoral examination and radiographs. The study attempted to determine the most frequent sequence of eruption and the final effects of the different sequences on occlusion (according to Angle's classification). The researchers found 18 different sequences in the maxilla. The most frequent sequence was 6 1 2 4 5 3 7, in 48.72% of children studied, and the second most frequent sequence was 6 1 2 4 3 5 7, in 16.01% of subjects (1= central incisor, 2 = lateral incisor, 3 = canine, 4 = first premolar, 5 = second premolar, 6 = first molar, and 7 = second molar). In the mandibular arch, 17 eruption sequences were observed. The most common sequence was 6 1 2 3 4 5 7, occurring in 45.77% of children, and the second most common sequence was 6 1 2 3 4 7 5, seen in 18.64% of children.[45]

In a cross-sectional examination of 6,000 children, Garn and Smith[46] studied the timing of tooth emergence and reported that the most common sequence in the maxilla was 6 1 2 4 3 5 7 and the most common sequence in the mandible was 6 1 2

3 4 5 7.

Different alterations in the sequence of eruption of the permanent teeth can have different clinical signs and different harmful effects on the occlusion, which are discussed in the following sections.

Eruption of the second molars before the premolars

If the second molars erupt prior to the premolars, the mesial force of second molar eruption can push the permanent first molar and decrease the space available for the second premolar. This may block the second premolar from the arch.

Eruption of the maxillary canines before the premolars

If the maxillary canine erupts before the maxillary first premolar, it can cause crowding or impaction of the second premolar. If the maxillary canine erupts at the same time as the first premolar, the canine will be forced labially. This condition can develop into a more complicated condition if the maxillary molar also shifts forward and the problem of space loss is added to the abnormal sequence of eruption.

Eruption of the mandibular first premolars before the canines

In normal occlusion, the mandibular and maxillary incisors are positioned in such a way that the mandibular dentition is confined within the parameters of the maxillary arch. After premature loss of the mandibular primary canines or primary first molar, the mandibular incisors tip lingually, and arch length is decreased anteroposteriorly. A normal eruption sequence and positioning of the mandibular permanent canines with a strong root and slightly distal position provide a strong support for normal mandibular incisor positioning. Any disturbances in the sequence between mandibular canine and first premolar can cause lingual tipping of the mandibular incisors and lingual or buccal displacement of the mandibular canines.

When the sequence of eruption in the mandibular arch is normal (3 4 5) and slight crowding is observed in the mandibular incisor area, the canine erupts into the space created by the exfoliation of the primary canine and moves slightly distally, creating a small amount of space for those crowded incisors.

Sampson and Richards[47] stated that, depending on the timing and sequence of eruption of the canine, various contact points are formed between the lateral incisor and the canine. They reported that incisor crowding seems to be greatest when the canine erupts buccally.

When incisor crowding is severe and tooth size–arch size discrepancy is present, the canine may be impeded in its eruption by the primary first molar, or exfoliation of the primary first molar may be accelerated, and the first premolar consequently may erupt before the canine. In this situation, the first and second premolars will occupy leeway space, causing canine impaction. If mandibular canines and first premolars erupt, occupying some leeway space, impaction or lingual eruption of the second premolars is possible.

Eruption of the maxillary canines before the first premolars

The sequence of eruption of the maxillary canine and premolar usually proceeds from the posterior to the anterior region of the arch. If the canine erupts before the premolar and space is deficient, the canine can push the maxillary incisors forward to create space or push the first premolar out of the arch. If the maxillary primary second molar is exfoliating, it can cause second premolar impaction.

Eruption of the maxillary second molars before the mandibular second molars

This abnormal sequence can push the maxillary first molar mesially and cause a Class II molar relationship. This condition occurs more easily if the maxillary and mandibular permanent first molars are in an end-to-end relationship.

Eruption of the maxillary lateral incisors before the central incisors

There are some occasions when the lateral incisors may erupt before the central incisors, such as in the presence of overretained primary central incisors, when there is very premature loss of primary central incisors, after development of sclerotic tissue, in the presence of a dense bone barrier, or in the presence of a mesiodens.

Any of these conditions and the consequent potential for eruption of lateral incisors before central incisors requires early detection and intervention. It is important to eliminate the cause and maintain the space for the central incisors, or crowding, uneruption, and impaction of the central incisors are inevitable.

Asymmetric eruption in the left and right sides of the arch

In all types of eruption sequences, usually there is a symmetric pattern between the left and right sides of the arch with some time variation among different individuals. Most investigators accept a maximum of 6 months' difference between left and right sides of the arch as normal. Asymmetric eruption between the left and right sides of the arch that persists for more than 6 months indicates the presence of some problem and requires a careful radiographic evaluation (Fig 10-11).

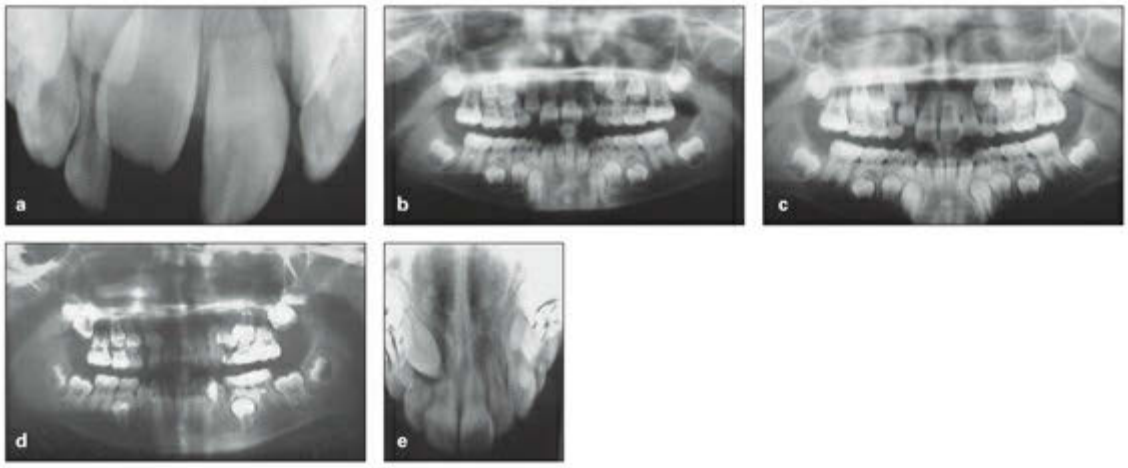


Fig 10-11 Asymmetric eruption that, due to negligence, was not evaluated at the proper time and caused various occlusal problems. *(a)* Occlusal radiograph taken in 1999, showing asymmetric eruption of the central incisors. *(b)* Panoramic radiograph taken in 2000, showing asymmetric eruption continuing with the lateral incisors. *(c)* Panoramic radiograph taken in 2003, showing the continued asymmetric eruption of the lateral incisors and the proximity of the maxillary right canine to the lateral incisor root. *(d)* Panoramic radiograph taken in 2004, showing complete resorption of the lateral incisor root. *(e)* Occlusal radiograph taken in 2004.

Application of longitudinal panoramic radiograph monitoring during the transitional dentition can be very helpful in the early detection of asymmetric eruption or abnormal sequence of eruption, and careful intervention can prevent damage to the occlusion. Utilization of permanent molar anchorage, such as a lower holding arch, Nance appliance, or transpalatal arch, and guidance of eruption by selective grinding or primary tooth extraction can change the pattern of tooth

eruption and intercept many future occlusal problems (see chapters 3 and 5).

The case shown in Fig 10-11 is an example of asymmetric eruption that, due to negligence, was not evaluated at the proper time and caused various occlusal problems. Two early radiographs show evidence of asymmetric eruption of the central and lateral incisors (see Figs 10-11a and 10-11b). The next panoramic radiograph, taken 3 years after the first one, shows continuation of the asymmetric eruption of the lateral incisors and the close proximity of the maxillary right canine crown to the lateral incisor root (see Fig 10-11c). No early intervention was initiated at this time, and the parents were asked to bring the child back for a checkup within 6 months. Unfortunately, they returned 2 years later. The final radiographs, taken 2 years later when the child was brought to clinic with a mobile maxillary right lateral incisor, show the complete resorption of the root (see Figs 10-11d and 10-11e).

Ectopic Eruption

Ectopic eruption, or *ectopia*, is defined as eruption that is out of the normal position. Ectopic eruption is a developmental disturbance in the eruption pattern of the teeth causing alteration of the tooth's eruption pathway and creating problems for adjacent teeth.

Prevalence

According to Weinberger,[48] the incidence of ectopic eruption ranges from 2% to 4.3% of the population. It occurs more often in the maxilla and more often unilaterally. The teeth that are most frequently found to be ectopic are maxillary first molars, maxillary canines, mandibular second premolars, and mandibular canines.

Ectopic eruption of permanent first molars

Ectopic eruption of the permanent first molar is a common problem during the early mixed dentition. In this situation, the permanent first molar erupts at a mesial angulation toward the primary second molar, resulting in a cessation of eruption that causes atypical resorption of the adjacent primary molar (Fig 10-12a). The ectopic permanent tooth may get locked in this position or can correct itself without treatment and erupt into a normal position later. These two types of ectopia are

classified as reversible (jump) and irreversible (hold) ectopic eruption.

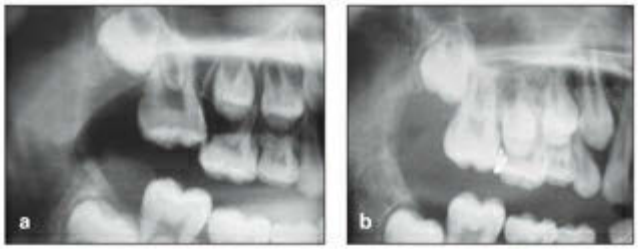


Fig 10-12 *(a)* Ectopic eruption of the maxillary right first molar, causing resorption of the primary second molar. *(b)* Wedging technique for distalization of the molar. Brass ligature wire has been placed between the first molar and adjacent teeth.

Etiology of ectopic molar eruption

Reports indicate some familial tendency for ectopic molars.[49] Several local factors can contribute to this anomaly:

- Large primary or permanent molars
- Convexity of the distal surface of the maxillary primary second molar
- Convexity of the mesial surface of the maxillary permanent first molar
- Missing maxillary second premolar
- Growth deficiency of the maxillary tuberosity and posterior positioning of the maxilla
- Mesially directed path of eruption of the permanent first molar
- Delayed development of the permanent first molar

Sequelae

Early detection and proper intervention can prevent many destructive sequelae. Delayed correction of this anomaly can have consequences such as pulpal infection of the primary second molar, abscess formation, premature loss of the primary second molar, space loss and tipping of the permanent first molar, and impaction of the second premolar.

Treatment options for ectopic molars

Traditionally, three different strategies have been applied in the management of ectopic permanent molars: observation, extraction of the primary molar, and distalization of the permanent first molar. An alternative treatment for mandibular

molar ectopia involves disking of the distal surface of the primary second molars, which allows the permanent molars to erupt.

Observation. One option is to observe the area for 5 to 6 months; not all ectopic molar eruptions require treatment. For example, treatment is not necessary if the permanent first molar has moved less than 2 mm toward the primary second molar and the long axis of the permanent molar shows minimal tipping.

Extraction of the primary molar. The second option is extraction of the primary molar—if pain or mobility is present— or waiting for exfoliation of the primary second molar, followed by an orthodontic approach, which involves distalization of the permanent molar and space regaining.

Distalization of the permanent first molar. The third option is distalization of the permanent molars through the use of orthodontic appliances and salvaging of the primary molar. There are two general strategies for distal movement of ectopic molars: the interproximal wedging technique and distal tipping of the ectopic molar.

The interproximal wedging technique can be applied when the ectopic tooth is not severely inclined and there is minimal impaction of the permanent first molar on the distal aspect of the primary second molar. Application of brass ligature wire is an old interproximal wedging technique and a frequently used treatment method for distalization of ectopic molars (Fig 10-12b); separating elastic (O-ring) can also be used in a wedging technique (Fig 10-13).

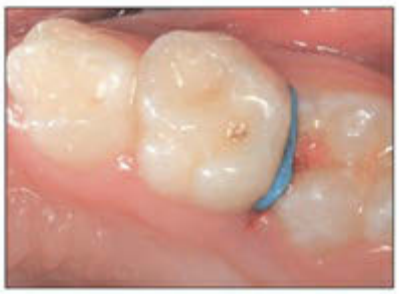


Fig 10-13 Distalization of an ectopic molar with a separating elastic (O-ring) used for the interproximal wedging technique.

When impaction is severe, the best option for ectopic treatment is distal tipping with active appliances, whether fixed or removable. The length of treatment will vary among patients. A treatment period of 1 to 3 months would be considered normal. The treatment goal is to upright and achieve normal angulation of the long axis of the permanent first molar.

Different fixed and removable appliances have been used for distalization and uprighting of ectopic molars. These include the elastic Halterman appliance, the Weinberger appliance, the de-impactor spring, and removable appliances.

Elastic Halterman appliance. This appliance consists of a band adjusted to the primary molar and a 0.036-inch stainless steel wire soldered to the buccal side of the band. The wire is extended distally behind the ectopic permanent molar and has a hook for attachment to a power chain. The other end of the power chain is fixed to an attachment bonded to the occlusal surface of the ectopic molar for distalization of the molar (Figs 10-14 and 10-15).

Fig 10-14 Halterman distalizer. (Courtesy of Great Lakes Orthodontics.)

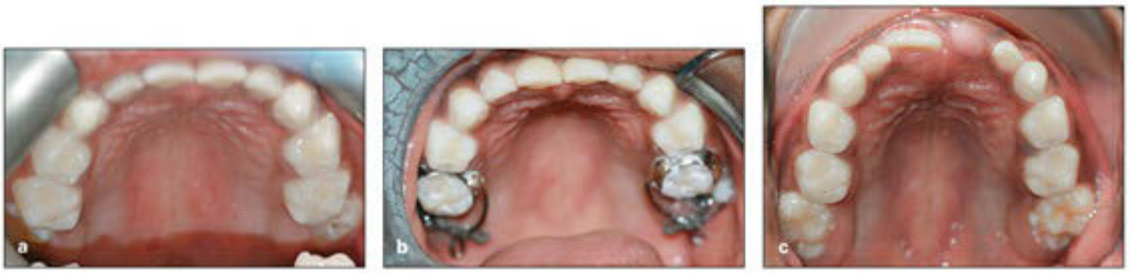


Fig 10-15 Management of two ectopic maxillary molars. *(a)* Pretreatment occlusion. *(b)* Treatment with two modified Halterman appliances. *(c)* Posttreatment occlusion after distalization of the molars.

Weinberger appliance. Designed by Weinberger[48] in 1992, this appliance is a bilateral distalizer and has distal extensions on both sides. Both extensions are fabricated using 0.036-inch wire and have small hooks on their distal aspect. The primary first molars are banded, and the bands are soldered to a transpalatal bar. An acrylic button is incorporated in the appliance for anchorage. Buttons are bonded on the disto-occlusal aspect of the ectopic molars. To provide distal force on both permanent molars, chain elastics are attached to both buttons and placed over the hooks on the distal aspect of both extensions (Fig 10-16).

Fig 10-16 Weinberger appliance for bilateral distalization. (Courtesy of Great Lakes Orthodontics.)

The force of this kind of distalizer can be activated every 2 to 3 weeks by shortening the elastics. If the primary second molar has already exfoliated and some space loss has occurred, activation can be continued until the lost space is regained. If necessary, this procedure can be followed by maintenance of the permanent molar with a Nance appliance or transpalatal arch.

De-impactor spring. The de-impactor spring is a kind of separating spring inserted between molars for tooth separation and band adjustment; it is also an efficient device to upright a locked permanent molar and can be used easily for minor molar distalization. The position of the spring can be checked every 3 to 4 weeks to ensure that the loops are well engaged. It is usually not necessary to adjust the spring during each visit, if the initial placement was tight (Fig 10-17).

Fig 10-17 De-impactor separating spring.

Removable appliances. Removable appliances such as a Hawley appliance with jackscrew or spring have also been applied for distalization of ectopic molars.

Ectopic eruption of permanent canines

Excluding the third molars, the maxillary canines are the permanent teeth with the highest tendency for ectopia, because maxillary canines are the last permanent teeth

to erupt (excluding third molars) and have the longest and most tortuous eruption route.

Ectopic canines cause various complications, such as impaction, resorption of adjacent teeth, transposition, and formation of dentigerous cysts. Therefore, for early intervention, it is important to evaluate a patient's canine positions no later than 10 years of age by a thorough clinical and radiographic examination, including visual inspection and palpation of the buccal sulcus and palatal mucosa. Longitudinal panoramic radiograph monitoring, careful supervision of the developing dentition during the transitional dentition, and proper intervention may prevent complications (for more detail, see the section on canine impaction later in this chapter and chapter 3).

Figure 10-18 illustrates treatment of a 13-year-old girl with an ectopic canine that resulted in resorption of the maxillary right central incisor root and then exfoliation of the central incisor.

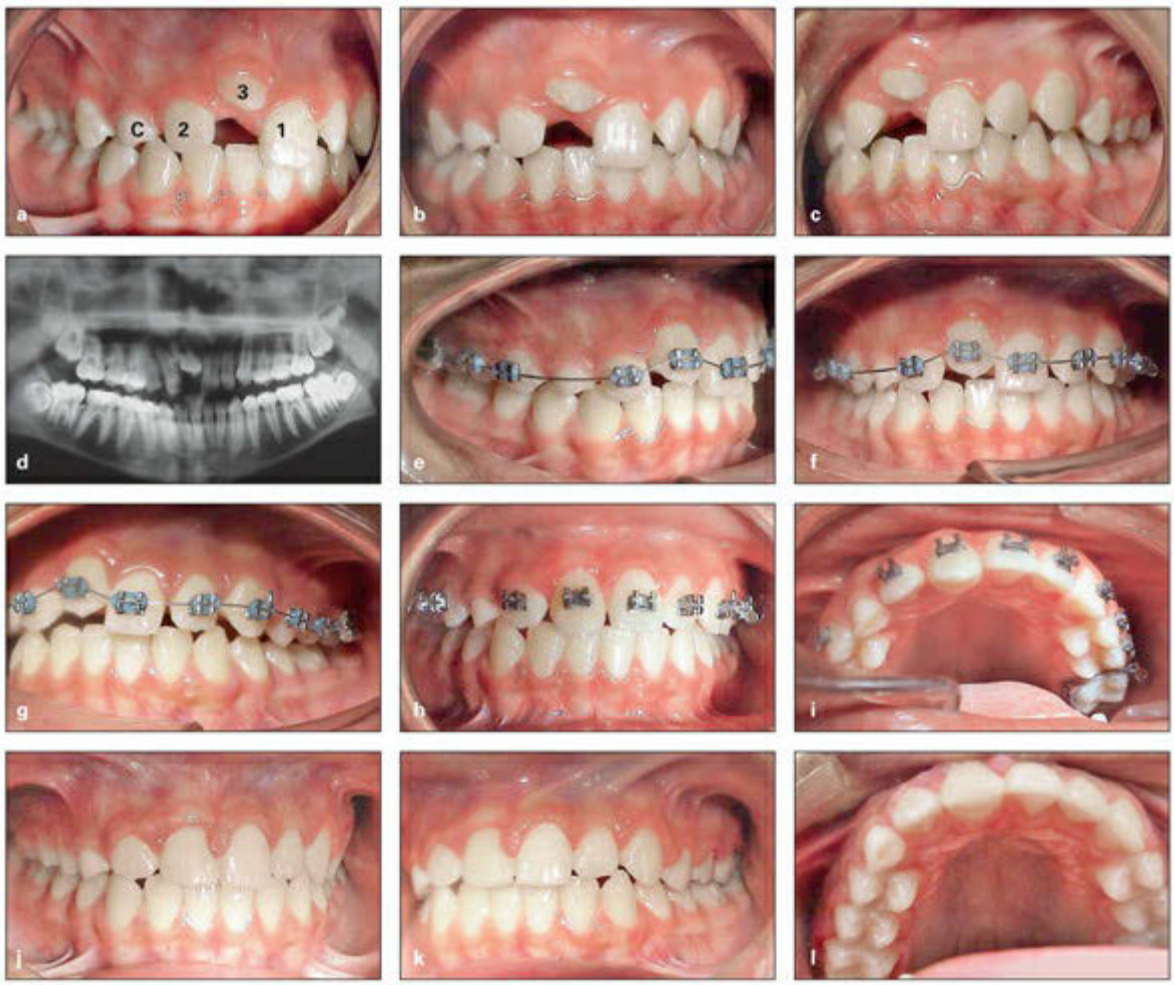


Fig 10-18 Management of an ectopic maxillary canine that has caused resorption of the permanent central incisor root and subsequent exfoliation. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e to h)* Occlusion during active treatment and leveling. The

canine bracket has a higher K distance to achieve elongation. *(i to l)* Posttreatment occlusion, after end of active treatment and reshaping of the canine to mimic the central incisor. 1—permanent central incisor; 2—permanent lateral incisor; 3—permanent canine; C—primary canine.

Tooth Transposition

Another kind of eruption disturbance is tooth transposition, or positional interchange of two adjacent teeth, especially their roots. Tooth transposition is a rare but clinically difficult developmental anomaly. Depending on the transposed teeth and their position, normal eruption of adjacent teeth can be affected, root anatomy can be damaged, and eruption of the affected teeth can be delayed. This eruption disturbance was first defined in 1849 by Harris,[50] who described tooth transposition as an "aberration in the position of the teeth."

Transposed teeth are classified into two types of tooth displacement: complete transposition and incomplete transposition (Fig 10-19). In complete transposition, both the crowns and the entire root structures of the involved teeth are displaced to abnormal positions. In incomplete transposition, only the crown of the involved tooth is transposed, and the root apices remain in place.

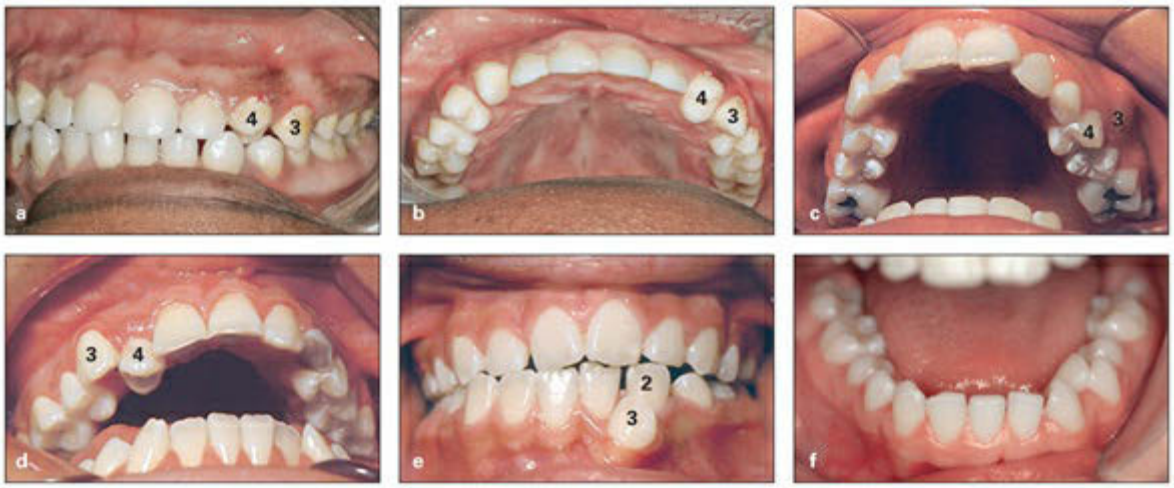


Fig 10-19 *(a to f)* Different varieties of tooth transposition. 2—lateral incisor; 3—canine; 4—premolar.

Transposition is sometimes accompanied by other dental anomalies, such as peg-shaped lateral incisors, congenitally missing teeth, crowding, overretained primary teeth, dilacerations, and rotation of adjacent teeth.

Displacement of one tooth from one quadrant across the midline to the other side of the arch has very rarely been reported, but according to Shapira and Kuftinec[51]

these types of anomalies should be considered ectopically erupted teeth, not transposed teeth.

Incidence

Transposition may occur in either jaw but appears with more frequency in the maxilla; it is usually unilateral and seldom bilateral. It may be associated with other dental anomalies, and it can affect both sexes but has a higher frequency in females.

The teeth most frequently involved in the maxilla are canines, premolars, and lateral incisors. The most commonly transposed tooth in the mandible is the lateral incisor. Transposition has never been reported in both jaws simultaneously or in the primary dentition. The canine seems to be involved in almost all kinds of transposition. Shapira and Kuftinec[51] reported an approximate ratio of 12 to 1. Huber et al[52] reported that the incidence of transposition in maxillary teeth is approximately 1 in every 300 patients.

In 85 cases of dental transposition, Ely et al[53] used panoramic radiographs and clinical records to assess 75 subjects (27 boys and 48 girls with a mean age of 12.25 years). They reported that 76% of the transpositions occurred in the maxillary dentition and 24% in the mandibular dentition. Unilateral transposition accounted for 88% of cases.

Overall, they found that the most common transposition involved the canine and first premolar (58%); in the maxilla, 84% of cases involved canine-premolar transposition. In the mandible, the canines and lateral incisors were most commonly transposed (73%). In the patients with unilateral transposition, they found no significant difference between left-side and right-side distribution.[53]

Shapira and Kuftinec[54] evaluated the records of 65 orthodontically treated individuals with maxillary tooth transpositions (40 female and 25 male subjects with an age range of 9 to 25 years). The aim was to determine the distribution of maxillary transposition and evaluate accompanying dental anomalies. They reported that 55% of the transpositions involved maxillary canines and first premolars, 42% involved canines and lateral incisors, and 3% involved central and lateral incisors. Females had 60% more transpositions than did males, and more transpositions were unilateral (88%), with moderate left-side dominance (58%).

Dental anomalies associated with transposition included missing lateral incisors and second premolars, undersized lateral incisors, overretained primary canines, and impaction of permanent canines and central incisors. Severely rotated adjacent

teeth were observed on the side of the transposition. Shapira and Kuftinec[54] also proposed that the dominance of the affected side suggested that unknown local factors may play a major role in these dental anomalies.

Shapira et al[55] evaluated the prevalence of tooth anomalies in a study of 34 individuals with Down syndrome. They assessed 15 female patients and 19 male patients ranging in age from 11 to 24 years using standardized records, which included a clinical examination, dental casts, and a panoramic radiograph. The results showed notably high prevalence rates for third molar agenesis (74% of individuals older than 14 years), canine impaction (15%), and maxillary canine–first premolar transposition (15%) compared with published figures from random population samples. Maxillary canine–first premolar transposition was found in five patients (three male patients and two female patients [15%]). Two of these patients were bilaterally affected, making a total of seven transpositions in all patients.

In a systematic review, Papadopoulos et al[56] initially retrieved 590 papers and then applied inclusion and exclusion criteria to select nine studies to review. Meta-analysis of the studies found the mean prevalence of tooth transposition to be 0.33%; the prevalence rates were statistically the same in both sexes. The review also confirmed that tooth transposition occurs more frequently in the maxilla than in the mandible and presents unilaterally more often than bilaterally.

Etiology

The etiology of tooth transposition is not fully explained; a heredity background and local factors such as overretained primary teeth, abnormal eruptive path, trauma, and migration of tooth buds have been proposed.

As a potential explanation for tooth transposition, Shapira and Kuftinec[51] stated that transposition results from an interchange in location between the anlagen of the developing teeth. They believed that transposition of the maxillary canine is due to its high position and its long eruption path through the region. The permanent canine usually erupts through the labial maxillary alveolar bone, and any bony obstruction, crowding, or resistance of the adjacent teeth, such as an overretained primary canine or supernumerary tooth, may deflect the canine. If deflection is toward the palate, the canine becomes impacted palatally; if the canine is displaced mesially, it becomes transposed with the lateral incisor. When it is displaced distally, the canine can be transposed with the premolar.

Regarding overretained primary canines, Shapira and Kuftinec[51] stated that there

may be a cause-and-effect relationship between retained primary teeth and the ectopic eruption of permanent teeth. They reported that in the majority of cases with canine transposition, primary canines and lateral incisors were retained. It is not really clear whether the overretained primary canine deflects the permanent canine or an abnormal path of eruption of the permanent canine causes overretention of the primary tooth.

Trauma to the primary dentition causes displacement of permanent tooth germs. Jaw fracture can also cause tooth bud displacement (Fig 10-20). The presence of a supernumerary tooth that changes the path of eruption of an affected tooth also seems to be a conceivable factor in permanent tooth transposition. The incidence of bilateral transposition, especially in siblings, suggests a hereditary cause, according to Allen[57] and Payne.[58] An abnormal eruptive path and migration of the tooth bud for unknown reasons can also cause tooth transposition.

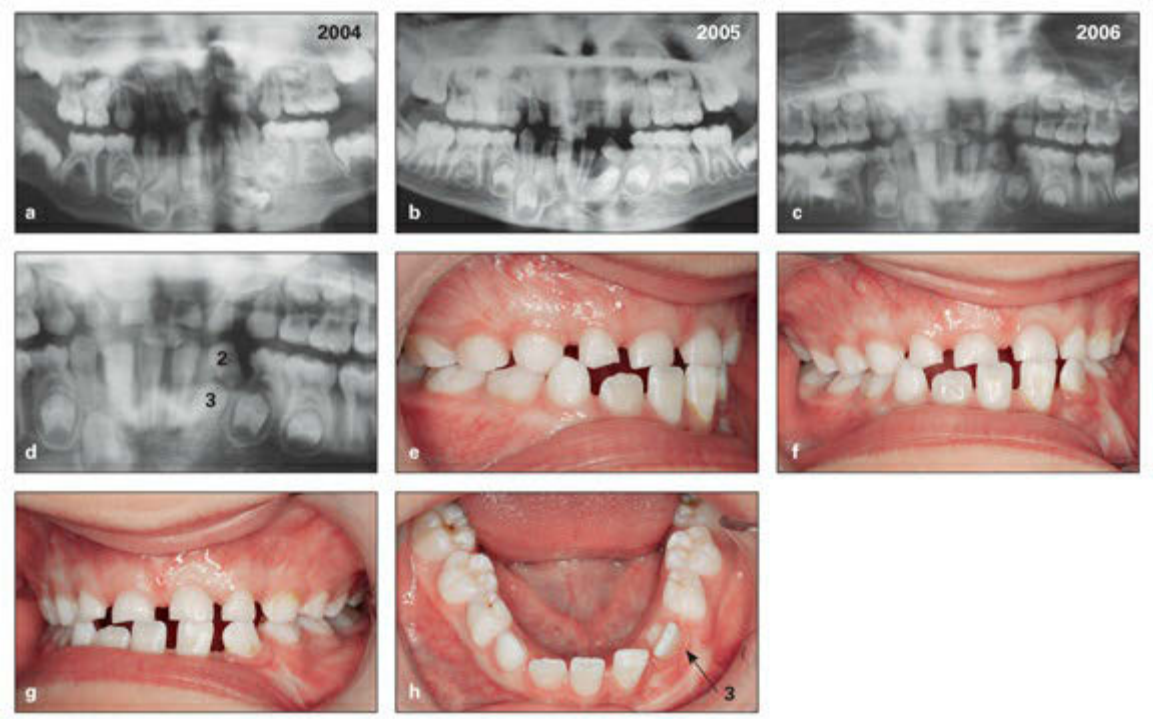


Fig 10-20 Tooth transposition in a child who had a mandibular fracture during the early mixed dentition. *(a to c)* Panoramic radiographs taken annually after the mandibular fracture. *(d)* Magnification of the 2006 panoramic radiograph, revealing the transposition of the mandibular left lateral incisor (2) and canine (3). *(e to h)* Occlusion after eruption of the lateral incisor and during emergence of the canine (3).

Through longitudinal panoramic radiograph monitoring during the transitional dentition, clinicians not only can detect transpositions earlier but also can recognize unusual transitional behavior and migration of the tooth germs that occur for unknown reasons. Having this goal in mind, the author has investigated

retrospectively many panoramic radiographs of different patients presenting with several kinds of tooth abnormalities in Iran and in Rochester, New York, over the years. The process of tooth eruption, exfoliation, and transition of the dentition is a complex phenomenon that is not clear, but it seems that several genetic and environmental factors can play roles in this process. What can be concluded from this investigation is that any change to the site of tooth eruption or transition—whether of genetic, physical, pathologic, or traumatic origin—can affect the normal eruption path of the permanent tooth buds.

The author has observed several types of abnormal tooth migration and transposition in his research. Figure 10-21a is a panoramic radiograph taken after extraction of a mandibular permanent first molar. Periapical radiographs taken 1 year later revealed distal migration of the premolar and distal root resorption of the primary second molar (Figs 10-21b and 10-21c).

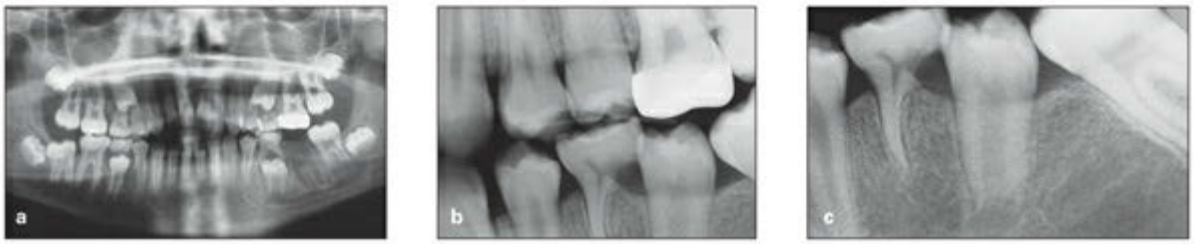


Fig 10-21 Transposition of the second premolar after tooth extraction. *(a)* Panoramic radiograph taken after extraction of the mandibular permanent first molar. *(b and c)* Periapical radiographs taken 1 year after extraction, revealing distal migration of the premolar and resorption of the distal root of the primary second molar.

Figure 10-22 shows a child in whom both mandibular permanent first molars were congenitally absent, causing migration of both premolars.

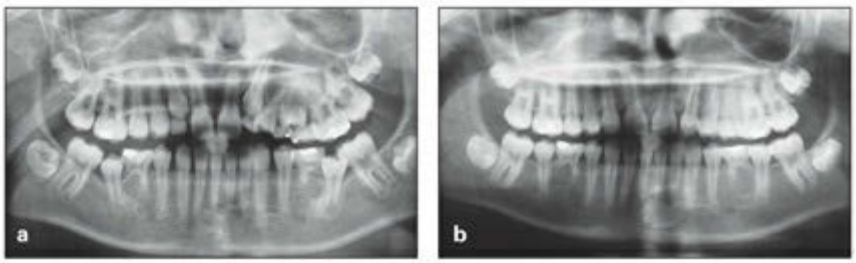


Fig 10-22 *(a and b)* Tooth transposition of the mandibular premolars in a patient with congenitally missing mandibular permanent first molars. The maxillary lateral incisors are also congenitally absent.

Figure 10-23 shows three significant processes at work during development of tooth transposition over the course of 18 months:

- The first process was an abnormal position of the mandibular left second premolar, which was preventing normal eruption of the mandibular permanent left first molar; the first molar on the right side had completely erupted (see Fig 10-23a).
- The second process was a positive intervention that was performed through extraction of the primary second molar and guidance of eruption of the abnormally positioned premolar (see Fig 10-23b).
- The third process was migration of both mandibular permanent canines, which occurred when the primary canines were restored with provisional crowns but not properly monitored, leading to infection and abscess formation (see Fig 10-23b).

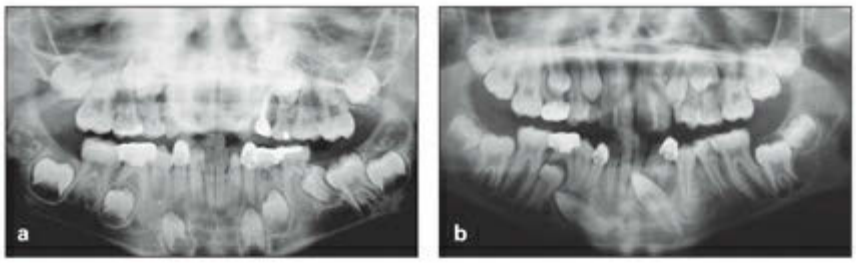


Fig 10-23 Development of tooth transposition over 18 months. *(a)* The displaced mandibular left premolar is preventing molar eruption on that side. *(b)* Positive intervention has taken place in the form of extraction of the primary left second molar and guided eruption of the abnormally positioned premolar. However, after the primary canines were restored with provisional crowns, they were not properly monitored, leading to infection and abscess formation. As a result, both mandibular permanent canines are displaced.

Diagnostic procedures

Tooth transposition, like many other developmental anomalies, takes place over a long period of time during the transitional dentition. Early diagnosis of a developing transposition is based on a thorough intraoral examination and complete radiographic analysis, especially during the first and second stages of dental transition (6 to 10 years of age). Longitudinal panoramic radiograph monitoring of patients at ages 6, 8, and 10 years can identify tooth transposition at an early stage of development. Proper intervention can prevent problems or reduce their severity.

When incipient transposition is recognized at an early stage of development, early intervention, such as extraction of a supernumerary tooth, removal of a cyst, extraction of an overretained primary canine, or space opening, can guide the ectopic eruption to a normal pathway. Delayed intervention of an incompletely transposed tooth can allow complete transposition and lead to a more complicated treatment situation.

Diagnosis of transposition at a later age, when the anomaly has already developed, consists of a thorough clinical evaluation by observation and palpation around the affected area and radiographic evaluation at different angulations. Sometimes computed tomography (CT) can be helpful in detecting the exact angulations and positions of the affected teeth. Good diagnostic procedures reveal the teeth involved and whether it is a complete or incomplete transposition. These factors play an important role in treatment planning, understanding the type of tooth movement, and prognosis.

Treatment considerations

Detection of transposed teeth at an early stage of development and recognition of etiologic factors make it possible to intervene and correct the eruptive path of the transposed tooth. The following interceptive actions can be effective toward this goal:

- Removal of any obstacle preventing a normal path of eruption of the affected tooth, such as an overretained primary tooth, supernumerary tooth, or cyst
- Opening of space in the arch if space deficiency is preventing a normal path of eruption
- Surgical uncovering of the transposed tooth and subsequent orthodontic movement of the tooth

Treatment options

Depending on the type of transposition (complete or incomplete), the stage of development of the anomaly, the amount of alveolar bone support (which is different in the maxilla and the mandible), and the patient's type of occlusion, the transposed tooth can be treated by one of the following three options:

1. Orthodontic treatment can be used to move the transposed tooth to its normal position.
2. The transposed tooth can be left in its present location, and the adjacent tooth can be aligned accordingly. Restorative procedures can be used to improve the esthetics of the teeth.
3. If neither of the other options can be applied, the transposed tooth may be

extracted. This option is commonly selected if the transposed tooth is deeply displaced and has an incorrect inclination.

Correction of incompletely transposed teeth, where the crowns are transposed but the root apices are in their normal position, is usually easier than correction of completely transposed teeth and will have an esthetically and functionally good outcome.

The possibility of moving the transposed tooth to its normal position (option 1) is much greater in the maxilla than in the mandible because the maxilla provides a wider area of alveolar bone support. The prognosis for exchanging the affected teeth is much better when the transposition occurs in the anterior segment of the maxilla, such as the lateral incisor and canine or the canine and premolar. Successful application of this option is almost impossible in the posterior segment (eg, between the premolar and molar).

Case 10-1

A 12-year-old girl presented with complete transposition of the mandibular left canine and mandibular left lateral incisor and mesial tipping of the mandibular canine. The mandibular left primary canine was still present. She had good occlusion on the right side and space loss on the maxillary left side. She had a straight profile. Figures 10-24a and 10-24b show pretreatment photographs after exfoliation of the primary canine and eruption of the permanent canine. Figure 10-24c shows the pretreatment panoramic radiograph.

Treatment:
Because of the divergency between the roots of the permanent canine and the lateral incisor and because of the volume of mandibular basal and alveolar bone, the treatment plan was to keep the canines and lateral incisors in their new positions, close the space of the missing second premolar on the same side, and achieve complete root parallelism.

The treatment sequence was leveling, uprighting, and root parallelism for the mandibular left canine, lateral incisor, and first premolar; space regaining for the maxillary left second premolar; and Class III mechanics against a maxillary solid arch to mesialize the mandibular left molars and close the space where the mandibular left premolar was missing (Figs 10-24d to 10-24g).

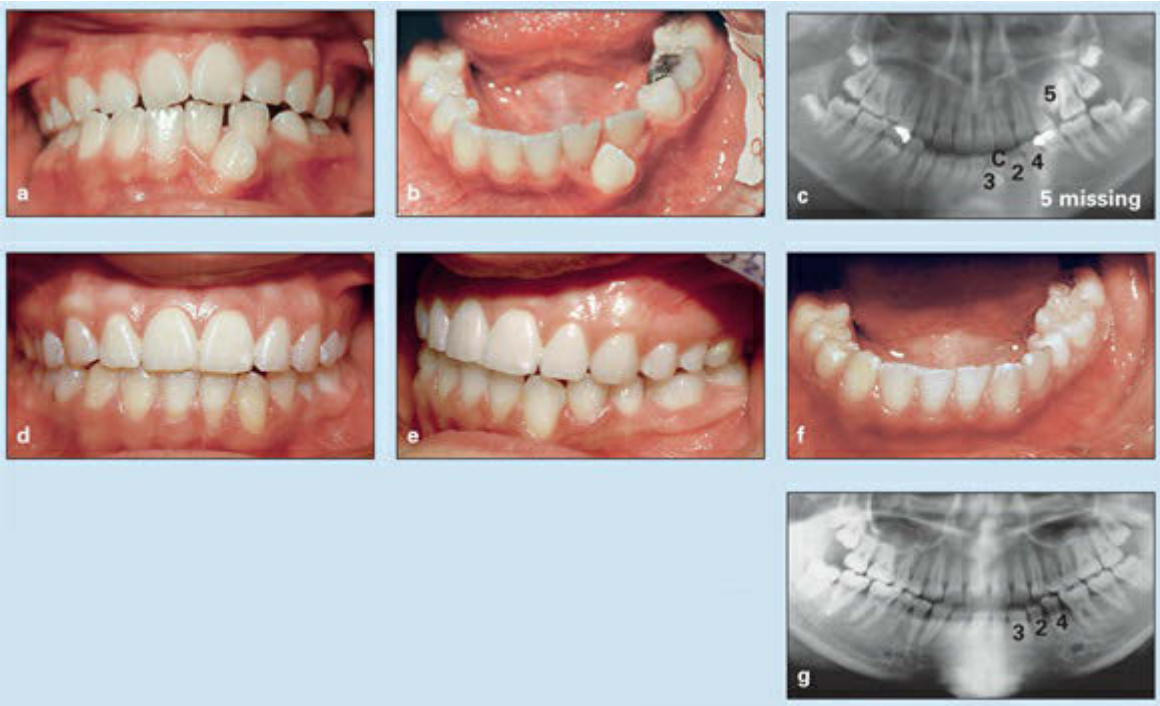


Fig 10-24 Management of lateral incisor–canine transposition in a 12-year-old girl with a Class I occlusion. *(a and b)* Pretreatment occlusion. *(c)* Pretreatment panoramic radiograph. The mandibular left permanent lateral incisor (2) and canine (3) are transposed. The left primary canine (C) is still present. The mandibular left first premolar (5) is missing. 4—first premolar. *(d to f)* Posttreatment occlusion. *(g)* Post-treatment panoramic radiograph.

Case 10-2

A 12-year, 8-month-old girl presented with a Class I occlusion and complete transposition of the maxillary left lateral incisor and canine (Figs 10-25a to 10-25e). The primary canine was still present, and an ectopic canine crown was located palatally, close to the central incisor root. There was a midline diastema of about 2.5 mm, and the left lateral incisor was in crossbite.

Treatment:
Considering the maxillary bone structure and the adequate volume of alveolar bone, treatment was designed to place the canine and lateral incisor in their normal positions. The treatment sequence was exposure of the impacted canine, insertion of an attachment, and then traction and separation of the canine from the central incisor root; correction of the lateral crossbite and alignment; and alignment of the canine and achievement of root parallelism and then closure of the diastema and shifting of the midline. Figures 10-25f to 10-25i show active alignment, root parallelism, and space closure at the final stage of active treatment. Figures 10-25j to 10-25m illustrate the posttreatment occlusion.

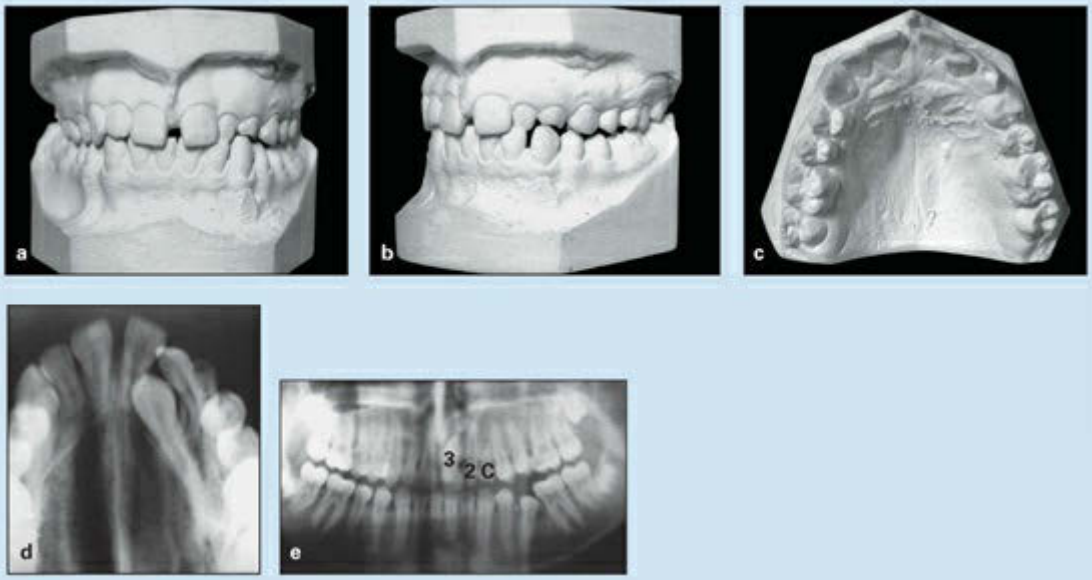


Fig 10-25 Management of complete transposition of the maxillary left lateral incisor and canine in a 12-year, 8-month-old girl. *(a to c)* Pretreatment casts of the occlusion. *(d)* Pretreatment occlusal radiograph. *(e)* Pretreatment panoramic radiograph. 2—lateral incisor; 3—canine; C—primary canine.

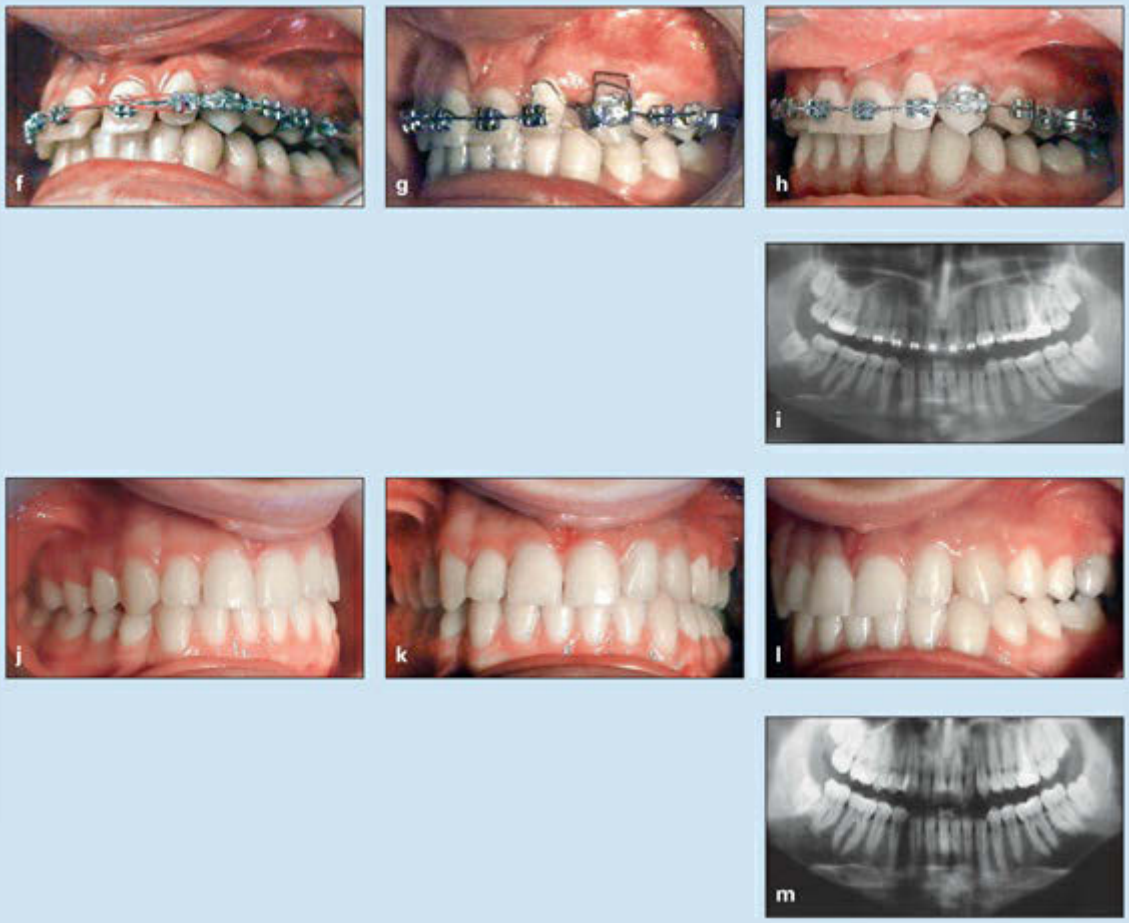


Fig 10-25 *(cont) (f to h)* Active alignment, root paralleling, and space closure at the end of active

treatment. *(i)* Panoramic radiograph taken during active treatment. *(j to l)* Posttreatment occlusion. *(m)* Posttreatment panoramic radiograph.

Case 10-3

A 7-year-old girl in the early mixed dentition had a Class I malocclusion, minor anterior crowding, rotation of the maxillary incisors, and end-to-end dental relationships. The partially erupted mandibular left lateral incisor had the potential to erupt in crossbite. She presented with an incomplete transposition of the mandibular permanent right lateral incisor and canine (Figs 10-26a to 10-26e).

Treatment:
Treatment phases were extraction of the mandibular primary canine, leveling and uprighting of the displaced mandibular lateral incisor, and alignment of the mandibular incisors. The next step was guidance of eruption of the displaced canine. This was followed by maxillary 2× 4 bonding to align the maxillary incisors and some proclination to increase the arch circumferences, thereby providing normal overbite and overjet and creating more space for the maxillary canines. Figures 10-26f to 10-26i illustrate different phases of mechanotherapy. Figures 10-26j to 10-26o show the posttreatment condition of the dentition.

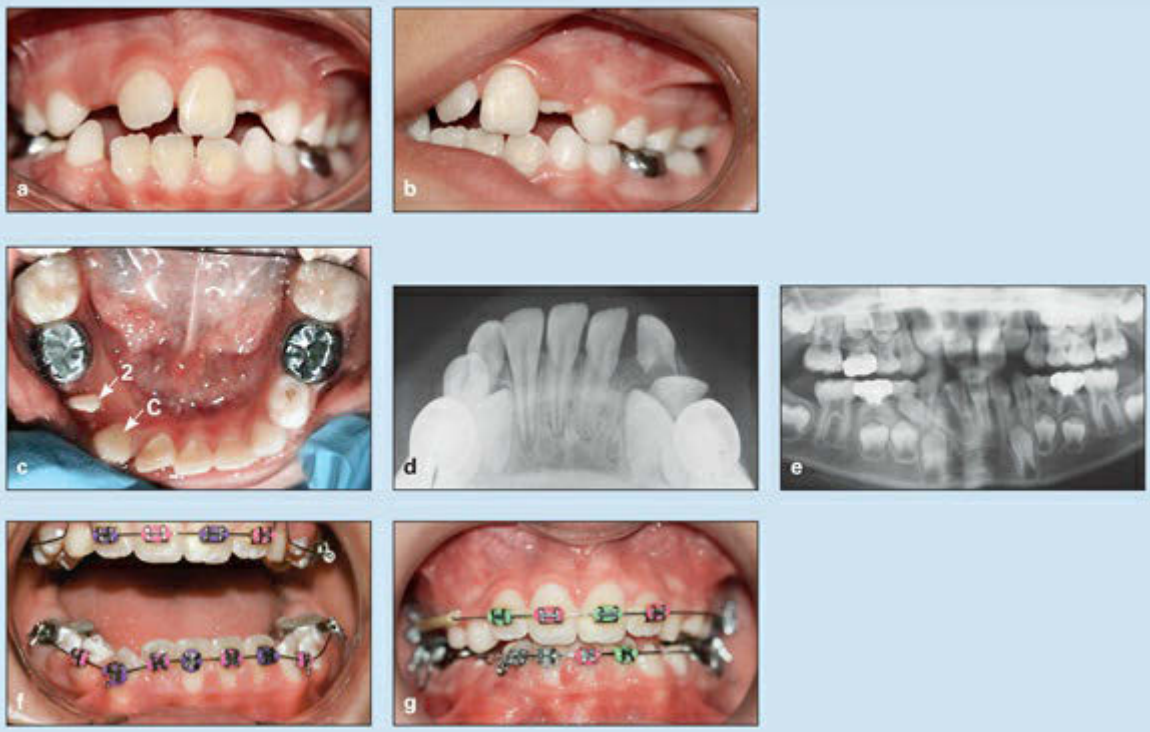


Fig 10-26 Management of incomplete lateral incisor–canine transposition in a 7-year-old girl in the early mixed dentition. The partially erupted mandibular left lateral incisor has the potential to erupt in crossbite. *(a to c)* Pretreatment occlusion. C— primary canine; 2—permanent lateral incisor. *(d)* Pretreatment occlusal radiograph. *(e)* Pretreatment panoramic radiograph. *(f and g)* Different stages of mechanotherapy, including leveling, incisor alignment, and space creation.

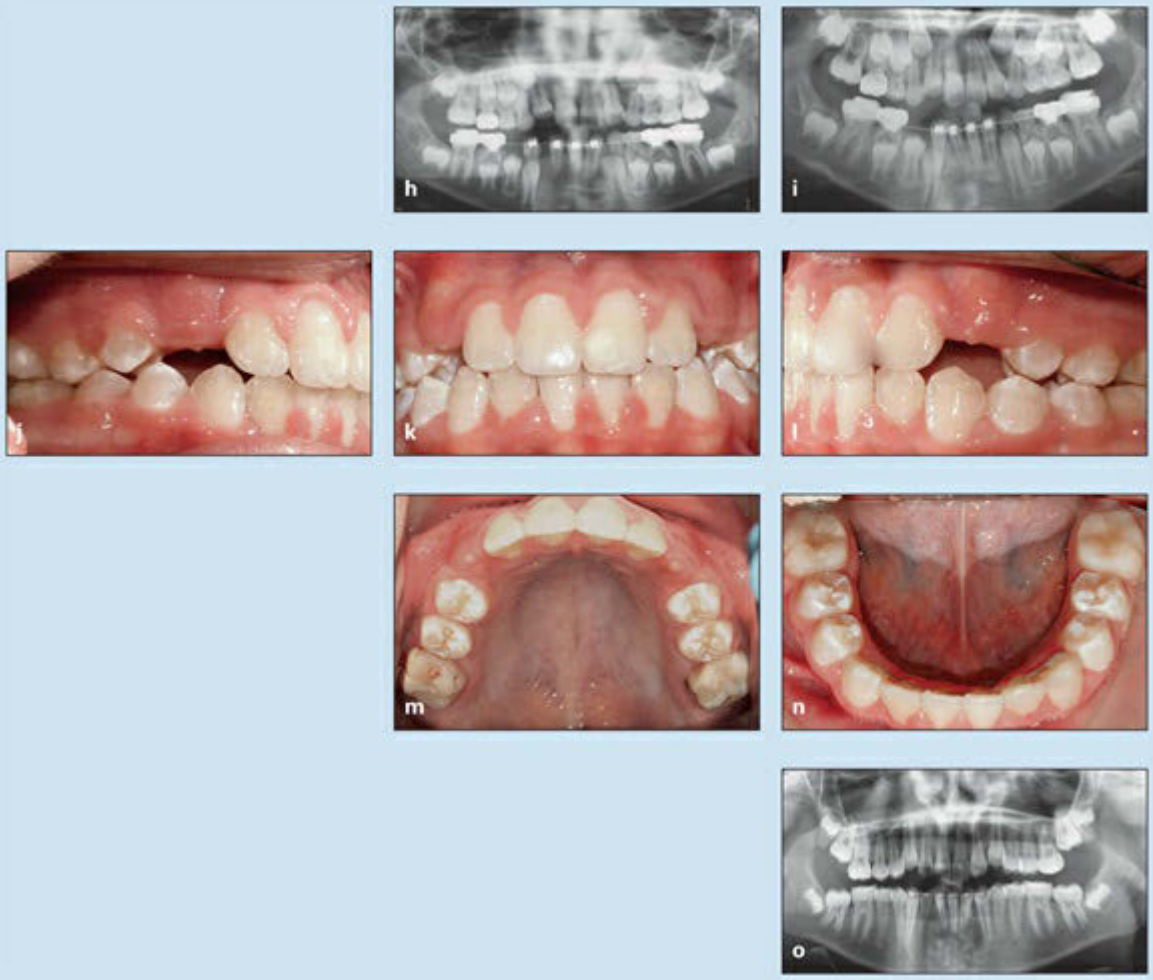


Fig 10-26 *(cont) (h and i)* Panoramic radiographs taken during active treatment. *(j to n)* Posttreatment occlusion. *(o)* Posttreatment panoramic radiograph.

Impaction

Tooth impaction is an intraosseously displaced position of the teeth and an abnormal eruption pathway that results in a prognosis of delayed or failed eruption. Impacted teeth are a commonly found anomaly in dental practice. They pose a problem for the maintenance and continuity of the patient's dental health and sometimes represent a risk-management threat for the practitioner. Any tooth can be impacted, but the following are the most frequently impacted teeth:

- Mandibular third molars
- Maxillary canines
- Maxillary third molars
- Maxillary and mandibular second premolars

• Maxillary central incisors

Maxillary Canine Impaction

The role of the canines in the development of occlusion was described by Broadbent[59] as a major factor in the "ugly duckling" stage of normal dental development. Today it is known that canines play several important vital roles in facial appearance and function, such as their role in dental esthetics of the smile, as an important part of the occlusal foundation in dental arch development, and as important dental units in functional occlusion.

Canines are prone to ectopic eruption and impaction because of their high and deep position in alveolar bone, their extended developmental period, and their long and tortuous eruption route.

Adjacent teeth or other local factors may also narrow the eruption path of the canine because of its late appearance in the eruption sequence. Early clinical and radiographic evaluation of canine position and proper intervention can guide the canine to a normal path of eruption and prevent many complications and risks.

Prevalence

Maxillary canines are the most commonly impacted teeth, second to mandibular third molars. The incidence of canine impaction in the maxilla is more than three times that in the mandible. Bishara's review[60] of the literature reported that the incidence of maxillary canine impaction ranges between 1% and 3% of patients. For the mandibular canine, the incidence of impaction is about 0.35%. This anomaly is twice as common in females as it is in males. Most affected patients have unilaterally impacted maxillary canines, and only 8% have bilateral impactions. Canine impaction has a high family association and is five times more common in Europeans than in Asians.[61]

Despite the fact that maxillary canine buds develop high near the orbit and sinus and buccal to adjacent tooth roots, the prevalence of palatal impaction is much higher than that of buccal impaction. Approximately two-thirds of impacted maxillary canines are impacted palatally and one-third labially.[62] Jacoby[63] reported that 85% of impacted canines are located palatally and 15% buccally. Fournier et al[64] reported a palatal-to-buccal impaction ratio of 3 to 1, and Jacoby[63] reported a

ratio of 12 to 1.

Buccal canine impactions are mostly associated with inadequate space and are positioned more vertically, while palatally impacted canines are positioned more horizontally. Because palatal bone is denser and palatal mucosa is thicker, palatally impacted canines rarely erupt without orthosurgical treatment.

In a clinical and radiographic evaluation of individuals with Down syndrome, Shapira et al[55] reported that prevalences of canine impaction (15%) and maxillary canine–first premolar transposition (15%) were high compared with published figures from random population samples. Warford et al[65] reported that maxillary canine impaction has an incidence of 1 in 100 among the general population.

Etiology of palatal canine impaction

Palatal canine impaction and buccal canine impaction differ in etiology, morphogenesis, and management. Two general theories have been proposed to explain the occurrence of palatally displaced and impacted maxillary canines: guidance theory and genetic theory.

Guidance theory

Guidance theory contends that this anomaly develops because of the presence of local predisposing causes. For example, the canine erupts along the root of the lateral incisor; thus, the presence of normal lateral incisor roots serve an important role in guidance of canine eruption. If the lateral incisor root is malformed or the lateral incisor is missing or misshaped (peg lateral), extra space in the apical part of the maxilla can deflect the eruption path of the canine.

In a retrospective study of the records of 182 orthodontic patients with palatally displaced canines, Mossey et al[66] measured the tooth length of lateral and central incisors on radiographs and the crown widths of lateral incisors on study casts. For more accuracy, the crown widths and root lengths of 160 extracted maxillary lateral incisors were also measured. The study supported the hypothesis that there is a weak association between palatally displaced maxillary canines and lateral incisors of smaller than average crown width and found weak support for the association between palatal canines and absence of the adjacent lateral incisor.

Becker[67] and other advocates of this theory have stated that even if absence of the lateral incisor is genetically determined, the palatal canine displacement has no

genetic association but occurs as a result of local environmental disturbances (Fig 10-27; see also Fig 10-30).

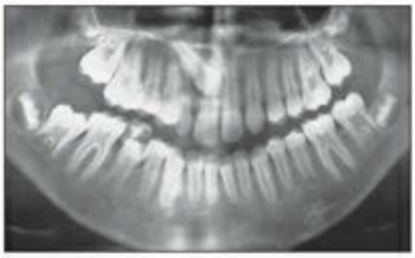


Fig 10-27 Palatal canine impaction. The impaction is related to the missing lateral incisor.

Arch length deficiency is another local factor that has been mentioned in the literature. According to Jacoby,[63] space deficiency is a valid explanation for most impactions but not for palatal impaction of the maxillary canine. His study showed that 85% of palatally impacted canines have sufficient space for eruption.

The buds of the lateral incisor and the first premolar are located behind the canine's palatal surface, and the maxillary canine bud is wedged between the nasal cavity, the orbit, and the anterior wall of the maxillary sinus. When there is arch length discrepancy or space loss in the maxillary canine area, the canine becomes impacted buccally; when the maxillary lateral incisor is missing, the canine becomes impacted palatally. Jacoby[63] indicated that this space can be provided by excessive growth in the base of the maxillary bone, by agenesis of lateral incisors or peg-shaped lateral incisors, or by stimulated eruption of the lateral incisor or the first premolar. He added that dysplasia in the maxillary-premaxillary suture can also modify the direction of eruption of the maxillary canine.

The author's clinical experience indicates that space deficiency can also cause palatal impaction in this situation. The radiographic evaluation also shows some palatal positioning of the canine.

The presence of a supernumerary tooth or an odontoma can also deflect the eruption path of the canine and cause impaction (see case 10-7). If the problem is detected early, interceptive treatment and preservation of space can guide the normal eruption pattern, and canine impaction can be prevented.

Al-Nimri and Gharaibeh[62] studied the etiology of palatal impaction by assessing the pretreatment dental casts of 34 patients with unilateral palatal canine impaction. The casts of the subjects were compared with the pretreatment dental casts of unaffected orthodontic patients, which were matched according to age, sex, and type of malocclusion. They found that palatal canine impaction occurred most frequently in subjects with Class II division 2 malocclusions and in those who were missing a

lateral incisor. They also found that the transverse arch dimension was significantly wider in the impaction group.

Zilberman et al[68] established a strong link between small, peg-shaped, or missing lateral incisors and palatally displaced maxillary canines. They also proposed that, because anomalies of the lateral incisor develop under strong genetic control, the first-degree relatives of patients with palatally impacted canines would themselves show an increased prevalence. They reported that anomaly of the lateral incisors among the relatives was found to be four times that of the general population.

Therefore, the following are the most common local factors that can cause canine impaction:

- Missing lateral incisor
- Peg-shaped lateral incisor
- Supernumerary tooth or odontoma
- Space deficiency
- Dysplasia in the maxillary-premaxillary suture
- Some Class II division 2 malocclusions

Genetic theory

According to genetic theory, disturbance of maxillary permanent canine eruption is due to an abnormal developmental condition of the dental lamina. Advocates of this theory cite evidential categories related to genetic origin such as familial and bilateral occurrence of impaction as well as increased occurrence of other associated anomalies, such as lateral incisor and second premolar hypodontia, ectopic eruption of first molars, and infraocclusion of primary molars.

Baccetti[69] studied an untreated orthodontic population to reveal patterns of association among seven types of dental anomalies, including small maxillary lateral incisors and palatal displacement of maxillary canines. The study found significant reciprocal associations among five of these anomalies, including size of the maxillary lateral incisors and palatal displacement of the maxillary canines and suggested a common genetic origin for these conditions. The existence of associations between different tooth anomalies is clinically relevant, because the early diagnosis of one anomaly may indicate an increased risk for others.

Peck et al[70] demonstrated that 33% of palatal canine impaction has some association with missing or peg-shaped lateral incisors. They examined the

specificity of tooth agenesis sites associated with the occurrence of palatally displaced canines, mandibular lateral incisor–canine transposition, and maxillary canine–first premolar transposition. They suggested that coupling these new clinical findings with results from recent molecular studies indicates that transcription factors such as MSX1 and PAX9, which have been associated with agenesis of molars, might be involved in the genetic control of mandibular lateral incisor–canine transposition and palatal canine impaction. However, it remains uncertain whether the anomalous or missing lateral incisor is a local causal factor or an associated genetic development.

Pirinen et al[71] examined 106 patients who had been treated orthodontically for palatally impacted canines and 110 first-degree and 93 second-degree relatives. The prevalence of hypodontia affecting permanent teeth in examined patients was 36%, which is 4.5 times the prevalence in the general population. The prevalence of hypodontia in both first- and second-degree relatives was 19% to 20%, which is 2.5 times the prevalence in the general population. From these findings, they concluded that the palatally displaced canine belongs to the spectrum of dental abnormalities related to hypodontia. Palatal displacement of canines is genetic and related to genetic incisor-premolar hypodontia and peg-shaped incisors. They also stressed the importance of screening for impacted canines in patients with hypodontia or peg-shaped teeth as well as their families.[71]

To summarize, the exact etiology of maxillary impaction is not clear; it may be a multifactorial disturbance, in which either genetic or systemic factors can play as predisposing factors, and local factors prepare the environment for canine deflection and an abnormal pathway. Regardless of the cause, because the maxillary canine has the longest and most tortuous path of eruption of any tooth, the likelihood of disturbances during the developmental path toward occlusion is great. At the age of 3 years, the canine bud is located very high in the maxilla close to the orbit. The crown is directed mesiolingually. From this point, it starts moving gradually toward the occlusal plane, while it is uprighting itself, until it reaches the distal aspect of the root of the lateral incisor. It then seems to be deflected to a more vertical position, until it reaches the occlusal contact at approximately age 13 years, with a marked mesial inclination. However, this long, complicated journey does not happen overnight; it takes about 10 years to be completed, and it is the responsibility of dental practitioners to monitor these events (by longitudinal panoramic radiographic monitoring) in order to detect problems early and to prevent complications through proper intervention.

Factors associated with palatal impaction

The following factors have been reported to be associated with palatal impaction:

- Space deficiency
- Trauma
- Overretention of the primary canine
- Premature loss of the primary canine
- Failure of the primary canine root to resorb, creating a potential mechanical obstacle for canine eruption
- Abnormal position of the tooth bud
- Disturbances in tooth eruption
- Localized pathologic lesions such as cysts and odontomas
- Abnormal sequence of eruption
- Missing lateral incisors
- Anomalous abnormal size of the lateral incisor or abnormal form of the lateral incisor roots (dilacerations of the root)
- Variation in timing of lateral incisor root formation
- Ankylosis of the permanent canine
- Presence of a supernumerary tooth
- Presence of an alveolar cleft
- Idiopathic conditions

Etiology of labial canine impaction

Labial impaction of a maxillary canine is most often the result of space deficiency during the transitional dentition (Fig 10-28). This deficiency can be caused by premature loss of the primary canine followed by a midline shift that results in insufficient space for and buccal eruption of the canine; anterior dental crossbite that decreases the anterior arch circumferences, deflecting the canine buccally; ectopic migration of the permanent canine over the adjacent teeth; or overretention of the primary canine.

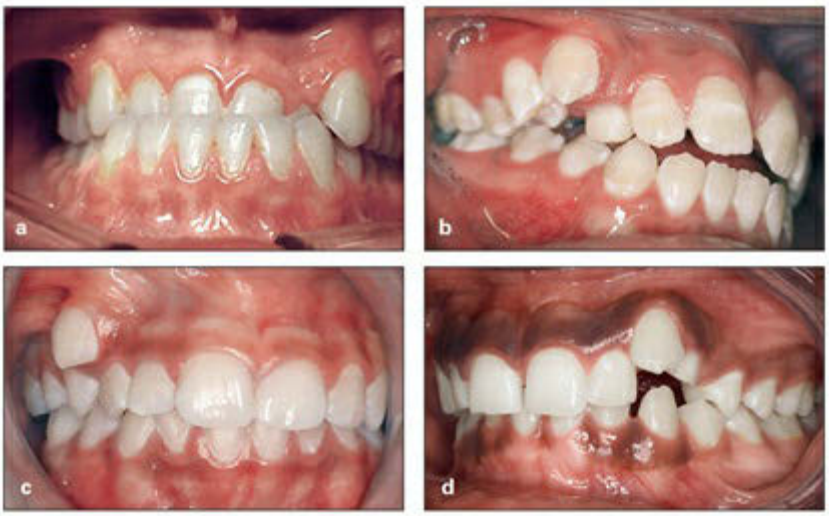


Fig 10-28 Buccal impaction caused by anterior crossbite *(a)*, insufficient space *(b)*, and overretained primary canine *(c and d)*.

Consequences of impacted maxillary canines

Most eruption problems, including canine impaction, are asymptomatic, and patients are unaware of their existence. If practitioners do not monitor their patient's eruption patterns, the time of discovery is usually late, and the problem has already completely developed. In some situations, adjacent teeth and surrounding structures have already been damaged. Abnormal eruption paths within the dentoalveolar process can cause migration of the adjacent teeth and loss of arch length, development of cystic lesions and infection, root resorption of the nearby lateral incisors, and overretention of primary lateral incisors.

Resorption of the incisor roots is also asymptomatic and cannot be recognized without clinical examination. Ericson and Kurol[72] investigated lateral root resorption caused by ectopic eruption of the maxillary canines. They assessed two groups, one with 40 cases of lateral incisor root resorption and a control group of 118 ectopic eruption cases without lateral incisor root resorption. When the cusp of the well-developed canine was positioned mesial to the lateral incisor root, the risk of complications was three times greater. Furthermore, the risk of resorption increased by 50% when the mesial eruption angle exceeded 25 degrees compared with the controls. Lateral incisor root resorption was three times more common in girls than in boys.

Walker et al[73] assessed the spatial relationship of impacted canines by using three-dimensional images of 27 unilaterally and bilaterally impacted canines from 19 consecutive patients (15 female and 4 male subjects). The study found that 92.6%

of impactions were palatal. Incisor resorption was present in 66.7% of the lateral incisors and in 11.1% of the central incisors adjacent to the impacted canine. Follicle size did not have a major influence on the position of the impacted canine. The alveolus was narrower on the side of the impacted canine than on the side of the erupted canine; however, the width of the alveolus on the impacted canine side is independent of the primary canines.

The potential complications emphasize the need for practitioners to closely monitor the development and eruption of the dentition during the stage of dental transition. In brief, the following complications can result when canine impaction is not treated promptly:

- Resorption of the roots of adjacent teeth
- External resorption
- Infection
- Cyst formation
- Loss of arch length
- Transposition
- Periodontal defects

Early detection of canine impaction

Because canine impactions generally require longer treatment times, depending on the location of the impacted tooth, early identification of impaction is of critical interest to the orthodontist and to all practitioners. Potentially impacted canines can be detected at an early age. With proper clinical diagnosis and radiographic evaluation and monitoring (longitudinal panoramic radiograph monitoring), the clinician might be able to prevent impaction through timely interceptive treatment.

Because of the wide variation that exists in the timing of eruption of permanent teeth among members of different ethnic groups, different investigators have recommended an age range of 8 to 10 years for early detection of maxillary canine impaction. Diagnostic procedures at these ages are a combination of careful clinical and complete radiographic examinations (longitudinal monitoring of panoramic radiographs).

Clinical examination

The clinical examination consists of digital palpation of the buccal bulge of the canine in the sulcus above the primary canine root, which can reveal the position of the maxillary permanent canine even in an 8- to 10-year-old patient. The clinician's index fingers can be used to palpate the area above the primary canine on both the buccal and palatal aspects of both sides of the arch.

This clinical examination should be repeated every 2 to 3 months. Impaction of the permanent canine is indicated if the buccal canine bulge is not palpable in children older than 10 years and the primary canine is retained and shows no signs of mobility. This preliminary diagnosis should be followed by careful radiographic examination for confirmation.

Another clinical finding, which may be observed after eruption of the permanent lateral incisors at around 9 to 10 years of age, is distobuccal inclination of the lateral incisors. Broadbent[59] called this normal transitional stage of dentition the "ugly duckling" stage preceding the eruption of the permanent canines. Tipping may cause spacing of the incisor crowns, despite the crowding of the roots (Fig 10-29). Severe distal tipping of the lateral incisor crowns might be due to pressure from the mesially displaced and abnormally positioned canine and indicate the potential for canine impaction. This situation requires careful radiographic evaluation and monitoring and possibly early intervention.

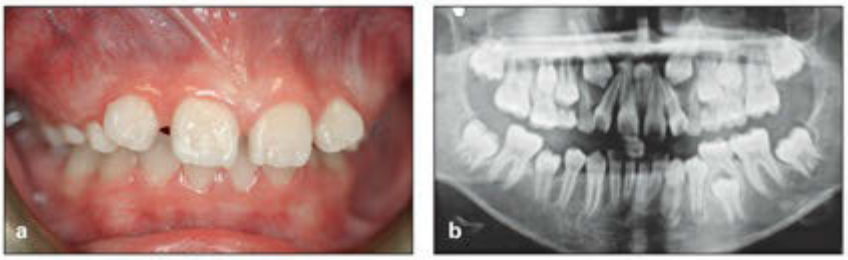


Fig 10-29 *(a and b)* Ugly duckling stage of the dentition.

Likewise, a labially inclined lateral incisor can be the result of a displaced canine lying on the labial aspect of the lateral incisor root. In all of these situations, careful monitoring of the permanent canine is recommended.

No orthodontic movement should be applied to the lateral incisors until the pressure of the canine crown is eliminated from the lateral incisor root. Extraction of the remaining primary canine can help the permanent canine crown toward its normal position. Untreated cases may cause incisor root resorption.

Clinical signs of canine impaction

The following are signs of canine impaction:

- Delayed eruption of the permanent canine
- Prolonged retention of the primary canine, especially asymmetric exfoliation of the primary canine
- Absence of a normal labial canine bulge
- Presence of a palatal bulge
- Distal tipping of the lateral incisor crown
- Retroclined position of the lateral incisor (if its root is forced buccally and its crown palatally by the palatally impacted canine)

Radiographic evaluation

Radiographic evaluation is essential for detecting potentially impacted canines and an important tool to determine whether the canine is positioned palatally or buccally. Different radiographic techniques can be used, including periapical, occlusal, panoramic, and cephalometric radiographs (lateral or posteroanterior).

Periapical radiographs. Clinicians can use different kinds of periapical radiographs for localization of a displaced canine, including the standard periapical or Clark's rule technique. Also termed the *image shift principle*, in the Clark's rule technique two different angulations are taken in the same horizontal plane. If the object moves to the same side of the angulation, the subject (in this case, the canine) is located more lingually (palatally); if it moves to the opposite direction, the object is more buccally positioned.

Occlusal radiographs. Occlusal radiographs are recommended to determine the position of the involved tooth in the buccolingual and mesiodistal directions. Various techniques can be applied to obtain occlusal radiographs for localizing the impacted tooth. Occlusal radiographs or vertex occlusal view radiographs taken with the x-ray beam directed along the long axis of the central incisors are useful for identifying the position of the palatally displaced tooth relative to adjacent teeth (Fig 10-30).

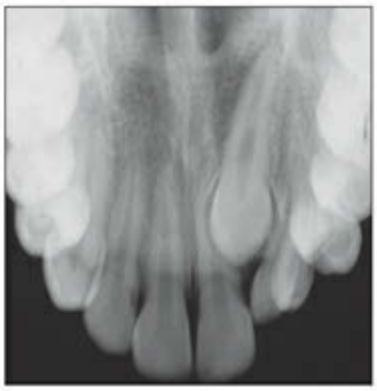


Fig 10-30 Occlusal radiograph showing the position of a palatally impacted canine.

Lateral and posteroanterior cephalometric radiographs. Lateral and posteroanterior (frontal) cephalometric radiographs are also useful techniques for localizing and identifying the inclination of impacted teeth. Lateral cephalometric radiographs can show the anteroposterior position of the canine relative to the incisors. Frontal radiographs can show the vertical and lateral positions of the canines relative to the incisors.

Williams[74] has shown that the permanent canine, during its pre-eruptive development, should be inclined slightly mesially, with the crown below the level of the apices of the lateral incisors and well below the lateral border of the nasal cavity. The canine root should lie distal to the lateral border of the nasal cavity. If the frontal radiograph reveals that the canine is mesially tipped, with its crown medial to the lateral border of the nasal cavity, and there is no evidence of primary canine root resorption, impending impaction of the displaced maxillary canine should be suspected.

Computed tomography scans. A CT scan is an expensive, routine radiographic technique in medicine and occasionally is used in dentistry. Scanning radiography can accurately locate the object in three dimensions.

In a recent study comparing conventional radiographic procedures (a single two-dimensional panoramic radiograph) with cone beam CT scans, Alqerban et al[75] showed that the cone beam CT is more sensitive than conventional radiography for both canine localization and identification of root resorption of adjacent teeth.

The three-dimensional images provided by this technique show the buccopalatal position of the impacted canine. The extent and exact location of lateral incisor root resorption can be detected; no other technique has the ability to show these aspects. Walker et al[73] stated that three-dimensional volumetric imaging of impacted canines can show the presence or absence of the canine, the size of the follicle, the

inclination of the long axis of the tooth, the relative buccal and palatal positions, the amount of bone covering the tooth, the three-dimensional proximity and resorption of roots of adjacent teeth, the condition of adjacent teeth, local anatomical considerations, and the overall stage of dental development. They concluded that three-dimensional imaging is clearly advantageous in the management of impacted canines.

Panoramic radiographs. The panoramic radiograph has been used extensively in dentistry; it is a useful tool that shows a general view of the combined permanent and primary dentitions and allows clinicians to monitor all abnormalities developing during the transitional dentition. Panoramic radiographs can also show other developmental anomalies, such as supernumerary teeth, missing teeth, odontomas, other pathologic conditions, the conditions of the primary roots and their successors, and the conditions of permanent root development. Moreover, panoramic radiographs have been used extensively for detecting canine inclination during the transitional dentition.

The general perception is that routine panoramic radiographs taken during the mixed dentition are useful for detecting only the mesiodistal position and inclination of the displaced unerupted canine; however, careful evaluation of high-quality panoramic radiographs can help in the detection of buccal or palatal positioning also.

Distinguishing buccopalatal position of an impacted canine by panoramic radiograph. Evaluation of the position of the impacted canine (buccal or palatal) depends on the relative radiopacities of the two superimposed objects. The film or the screen that captures the image is located in front of two objects, the canine and incisors. If the area of superimposition of the two images is dominated by the opacity representing the shape of the lateral incisor, it means that the lateral incisor is closer to the film, indicating that the canine is located more palatally. If the area is dominated by the opaque contours of the canine, it means that the canine is closer to the film, indicative of a more buccally positioned canine.

Figure 10-31 is a good example of this radiographic phenomenon for comparison of the different appearances of the two impacted canine positions. The panoramic radiograph clearly shows the position of the canines relative to the lateral incisors on the left and right sides; the palatal positioning of the right canine is indicated by the dominant contours of the lateral incisor in that area, and the buccal positioning of the left canine is indicated by the dominant contours of the canine on that side.

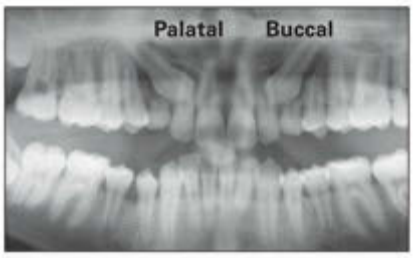


Fig 10-31 Panoramic radiograph showing two impacted canines. The right canine is palatally impacted and the left canine is buccally impacted.

Longitudinal monitoring of panoramic radiographs can help in early detection and prediction of maxillary canine impaction. Two important signs for prediction and early detection of maxillary canine impaction on panoramic radiographs are the position of the impacted tooth and the inclination of the impacted tooth. As mentioned earlier, Williams[74] described the normal position of the permanent canine during pre-eruptive development: inclined slightly mesially with the crown below the level of the apices of the lateral incisors and well below the lateral border of the nasal cavity. The canine root should lie distal to the lateral border of the nasal cavity. If the canine is mesially tipped in the frontal radiograph with its crown medial to the lateral border of the nasal cavity and there is no evidence of primary canine root resorption, an impending impaction of the displaced maxillary canine should be suspected.

Figure 10-32 is a retrospective evaluation of three panoramic radiographs taken during different stages of the dentition. The panoramic radiograph taken when the patient was 9 years old indicated that the maxillary left canine had a higher position and greater inclination than the right canine. No early intervention was attempted, and panoramic radiographs taken 1 and 4 years later revealed the incorrect eruption path of the palatally impacted canine. Anchorage preparation for the maxillary molars and early extraction of the primary canines and primary first molars might have guided the canine to a normal path of eruption or at least might have reduced the severity of the problems.

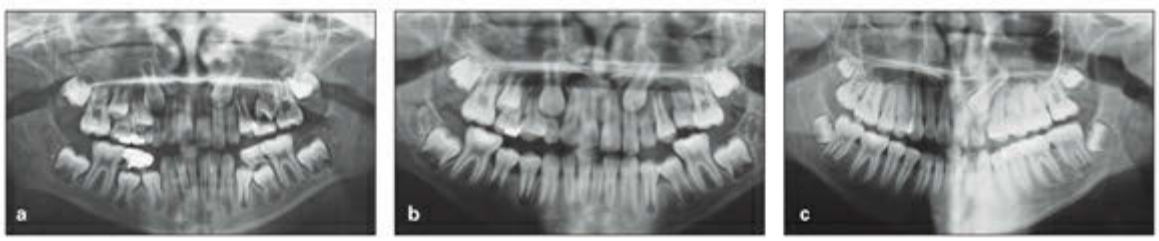


Fig 10-32 Retrospective evaluation of an incorrectly positioned maxillary left canine. *(a)* Panoramic radiograph taken at age 9 years. The left canine is positioned higher and is more inclined than the right canine. *(b)* Panoramic radiograph taken 1 year later. *(c)* Panoramic

radiograph taken 4 years later. No early intervention has been attempted to correct the path of eruption.

The proximity of impacted canine to adjacent teeth is another important point to be assessed before treatment planning. Depending on the position of the impacted tooth, the complexity of treatments differ. Impacted maxillary canines are usually positioned distal and either buccal or palatal to the lateral incisor root, with some degree of increased inclination. They are in a lower or higher position than the lateral incisor root.

Impacted canines have more mesial inclination than normal, and sometimes they are displaced from their normal position, with close proximity to the roots of adjacent teeth, and therefore are also ectopic. Ectopic impacted teeth are usually located close to the lateral or central incisor roots and hence have the potential to damage those roots. Canines that are both impacted and ectopic are more difficult to manage, require longer treatment time, can be more destructive to adjacent teeth during treatment, and demand much more percussion during treatment planning and precision during the treatment process (see case 10-4).

The other important factor for prediction of impaction from a panoramic radiograph is the inclination of the impacted tooth. The mesiodistal inclination of the maxillary canine is another predictor of impaction as well as a sign of complexity and an indicator of the potential success of treatment. Some investigators have proposed different techniques for evaluating potential outcomes of palatally impacted canines. Ericson and Kurol[76] found that the more mesially located and horizontally inclined the crown is, the less the likelihood of eruption after extraction of the primary canine. Power and Short[77] also looked at angulation as a predictor and found that if the tooth is angled more than 31 degrees relative to the midline, its chances of eruption after primary extraction are decreased.

Warford et al[65] drew a horizontal reference line on panoramic radiographs, across the most superior point of both condyles, and measured the mesial angle formed by the constructed horizontal line and the long axis of the unerupted tooth. They found that the angulation for nonimpacted teeth, a mean of 75.12 degrees, was greater than that for impacted teeth, 63.20 degrees. Figure 10-33 presents a Warford analysis of a panoramic radiograph, indicating that the left canine is much more severely impacted than the right canine (see case 10-4).

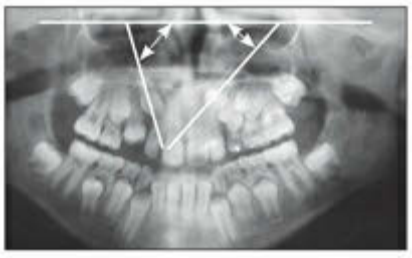


Fig 10-33 Inclination of both canines assessed with the Warford analysis.[65] The angulation indicates the severity of the problem affecting the left canine.

Treatment options for impacted maxillary canines

The duration of the long and problematic transitional process of permanent canines is usually about 10 years, from the beginning of eruption to the complete occlusal position (from ages 3 to 13 years). Thus, for any type of preventive or interceptive treatment, clinicians must monitor this long process during the mixed dentition. Depending on the age of the patient, the dental developmental age, the stage of eruption disturbances, and the type of impaction, there are a number of treatment options for the management of impacted teeth:

- Observation during the transitional dentition
- Interceptive treatment
- Surgical exposure of the canine and orthodontic treatment
- Autotransplantation of the canine
- Extraction of the impacted canine and space closure
- Extraction of the impacted canine and prosthetic or implant replacement
- Periodic radiograph observation (in adults when the impacted canine is located very high or horizontally and because of either high risk or the patient's desire for no treatment)

Some kind of interaction among these options might be necessary, such as selective primary tooth extraction, removal of any obstacle deflecting the canine, or space creation.

The most desirable approach in managing impacted maxillary canines is early detection and interception of a deflected canine that has the potential for impaction at its initial stage, at around 8 to 10 years of age, when the canine starts its long intrabony movement toward its final position.

Observation

During the transitional dentition, such as during the ugly duckling stage or when the permanent canine is tipped mesially and is close to the lateral incisor apex, orthodontic movement of the lateral incisor crown will cause root resorption of the lateral incisor. In this situation, the clinician should only observe the situation until the canine crown changes its position, away from the apex. If the canine does not show any change in position after 10 to 12 months and the possibility of lateral incisor root resorption is evident, exposure of the canine and bonding attachment for orthodontic traction is indicated.

Williams[74] stated that age 8 years is the best time to begin observation of the maxillary canines and that the positional changes over the next 2 years require careful observation.

Observation of the transitional dentition from the beginning of the mixed dentition to complete eruption of the canines can predict tooth impaction and many other eruption disturbances (see Figs 10-32 and 10-33).

Interceptive treatment

Interceptive treatment, or guidance of eruption, for canine impaction is a series of procedures that should begin when the canine starts its long intrabony movement toward its final position.

Many investigators suggest that the best time to begin assessing a patient for potential maxillary canine impaction is around 8 to 10 years of age. It is believed that after the patient is 11 to 12 years of age the only remaining option is to correct the anomaly by orthosurgical intervention, which is sometimes risky, unpredictable, or unsuccessful.

During this period, the permanent canine begins its intrabony descent from its lingual position relative to the root apex of the primary canine and the primary canine root starts to resorb. Dewel[78] and Newcomb[79] concluded that adequate observation, diagnosis, and treatment planning can be critical during this stage of canine transition.

Interceptive options. After thorough clinical and radiographic evaluation, when the potential for canine impaction is confirmed, depending on the etiologic factors and the patient's age and type of occlusion, several interceptive and guidance procedures can be applied:

- Removal of the obstacle causing deflection of the canine, such as a supernumerary tooth, odontoma, cyst, or ankylosed primary tooth.
- Creation of space, if space deficiency is the cause of this anomaly. Depending on the occlusion and age of the patient, different tactics can be applied for providing the necessary space:
  - Distalization of the buccal segment in some Class II mixed dentition treatment
  - Correction of maxillary anterior dental crossbite if retroclination of the maxillary incisors has resulted in space deficiency for maxillary canines or if correction of crossbite increases the arch circumference in the anterior segment
  - Closure of the space that sometimes remains between the incisors, even after complete eruption of the lateral incisors
- Extraction of the primary canine at the right time is advocated by many investigators, who believe this action facilitates and promotes permanent canine eruption.

Primary canine extraction procedure. Primary canine extraction is an interceptive treatment for guiding the permanent canine eruption to prevent impaction; the best age for this procedure is when the child is between 8 and 10 years and not after the age of 11 years.

Shapira and Kuftinec[80] concluded that early diagnosis and detection of a potentially impacted maxillary canine and timely interceptive removal of the primary canine reduces the need for complicated orthodontic treatment. Ericson and Kurol[76] reported that favorable eruption occurred 78% of the time in their sample. In an investigation into the response of palatally displaced canines to the removal of primary canines, Power and Short[77] reported success 62% of the time.

The best situation for this procedure is when the clinical examination and radiographic evaluation of an 8- to 10-year-old child indicate the presence of the following conditions:

- The permanent canine is mesially displaced and does not follow the guidance of the lateral incisor root.
- The permanent lateral incisor crown is somewhat distally inclined, indicating the presence of permanent canine crown force.
- The buccal canine bulge cannot be palpated.
- An overretained primary canine is present and shows no signs of mobility.

Timely extraction of the primary canine in the presence of any of the aforementioned conditions allows the permanent canines to become upright and erupt into their proper positions in the arch and provides the best long-term results, without many of the complications that can arise if intervention is delayed.

Before this interceptive treatment is initiated, the proximity of the permanent canine crown and lateral apex must be evaluated. If distal tipping of the lateral incisor crown is clinically and radiographically evident, no orthodontic force can be applied to the lateral incisor, because the force will eventually cause severe root resorption. In this situation, after extraction of the primary canine, the most cautious treatment plan is use of some kind of anchorage device, such as a transpalatal arch, and then monitoring the permanent canine crown and lateral incisor apex until some uprighting of the permanent canine has occurred and the canine crown is located distal to the lateral incisor root.

The next step in any case of canine guidance would be 2 × 4 bonding, slow alignment of the maxillary incisors, and closure of any diastema or dental spacing present between incisors to facilitate eruption of the permanent canine.

Olive[81] evaluated 30 palatally impacted canines that emerged following orthodontic space opening without surgical exposure and tried to determine factors influencing the time of eruption. The sector of impaction (severity of canine inclination) was the best guide to the duration of orthodontic treatment prior to emergence.

Jacobs[82] stated that extraction of the primary canine for prevention or interception of a palatally displaced canine is best carried out as early as the displacement is detected, soon after 10 years of age.

Ericson and Kurol[76] stated that successful eruption of the impacted canine after extraction of the primary canine depends on the horizontal angulation of the permanent canine and the position of the canine crown relative to the root of the lateral incisor. If radiographs showed that the crown of the permanent canine was positioned over the root of the maxillary lateral incisor but not past the mesial surface of the root, self-correction of the ectopic canine occurred with high predictability. However, if the permanent canine was positioned well beyond the mesial surface of the lateral incisor root, the chance of self-correction and eruption of the permanent canine was very low.

Early diagnosis and intervention prevent resorption of the adjacent incisor root, save time, reduce expense, avoid the surgical and orthodontic treatment needed to align a palatally impacted canine, and eliminate complications associated with

surgical exposure and orthodontic procedures. These complications include long treatment time, bone loss, root resorption, and gingival recession around the treated teeth.

Surgical exposure of the canine and orthodontic treatment

If impacted canines are in their final stage of development in the permanent dentition, the clinician must consider orthodontic treatment combined with surgical exposure of the canine to bring it into occlusion. Orthosurgical approaches vary greatly depending on place of impaction, whether the impaction is labial or palatal, the age of the patient, the proximity of the impacted tooth to adjacent teeth, etiologic factors, and the morphology of impacted teeth. These approaches depend on the clinical judgment and experience of the orthodontist and surgeon. To ensure that appropriate surgical and orthodontic techniques are applied, the first step is coordination between the orthodontist and the oral surgeon.

Next, before any treatment planning is completed, a thorough clinical and radiographic evaluation must be performed to precisely locate the position and inclination of the impacted tooth and the proximity to adjacent teeth and to determine the type of surgical approach and the method and direction of orthodontic traction. Important points of clinical examination and different radiographic techniques that can help in diagnosis and precise localization have been discussed previously.

Pretreatment considerations. An impacted canine can be located palatally or buccally, can be ectopic or nonectopic, and can be unilateral or bilateral; each of these situations requires different surgical techniques and different orthodontic approaches. Among the factors that must be considered are:

- The inclination of the impacted tooth
- The position of the impacted crown and its proximity to adjacent teeth
- The cause of impaction
- The age of the patient and the amount of root development

As discussed earlier, the inclination of the canine is a sign of impaction as well as a predictor of treatment complexity and success. The more mesially located and horizontally inclined the crown is, the less likelihood of eruption after extraction of the primary canine.

The position of the impacted crown and its proximity to adjacent teeth not only

influence the amount of damage that the canine may cause to other teeth but also affect the complexity of treatments and dictate the technique of orthodontic traction that must be used. Impacted canines usually have more mesial inclination than normal and are positioned distal and either buccal or palatal to the lateral incisor root. They also can have a lower or higher position than the lateral incisor root. Sometimes impacted canines also are in close proximity to the roots of adjacent teeth and therefore are also ectopic, which represents a treatment challenge (Fig 10-34).

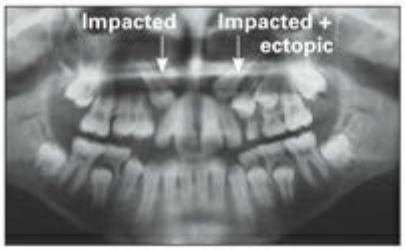


Fig 10-34 Difference between impaction and ectopic impaction.

Another point to be considered before treatment planning is the dental age of the patient and the stage of root development. Proper intervention before root completion has a better prognosis.

There are several factors that can cause canine impaction. Early detection and recognition of the problem is helpful in treatment planning and can indicate the complexity of the treatment and the possibility of success. If the cause is clear-cut, such as space deficiency or a supernumerary tooth, the prognosis is more predictable; if the cause is unknown and ankylosis of the impacted tooth is suspected, the prognosis is unknown, and treatment is risky, the treatment plan must be adjusted accordingly.

For example, if extraction of a premolar is part of the treatment plan and ankylosis of the impacted canine is also suspected, it is recommended that the impacted canine be exposed and orthodontic traction started first to ascertain impacted tooth movement. If the canine shows resistance to eruption and ankylosis is confirmed, the impacted tooth can be extracted, and the premolar can be substituted for the canine in the dentition (see case 10-8).

Surgical approach. After careful clinical and radiographic evaluation and confirmation of the exact location and inclination of the impacted tooth, the feasibility of surgical exposure and proper access for the surgical approach are determined.

In the past, surgical approaches to access impacted teeth consisted of wide resection of gingival and osseous tissues to uncover the impacted tooth for attachment of the traction device or wires. These approaches were often accompanied by surgical problems such as bleeding, difficulty in the placement of the attachment device, and, most significant, excessive removal of bone and soft tissues. Figure 10-35 shows the type of attachment that was used before bonding techniques were available. Many reports indicate various periodontal complications after this kind of surgical technique, including bone loss, gingival recession, decreased width of keratinized tissue, delayed periodontal healing, and gingival inflammation.[83]

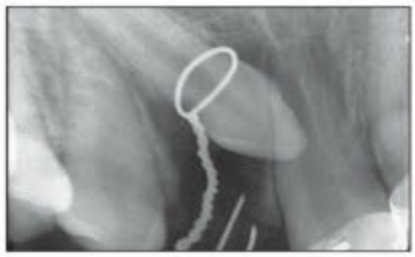


Fig 10-35 Surgical attachment used before the bonding technique was available.

Now surgical techniques are improved and less aggressive, and the availability of direct bonding with delicate attachments means that the surgeon can bond to the impacted crown directly and then replace and suture the flap. These procedures can be accomplished with few complications.

Palatal versus buccal impaction. Palatal canine impaction and buccal canine impaction differ in morphogenesis, etiology, and anatomical position, and therefore their treatment, whether interceptive or corrective via surgical procedure, is also different. The most common cause of buccal impaction is crowding. As discussed previously, many other etiologic factors also have been proposed for palatal impaction.

Both orthodontic and orthosurgical approaches are more predictable and easier in treatment of buccal impaction than palatal impaction. Buccally impacted canines reach occlusion faster and with less complexity. Palatally impacted canines also can vary in terms of complexity and difficulty of management, and there are some variations that must be considered before treatment planning.

Surgical considerations in palatal canine impaction. Early extraction of primary maxillary canines, around the age of 8 to 9 years, when the crown of the permanent canine is positioned over the root of the maxillary lateral incisor but not past the

mesial surface of the root, will result in normal eruption of ectopically displaced permanent maxillary canines with high predictability. However, if early intervention was not performed at the proper age or the permanent canine is positioned well beyond the mesial surface of the lateral incisor root, the permanent canine will become impacted and self-correction of the permanent canine is not possible; these situations must be treated by orthosurgical procedures.

Careful surgical management requires thorough clinical and radiographic evaluations; accurate localization of the palatally impacted tooth with regard to labiopalatal position, vertical position, inclination, depth of impaction, and proximity to adjacent teeth; and design of an accurate surgical approach according to the type of impaction.

Surgical considerations in buccal canine impaction. In any type of mucogingival intervention, an adequate zone of keratinized tissue must be maintained. If this procedure is not done properly and the tooth is moved through alveolar mucosa, it is likely that gingival recession will occur, leaving a periodontally compromised gingival margin. All orthodontic and surgical techniques in the management of labial impaction must be designed to maintain or ensure the presence of adequate amounts of keratinized gingiva.

To determine the correct surgical technique for exposing a maxillary canine that is labially or intra-alveolarly impacted, four criteria have been proposed:

1. The vertical position of the canine relative to the mucogingival junction
2. The amount of gingiva in the area of the canine
3. The labiolingual position of the canine crown
4. The mesiodistal position of the canine crown

Depending on the vertical position of labially impacted teeth relative to the mucogingival junction and the amount of gingiva, Kokich[84] described three techniques for uncovering a labially impacted maxillary canine: excisional uncovering, apically positioned flap, or closed-eruption techniques. He recommended simple excisional uncovering if the tooth is positioned labially and the crown is below the mucogingival junction. If the canine crown is positioned slightly apical to the mucogingival junction, an excisional technique would be inappropriate, because it would not result in any gingiva over the labial surface of the tooth after it had erupted. If the crown is positioned significantly apical to the mucogingival junction, an apically positioned flap would also be inappropriate, because it would result in instability of the crown and possible reintrusion of the tooth after

orthodontic treatment. In this situation, a closed-eruption technique will provide adequate gingiva over the crown and will not result in reintrusion of the tooth over the long term.

Basic orthodontic procedures for correction of canine impaction. Orthodontic strategy must be based on the position and complexity of the impacted tooth. It is crucial that the orthodontist establishes accurate appliance design for anchorage preparation, proper force delivery, and especially correct direction of force. Box 10-3 provides orthodontists with guidelines for step-by-step management of canine impaction.

Box 10-3	Step-by-step management of impacted maxillary canines
1. Prepare complete records, including clinical and paraclinical examinations, to enable clear identification of the problem and design accurate surgical and orthodontic techniques. 2. Accomplish orthodontic preparation prior to surgical intervention; this can include leveling, correction of rotation, elimination of crowding, and creation of space for the impacted tooth if tipping of adjacent teeth has resulted in insufficient space. 3. After all preliminary orthodontic movement is completed, refer the case for surgical intervention. The surgical approach should be based on the buccal or palatal inclination and vertical position of the impacted tooth and its proximity to adjacent teeth. 4. After a week or more of postsurgical healing, initiate orthodontic movement of the crown toward the edentulous site.	5. Design the direction and the amount of force delivered for traction of the impacted tooth to smooth the eruption path and prevent any damage to the impacted tooth and its supporting structure as well as the adjacent teeth. Follow biomechanical principles with regard to the specific conditions of each case. Among the appliance designs appropriate for anchorage preparation are the modified transpalatal arch, mini-implant or vertical and horizontal loop, auxiliary labial archwire, spring or chain, or elastic traction. For example, when the crown of a palatally impacted canine is in intimate contact with the lingual surfaces of the incisor roots, the first direction of movement should be to shift the impacted tooth away from the adjacent teeth to prevent root resorption.

Some investigators have reported on early uncovering of palatally impacted canines during the mixed dentition, so that the canines can erupt autonomously, without orthodontic intervention. They believe that when the impacted crown is available, regular bonding of brackets and movement into the dental arch can be accomplished more efficiently, and the overall active treatment time for the patient is reduced. Periodontal and esthetic results are also excellent. This procedure is not recommended if the canine is deeply impacted and its proximity to other tooth roots is harmful; in such cases, the need to move the impacted tooth quickly away from the roots is urgent (see Case 10-7).

When severe incisor crowding or protrusion is present and extraction of

premolars is indicated to correct general crowding and create space for the impacted tooth, the treatment plan must be designed very cautiously. One of the factors that can cause impaction of canines is ankylosis of the affected tooth; therefore, it is prudent to postpone extraction of the premolar in the quadrant with the impacted canine.

The surgical procedure and bonding of the attachment to the impacted tooth should be performed first, and orthodontic traction should be initiated. If tooth movement is not detectable after application of reasonable orthodontic force for 2 to 3 weeks (and in some cases intrusion of adjacent teeth might be noticed), ankylosis of the impacted tooth is confirmed. The best option in this situation is extraction of the ankylosed canine and substitution with the first premolar in that quadrant. Another option is autotransplantation of the impacted canine.

Complications in orthosurgical treatment of impaction. Complications may arise following orthosurgical treatment of impacted teeth if surgical exposure or orthodontic mechanotherapy is improperly designed or executed. Possible complications include gingival recession, gingivitis, ankylosis, devitalization, and external root resorption.

Autotransplantation of the canine

Surgical repositioning, or autotransplantation, of the impacted canine can be considered when interceptive or corrective treatment fails to improve the severely malpo-sitioned or horizontally inclined impacted tooth. Autotransplantation also can be considered as a proper option in other situations:

- When the impacted tooth is ankylosed
- If the patient desires to minimize or eliminate orthodontic treatment
- When the patient has a physical or mental disability and may benefit from limitation or elimination of orthodontic treatment

Complications associated with autotransplantation include root resorption, devitalization, pulpal obliteration, and ankylosis of the transplanted tooth.

Extraction of the impacted canine and space closure or prosthetic replacement

Another option in management of impacted maxillary canines is extraction of the impacted tooth, followed by orthodontic space closure or replacement with an

implant- or tooth-supported prosthesis. It is not always possible to recover all deeply impacted canines and reposition all impacted teeth within the alveolus. Removal might be indicated, for example, when the tooth is impacted very deeply and severely inclined, when the tooth's position and proximity to incisor roots contraindicate any tooth movement, or when the impacted tooth is ankylosed and patient compliance is not sufficient for transplantation.

Mandibular Canine Impaction

Mandibular canine impaction is rare compared with maxillary canine impaction. Grover and Lorton[85] found only 11 impacted mandibular canines in 5,000 individuals (0.22%). Röhrer[86] examined 3,000 patients radiographically and found 62 impacted maxillary canines (2.06%) and only 3 impacted mandibular canines (0.1%), a 20:1 ratio.

Many causes for impacted mandibular canines have also been proposed, including inadequate space, the presence of a supernumerary tooth or odontoma, premature loss of the primary canine, hereditary factors, functional disturbances of the endocrine glands, tumors, cysts, and trauma. Impaction, failure of eruption, and horizontal displacement of the mandibular canine are a common occurrence in patients with cleidocranial dysostosis.

Early loss of the primary canine or arch length deficiency in the anterior segment of the mandible is a common cause of buccal or lingual eruption of the mandibular permanent canine. However, this type of canine position cannot be considered impaction and may be treated by space provision or extraction.

In some situations, the tooth bud of the mandibular canine may become displaced or rotated in the alveolus because of some obstacle or for idiopathic reasons, and the canine root can develop in a horizontal direction (see Fig 10-23). In this situation, if the impacted mandibular canine is displaced horizontally below the apices of the incisors, the only option is extraction of the impacted canine to prevent damage to the roots of the incisors.

Depending on the patient's age, the position of the impacted canine, and the condition of the occlusion, there are several treatment options for mandibular impaction: observation, exposure, orthodontic traction, transplantation, and extraction.

Observation of the mandibular impacted canine is indicated if the canine is deeply impacted and asymptomatic and no associated pathosis is evident. Periodic

radiographic follow-up is necessary. In this situation, if the primary canine is in good condition and has a good root length, it can be maintained in the arch.

Case Reports

The following examples of different types of canine impaction illustrate orthodontic and orthosurgical approaches to the treatment of impacted teeth.

Case 10-4

Figures 10-36a and 10-36b show panoramic radiographs of an 11-year-old girl with a Class II malocclusion. Both maxillary canines were impacted. The panoramic radiographs were taken by her dentist 18 months apart, and no treatment was attempted in the interval. The right canine was impacted, and the left canine was ectopic and impacted. The radiographs revealed the proximity of the canine crown to the left lateral incisor root and some damage to the root. The cause of this abnormality was diagnosed as space deficiency on both sides.

Treatment:
The first step of the intervention was extraction of both maxillary left primary molars and creation of space for both canines by distalization of the molars and premolar with headgear (Fig 10-36c). The impacted right canine erupted automatically after space opening, but the ectopic and impacted left canine remained positioned over the lateral incisor roots. The next step was surgical exposure of the left canine and orthodontic traction. Figure 10-36d shows the patient's posttreatment occlusion, and Fig 10-36e reveals the resorption of the lateral incisor root.

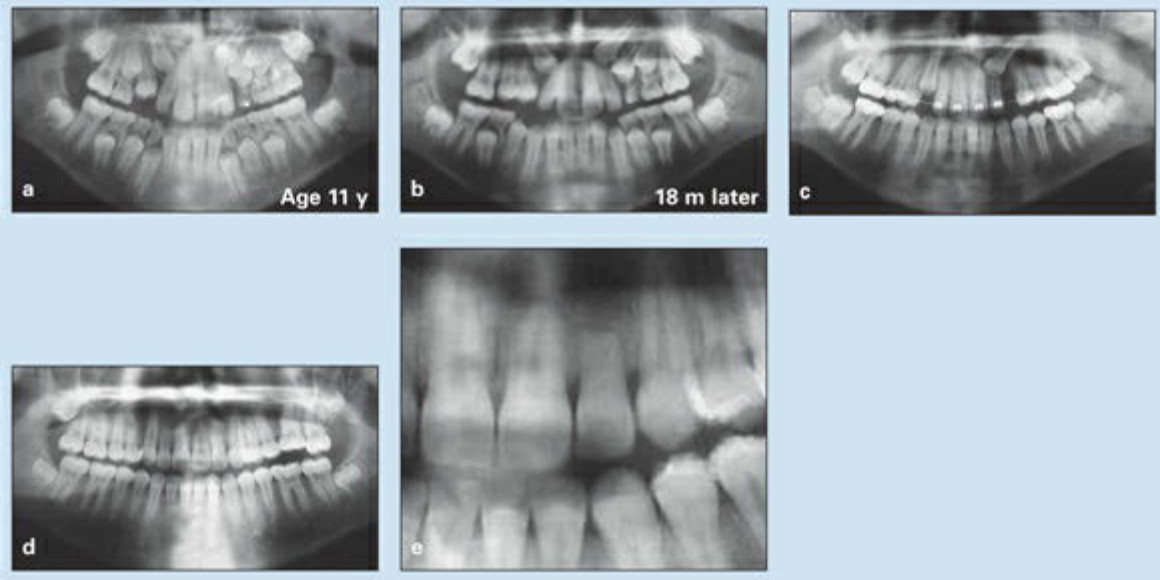


Fig 10-36 *(a and b)* Right and left maxillary canines impacted because of space deficiency. The left canine is also ectopic. *(c)* Panoramic radiograph taken after distalization of both maxillary segments to provide space for the canines. *(d)* Posttreatment panoramic radiograph. *(e)* Lateral incisor root resorption.

Case 10-5

An 11-year-old girl with a Class II division 1 malocclusion presented with displacement of both maxillary canines and maxillary space deficiency (Figs 10-37a to 10-37c). The mandibular dentition was normal. The left canine was displaced buccally and slightly overlapped the lateral incisor, while the right canine was severely impacted and ectopic.

Treatment:
Cervical headgear was used to correct the Class II molar relationships and create space for both maxillary canines. Figure 10-37d illustrates the patient's occlusion after molar retraction and space creation and before surgical exposure and bonding attachment. The left canine erupted spontaneously and was aligned. A spring was soldered to the palatal side of the molar buds for vertical traction and separation of the ectopic canine from the lateral incisor root as the first step of orthodontic traction (Fig 10-37e). Figures 10-37f to 10-37h show the posttreatment occlusion.

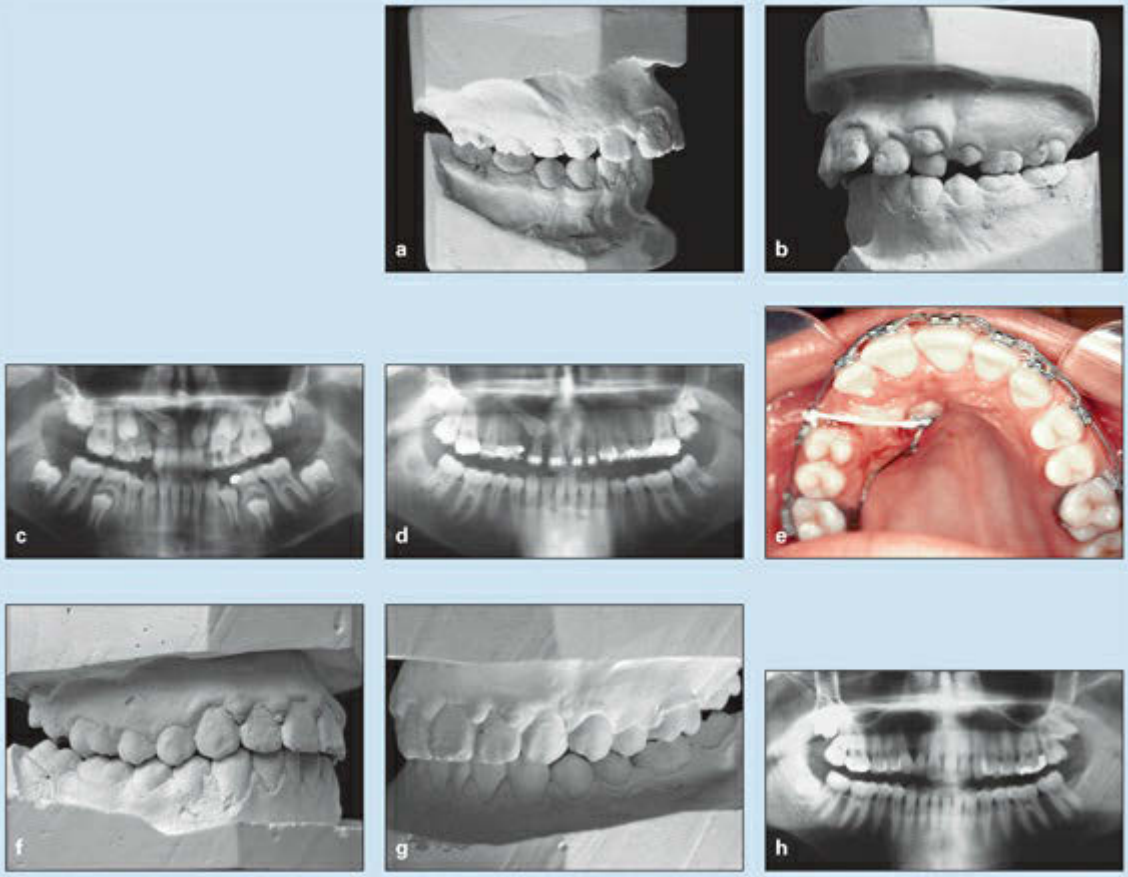


Fig 10-37 Management of maxillary canine displacement and space deficiency in an 11-year-old girl. *(a and b)* Pretreatment casts of the occlusion. *(c)* Pretreatment panoramic radiograph. *(d)* Panoramic radiograph taken after space creation and before surgical attachment. The left canine has erupted. *(e)* Spring used for vertical traction. *(f and g)* Posttreatment casts of the occlusion. *(h)* Posttreatment panoramic radiograph.

Case 10-6

A 15-year-old girl presented with a Class II severe deep bite and oligodontia; she was missing both maxillary lateral incisors and three mandibular permanent incisors. The maxillary left permanent canine was impacted and ectopic. The maxillary left primary canine and three primary incisor roots were still present (Figs 10-38a to 10-38d).

Treatment:
After consideration of the patient's age and evaluation of her complicated orthodontic problems, the following treatment plan was designed:

1. Extraction of the primary canine.
2. Alignment of the maxillary anterior teeth and closure of the diastema.
3. Surgical exposure and insertion of an attachment, which was a soft 0.022-inch stainless steel wire (see Fig 10-35). This was the technique available at the time, before bonding techniques had been developed.
4. Correction of the impacted canine.
5. Substitution of both maxillary canines for the lateral incisor positions.
6. Extraction of the three mandibular primary incisor roots, leveling of the mandibular arch, and intrusion of the maxillary central incisors to reduce overbite.
7. Prosthetic restoration to replace the missing mandibular incisors. The mandibular fixed prosthesis extended from canine to canine and included the single permanent incisor present.

Figures 10-38e to 10-38h show the patient's occlusion after completion of treatment.

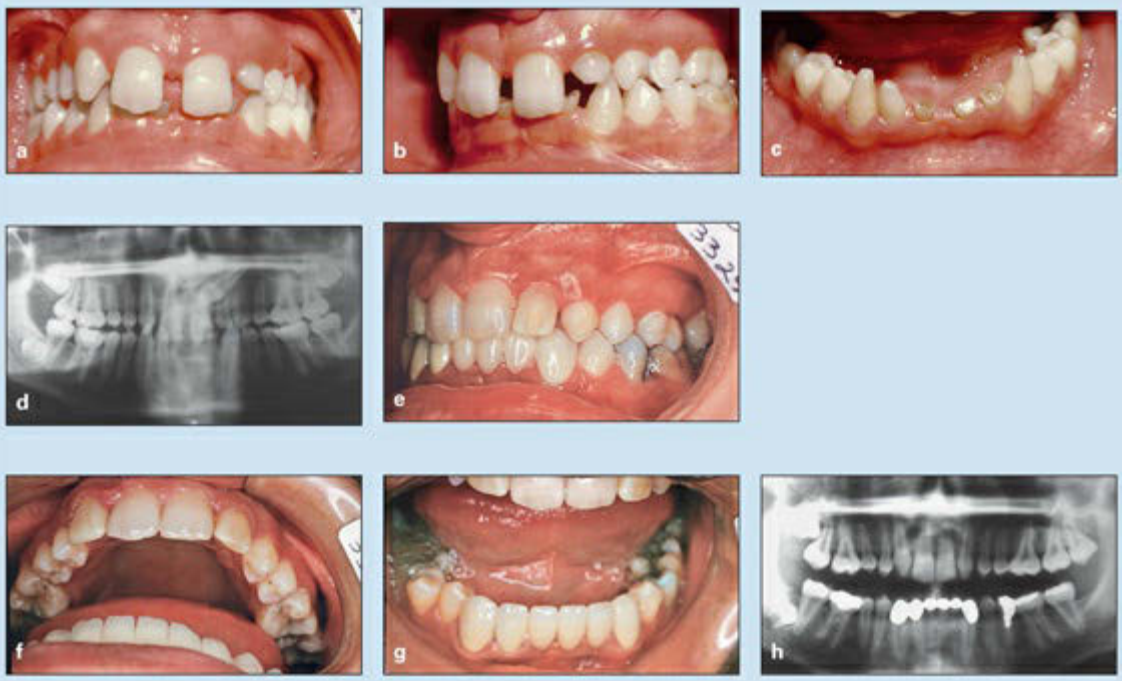


Fig 10-38 Management of an impacted and ectopic maxillary left permanent canine in a 15-year-old girl. She exhibits a Class II severe deep bite malocclusion and oligodontia. Both maxillary lateral incisors and three mandibular permanent incisors are missing. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e to g)* Posttreatment occlusion. *(h)* Post-treatment panoramic radiograph.

Case 10-7

A 13-year, 7-month-old girl presented with Class I malocclusion and minor anterior crowding. The crown of the maxillary right lateral incisor was tipped buccally due to force from the impacted canine. The maxillary right primary canine and first molar were still present. There was no canine bulge. The maxillary right canine and first premolar were deeply impacted because of a compound odontoma (Figs 10-39a to 10-39h).

Treatment:

After complete clinical and paraclinical evaluations, the patient was referred to an oral surgeon for removal of the odontoma, extraction of the overretained maxillary primary canine and first molar, and surgical attachment to the impacted canine and premolar. Because the odontoma tissue was extensive, removal was accomplished in two procedures (Figs 10-39i and 10-39j).

Orthodontic tooth traction was started with vertical movement of the impacted first premolar to prepare space for the impacted and ectopic canine, which had to be moved horizontally first because of its proximity to the incisor roots. Figures 10-39k to 10-39n show different phases of orthodontic traction: Fig 10-39k was taken during premolar traction; Fig 10-39l was taken after separation of the premolar and at the beginning of canine traction; and Figs 10-39m and 10-39n were taken after premolar eruption and during application of distal force to the canine to move it horizontally and separate it from the incisors.

Figure 10-39o shows the horizontal direction of force used to separate the impacted canine from the incisor roots. Figures 10-39p and 10-39q show canine traction. Figures 10-39r to 10-39t show the different stages of canine movement after its emergence. The results of treatment are shown in Figs 10-39u to 10-39x.

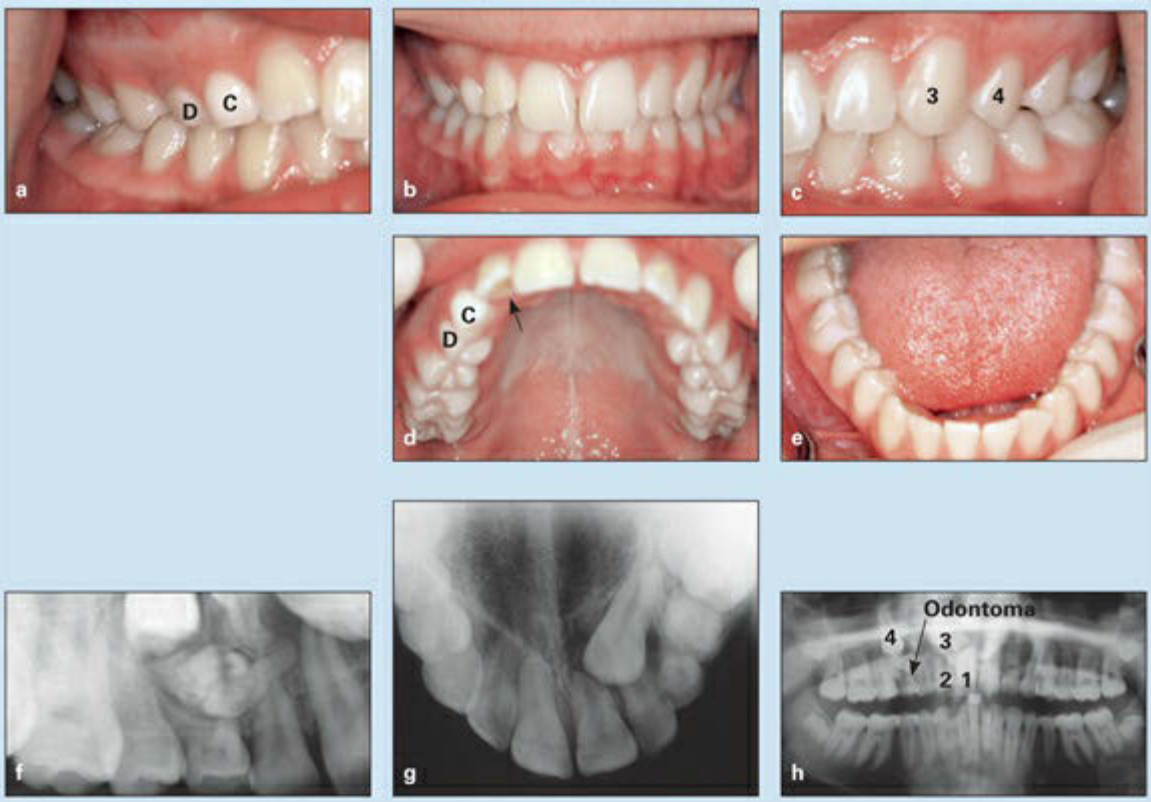


Fig 10-39 Management of maxillary canine impaction caused by an odontoma. This 13-year, 7-month-old girl had a Class I malocclusion, minor anterior crowding, and asymmetric canine and premolar eruption. *(a to e)* Pretreatment occlusion. C—maxillary right canine; D—maxillary right first premolar; 3—maxillary left canine; 4—maxillary left first premolar. *(f)* Pretreatment periapical

radiograph. *(g)* Pretreatment occlusal radiograph. *(h)* Pretreatment panoramic radiograph. The odontoma has caused displacement of the maxillary right canine (3) and first premolar (4). The impacted and ectopic canine is resorbing the roots of the lateral (2) and central (1) incisors.

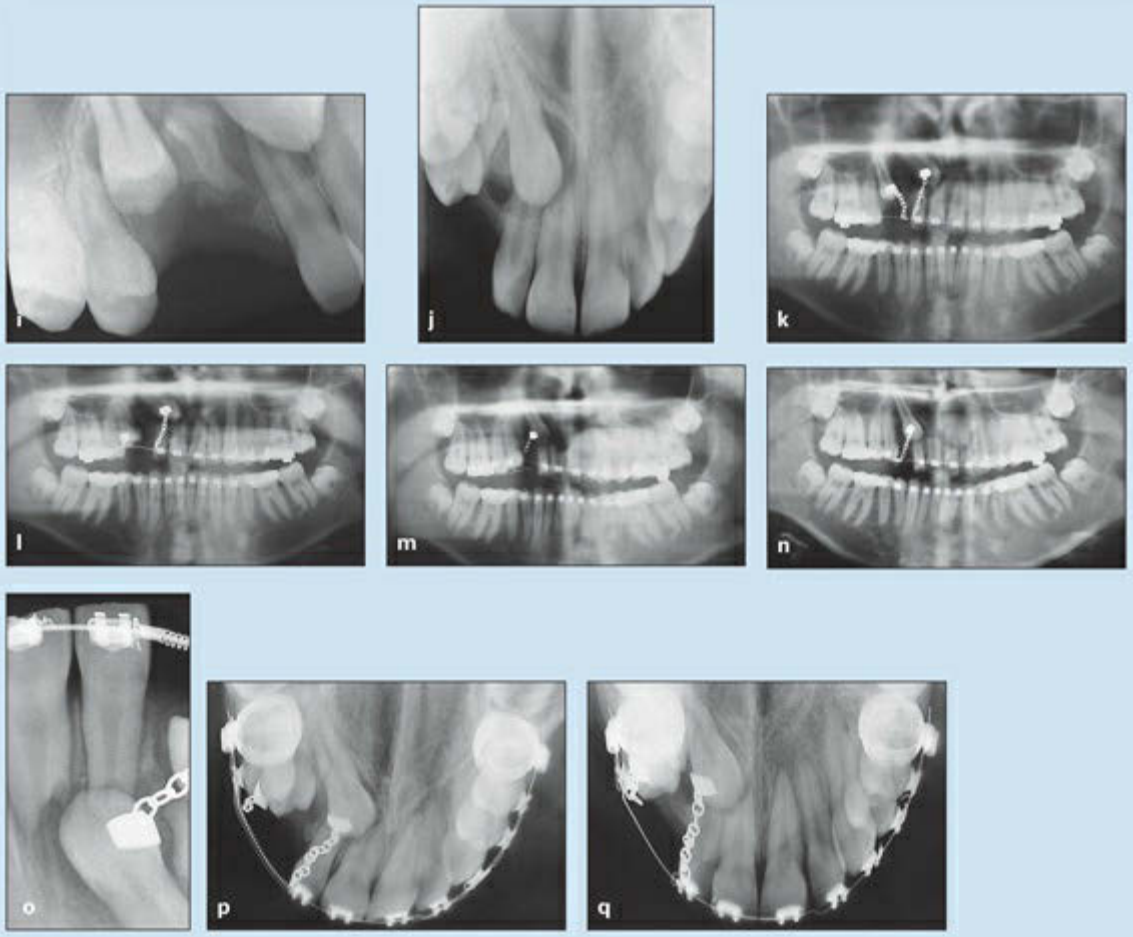


Fig 10-39 *(cont)* *(i)* Periapical radiograph taken after the second surgery to remove the odontoma. *(j)* Occlusal radiograph taken after the second surgery to remove the odontoma. *(k)* Panoramic radiograph taken during premolar traction. *(l)* Panoramic radiograph taken after premolar separation and at the beginning of canine traction. *(m and n)* Panoramic radiographs taken after premolar eruption and during application of distal force to move the canine horizontally and separate it from the incisor roots. *(o)* Horizontal traction of the canine. *(p and q)* Occlusal radiographs taken during canine traction.

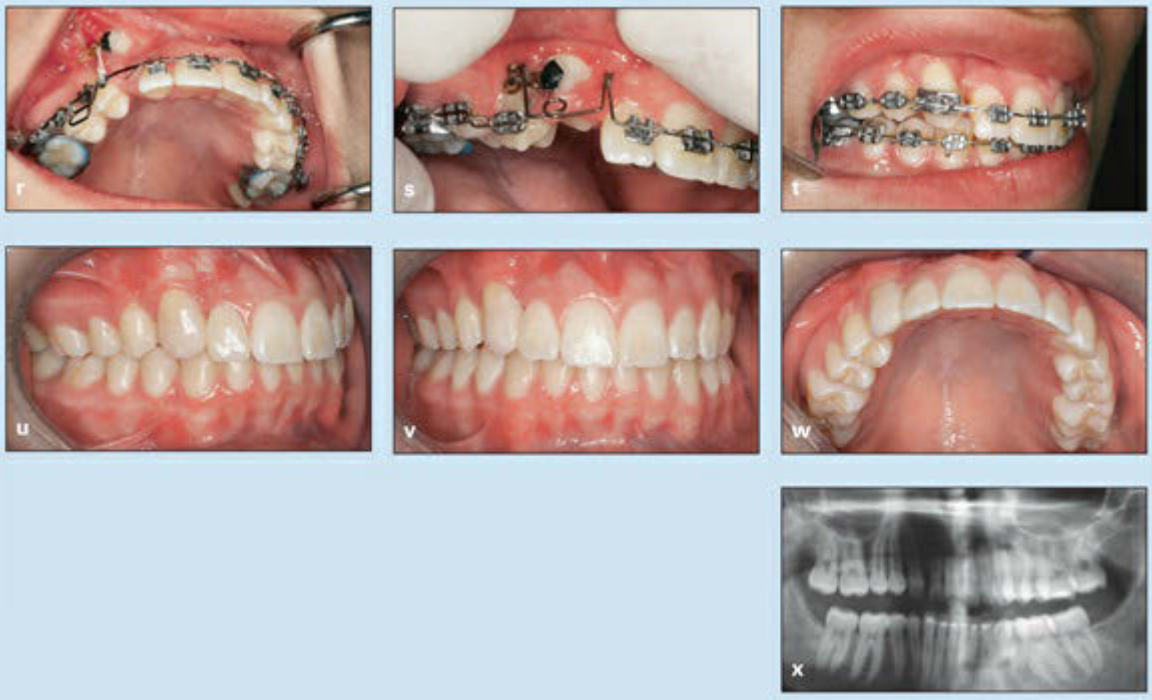


Fig 10-39 *(cont) (r to t)* Different stages of canine movement: traction, alignment, and uprighting. *(u to w)* Posttreatment occlusion. *(x)* Posttreatment panoramic radiograph.

Case 10-8

The possibility that an impacted canine is ankylosed must be carefully evaluated before a treatment plan is designed, especially when extraction of a premolar is an inevitable part of the orthodontic management. As discussed earlier, many factors can cause canine impaction; although it is rare, ankylosis is one of these factors. If the possibility of canine ankylosis is present, extraction of the premolar must be postponed until orthodontic traction of the surgically exposed canine has been attempted; if the orthodontic traction is ineffective, the best option would be extraction of the impacted ankylosed canine and substitution by the first premolar. This case is an example of this situation.

A 14-year-old girl presented with a Class I malocclusion, moderate anterior crowding, maxillomandibular dental protrusion, and an impacted maxillary right canine (Figs 10-40a and 10-40b).

Treatment:

Because of the maxillomandibular dental protrusion and moderate crowding, the treatment plan included extraction of the four first premolars. Because of the high position of the impacted canine in the absence of any obstacle preventing its eruption, ankylosis of this tooth was suspected. The first premolar on the maxillary left side was left in place. Three other premolars (maxillary right and mandibular left and right) were removed.

A surgical attachment was placed on the impacted canine, and orthodontic force was applied (Fig 10-40c). After a few weeks, no movement of the canine was noted, but intrusion of the adjacent teeth (lateral incisor, central incisor, and premolar) was produced (Figs 10-40d and 10-40e). Therefore, treatment was continued with extraction of the ankylosed canine and substitution of the premolar for the canine (Figs 10-40f to 10-40k).

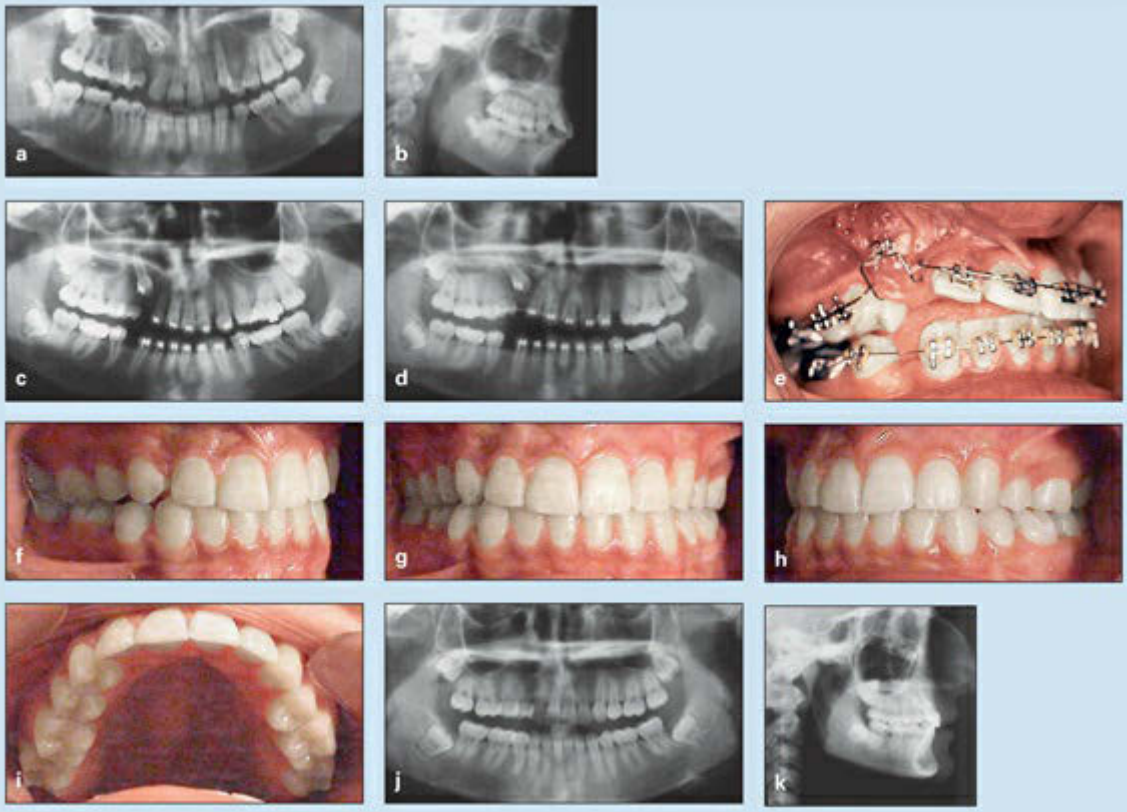


Fig 10-40 Management of an impacted maxillary right canine in a 14-year-old girl with a Class I malocclusion, moderate anterior crowding, and maxillomandibular dental protrusion. *(a)* Pretreatment panoramic radiograph. Ankylosis of the canine is suspected. *(b)* Pretreatment cephalometric radiograph. *(c)* Panoramic radiograph taken after surgical attachment and traction of the canine. *(d)* Panoramic radiograph taken after application of force, resulting in intrusion of the central incisor, lateral incisor, and canine. *(e)* Occlusion after unintended intrusion of the adjacent teeth. *(f to i)* Posttreatment occlusion after extraction of three premolars and the ankylosed maxillary right canine and substitution of the canine with the first premolar. *(j)* Posttreatment panoramic radiograph. *(k)* Posttreatment cephalometric radiograph.

Incisor Impaction

Eruption disturbances affecting the maxillary incisors during the early or middle mixed dentition are not uncommon. Any negligence in diagnosis and early intervention usually will result in other complications during the permanent dentition, including impaction, which is a rare condition. Impaction of the anterior teeth creates occlusal problems and can have serious dental, esthetic, and psychologic consequences for patients.

Etiology of incisor impaction

Several local etiologic factors can harm normal development and eruption of the anterior region. These factors include trauma, supernumerary teeth or odontomas, hypodontia, anomaly of tooth shape and size (peg lateral incisors), bone barriers or fibrotic tissue, space loss or space inadequacy, and delayed resorption of primary roots.

Trauma

Trauma to the maxillary primary anterior teeth is a common cause of disruption of maxillary incisor eruption. Various accidents during early stages of the primary dentition can have negative consequences due to the proximity of the roots of the primary teeth to their permanent successors. Several incisor disturbances have been attributed to trauma to the primary incisors,[87–90] including permanent tooth bud displacement, root dilaceration, complete arrest of root formation, enamel dysplasia, retarded eruption, failure of eruption, impaction, and transposition.

Da Silva et al[87] evaluated 389 pediatric records for a total of 620 traumatized primary teeth that received damage between 6 and 36 months of age. Clinical and radiographic examinations were carried out after eruption of the permanent successors. Developmental disturbances were detected in 126 teeth (20.2%). White or yellow-brown discoloration of enamel was found in 78.0% of teeth, and 86% of the teeth showed hypoplasia detected through radiographic analysis. Root alterations were rare; root dilaceration was observed in only one tooth. The researchers found that intrusive luxation and avulsion were associated with most of the cases of sequelae in the successor permanent teeth and concluded that special attention is required after such injuries. Severe injury to the periodontal ligament during intrusion may lead to ankylosis of the primary incisor and consequently to delayed or ectopic eruption of its permanent successor.

To examine the relationship between maxillary incisor impaction and canine displacement, Chaushu et al[91] retrospectively assessed 75 unilateral cases of incisor impaction using initial panoramic radiographs. They evaluated the positions of the ipsilateral and contralateral canines and the lateral incisors as well as the eruption progress of the canines. They found a significant increase in the prevalence and severity of displaced canines on the ipsilateral side (41.3%).

Considering the consequences of trauma to primary incisors, special attention and radiographic follow-up for children who suffered dental trauma at an early age are important to monitor possible sequelae in the permanent successors even before their eruption (Fig 10-41).

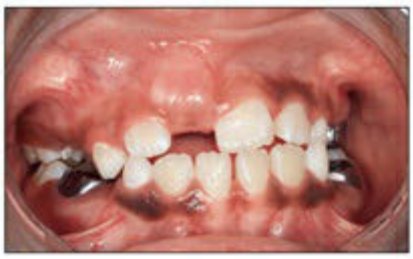


Fig 10-41 Impaction of the maxillary right central incisor caused by trauma to the primary incisors.

Supernumerary teeth or odontomas

Supernumerary teeth (mesiodentes) are common in this region and can cause several problems for incisors and canines, including incisor crowding and rotation, severe diastemata, incisor displacement, delayed incisor eruption, failed incisor eruption, incisor root resorption, and incisor impaction (Fig 10-42). The best management is early detection and extraction of any supernumerary and primary teeth, if present, and maintaining the space for permanent tooth eruption. After removal of the supernumerary tooth and space maintenance, spontaneous eruption of the permanent incisors occurs.

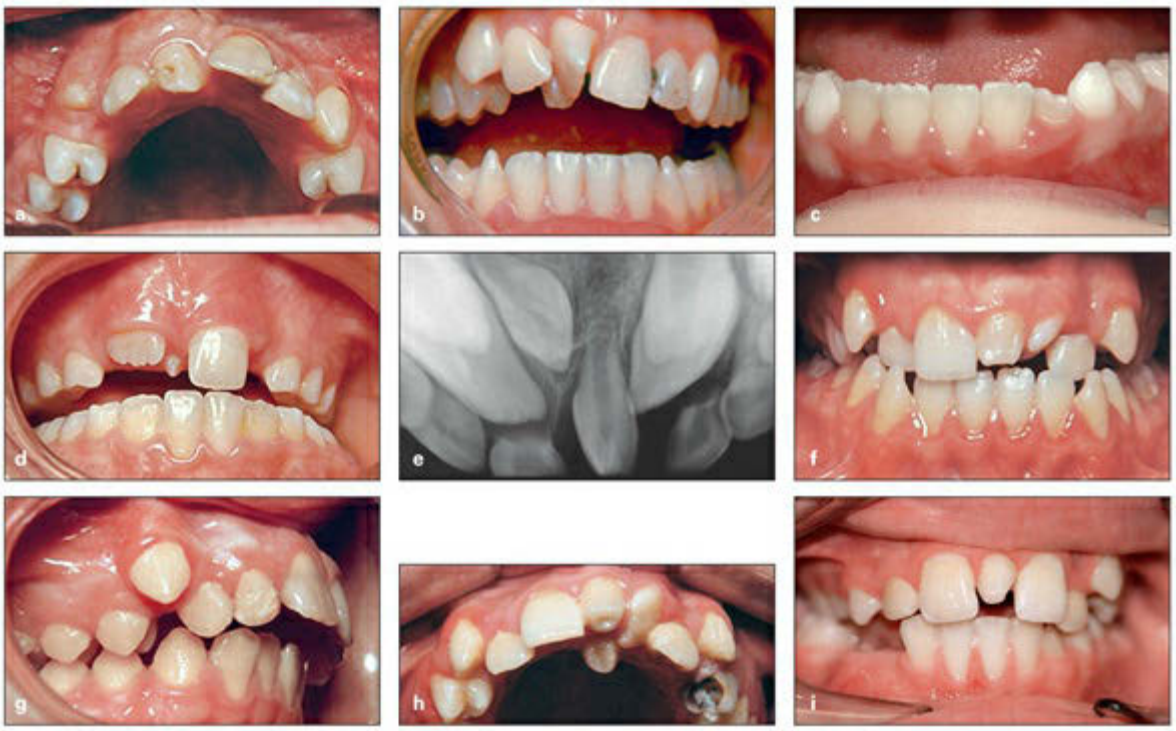


Fig 10-42 *(a to i)* Eruption problems, including incisor impaction, caused by supernumerary teeth in the incisor region.

Hypodontia or peg-shaped lateral incisors

Congenital absence or abnormal shape of the lateral incisors also disrupts anterior tooth eruption, resulting in problems such as diastema and canine impaction.

Bone barriers or fibrotic tissue

Sclerotic tissue or dense bone that covers incisors, which is common following premature loss of primary incisors, can interfere with the normal eruption pattern of incisors. It can result in delayed eruption, space loss and consequently crowding of the incisors, abnormal sequence of eruption, and even impaction of the incisors.

Space loss or space inadequacy

Space loss in the incisor region following premature loss of the primary teeth to trauma or caries disrupts normal eruption of permanent incisors and can result in crowding of the permanent incisors, delayed eruption, failed eruption, and even impaction of the incisors.

Overretained primary incisors

Primary incisors that are overretained because of ankylosis or infection and necrotic pulp can deflect erupted teeth, delay or prevent eruption, and cause impaction of the permanent incisors.

Early detection and diagnostic considerations

The best means of early detection of these anomalies is periodic radiographic evaluation during the transitional dentition. There are variations in the time of shedding of primary teeth and emergence of their permanent successors, but significant deviation from the established norms is a sign of potential impaction. The following clinical signs should alert clinicians who are examining a child during the mixed middle dentition to suspect incisor impaction:

- Delayed exfoliation of primary incisors
- Delayed eruption of permanent incisors
- Asymmetric eruption of permanent incisors
- Severe diastemata
- Abnormal sequence of eruption (eg, eruption of maxillary lateral incisors before

central incisors)

Interceptive treatment for incisor impaction

The best management for incisor eruption problems is early intervention to eliminate etiologic factors and guide eruption. This interceptive management includes the following steps:

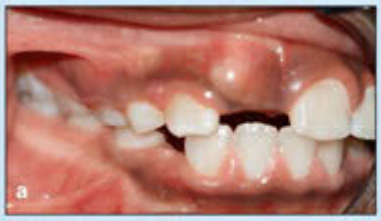
1. Recognition of the cause through careful clinical and radiographic examination.
2. Elimination of the cause, such as extraction of a supernumerary tooth or an odontoma, maintenance or regaining of necessary space for the permanent incisors, or removal of bone barriers or fibrotic tissue that is preventing eruption.
3. After removal of the obstacle or overretained primary tooth, maintenance of the space for permanent eruption and prevention of a midline shift. If there is no significant progress of permanent tooth eruption after the elimination of the cause and maintenance of the necessary space for 6 to 12 months, surgical exposure and orthodontic traction of the impacted incisor to the normal position is the only option. The surgery and bonding attachment are usually accomplished through a closed-eruption technique.

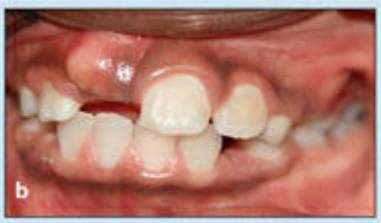
Case 10-9

An 8-year-old boy with a Class I malocclusion and minor maxillary anterior crowding had a chief complaint of an unerupted maxillary right central incisor (Figs 10-43a to 10-43c). His mother indicated that the left incisor had erupted more than 1 year earlier. He had a history of trauma to the primary incisors.

Treatment:
The first step in treatment was maxillary 2 × 4 bonding to align the maxillary incisors and provide sufficient space for the unerupted central incisor and placement of a lower holding arch to preserve the mandibular leeway space for the integrity of the mandibular permanent dentition.

The next step was exposure of the unerupted incisor and placement of a bracket for orthodontic traction. Figures 10-43d to 10-43f illustrate the different phases of active treatment. Figures 10-43g to 10-43j show the results of treatment. The only appliances used were a lower holding arch and maxillary 2 × 4 bonding.





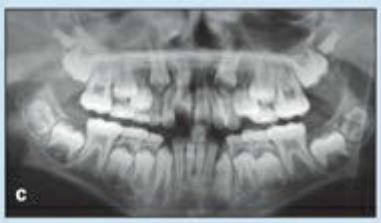


Fig 10-43 Management of permanent central incisor displacement and impaction in an 8-year-old boy with a history of trauma to the primary incisors. *(a and b)* Pretreatment occlusion. *(c)* Pretreatment panoramic radiograph.

Fig 10-43 *(cont) (d)* Lower holding arch to preserve leeway space. *(e)* Tooth exposure and traction. *(f)* Final phase of active treatment. *(g to i)* Posttreatment occlusion. *(j)* Posttreatment panoramic radiograph.

Case 10-10

A 9-year-old boy presented with an unerupted maxillary right central incisor, end-to-end anterior relationships, and some space deficiency (Figs 10-44a to 10-44d).

The author always suggests retrospective evaluation of a patient's record to find the initial cause of the problem; Figs 10-44e to 10-44g clearly answer this question. The first periapical radiograph reveals neglected deep caries in the primary dentition; the second radiograph, taken at the beginning of permanent incisor eruption, reveals the remnants of the pulpless primary incisors and developing periapical cyst; consequently, the third radiograph reveals asymmetric eruption.

Treatment:
Figures 10-44h to 10-44j show the different phases of mechanotherapy, and Figs 10-44k and 10-44l show the guidance of eruption. Figures 10-44m to 10-44q show the tooth alignment that has been achieved through maxillary 2 × 4 bonding and a mandibular lingual arch appliance.

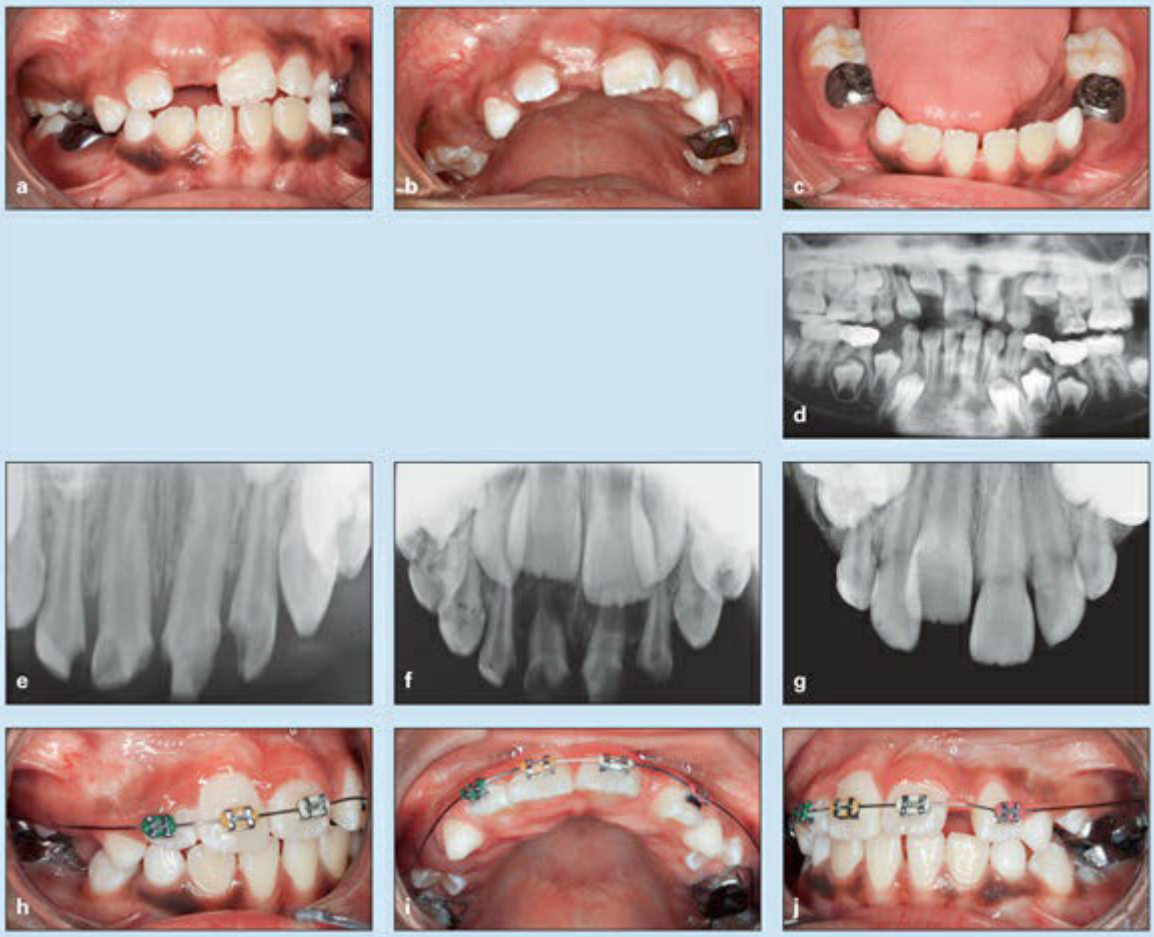


Fig 10-44 Management of an unerupted maxillary right central incisor in a 9-year-old boy with end-to-end anterior relationships and space deficiency. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e to g)* Pre-referral occlusal radiographs showing neglected necrotic primary incisors and cyst formation, causing asymmetric eruption. *(h to j)* Phases of mechanotherapy, with *(j)* showing the occlusion before midline correction.

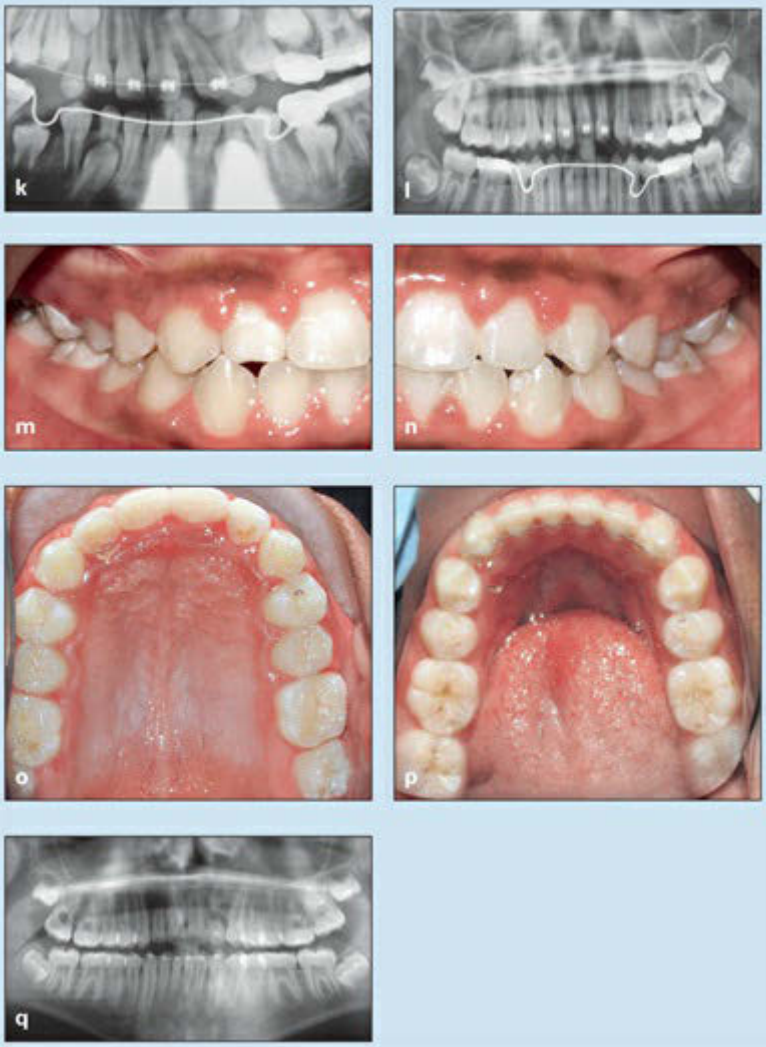


Fig 10-44 *(cont) (k and l)* Guidance of eruption. *(m to p)* Posttreatment occlusion. *(q)* Posttreatment panoramic radiograph.

Case 10-11

Mesiodentes, or supernumerary teeth in the maxillary incisor area, are one of the most common types of supernumerary tooth. They can cause various problems for adjacent teeth, including displacement, cyst formation, structural damage, delayed eruption, failed eruption, and impaction. Early detection and proper management is the key to a successful treatment result (see chapter 8).

After extraction of the supernumerary tooth, maintenance of space for the incisors is important, especially if the unerupted permanent incisor is positioned high and early eruption is not predicted. If space is not maintained after extraction, other problems can arise in the incisor area. This case is an example of this condition.

A 7-year-old girl had two mesiodentes preventing eruption of the maxillary right central incisor (Fig 10-45a). After extraction of the mesiodentes, the space was not maintained and no treatment was performed. Because of crowding around both the maxillary and the mandibular incisors, space loss was rapid. Figures 10-45b to 10-45d show the patient's occlusion about 1 year after extraction of the mesiodentes. Unfortunately, the parents did not want to start the treatment at this stage of the problem

because of a lack of insurance coverage.

The patient returned 1 year later, when the parents were willing to start treatment. Figures 10-45e to 10-45h reveal the patient's occlusion at the second referral before treatment. The long period of neglect and delayed treatment clearly complicated the patient's problems, which could have been prevented by proper space maintenance immediately after extraction of the mesiodentes. In the absence of prevention, the second choice would have been simple 2 × 4 bonding and interceptive treatment when the patient was referred at the earlier age, before eruption of all the incisors.

The problems at this late stage consisted of severe maxillary crowding, rotation, and tooth displacement; moderate mandibular crowding; locked crossbite of the right lateral incisor; and dental midline shift.

Treatment:

The first step of treatment was complete maxillary bonding and placement of occlusal composite resin to disocclude the anterior crossbite and locked occlusion. After crossbite correction, the second step was removal of the mandibular occlusal composite resin and insertion of mandibular bonding. Figures 10-45i to 10-45l show different phases of treatment. Figures 10-45m to 10-45q show the posttreatment occlusion.

Fig 10-45 Management of an unerupted maxillary right central incisor in a 7-year-old girl. Eruption of the central incisor was prevented by two mesiodentes. *(a)* Panoramic radiograph taken before extraction of the mesiodentes. *(b to d)* Occlusion 1 year after extraction of the mesiodentes. Space loss has been rapid.

Fig 10-45 *(cont) (e to g)* Occlusion an additional 1 year later, revealing the consequences of delayed treatment. *(h)* Pretreatment panoramic radiograph. *(i to l)* Phases of mechanotherapy. *(m to q)* Posttreatment occlusion.

Ankylosis

Ankylosis is a specific abnormality of tooth eruption and development that is caused by the fusion of cementum with the alveolar bone; it is a progressive anomaly of tooth eruption that can occur before, during, or after tooth eruption and usually has a profound effect on occlusion.

Both permanent and primary teeth may become ankylosed, but primary teeth become ankylosed far more frequently than do permanent teeth; the ratio is about 10 to 1. Mandibular teeth are ankylosed more than twice as often as maxillary teeth. Almost all infraoccluded primary molars are the result of ankylosis. The most frequent ankylosed teeth are mandibular primary second molars, then first molars, and then maxillary primary molars.

Ankylosis may occur at any time during the eruption process and as soon as the fusion between alveolar bone and cementum occurs. Ankylosis leads to cessation of the eruptive movement. As the vertical growth of the jaw and alveolar process of the adjacent occlusal plane continues, it gives the impression that the ankylosed tooth is submerged (Fig 10-46).

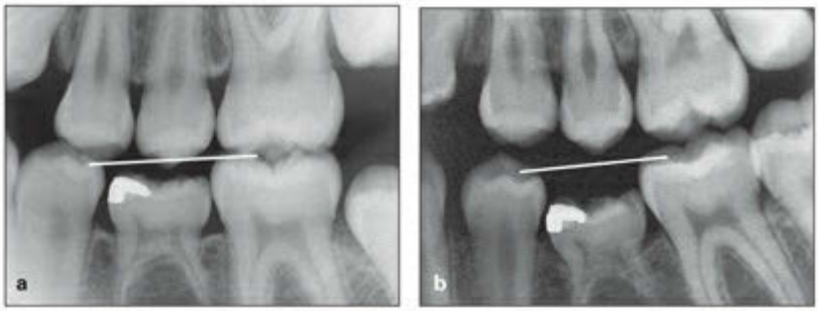


Fig 10-46 *(a and b)* Periapical radiographs showing the increasing submergence of the primary molar over 16 months.

Tooth ankylosis, especially in the primary dentition, deserves much more attention by the profession because of the different types of damage that may be associated with this anomaly. Early recognition and proper intervention are important.

Prevalence

The frequency of infraerupted or ankylosed teeth reportedly ranges from 1.3% to 38.5%; it seems this variation is related to the age of patients. Kurol[92] evaluated 1,059 Swedish children aged 3 to 12 years. The age groups were evenly distributed. Infraocclusion was found beginning at age 3 years, and the overall prevalence of infraocclusion for the whole sample was 8.9%. Prevalence varied between age groups; the maximum, 14.3%, was found in 8- and 9-year-old children, and the

minimum, 1.9%, was found in 12-year-old children. The mandibular primary molars were affected more than 10 times as often as the maxillary molars. The highest rate was found in mandibular second molars. The second molar was the tooth most commonly found in infra-occlusion. Supporting the hypothesis that there is a familial tendency in infraocclusion of primary molars, Kurol[92] found that the prevalence of infraocclusion was 18.1% in 138 siblings aged 3 to 12 years, more than double the frequency in the total sample (8.9%).

Biederman[93] reported that primary molar ankylosis is most prevalent during the middle mixed dentition stage of development and that the primary second molars are affected more often than the primary first molars. He also reported that the prevalence of ankylosed primary molars was greater in the mandible than in the maxilla.

Krakowiak[94] reported a higher prevalence of ankylosis among white children (4.10%) and a lower prevalence among black children (0.93%).

Etiology

The biologic mechanism by which a tooth can become ankylosed has not been demonstrated. However, as long as the periodontal membrane remains intact, there is no possibility of contact between cementum and alveolar bone and therefore no fusion can develop. Among the possible causes are a genetically determined gap in the membrane that is followed by a filling in of the bone; some trauma or other environmental factor that has created a rupture; or a direct local ossification of the membrane itself, which is not likely. In radiographic evaluation, no trace of the lamina dura or periodontal membrane can be found at the site of ankylosis.

Several theories regarding tooth ankylosis have been proposed. The three main theories are heredity, trauma, and intermittent primary root resorption, although other causes have been suggested.

Heredity

The conclusion that heredity is responsible has been drawn from the observation of ankylosis in several members of the same family. This lends support to the theory that it follows a familial pattern; likewise, the presence of other dental anomalies in conjunction with ankylosis supports a genetic etiology for this anomaly.[69,95–97]

Trauma

Another etiologic factor that has been proposed by different investigators is trauma.[92,98] This perception is partially drawn from clinical experience, such as the high frequency of ankylosis that can be seen in reimplanted avulsed incisors. Traumatically induced ankylosis in experimental animals also suggests that trauma to the periodontal membrane may be an etiologic factor in fusion of the root cementum to the adjacent alveolar bone.[93]

Intermittent primary root resorption

Another theory is that resorption of the primary roots is an intermittent process; that is, there is a period of active resorption followed by a period of repair, so that the primary teeth during root resorption periodically loosen and then tighten again before the whole root is resorbed. According to this theory, when the resorption bays are filled in with secondary cementum or bone, the periodontal membrane can be reattached and hold the teeth tightly again. In some cases, the reparative process may extend and not only fill in the resorption bays but overfill them, causing ankylosis between the root and the alveolar bone. When this occurs, the primary tooth becomes locked to the bone and cannot erupt any further. At the same time, adjacent teeth continue their eruption, leaving the ankylosed tooth below the plane of occlusion. In some severe conditions, they become submerged (see Fig 10-46).

Other theories

Other hypotheses for tooth ankylosis have also been proposed, such as disturbed local metabolism, localized infection, or chemical or thermal irritation. The disturbed local metabolism theory, as Biederman[99] stated, is based on the belief that root resorption in primary teeth normally precedes disappearance of the periodontal ligament. Disruption in local metabolism leads to obliteration of the periodontal ligament first, and the resulting close contact of the bone to the tooth structure allows union.

Effects on the dentition

Association of primary molar ankylosis with various developmental disturbances in the permanent dentition has been routinely reported; these anomalies include aplasia of the succedaneous tooth, ectopic eruption or impaction of the premolar,

infraocclusion of the ankylosed tooth (leading to tipping of the adjacent teeth), overeruption of the opposing tooth, and taurodontism of the permanent first molar. Ankylosed primary teeth that are located below the occlusal plane, especially submerged ankylosed primary molars, need special attention at their early stage of development before complications arise:

- They may interfere with the eruption of succeeding permanent teeth, causing ectopic eruption.
- Arch length loss can result from tipping of adjacent teeth (Fig 10-47).
- Extrusion of teeth in the opposing arch may occur (Fig 10-48).
- Submergence of primary teeth damages the occlusion and can be harmful to the jaws and health of the patient (Fig 10-49).
- Transposition of the second premolar to the place of the missing permanent first molar is a rare consequence of primary second molar ankylosis (Fig 10-50).
- Submerged primary teeth not only prevent eruption of the permanent teeth but also can prevent vertical alveolar growth and cause lateral open bite (see Fig 10-48).

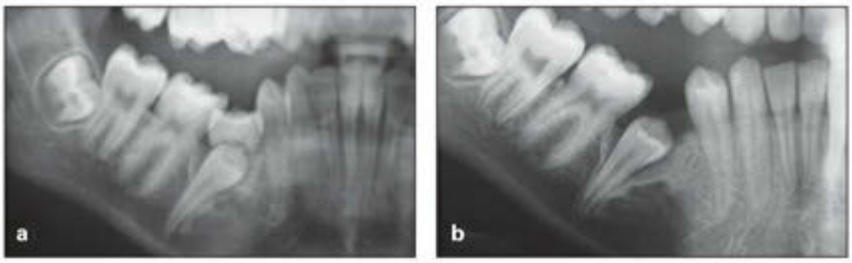


Fig 10-47 *(a and b)* Ankylosed primary molars causing space loss.

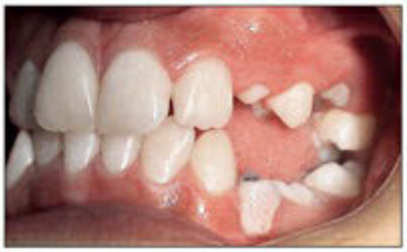


Fig 10-48 Ankylosis preventing eruption of permanent teeth and causing lateral open bite and the possibility of overeruption of the opposing teeth.

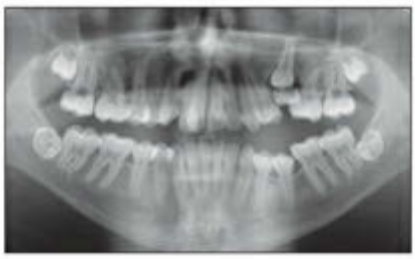


Fig 10-49 Submerged maxillary left primary first molar causing impaction of the permanent second premolar.

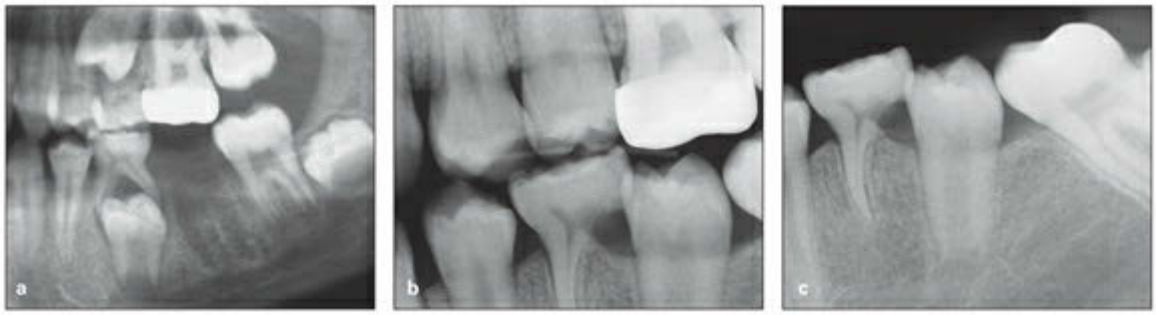


Fig 10-50 *(a to c)* Transposition of the second premolar to the site of the permanent first molar as a consequence of extraction of the first molar and primary second molar ankylosis.

Diagnosis

Recognition of primary tooth ankylosis is not difficult; the condition can be diagnosed through careful clinical and radiographic examinations; histologic changes in the area of fusion have also been reported.[100,101]

Clinical examination

Ankylosed primary molars show the following clinical signs:

- Ankylosed primary molars often present with infraocclusion relative to adjacent teeth; however, depending on the time of fusion, they may be at the same occlusal level as adjacent teeth.
- On percussion, an ankylosed tooth gives a solid sound, whereas the normal adjacent teeth give a cushioned or dull sound.
- Ankylosed teeth lack mobility even when root resorption is advanced.

Radiographic evaluation

Radiographs can show obliteration of the periodontal ligament space and absence of

continuity of periodontal ligament in some areas. The roots are less radiopaque, and in some cases of advanced fusion, the root is less distinguishable from surrounding bone.

Radiographic assessment is useful only when the area of fusion is extensive and when the site of fusion is located on the mesial or distal surface of the roots. Detection of fusion on the palatal and buccal aspects is impossible because of the superimposition of other structures.

Histologic findings

Mancini et al[100] and Haselden et al[101] reported histologic changes in areas of fusion of cementum and bone, fibrotic periodontal ligament remnants with very few cells, and absence of mucopolysaccharidase activity, which is essential for the normal process of root resorption during eruption of the permanent successor.

Management of ankylosed primary teeth

Early recognition is extremely important in the management of an ankylosed tooth. Although the treatment eventually may involve surgical removal of the affected tooth, early detection and proper management facilitate normal eruption and guidance of the successor tooth and prevent potential complications.

Treatment considerations

The treatment procedure depends on the following factors:

- Whether the ankylosed tooth is a primary or a permanent tooth
- The time of onset and stage of eruption; early fusion and submergence result in more complications
- The degree of infraocclusion
- The patient's chronologic and dental ages
- The location of the affected tooth and the normal time of exfoliation
- The presence or absence of a permanent successor
- The root development of the successor tooth, the proximity of the permanent tooth to the ankylosed primary tooth, and the possibility of deflection of the permanent tooth

- The patient's occlusion and presence of crowding or spacing

The importance of the presence of a permanent successor for normal exfoliation of a primary molar has been emphasized by Kurol and Thilander.[98] They indicated that there was no spontaneous exfoliation or very slow root resorption for most of the ankylosed teeth of primary molars without a permanent successor.

There are controversies regarding the timing of extraction of severely infraoccluded and ankylosed primary molars. Messer and Cline[102] emphasized proper timing of extraction of severely infraoccluded ankylosed primary molars and the risk of future periodontal damage to the mesial alveolar support of permanent first molars.

However, Kurol and Olson[103] studied 143 permanent molars adjacent to 119 infraoccluded primary molars and 24 normal contralateral primary molars in 68 individuals. The subjects were reexamined 8 years after the exfoliation or extraction of the infraoccluded primary second molars. The alveolar bone level mesial to the permanent first molars was measured on bitewing radiographs. They reported that all permanent first molars showed a normal alveolar bone level mesially except two that showed 3- to 4-mm bone loss but no pocket. They concluded that infraocclusion and ankylosis of primary molars does not constitute a general risk for future alveolar bone loss mesial to the permanent first molars.

Treatment options

Treatment options include periodic clinical and radiographic observation, an attempt at luxation, extraction of the involved tooth, extraction and space maintenance, extraction and implant or prosthetic substitution, or extraction and orthodontic intervention and space closure:

- If the ankylosed tooth is interfering with eruption of the successor tooth, immediate extraction is recommended. If the emergence time of the permanent successor is predicted to be more than 6 months later, a proper space maintainer must be provided.
- If the successor is present, the onset of ankylosis in the primary tooth is late, and the permanent successor is usually not affected by the ankylosed tooth, periodic evaluation and removal of the ankylosed tooth at the proper time is recommended.
- If the successor is present, the onset of ankylosis is early, the occlusal level is low, and the possibility of submergence and disturbed eruption of the permanent

tooth is predictable, extraction of the ankylosed tooth and space maintenance is recommended.

- If the successor is missing, depending on the patient's type of occlusion and the situation of the ankylosed tooth, different procedures can be applied:
  - If the ankylosed tooth has a good prognosis, it can be left in place. It should be restored with composite resin buildup or a crown if it is below the occlusal level to prevent overeruption of the opposing tooth.
  - The ankylosed tooth can be extracted if the patient has crowding and needs extraction in the other segments of the arch. The space can be closed orthodontically.
  - If the ankylosed tooth has poor structure and the patient has good occlusion and no need for orthodontic treatment, the ankylosed primary tooth can be extracted at an early age and a provisional bonded restoration can be placed. The permanent tooth should then be replaced by an implant or prosthetic treatment at a proper age.
- If the ankylosed tooth is a permanent tooth and the onset of ankylosis is early, surgical luxation might help.
- If repeated luxation is not useful, extraction is recommended to prevent submergence of the tooth.
- Reimplantation of an ankylosed permanent tooth is another option.
- If the onset of ankylosis in a permanent tooth is late, luxation is not effective, and there is no evidence of submergence, the tooth can be built up later to prevent overeruption of the opposing tooth.

Therefore, the general treatment recommendation for an ankylosed primary tooth that is not disturbing the patient's occlusion or its permanent successor is to wait and monitor for normal exfoliation of the primary tooth and eruption of the successor. If an abnormality is associated with the succedaneous tooth (such as agenesis or ectopic eruption), early intervention will most likely be the best option to follow.

Case 10-12

This case is an example of proper intervention and guidance of eruption of a permanent dentition that was disrupted by submerged primary molars.

A 14-year-old girl with a Class I malocclusion and normal skeletal pattern presented with four primary molars that were ankylosed and submerged, preventing eruption of the premolars, which had

almost completely developed roots (Figs 10-51a to 10-51c).

The proper timing of intervention for this patient would have been around the age of 10 years, when the premolar roots lengths were at least half formed and before there was any effect on the dentition.

Treatment:

The first step of treatment was anchorage preparation for the maxillary and mandibular molars by placement of a maxillary Nance appliance and a lower holding arch and extraction of the ankylosed primary molars (Figs 10-51d and 10-51e). Lateral cephalometric and panoramic radiographs show guidance of the premolars (Figs 10-51f and 10-51g); spontaneous eruption was achieved without any mechanotherapy. Figures 10-51h to 10-51n show the treatment results.

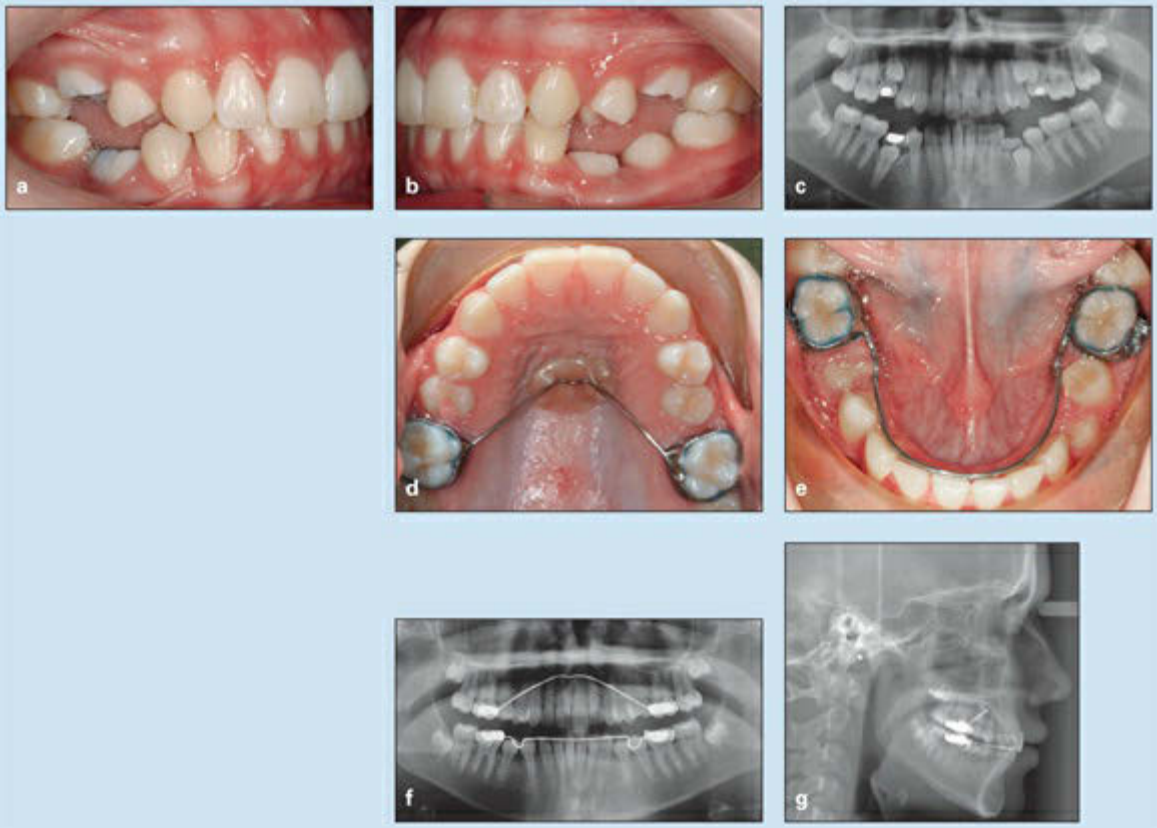


Fig 10-51 Proper intervention and guidance of eruption in a 14-year-old girl with submerged primary molars. The ankylosed primary molars prevent eruption of the premolars, which have almost completely developed roots. *(a and b)* Pretreatment occlusion. *(c)* Pretreatment panoramic radiograph. *(d and e)* Anchorage preparation and extraction of all ankylosed primary molars. *(f)* Panoramic radiograph during premolar eruption, which occurred spontaneously without mechanotherapy. *(g)* Cephalometric radiograph during premolar eruption.

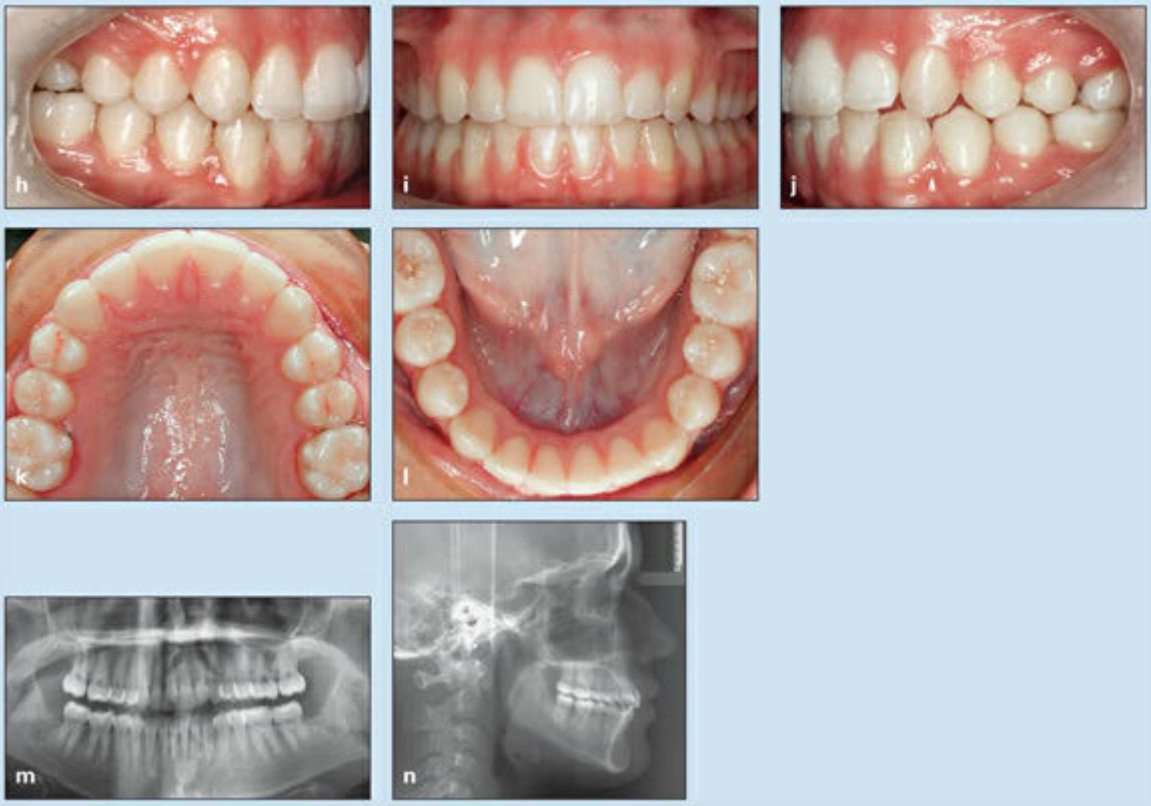


Fig 10-51 *(cont) (h to l)* Posttreatment occlusion. *(m)* Posttreatment panoramic radiograph. *(n)* Posttreatment cephalometric radiograph.

Summary

- *Eruption* is the movement of a tooth from its developmental site within the alveolar process to its functional position in the oral cavity; this process takes about 5 years for the permanent dentition.
- During this prolonged path of movement, many events can disturb normal tooth eruption.
- Eruption disturbances can be broadly classified into two general categories: disturbances related to the time of eruption of the affected tooth and disturbances related to position of the affected tooth.
- Defects in tooth formation and eruption have been attributed to systemic, genetic, and local factors.
- During different stages of eruption, these factors can cause problems such as delayed tooth eruption, early exfoliation, premature tooth eruption, primary and secondary failure of eruption, retarded tooth development, abnormal sequence of eruption, ectopic eruption, transposition, abnormal path of

eruption, impaction, and ankylosis.

- Many theories about tooth eruption have been proposed; recent reports indicate that the tooth itself does not contribute to the eruption process. Rather, osteoclastogenesis and osteogenesis needed for eruption are regulated by differential gene expression in the dental follicle, both chronologically and spatially. Osteoclastic activity causes occlusal bone resorption in the bony crypt and creates an eruption tunnel to facilitate eruption. Simultaneously, new bone formation fills the space left behind the crown and growing root.
- Tooth eruption requires at least the presence of three important conditions: *(1)* the dental follicle, *(2)* alveolar bone resorption to produce an eruption pathway, and *(3)* alveolar bone formation at the base of the bony crypt.
- Longitudinal monitoring of the transitional dentition with panoramic radiographs is an accurate means for early detection of eruption problems. The following are the consecutive steps of primary tooth replacement by permanent successors that can be seen in a normal site of dental transition. Any disturbance of these steps can be a sign of problems:

- Resorption of the primary tooth root
- Lengthening of the permanent root
- Exfoliation of the primary crown
- Resorption of overlying bone to clear the path of eruption
- Bone apposition under the moving tooth germ to fill the space left behind
- An increase in alveolar height
- Movement of the permanent tooth through bone
- Piercing of the alveolar crest at two-thirds' root formation
- Piercing of the gingival margin at three-quarters' root formation
- Root completion after several months in occlusion

- Basic knowledge of the embryology, histology, and morphogenesis of tooth formation, eruption, and exfoliation and careful clinical and radiographic evaluation of these changes can help the clinician to identify factors that may cause eruption problems, such as bone barriers, fibrotic tissue, supernumerary teeth, odontomas, remaining primary roots, ankylosis, and cyst formation or other pathologic lesions.
- Delayed eruption is a problem that ranges from delayed eruption to complete failure of eruption, including tooth ankylosis, PFE, or secondary failure of

eruption. It can be a simple inability to erupt due to space loss or it can be a failure of eruption. The morphology and etiologic causes can differentiate the kind of delayed or failure of eruption. PFE is an abnormality that affects all teeth distal to the most mesially involved tooth, while mechanical failure of eruption affects only the involved tooth or teeth. Recent studies show that the former is an inherited, autosomal-dominant mode of nonsyndromic primary failure of eruption.

- Retarded eruption is a different form of delayed eruption, commonly observed in the second premolar area. This kind of anomaly is associated with late development of tooth germs. Radiographic evaluation of retarded tooth formation can even be misleading because of its similarity to congenital absence of premolars.
- An abnormal sequence of eruption can have direct effects on the development of occlusion. The sequence of shedding and subsequent eruption of the maxillary and mandibular posterior teeth and the way that the leeway space is utilized have important implications for molar occlusion. Likewise, the normal sequence of eruption of mandibular and maxillary canines relative to the premolars plays an important role in the anterior part of occlusion. In all types of eruption sequences, there is usually a symmetric pattern between the left and right sides of the arch, although there is some time variation among different individuals. More than 6 months' difference between the left and right sides of the arch can be an important sign of eruption problems.
- *Ectopic eruption* is tooth eruption that is out of normal position. Ectopic eruption may lead to various complications, including impaction, loss of bone, formation of dentigerous cysts, and resorption of adjacent teeth. One common form is ectopic eruption of the permanent first molar during the early mixed dentition. A mesial angulation of the permanent first molar results in a cessation of its eruption and causes atypical resorption of the primary second molar.
- *Tooth transposition* is the positional interchange of two adjacent teeth. The teeth most frequently involved in the maxilla are canines, premolars, and lateral incisors, and the most commonly transposed teeth in the mandible are lateral incisors. Early detection of transposed teeth at an early stage of development and recognition of the etiologic factors makes it possible to intervene and change the abnormal eruptive path of the transposed teeth through proper guidance of eruption.
- Impacted teeth are a commonly found anomaly. After mandibular third

molars, maxillary canines are the most commonly impacted teeth. Palatal canine impaction and buccal canine impaction are not exactly the same in etiology, morphogenesis, or management. Several etiologic factors that can cause canine impaction require early detection and intervention. These include space deficiency, trauma, prolonged retention of the primary canine, premature loss of the primary canine, abnormal position of the tooth bud, localized obstacles such as cysts and odontoma, supernumerary teeth, abnormal sequence of eruption, missing lateral incisors, and ankylosis of the permanent canine.

- Ankylosis is a specific abnormality of tooth eruption and development caused by the fusion of some part of cementum with the alveolar bone. Primary tooth ankylosis is a progressive anomaly of tooth eruption that can occur before, during, or after tooth eruption and usually has a profound effect on successor teeth, alveolar bone growth, and occlusion.
- Careful clinical examination and radiographic monitoring of changes during the transitional dentition can help to detect incipient problems and ensure that proper intervention is initiated.
- The following are important clinical signs that indicate the possibility of eruption problems:

- Overretained primary teeth
- Early exfoliation of primary teeth
- Asymmetric permanent eruption (left and right sides of the arch)
- Asymmetric root development
- Abnormal sequence of eruption
- Failure of eruption or delayed eruption
- Sustained partial eruption
- Displacement of an erupted permanent tooth (lateral incisor tilting)
- Absence of a crown bulge

References

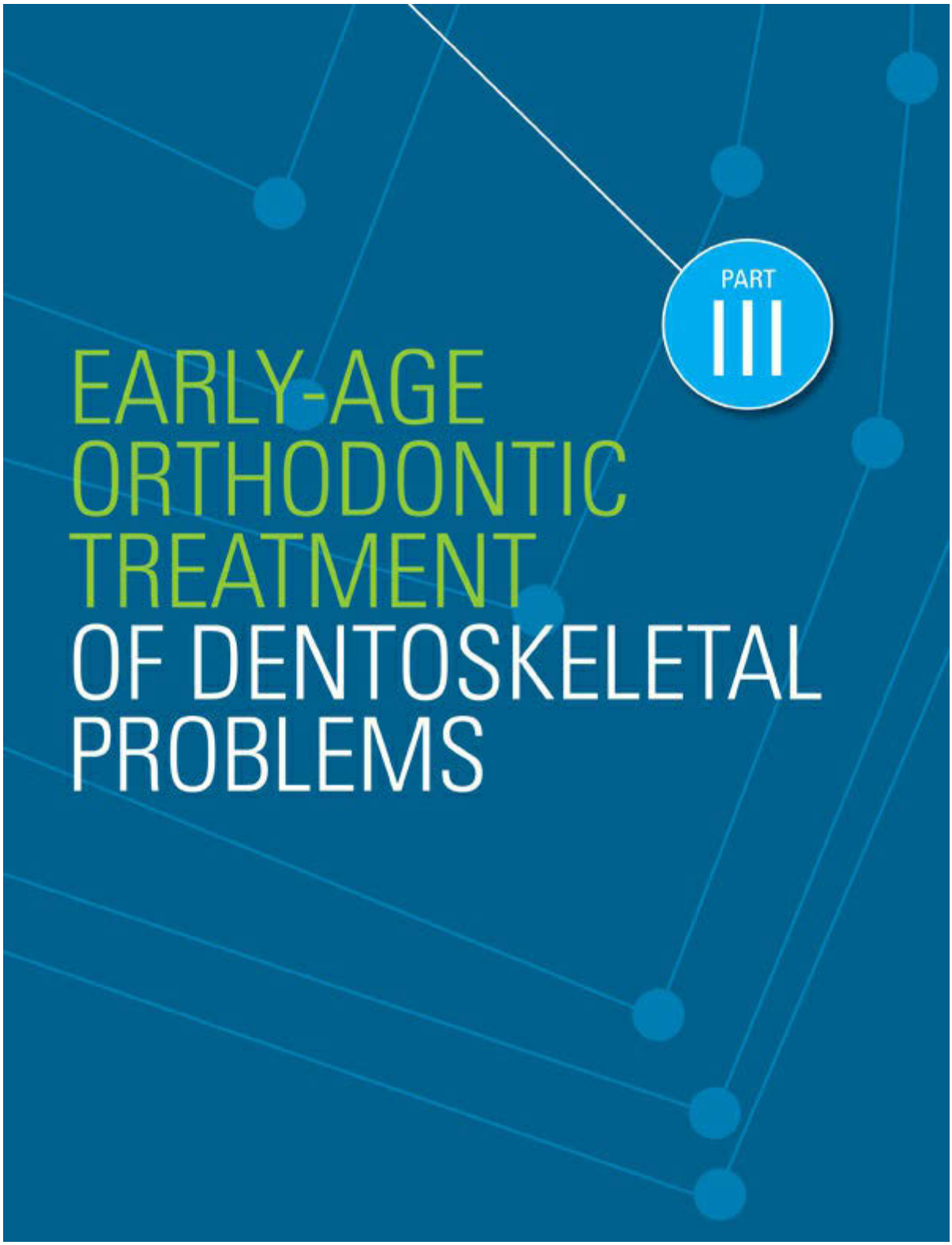
1. Massler M, Schour I, Poncher HG. Developmental pattern of the child as reflected in the calcification pattern of the teeth. Am J Dis Child 1941;62:33–67.
2. Bosshardt DD, Schroeder HE. Cementogenesis reviewed: A comparison between human premolars and rodent molars. Anat Rec 1996;245:267–292.

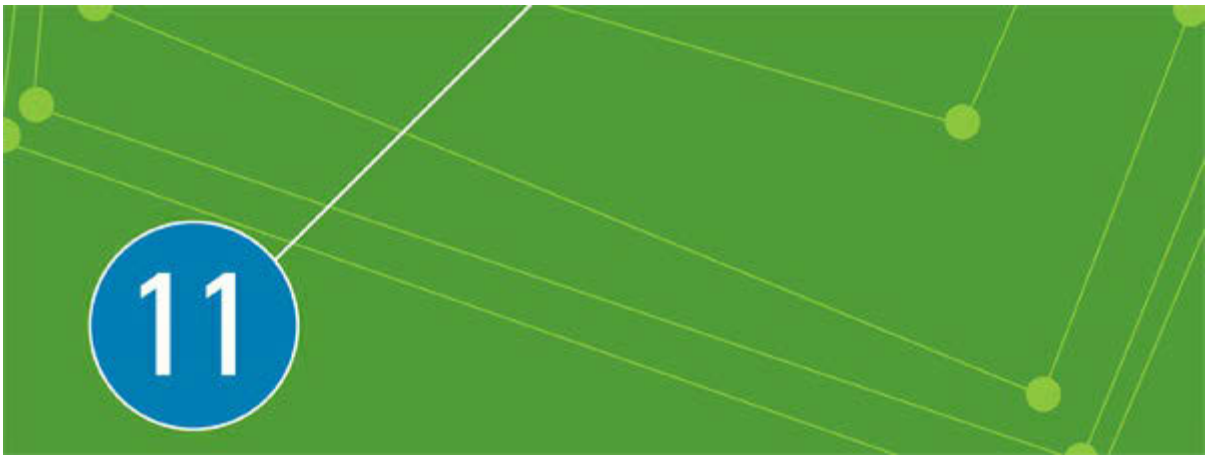
3. Shumaker DB. A comparison of chronologic age and physiologic age as predictors of tooth eruption. Am J Orthod 1974;66:50–57.
4. Marks SC Jr, Cahill DR. Experimental study in the dog of the non-active role of the tooth in the eruptive process. Arch Oral Biol 1984;29:311–322.
5. Berkovitz BK, Thomas NR. Unimpeded eruption in the root-resected lower incisor of the rat with a preliminary note on root transection. Arch Oral Biol 1969;14:771–780.
6. Carl W, Wood R. Effects of radiation on the developing dentition and supporting bone. J Am Dent Assoc 1980;101:646–648.
7. Van Hassell HJ, McMinn RG. Pressure differential favouring tooth eruption in the dog. Arch Oral Biol 1972;17:183– 190.
8. Chiba M, Ohshima S. Effects of colchicine and hydrocortisone on unimpeded eruption rates of root-resected mandibular incisors of rats. Arch Oral Biol 1985;30:147–153.
9. Marks SC Jr. The basic and applied biology of tooth eruption. Connect Tissue Res 1995;32(1–4):149–157.
10. Wise GE, Yao S, Henk WG. Bone formation as a potential motive force of tooth eruption in the rat molar. Clin Anat 2007;20:632–639.
11. Cahill DR, Marks SC Jr. Tooth eruption: Evidence for the central role of the dental follicle. J Oral Pathol 1980;9:189– 200.
12. Ten Cate AR. Oral Histology: Development, Structure, and Function, ed 8. St Louis: Mosby-Year Book, 2012.
13. Ten Cate AR, Deporter DA, Freeman E. The role of fibroblasts in the remodeling of periodontal ligament during physiological tooth movement. Am J Orthod 1976;69:155– 168.
14. Berkovitz BK. How teeth erupt. Dent Update 1990;17:206– 210.
15. Brash JC.The growth of the alveolar bone and its relation to the movements of the teeth, including eruption. 1. Int J Orthod Oral Surg Radiogr 1928;14:196–223.
16. Brash JC.The growth of the alveolar bone and its relation to the movements of the teeth, including eruption. 2. Int J Orthod Oral Surg Radiogr 1928;14:283–293.
17. Brash JC.The growth of the alveolar bone and its relation to the movements of the teeth, including eruption. 3. Int J Orthod Oral Surg Radiogr 1928;14:398–405.
18. Brash JC.The growth of the alveolar bone and its relation to the movements of the teeth, including eruption. 4. Int J Orthod Oral Surg Radiogr 1928;14:487–504.
19. Larson EK, Cahill DR, Gorski JP, Marks SC Jr. The effect of removing the true dental follicle on premolar eruption in the dog. Arch Oral Biol 1994;39:271–275.
20. Wise GE. Cellular and molecular basis of tooth eruption. Orthod Craniofac Res 2009;12:67–73.
21. Philbrick WM, Dreyer BE, Nakchbandi IA, Karaplis AC. Parathyroid hormone-related protein is required for tooth eruption. Proc Natl Acad Sci U S A 1998;95(20):11846– 11851.
22. Suri L, Gagari E, Vastardis H. Delayed tooth eruption: Pathogenesis, diagnosis, and treatment. A literature review. Am J Orthod Dentofacial Orthop 2004;126:432–445.
23. Wise GE, King GJ. Mechanisms of tooth eruption and orthodontic tooth movement. J Dent Res 2008;87:414–434.
24. Baume LJ, Becks H. The effect of thyroid hormone on dental and paradental structures. Parodontopathies 1952;6:89–109.
25. Tse Mdo C, Boaventura MC, Fernandes GD, Merzel J. The effects of cerebral hemidecortication on the eruption rate and uptake of [3H]-glycine by the periodontal ligament of the rat incisor. Arch Oral Biol 1988;33:605–611.
26. Kosowicz J, Rzymski K. Abnormalities of tooth development in pituitary dwarfism. Oral Surg Oral Med Oral Pathol 1977;44:853–863.
27. Barbería Leache E, Marañes Pallardo JP, Mourelle Martínez MR, Moreno González JP. Tooth eruption in children with growth deficit. J Int Assoc Dent Child 1988;19(2):29–35.
28. Stellzig-Eisenhauer A, Decker E, Meyer-Marcotty P, et al. Primary failure of eruption (PFE)—Clinical and molecular genetics analysis. J Orofac Orthop 2010;71:6–16.

29. Frazier-Bowers SA, Simmons D, Koehler K, Zhou J. Genetic analysis of familial non-syndromic primary failure of eruption. Orthod Craniofac Res 2009;12:74–81.
30. Gron AM. Prediction of tooth emergence. J Dent Res 1962;41:573–585.
31. Posen AL. The effect of premature loss of deciduous molars on premolar eruption. Angle Orthod 1965;35:249–252.
32. Hartsfield JK Jr. Premature exfoliation of teeth in childhood and adolescence. Adv Pediatr 1994;41:453–470.
33. Dean JA, Avery DR, McDonald RE. Dentistry for the Child and Adolescent, ed 9. St Louis: Mosby, 2010:111.
34. Proffit WR, Vig KW. Primary failure of eruption: A possible cause of posterior open-bite. Am J Orthod 1981;80:173– 190.
35. Rasmussen P, Kotsaki A. Inherited primary failure of eruption in the primary dentition: Report of five cases. ASDC J Dent Child 1997;64:43–47.
36. Frazier-Bowers SA, Puranik CP, Mahaney MC. The etiology of eruption disorders—Further evidence of a 'genetic paradigm'. Semin Orthod 2010;16:180–185.
37. Ahmad S, Bister D, Cobourne MT. The clinical features and etiological basis of primary eruption failure. Eur J Orthod 2006;28:535–540.
38. Proffit WR, Frazier-Bowers SA. Mechanism and control of tooth eruption: Overview and clinical implications. Orthod Craniofac Res 2009;12:59–66.
39. Mc Cafferty J, Al Awadi E, O'Connell AC. Case report: Management of severe posterior open bite due to primary failure of eruption. Eur Arch Paediatr Dent 2010;11:155–158.
40. Lygidakis NA, Bafis S, Vidaki E. Case report: Surgical luxation and elevation as treatment approach for secondary eruption failure of permanent molars. Eur Arch Paediatr Dent 2009;10(suppl 1):46–48.
41. Taguchi Y, Yano Y, Kobayashi H, Noda T. Retarded eruption of maxillary second premolars associated with late development of the germs. J Clin Pediatr Dent 2003;27:321–326.
42. Peterka M, Tvrdek M, Müllerová Z. Tooth eruption in patients with cleft lip and palate. Acta Chir Plast 1993;35:154– 158.
43. Peterka M, Peterková R, Likovský Z. Timing of exchange of the maxillary deciduous and permanent teeth in boys with three types of orofacial clefts. Cleft Palate Craniofac J 1996;33:318–323.
44. Moorrees CF, Gron AM, Lebret LM, Yen PK, Fröhlich FJ. Growth studies of the dentition: A review. Am J Orthod 1969;55:600–616.
45. Lo R, Moyers RE. Studies in the etiology and prevention of malocclusion. 1. The sequence of eruption of the permanent dentition. Am J Orthod 1953;39:460–467.
46. Garn SM, Smith BH. Developmental communalities in tooth emergence timing. J Dent Res 1980;59:1178.
47. Sampson WJ, Richards LC. Prediction of mandibular incisor and canine crowding changes in the mixed dentition. Am J Orthod 1985;88:47–63.
48. Weinberger SJ. Correction of bilateral ectopic eruption of first permanent molars using a fixed appliance. Pediatr Dent 1992;14:382–383.
49. Bjerklin K. Ectopic eruption of the maxillary first permanent molar. An epidemiological, familial, etiological and longitudinal clinical study. Swed Dent J Supp l994;100:1–66.
50. Harris CA. A Dictionary of Dental Science: Biography, Bibliography and Medical Terminology. Philadelphia: Lindsay & Blakiston, 1849.
51. ShapiraY, Kuftinec MM. Tooth transpositions—A review of the literature and treatment considerations. Angle Orthod 1989;59:271–276.
52. Huber KL, Suri L, Taneja P. Eruption disturbances of the maxillary incisors: A literature review. J Clin Pediatr Dent 2008;32:221–230.
53. Ely NJ, Sherriff M, Cobourne MT. Dental transposition as a disorder of genetic origin. Eur J Orthod 2006;28:145–151.
54. Shapira Y, Kuftinec MM. Maxillary tooth transpositions: Characteristic features and accompanying dental anomalies. Am J Orthod Dentofacial Orthop 2001;119:127–134.
55. Shapira J, Chaushu S, Becker A. Prevalence of tooth transposition, third molar agenesis, and maxillary canine

impaction in individuals with Down syndrome. Angle Orthod 2000;70:290–296.
56. Papadopoulos MA, Chatzoudi M, Kaklamanos EG. Prevalence of tooth transposition. A meta-analysis. Angle Orthod 2010;80:275–285.
57. Allen WA. Bilateral transposition of teeth in two brothers. Br Dent J 1967;123:439–440.
58. Payne GS. Bilateral transposition of maxillary canine and premolars. Report of two cases. Am J Orthod 1969;56:45– 52.
59. Broadbent BH. Ontogenic development of occlusion. Angle Orthod 1941;11:223–241.
60. Bishara SE. Impacted maxillary canines: A review. Am J Orthod Dentofacial Orthop 1992;101:159–171.
61. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod 1994;64:249–256.
62. Al-Nimri K, Gharaibeh T. Space conditions and dental and occlusal features in patients with palatally impacted maxillary canines: An etiological study. Eur J Orthod 2005;27:461–465.
63. Jacoby H. The etiology of maxillary canine impactions. Am J Orthod 1983;84:125–132.
64. Fournier A, Turcotte JY, Bernard C. Orthodontic considerations in the treatment of maxillary impacted canines. Am J Orthod 1982;81:236–239.
65. Warford JH Jr, Grandhi RK, Tira DE. Prediction of maxillary canine impaction using sectors and angular measurement. Am J Orthod Dentofacial Orthop 2003;124:651–655.
66. Mossey PA, Campbell HM, Luffingham JK. The palatal canine and the adjacent lateral incisor: A study of a west of Scotland population. Br J Orthod 1994;21:169–174.
67. Becker A. In defense of the guidance theory of palatal canine displacement. Angle Orthod 1995;65:95–98.
68. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. Eur J Orthod 1990;12:135–139.
69. Baccetti T. A controlled study of associated dental anomalies. Angle Orthod 1998;66:267–274.
70. Peck S, Peck L, Kataja M. Concomitant occurrence of canine malposition and tooth agenesis: Evidence of orofacial genetic fields. Am J Orthod Dentofacial Orthop 2002;122:657–660.
71. Pirinen S, Arte S, Apajalahti S. Palatal displacement of canine is genetic and related to congenital absence of teeth. J Dent Res 1996;75:1742–1746.
72. Ericson S, Kurol J. Resorption of maxillary lateral incisors caused by ectopic eruption of the canines. A clinical and radiographic analysis of predisposing factors. Am J Orthod Dentofacial Orthop 1988;94:503–513.
73. Walker L, Enciso R, Mah J. Three-dimensional localization of maxillary canines with cone-beam computed tomography. Am J Orthod Dentofacial Orthop 2005;128:418–423.
74. Williams BH. Diagnosis and prevention of maxillary cuspid impaction. Angle Orthod 1981;51:30–40.
75. Alqerban A, Jacobs R, Fieuws S, Willems G. Comparison of two cone beam computed tomographic systems versus panoramic imaging for localization of impacted maxillary canines and detection of root resorption. Eur J Orthod 2011;33:93–102.
76. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. Eur J Orthod 1988;10:283–295.
77. Power SM, Short MB. An investigation into the response of palatally displaced canines to the removal of deciduous canines and an assessment of factors contributing to favourable eruption. Br J Orthod 1993;20:215–223.
78. Dewel BF. The upper cuspid: Its development and impaction. Angle Orthod 1949;19:79–90.
79. Newcomb MR. Recognition and interception of aberrant canine eruption. Angle Orthod 1959;21:161–168.
80. Shapira Y, Kuftinec MM. Early diagnosis and interception of potential maxillary canine impaction. J Am Dent Assoc 1998;129:1450–1454.
81. Olive RJ. Factors influencing the non-surgical eruption of palatally impacted canines. Aust Orthod J 2005;21:95–101.
82. Jacobs SG. The impacted maxillary canine. Further observations on aetiology, radiographic localization, prevention/interception of impaction, and when to suspect impaction. Aust Dent J 1996;41:310–316.
83. Frank CA, Long M. Periodontal concerns associated with the orthodontic treatment of impacted teeth. Am J Orthod Dentofacial Orthop 2002;121:639–649.

84. Kokich VG. Surgical and orthodontic management of impacted maxillary canines. Am J Orthod Dentofacial Or-thop 1980;126:278–283.
85. Grover PS, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. Oral Surg Oral Med Oral Pathol 1985;59:420–425.
86. Röhrer A. Displaced and impacted canines: A radiographic research. Int J Orthod Oral Surg Radiogr 1929;15:1003– 1020.
87. Da Silva Assunção LR, Ferelle A, Iwakura ML, Cunha RF. Effects on permanent teeth after luxation injuries to the primary predecessors: A study in children assisted at an emergency service. Dent Traumatol 2009;25:165–170.
88. Anthonappa RP, Ongtengco KL, King NM. A report of an impacted primary maxillary central incisor tooth [epub ahead of print 10 January 2013]. Dent Traumatol doi: 10.1111/edt.12031.
89. Pavoni C, Mucedero M, Laganà G, Paoloni V, Cozza P. Impacted maxillary incisors: Diagnosis and predictive measurements. Ann Stomatol (Roma) 2012;3:100–105.
90. Biagi R, Butti AC, Salvato A. Premature loss of maxillary primary incisor and delayed eruption of its successor: Report of a case. Eur J Paediatr Dent 2011;12:194–197.
91. Chaushu S, Zilberman Y, Becker A. Maxillary incisor impaction and its relationship to canine displacement. Am J Orthod Dentofacial Orthop 2003;124:144–510.
92. Kurol J. Infra-occlusion of primary molars: An epidemiologic and familial study. Community Dent Oral Epidemiol 1981;9:94–102.
93. Biederman W. The problem of the ankylosed tooth. Dent Clin North Am 1968;Jul:409–424.
94. Krakowiak FJ. Ankylosed primary molars. ASDC J Dent Child 1978;45:288–292.
95. Via WF Jr. Submerged deciduous molars: Familial tendencies. J Am Dent Assoc 1964;69:128–129.
96. Brearley LJ, McKibben DH Jr. Ankylosis of primary molar teeth. 1. Prevalence and characteristics. ASDC J Dent Child 1973;40:54–63.
97. Garib DG, Peck S, Gomes SC. Increased occurrence of dental anomalies, associated with second premolar agenesis. Angle Orthod 2009;79:436–441.
98. Kurol J,Thilander B. Infraocclusion of primary molars with aplasia of the permanent successor: A longitudinal study. Angle Orthod 1984;54:283–294.
99. Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670–684.
100. Mancini G, Francini E, Vichi M, Tollaro I, Romagnoli P. Primary tooth ankylosis. Report of a case with histological analysis. J Dent Child 1965;62:215–219.
101. Haselden K, Hobkirk JA, Goodman JR, Jones SP, Hemmings KW. Root resorption in retained deciduous canine and molar teeth without permanent successors in patients with severe hypodontia. Int J Paediatr Dent 2001;11:171– 178.
102. Messer LB, Cline JT. Ankylosed primary molars: Results and treatment recommendations from an eight-year longitudinal study. Pediatr Dent 1980;2:37–47.
103. Kurol J, Olson L. Ankylosis of primary molars—A future periodontal threat to first permanent molars? Eur J Orthod 1991;13:404–409.





Management of Sagittal Problems (Class II and Class III)

A major and important objective of early treatment during the primary and mixed dentitions is enhancing the normal growth and development of dental occlusion. Recent studies indicate that genetic mechanisms have more influence on the morphogenesis of craniofacial structure during embryonic life, while environmental factors influence the developing occlusion, especially during the early postnatal period.[1]

The first step in development of the vertical dimension of occlusion is established when the primary first molars reach occlusal contact. When the primary second molars reach occlusion, their distal surfaces (terminal plane) play an important role in the sagittal dimension of final occlusion.

It is also known that establishment of the occlusion is under the control of genetic and environmental influences and influenced by the interaction of growth and development of basal bone and other craniofacial structures. All of these structures are also under the influence of neuromuscular and soft tissue structures and their functional matrix. Disturbance of any of these components or the occurrence of disharmony or dysfunction during the development of occlusion can result in abnormal occlusion.

Many incipient malocclusions that are under the influence of environmental

factors during the primary or mixed dentition could be preventable; early recognition of these anomalies and interception can eliminate or reduce the severity of future problems. There are special orthodontic problems that require early intervention, even in very young patients during the primary or early mixed dentition; these include Class II malocclusion, anterior dental crossbite, and dentoskeletal Class III malocclusion, which will be discussed in this chapter, as well as posterior crossbite, especially if it is associated with a lateral mandibular shift (see chapter 12), open bite associated with persistent oral habits or other oral dysfunction (see chapter 13), space management (see chapter 4), severe incisor protrusion (see chapter 5), and craniofacial anomalies such as clefts or hemifacial microsomia (see chapter 7).

Understanding the mechanisms of early detection and orthodontic intervention requires a thorough understanding of the development of the dentition (see chapters 1 and 2). The major goal of early intervention is to eliminate or reduce the damage from etiologic factors that cause trauma to the occlusion or interfere with the normal growth and development of occlusion, thereby providing an environment that promotes normal jaw growth and an optimal eruption pattern during the transitional dentition.

Proper intervention at early ages both corrects the present abnormalities and eliminates or minimizes more severe problems that can occur later. Depending on the type of problem and the dental and skeletal age of the patient during treatment, some patients may need a second or third phase of treatment. However, the final phase is usually much easier and faster after early intervention. Some patients may not need any further treatment.

The timing of early treatment is controversial; some practitioners advocate for treatment during the primary dentition, and some orthodontists prefer to begin treatment during the mixed dentition. There is also controversy about whether the early, middle, or late mixed dentition is preferable. The American Association of Orthodontists[2] recommends that the initial orthodontic evaluation occur at the first sign of orthodontic problems or no later than the age of 7 years.

Examination of a child's dentition during the primary dentition does not always mean treatment; careful examination can detect and anticipate developing problems at this early age and clarify the possibility of early intervention. The orthodontic examination, especially during the primary and early mixed dentitions, is more than merely an examination of dental occlusion. The assessment should include diagnosis of facial or skeletal problems that are initiating and developing in a growing child. Problems such as upper airway obstruction (mouth breathing), temporomandibular

dysfunction, and abnormal myofunctional habits can have adverse effects on dentoskeletal structures. If problems are recognized early and preventive measures are instituted, many facial and dental problems may be minimized or averted.

Gugino and Dus[3] stated that the human face is anatomically and functionally one of the most complex regions of the human body. A sound understanding of the physiology of the stomatognathic system is an important requirement for clinicians who wish to provide their patients with the best orthodontic and orthopedic treatment results. Therefore, clinicians must understand the basic process of occlusal development, always be aware for cautious detection, and decide on a case-by-case basis when to provide orthodontic intervention.

Early treatments follow two major strategies:

1. Removal of etiologic factors and restoration of a normal environment to facilitate normal growth and development of the dentition
2. Guidance of tooth eruption and the growth pattern to a normal state to correct or reduce the severity of dentoskeletal malrelationships

This chapter discusses common sagittal abnormalities that initiate and develop during the primary or early mixed dentition, are not self-corrected, and in some situations worsen in later stages of the dentition:

- Class II malocclusion (Class II division 1 and Class II division 2)
- Anterior dental crossbite
- Dental and dentoskeletal Class III malocclusion

Class II Malocclusion

Two large-scale surveys carried out by the United States by the Division of Health Statistics of the US Public Health Service covered 6- to 11-year-old children and youths aged 12 to 17 years. The surveys indicated that Class II malocclusion accounts for about one-third of the total malocclusions of the US population,[4] with variable combinations of dental and skeletal factors contributing to the disharmony. Class II malocclusion usually causes esthetic and functional problems of varying severity, depending on the amount of anteroposterior discrepancy and its interaction with adjacent soft tissues.[5]

Long-term studies from the primary to the permanent dentition have shown that

Class II abnormalities are not self-correcting and in some cases may worsen with age.[6,7] Not all Class II malocclusions are alike; numerous types of skeletal and dental Class II malocclusions might be observed under Class II molar relationships.

A review of older literature indicates that Class II discrepancy has mainly been perceived as a sagittal problem. The characteristics of these anomalies have been classified under four sets of anteroposterior criteria: *(1)* maxillary skeletal position, *(2)* maxillary dentoalveolar position, *(3)* mandibular skeletal position, and *(4)* mandibular dentoalveolar position. The vertical and transverse dimensions were not considered to be related to Class II discrepancy; however, later research showed that either of these dimensions may be involved.

Schudy[8] showed that variations in facial height may conceal or intensify the clinical appearance of Class II malocclusion. An increased mandibular vertical dimension in association with Class II malocclusion usually rotates the mandible downward and backward, which manifests as a more retruded mandible and a more convex profile. Any mistake in treatment would worsen the situation. A decreased vertical dimension causes the mandible to rotate upward and forward, manifesting as a prominent chin point.

The transverse dimension also must not be overlooked in evaluation of Class II malocclusions. Most Class II patients with a narrow maxillary arch may show a normal relationship of buccal segments in centric occlusion. Tollaro et al[9] investigated posterior transverse interarch discrepancy (PTID), which was measured as the difference between the maxillary and mandibular intermolar width, in two Class II division 1 groups: one with PTID and one without PTID. In the Class II group with PTID, mandibular retrusion was associated with a posteriorly displaced mandible of normal size (functional mandibular retrusion). In the Class II group without PTID, mandibular retrusion was caused by a micrognathic mandible (anatomical mandibular retrusion).

Sometimes an underlying transverse discrepancy of 3 to 5 mm exists in the dental arch. This maxillary constriction can be unmasked clinically by having the patient bring the mandible forward to a Class I molar relationship.

Early intervention in transverse problems and proper expansion at earlier stages will facilitate forward growth of the mandible. Although Class II malocclusion is perceived as a sagittal problem, the vertical and transverse dimensions of each patient must also be considered in treatment planning.

Morphologic characteristics

The molar or canine relationships in patients with Class II malocclusion constitute only one part of a broader and extended syndrome and cannot clearly define treatment planning. Different morphologic characteristics of Class II malocclusion can be caused by malrelationships of the basal bone, malrelationships of the dentition, dentoalveolar or dentoskeletal malrelationships, or some combination of these factors (Box 11-1). In addition, discrepancies in other bony structures, such as different growth or different angulation of the anterior and posterior cranial bases, can have destructive effects on jaw harmony and occlusion (Box 11-2). Figure 11-1 shows different variations of skeletal Class II malocclusion that can develop, depending on the morphologic characteristics of basal bone.

Box 11-1	Morphologic characteristics of the jaws in patients with Class II malocclusion
• Maxillary basal protrusion • Maxillary dentoalveolar protrusion • Maxillary counterclockwise rotation • Overdevelopment of the maxillary length relative to the mandible • Vertical overgrowth of the maxilla • Mandibular basal retrognathism	• Mandibular dentoalveolar retrognathism • Underdevelopment of the mandibular length relative to the maxilla • Overgrowth of the ramus relative to the mandibular body • Underdevelopment of the ramus relative to the mandibular body

Box 11-2	Morphologic characteristics of other bony structures in patients with Class II malocclusion
• Increased anterior cranial base length, causing anterior positioning of the maxilla • Increased posterior cranial base length, causing posterior positioning of the mandible	• Increased saddle angle (nasion-sella-articulare, N-S-Ar), causing posterior positioning of the condyle • Increased articular angle (sella-articularegonion, S-Ar-Go), causing posterior positioning of the mandible

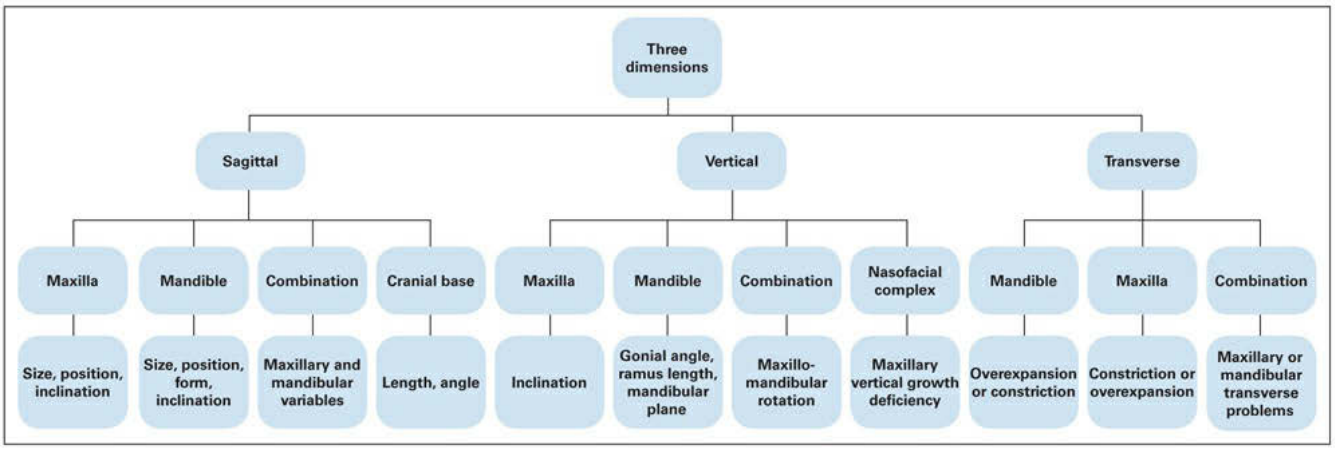


Fig 11-1 Possible variations of Class II malocclusions based on skeletal components.

Diagnostic procedures

Class II malocclusion is a multifactorial entity caused by hereditary factors or abnormally acquired environmental factors that produce aberrations in form and function during occlusal development. Therefore, thorough diagnostic evaluations are necessary for correct identification of etiologic factors and affected anatomical structures as well as proper treatment planning for correction of Class II malocclusion.

The dental occlusion and dental arch are not an isolated entity; rather, they are a part of an extensive system comprising the teeth, basal bone, and other skeletal structures of the face and cranium that interact with the neuromuscular system and surrounding soft tissues. All kinds of dentoskeletal malocclusions, including Class II types, are associated, to one degree or another, with the relationships of the following six dentoskeletal components:

1. Cranium and cranial base
2. Skeletal maxilla
3. Nasomaxillary complex
4. Maxillary teeth and alveolar process
5. Skeletal mandible
6. Mandibular teeth and alveolar process

These are independent functional units, but their interrelationships in three dimensions can produce several variations in the morphology of all dentoskeletal malocclusions, including Class II forms.

Cephalometric analysis

Proper treatment planning for dentoskeletal malocclusions cannot be established without a thorough evaluation of the interrelationships of these compartments relative to the dentition and dental occlusion. Since the introduction of the cephalometric technique by Broadbent[10] in 1931, a variety of analyses has been presented by different investigators.

One of the goals of cephalometric analysis is to establish the relationships of these functional components in both the anteroposterior and vertical planes of space in order to differentiate the type of malocclusion. Research reports that have evaluated the morphology of the maxillomandibular relationship in patients with Class II malocclusion have reached differing conclusions. Some reports have indicated a high percentage of maxillary base protrusion,[11] and some reports have indicated a higher percentage of mandibular base retrusion.[12] Variations also exist in dentoalveolar types. Although Class II is perceived as a sagittal problem, the vertical and transverse dimensions of each patient must also be considered.

It is not the intention of this book to discuss cephalometric analysis; there are many valuable sources for this information. However, the reader should note the importance of these tools for differential diagnosis in dentoskeletal malocclusion, including Class II malocclusion, especially in early treatment approaches aiming for growth modification.

In cephalometric evaluation of dentoskeletal malocclusions and differentiation of various components of each malrelationship, it is wise not to rely on any specific angle or linear measurement; no single measurement may be reliable all the time, and other data are available. For example, because of variability in length and inclination of sella-nasion in some patients, the sella-nasion–point A (SNA) and sella-nasion–point B (SNB) angles cannot always be accurate in anteroposterior evaluation.

In all sagittal evaluations of Class II dentoskeletal malrelationships, besides the SNA and SNB measurements, the Landes angle (nasion–point A to Frankfort plane) provides a high degree of reliability for the maxilla. The Wit analysis (perpendicular lines drawn from point A and from point B to the occlusal plane) can also be used to compare the anteroposterior relationships of the maxilla and the mandible.

Growth prediction

Another important aspect of cephalometric analysis is prediction of the growth pattern, which is critical in treatment planning, especially for growing patients. Many efforts have been made to develop methods for growth prediction from cephalometric radiographs. Successful prediction is achieved when the amount and the direction of growth are predicted before early-age treatment.

Orthopedic interventions for growing children should be preceded by careful cephalometric evaluation of all components of dentoskeletal structures, such as the cranium and cranial base, the skeletal maxilla and nasomaxillary complex, the skeletal mandible, the maxillary teeth and alveolar process, the mandibular teeth and alveolar process, and especially the potential direction of jaw growth for the child. Facial divergence and convergence, Frankfort plane–mandibular incisor angle (FMA), gonial angle, y-axis, anterior and posterior facial heights, and posterior and anterior cranial base lengths and angulations are examples of measurements that can indicate children's potential growth pattern.

Advantages of early treatment

Early orthodontic treatment for Class II malocclusion has the following benefits:

- It affords the greatest utilization of skeletal growth.
- Skeletal disharmony can be controlled in three dimensions (sagittal, vertical, and transverse).
- Observation and guidance of eruption during the transitional dentition are promoted.
- Disarticulation of the posterior segment during tooth eruption facilitates normal interdigitation.
- Leeway spaces within the dental arches are preserved.
- The need for tooth extraction is reduced.
- Better skeletal harmony is achieved.
- Results are more stable.
- Early treatment of unesthetic malocclusion can have an important impact on the psychosocial development of the patient.
- Fractures caused by trauma to protruded incisors are prevented.
- Patients exhibit better compliance at earlier ages.

The major goal of early treatment is to correct or reduce the adverse effects of

dentoskeletal and muscular imbalance by improving the environment to encourage optimal occlusal development.

Class II Treatment Options

A Class II malocclusion can have various anatomical, morphologic, and functional characteristics. Treatment strategies differ depending on the special characteristics of this anomaly. Class II treatment also varies according to the skeletal age of the patient. Growth modification strategy is entirely different from treatment tactics applicable after completion of the growth spurt. Depending on the type of Class II malocclusion present and the age of the patient, four treatment options are available for Class II correction:

1. Camouflage treatment
2. Orthognathic surgery
3. Growth modification and occlusal guidance
4. A combination of the above

Camouflage treatment

Camouflage treatment is used in the permanent dentition, usually after the growth spurt, to obtain optimum results within the physiologic limits of each patient. With camouflage treatment the goal is to produce acceptable dental relationships and disguise unacceptable skeletal relationships through tooth movement, which may or may not require serial extractions. This type of treatment is indicated in adolescent and adult patients with mild to moderate skeletal problems.

Orthognathic surgery

Orthodontic treatment combined with surgical procedures is applied in adult patients to correct dental malrelation-ships and skeletal disharmonies. These types of treatments are usually implemented in individuals with severe dentoskeletal malrelationships caused by heredity, skeletal disharmony, or congenital skeletal malformation.

Growth modification and occlusal guidance

Growth modification and occlusal guidance treatments are interceptive treatments that are applied in growing children during the primary or mixed dentition to eliminate or minimize the dentoalveolar and skeletal disharmonies that can interfere with the normal growth and development of occlusion. The main objective of this type of intervention is to prepare an environment that will enhance the positive effects of growth potential for improving occlusal development.

Early versus late orthodontic treatment, especially early treatment for Class II problems, is one of the greatest controversies in orthodontics. Randomized clinical trials have been specifically designed to address these important issues in Class II treatment. Keeling et al,[13] Tulloch et al,[14] and Ghafari et al[15] concluded that early treatment followed by later comprehensive treatment of Class II malocclusion associated with moderate to severe deformity does not produce major differences in jaw relationships or dental occlusion compared with later one-stage treatment.

On the other hand, many other experienced researchers and clinicians, including Gugino and Dus,[3] Ricketts,[16–18] Subtelny,[19] Bench et al,[20] Graber,[21] and McNamara,[22] insist that there are many disadvantages to waiting for eruption of the permanent dentition and second molars. In addition, many animal studies, carried out by pioneers such as Harvold et al,[23] Woodside et al,[24] and McNamara[25] have clearly shown the histomorphologic changes induced in animal jaw growth by orthopedic forces.

Most dentoskeletal anomalies develop during the primary and mixed dentitions, and, according to Carlson,[26] the midface and the mandible complete only 50% of their total growth by age 8 to 10 years; thus, a considerable amount of midface and mandibular growth occurs during the transitional dentition, which provides a good opportunity to intervene.

Long-term studies from the primary to the permanent dentition have also shown that Class II abnormalities are not self-correcting and in some cases may even worsen.[6,7] Why should dental practitioners allow an unfavorable dental, skeletal, or soft tissue relationship to remain for a number of years if it can be corrected completely or at least partially at an earlier age with minimal appliance therapy and treatment effort, by taking advantage of the child's growth potential?

Deviations in skeletal growth patterns accompanied by a disturbed functional matrix environment, such as mouth breathing, atypical swallowing, or other abnormal behaviors that can disturb jaw growth, require early intervention to

eliminate or modify the condition and give orthodontists the opportunity to guide dentoalveolar development.

History of growth modification tactics

The mandible is the only freely functioning bone in the body not connected to another bone. It is tied to its contiguous structures by 13 attached muscles. The mandibular position in three planes of space (sagittal, vertical, and transverse) plays an important role in harmony of the dental occlusion. These specifications of the mandible have drawn orthodontists for many years to the possibility of influencing the mandibular position by altering the structure of the temporomandibular joint.

In 1880, Kingsley[27] introduced the concept of *jumping the bite* for correction of mandibular retrusion. He designed a vulcanite palatal plate consisting of an anterior inclined surface to guide the mandible to a forward position. This idea influenced the development of functional jaw orthopedic treatment. Graber et al[28] provided a brief history of different functional devices developed in the past century. Hotz, by modifying the Kingsley device, introduced the Vorbissplatte for treatment of severe overbite, mandibular retrusion, and retroclined incisors by muscle hyperactivity. Pierre Robin, a French physician, introduced the monobloc in 1902 as a passive positioning device for his patients to prevent glossoptosis (blocking of the airway by the tongue).

Viggo Anderson was familiar with the appliances of Kingsley and Pierre Robin and, in 1908, without thinking of "guiding growth," applied a monobloc as a retainer for his daughter to eliminate adverse effects of abnormal function. He found that use of the appliance resulted in sagittal correction and profile improvement. Later, Anderson, in association with Haupl, named the appliance the *activator* because of its ability to activate the muscle forces.

In 1927, Emil Herbst introduced the fixed functional appliance (Herbst appliance). Later several modifications of the activator were introduced by different investigators, such as the Bionator by Balters in 1965, the functional regulator inducing muscle stretch by Fränkel[29] in 1966, functional headgear (combined headgear and Bionator) by Stockli[30] in 1973, fixed Herbst fully banded appliances by McNamara,[22] the twin block (two-piece functional) appliance by Clark[31] in 1977, and the magnetic activator device by Darendeliler and Joho.[32]

Several methods of Class II treatment that do not rely on significant patient compliance have become popular during the last decade, including several versions

of the Herbst appliance and the Pendulum or Pendex molar distalization appliance. Theoretically, however, these general approaches have opposite treatment effects, one presumably enhancing mandibular growth and the other moving the maxillary teeth posteriorly. In 2007, Seifi et al[33] introduced a two-piece functional appliance for Class II deep bite correction with a simple design, mandibular incisor capping, a palatal plate for maintaining the mandible in the protruded position, and without occlusal coverage so that no acrylic adjustment is necessary.

Functional appliances

Generally, functional appliances are removable devices designed to alter the neuromuscular environment of the orofacial region and to improve occlusal development and/or the craniofacial skeletal growth pattern through the following mechanisms:

- Utilization of muscle force to achieve dentoskeletal changes
- Disarticulation of the teeth to facilitate changes of inter-digitation
- Alteration in the eruptive paths of teeth
- Promotion of mandibular protraction to enhance forward mandibular growth
- Utilization of different pads to change the equilibrium of the perioral muscles

Functional therapy has several goals:

- Creation of a more favorable environment for occlusal development
- Enhancement of the normal growth of the basal and craniofacial skeletons
- Promotion of a normal growth direction of the craniofacial skeleton
- Selective inhibition or control of unfavorable growth
- Guidance of erupting teeth into more favorable positions

Extraoral traction

The use of extraoral devices to achieve growth modification in patients with Class II malocclusion goes back to the use of extraoral traction in the treatment of maxillary protrusion, first employed by Oppenheim[34–36] in 1936. In 1947, Kloehn[37] recommended headgear treatment in growing children to guide alveolar growth and eruption of the teeth, to reduce treatment time, and to produce a more balanced

dentition and face. Since then, other investigators have reported on the effects of headgear use in early treatment of Class II malocclusions and recommended the use of headgear as one of the most reliable methods for management of maxillary protrusion.[16–18,37,38]

Kopecky and Fishman[39] established that early orthodontic treatment and growth modification are best achieved before and during the growth spurt. Baccetti et al,[40] in a cephalometric study, evaluated the role of timing in relation to skeletal maturity on the outcomes of comprehensive, nonextraction Class II therapy. They concluded that treatment of Class II malocclusion before or during the pubertal growth spurt induces significant favorable skeletal changes. They also stated that use of headgear during the prepubertal stage restricts maxillary advancement and enhances mandibular growth. The only significant changes in patients treated after the pubertal growth spurt were dentoalveolar changes.

The effects of headgear alone or headgear combined with other devices on the dentition or jaws have also been reported by many investigators. These reports reveal several changes, such as molar relationships, SNA changes and downward tipping of the palatal plane, opening or closing of the bite, maxillary molar extrusion, intrusion, downward or backward rotation of the maxilla and consequently mandibular rotation, and increases in anterior facial height.[4,24,34–36,41–43]

Applications of headgear in combination with removable or fixed and functional appliances have also been reported. Cephalometric evaluation in Class II division 1 patients treated with the Cetlin method—use of cervical headgear, lip bumper, and maxillary removable plate with springs to distalize the molars— showed a significant distal tipping in 70% of the treated patients and significant limitation of maxillary forward growth but no significant change in the mandibular position.[42]

The lip bumper is another orthodontic device that has several indications in early orthodontic treatment, especially in some Class II malocclusions. Many investigators have reported the effect of lip bumper application on transverse, sagittal, and vertical changes in occlusion.[19,44,45] These reports indicated that the lip bumper has several effects: increase in arch length, uprighting of mandibular first molars, bodily dental expansion, alveolar bone remodeling, proclination of mandibular incisors by reduction of lip muscle force and abnormal dysfunction and thereby reduction of overjet, correction of the curve of Spee, opening of the bite, and maintenance of leeway space.[19,44,45]

The Hawley appliance also has many applications as a passive or active appliance in orthodontic treatment, including as an anterior bite plate for reducing

overbite and overjet by retroclination of maxillary incisors and as a slow expander by adding a jackscrew.

Early Class II Treatment with Headgear, Lip Bumper, and Hawley Appliance (HLH) Technique

This section introduces a technique for early treatment of Class II malocclusions during the mixed dentition. In this technique, a combination of headgear, a lip bumper, and a Hawley appliance, which the author calls the *HLH technique*, has been designed with certain modifications.

To explain the characteristics, mechanisms, and the reasons for the modifications that have been applied in the HLH technique, it is necessary to review briefly the application and original function of each of these three devices separately.

Headgear

As an extraoral device, headgear can exert different directions of force to the basal bone and dentition and produce different treatment effects on the occlusion. Success in the application of headgear depends on three important factors: application at the proper time of growth, the patient's cooperation, and proper mechanical management.

Proper timing

The proper age for starting early orthodontic treatment and achieving proper growth modification is just before and during the growth spurt, as has been advocated by some investigators.[40] Kopecky and Fishman[39] also indicated that this type of treatment has the best results when implemented before and during the growth spurt, which coincides with a value of 4 to 7 according to Fishman's method[46] of skeletal maturation assessment.

Patient's cooperation

The patient's cooperation is an important factor in treatment in any orthodontic treatment, especially in headgear wear, and must be considered in treatment planning. Practitioners have an important role in gaining the understanding of

patients and parents and must explain the patient's dentofacial problems as well as the necessity and advantages of orthopedic force.

Proper mechanical management

Contrary to perceptions that headgear application is an easy procedure, the mechanical management of headgear therapy is a procedure that requires precision. Headgear consists of three parts: the facebow, the anchorage pad, and a strap for traction. Depending on whether tipping, bodily, or combination movement is desired for the teeth or the maxilla, the following determinations must be made:

- Location of anchorage
- Location of the inner bow attachment
- Position of the outer bow relative to the inner bow (above, below, or at the same level)
- Type of outer bow (short, medium, or long)

The location of anchorage can be cervical, occipital, or a combination. A cervically located headgear pad with a long outer bow exerts a distal and low force on the teeth and the maxilla. This design has distalization and extrusion effects on the molars and a tipping effect on the palatal plane (counterclockwise rotation). The best indication for this configuration is Class II deep bite in a patient with a horizontal growth pattern.

High-pull headgear, in which the anchorage pad is located on the occipital area, exerts a vertical and distal force on the maxillary molars and maxillary base and causes intrusion and distal movement. Depending on the size of the outer bow (short or medium) used with high-pull headgear, different effects on the molars and the maxilla can be achieved. These include intrusion and distal tipping or intrusion and distal bodily movement.

Combination headgear has a combination of cervical and occipital pull that imparts a straight distal force to the molars for controlling extrusion and distal movement.

In all mentioned types of headgear, the size of the outer bow (short, medium, or long) and the position of the outer bow relative to inner bow (above, below, or at the same level) can change the direction of forces.

Another mechanical specification of headgear that can have different effects on the dentition and basal bone is the location of the inner bow attachment. The inner bow

is commonly connected to molar tubes, but in certain situations it can be connected to the anterior segment of the maxillary archwire (like J-hook headgear), such as in patients who need incisor retraction accompanied with intrusion.

In a headgear–functional appliance combination, the inner bow can be inserted in an acrylic resin pad in the anterior, molar, or premolar area, depending on the desired direction of orthopedic force.

The following are the mechanical functions of headgear, which may vary with different configurations:

- Establishment of normal molar relationships
- Restriction of forward maxillary growth (prevention of forward movement of point A)
- Restriction of vertical maxillary growth (high-pull headgear)
- Creation of minor space by distalization of molars (tipped or bodily, depending on the type of headgear)
- Extrusion of maxillary molars to reduce overbite
- Intrusion of maxillary molars to reduce open bite or open bite tendency
- Correction of molar rotation
- Expansion of molar region
- Constriction of molar region

Modified Hawley appliance

The second component of the HLH technique for early treatment of Class II malocclusion is a modified Hawley appliance (Fig 11-2). The modified appliance has the following specifications:

- Two C-clasps: These are simple clasps made with 0.032- or 0.036-inch stainless steel wire and designed to be inserted in an acrylic resin pad that runs from the distal surface of the molars over the molar buccal tube and extends to the premolar area for easy removal.
- An inclined acrylic resin pad: The pad is placed behind the maxillary incisors for mesial movement of the mandible during closure. The position of this pad varies, depending on the severity of overjet and sagittal discrepancy of the maxilla and the mandible.

- A standard labial bow: The bow is designed to maintain anterior retention and help incisor retraction if needed.
- In some cases, 2 × 4 bonding instead of a labial bow: Another modification might be necessary in some patients with maxillary incisor crowding, rotation, or overlap that cannot be managed by a labial bow. In this situation, the Hawley appliance is designed without a labial bow; instead, 2 × 4 bonding can be used simultaneously with the inclined acrylic resin pad to achieve leveling, unraveling, and correction of crowding in the incisors.
- Addition of a jackscrew for expansion of the posterior arch segment: The jackscrew is added when patients with Class II malocclusion also have transverse problems.
- Occlusal acrylic resin coverage in the posterior regions: Occlusal coverage is a modification that might be needed in patients with Class II malocclusion, anterior open bite or open bite tendency, and a vertical growth pattern, where all of the effort must be focused on controlling the vertical dimension.

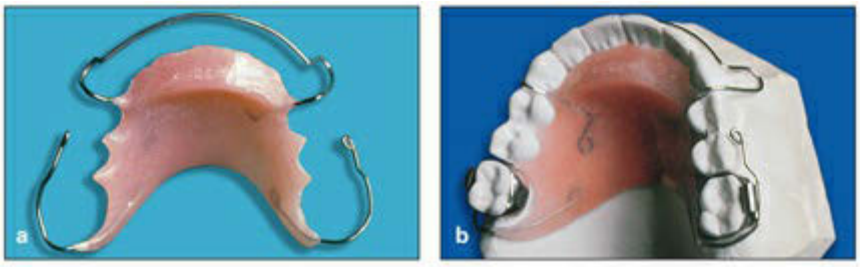


Fig 11-2 *(a and b)* Modified Hawley appliance designed for the HLH technique.

When the modified Hawley appliance is used in conjunction with headgear, the following treatment results can be achieved:

- Unilateral or bilateral distal movement of the molars by distal positioning of the C-clasp to achieve Class I molar relationships
- Distal maxillary traction, applied to the whole maxilla as a unit, for orthopedic action to control maxillary growth and correct point A–point B (A-B) discrepancy
- Mandibular forward displacement as an orthopedic force to achieve mandibular protraction
- Correction of deep bite by anterior placement of the acrylic resin pad and opening of posterior bite and extrusion of molars by cervically placed headgear.
- Correction of anterior open bite or open bite tendency by the addition of posterior occlusal acrylic resin coverage to the modified Hawley in conjunction with high-pull headgear

- Maxillary arch expansion, if needed, by the addition of a jackscrew to the modified Hawley appliance

Lip bumper

The third appliance used in the HLH technique is the lip bumper, which consists of two molar bands with a double buccal tube—one a round 0.040- or 0.045-inch tube for insertion of the lip bumper bow and one for insertion of the archwire—and an acrylic resin or plastic pad located behind the lower lip.

In patients that need some buccal expansion, the pad can be extended toward the buccal segments to separate perioral muscle force, facilitate natural arch expansion, and perhaps initiate bone remodeling. Many investigators have reported on the stability of this type of expansion that takes place during occlusal development.[19,45]

The lip bumper has several indications in early orthodontic treatment, especially in some types of Class II malocclusion. As mentioned earlier, many investigators have reported use of different types of lip bumper and the transverse and vertical changes they have on occlusion.[17,42,43]

As a part of the HLH technique, the lip bumper is used in conjunction with headgear and a modified Hawley appliance for the following conditions:

- Class II division 1 malocclusions associated with deep bite, deep curve of Spee, and retruded and sometimes crowded mandibular incisors are usually the result of early loss of mandibular primary canines or primary first molars, combined with lip dysfunction. These cases are ideal for lip bumper application.
- The lip bumper can upright permanent molars, procline mandibular incisors, correct curve of Spee, and reduce overbite in patients with Class II division 1 malocclusion who exhibit deep bite and tipped mandibular permanent molars due to early loss of primary molars.
- In patients with Class II division 1 malocclusion who have normal overbite or open bite tendency but minor mandibular incisor crowding and retrusion due to hyperactivity or dysfunction of the lower lip, a regular lip bumper cannot be applied because it can produce greater bite opening. In these patients, if it is necessary to release the incisors from lip pressure, the author recommends using the combination of a lip bumper and a lower holding arch, which will have the same effect on the mandibular incisors but prevent any changes in the molar area (Fig 11-3).

Fig 11-3 *(a)* Simple lip bumper. *(b)* Combination of lip bumper and lower holding arch for the HLH technique.

The lip bumper can achieve the following:

- Increase in arch length (distal molar movement; labial anterior movement)
- Disruption of the equilibrium between the perioral musculature and the tongue to increase arch width and promote alveolar growth
- Correction of the curve of Spee by uprighting the molars, proclining the anterior teeth, and facilitating eruption of the posterior segment
- Reduction of overjet by preventing lip muscle dysfunction and proclining the incisors

Strategy and Tactics of HLH Application

Application of triple appliances (HLH) with the proper modifications during the mixed dentition is a simple, effective way of achieving early Class II correction. Selection of the proper type of headgear, determination of the necessary design modifications for the Hawley appliance (such as anterior inclined surface, bite plane, or posterior occlusal coverage), and proper use of the lip bumper are critically important to the success of the HLH technique.

For early intervention in Class II division 1 malocclusion, the HLH approach may be applied as a two-phase or a one-phase treatment. For treatment of Class II division 2 malocclusion, the HLH technique has some differences in tactics, which are designed to address the specific characteristics of this anomaly.

Two-phase strategy for Class II division 1 malocclusion

Two-phase HLH application is used for management of Class II dental malocclusion

or Class II malocclusion with minor A-B discrepancy. This tactic usually is started during the middle mixed dentition to establish normal permanent molar relationships, normal overjet, and normal overbite. After the interceptive HLH therapy has established Class I molar relationships, an interim phase continues until all permanent canines and premolars erupt. The interim phase can include use of a Hawley retainer or be limited to observation. Phase 2 takes place during the permanent dentition and involves minor final adjustment.

The advantage of early HLH intervention during the mixed dentition is the possibility for normal eruption of the canines and premolars with normal interdigitation that may not need any further treatment (see cases 11-1 and 11-2). In the two-phase approach, phase 1 (HLH treatment) usually takes 1 to 1.5 years; the interim phase of observation and guidance of eruption takes about 1 year; and phase 2 (final alignment) takes about 1 year. However, permanent canines and premolars may exhibit normal interdigitation after eruption, eliminating the need for phase 2. This prospect is based on the finding that, if Class I molar relationships are achieved, normal interdigitation of buccal segments will follow, as Poulton[47] has shown. Canine and premolar tooth buds move distally and follow the root movement of the maxillary permanent first molars after they are distalized.

One-phase strategy for Class II division 1 malocclusion

The one-phase HLH treatment strategy is applied for correction of skeletal Class II malocclusions with moderate or severe A-B discrepancies, usually starting around the late mixed dentition or before the growth spurt (skeletal maturation assessment value of 4 to 7) and continuing until eruption of the permanent dentition (excluding third molars) is completed. It is a continuous treatment approach with no interim phase. The one-phase HLH treatment is followed by complete bonding for final tooth alignment. It usually takes about 2 to 2.5 continuous years of treatment from HLH to the end of comprehensive treatment.

The first step is establishment of normal permanent molar relationships combined with control of maxillomandibular discrepancy by controlling maxillary growth, promoting mandibular growth, or both. The second step starts with the bonding of premolars after they erupt and retraction, if needed, to establish normal interdigitation in the molar and premolar segments. The next step is canine alignment and retraction, which might be accompanied by Class II elastics. The final step is incisor retraction and final adjustment before retention. Depending on the severity of the skeletal jaw discrepancy, the use of headgear might be continued to the final

stage of treatment, even after normal molar relationships are established, or to provide anchorage.

A significant advantage of this type of early intervention is that sagittal abnormalities, whether the result of maxillary protrusion, mandibular retrusion, or a combination, can be controlled with the HLH approach, while the vertical and transverse dimensions of the malocclusion can be controlled by taking advantage of the patient's growth potential.

Strategy for Class II division 2 malocclusion

Early treatment of Class II division 2 malocclusions has some specific differences in tactics. The major characteristic of Class II division 2 malocclusions, besides Class II interdigitation, is deep bite and maxillary incisor crowding with severe retroclination of the maxillary central incisors. Therefore, the first steps in treatment are to control overbite with a thick anterior bite plate that will disarticulate the posterior dentition, encouraging eruption of those teeth, and to facilitate molar retraction with the cervical headgear. The next step is 2 × 4 maxillary bracketing to achieve anterior alignment and some proclination of the central incisors. This measure will facilitate mandibular growth and allow for accelerated dentoalveolar development that might have been restricted by locked occlusion caused by maxillary incisor retroclination and deep bite.

In the next step, after some overbite reduction, a lip bumper is applied if the mandibular incisors are also crowded and retroclined; later, this is followed by mandibular incisor bonding.

One important goal of early intervention in Class II division 2 malocclusion is to control the child's overbite at an early age by taking advantage of the patient's growth; this type of treatment is very difficult to manage later.

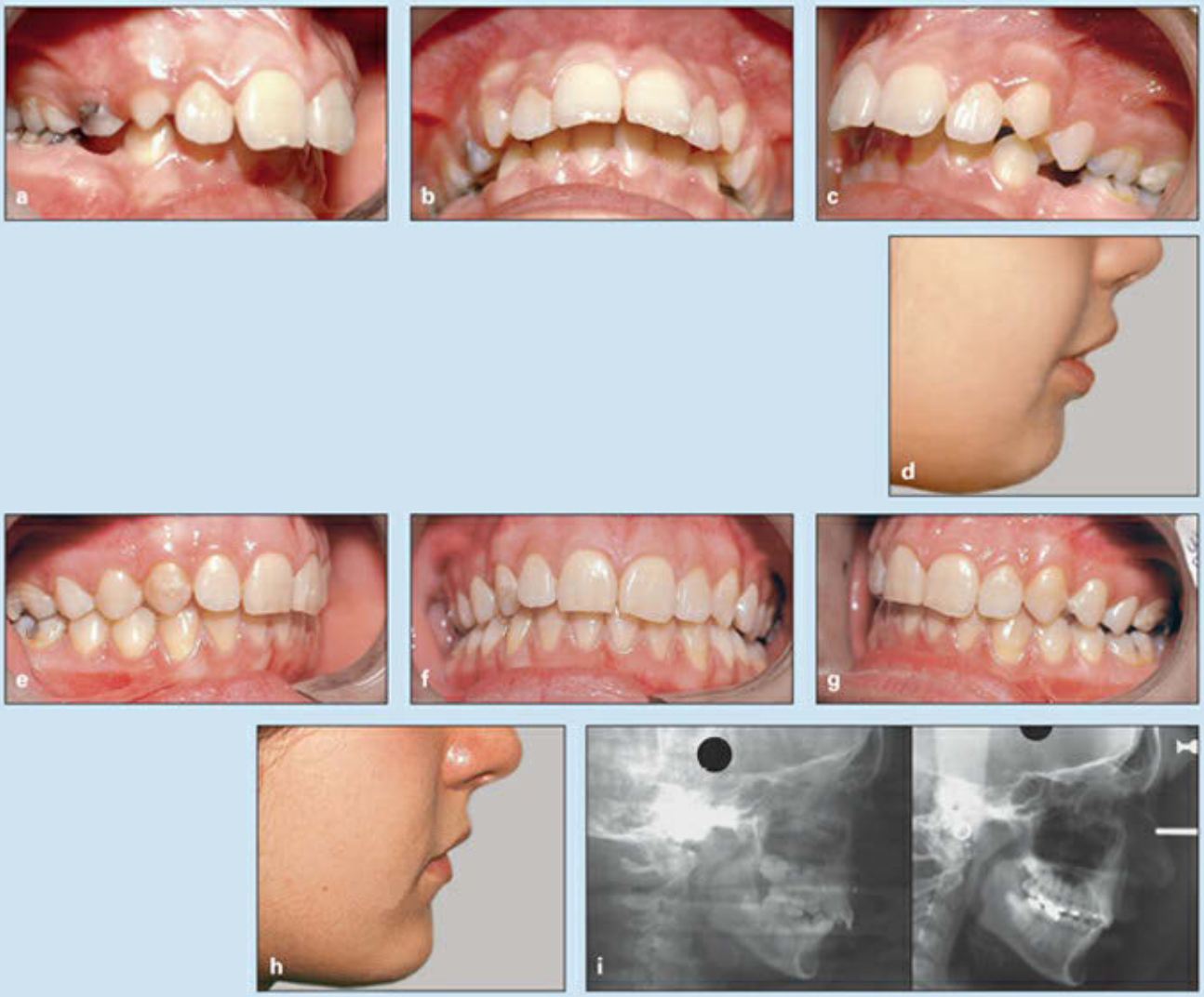
Case Reports

The following case reports present examples of early treatment with the HLH procedure, applied in either one phase or two phases of treatment.

Case 11-1: Two-phase interceptive treatment and observation

A 10-year, 5-month-old girl in the late mixed dentition had a Class II division 1 malocclusion, a 9.2-mm overjet, and deep overbite. The maxillary incisors had normal inclination, the mandibular incisors were slightly uprighted, and she had a large A-B discrepancy (point A–nasion–point B [ANB] angle = 7.4 degrees) resulting from mandibular retrognathia (Figs 11-4a to 11-4d).

Treatment:
The treatment plan was use of the HLH technique alone: cervical headgear, a maxillary modified Hawley appliance with anterior inclined surface, and a mandibular lip bumper. Treatment resulted in significant changes in the patient's occlusion, soft tissue profile, and molar relationships (Figs 11-4e to 11-4j). The A-B discrepancy (ANB) was reduced from 7.4 to 0.8 degrees and overjet from 9.2 to 2.9 mm.



j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.9	88.9
SNB (°)	78.0	73.5	76.6
SNA (°)	82.0	80.9	77.4
Maxillary depth (FH-NA) (°)	90.0	88.6	87.8
ANB (°)	2.3	7.4	0.8
FMA (MP-FH) (°)	25.5	26.8	21.0
Y-axis (SGn-SN) (°)	59.4	68.3	67.1
Interincisal angle (U1-L1) (°)	135.0	126.3	131.1
U1-FH (°)	116.2	111.7	114.6
IMPA (L1-MP) (°)	95.0	95.2	93.4
Overbite (mm)	2.5	5.0	2.4
Overjet (mm)	2.5	9.2	2.9

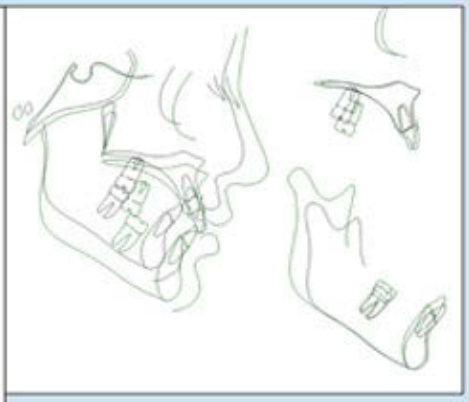


Fig 11-4 Management of severe overjet and deep overbite in a 10-year, 5-month-old girl in the late mixed dentition. She was treated only with the HLH technique. *(a to c)* Pretreatment

occlusion. *(d)* Pretreatment soft tissue profile. *(e to g)* Posttreatment occlusion. *(h)* Posttreatment soft tissue profile. *(i)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-2: Two-phase interceptive treatment and observation

A 9-year-old girl had a Class II division 1 malocclusion, minor overbite, a 7.9-mm overjet, a steep mandibular plane, and a slight open bite tendency (Figs 11-5a to 11-5d). Maxillary incisor protrusion had caused lip incompetence, but the mandibular dentition was good.

Treatment:
Treatment involved only the application of the HLH technique (combination headgear) with one stage of active treatment and a second stage of observation. Instead of a lip bumper, a lower holding arch was used to preserve leeway space and prevent molar eruption. A maxillary Hawley appliance was used for maxillary incisor retraction, and the headgear also aided maxillary retraction. Figures 11-5e to 11-5j show the results of treatment.

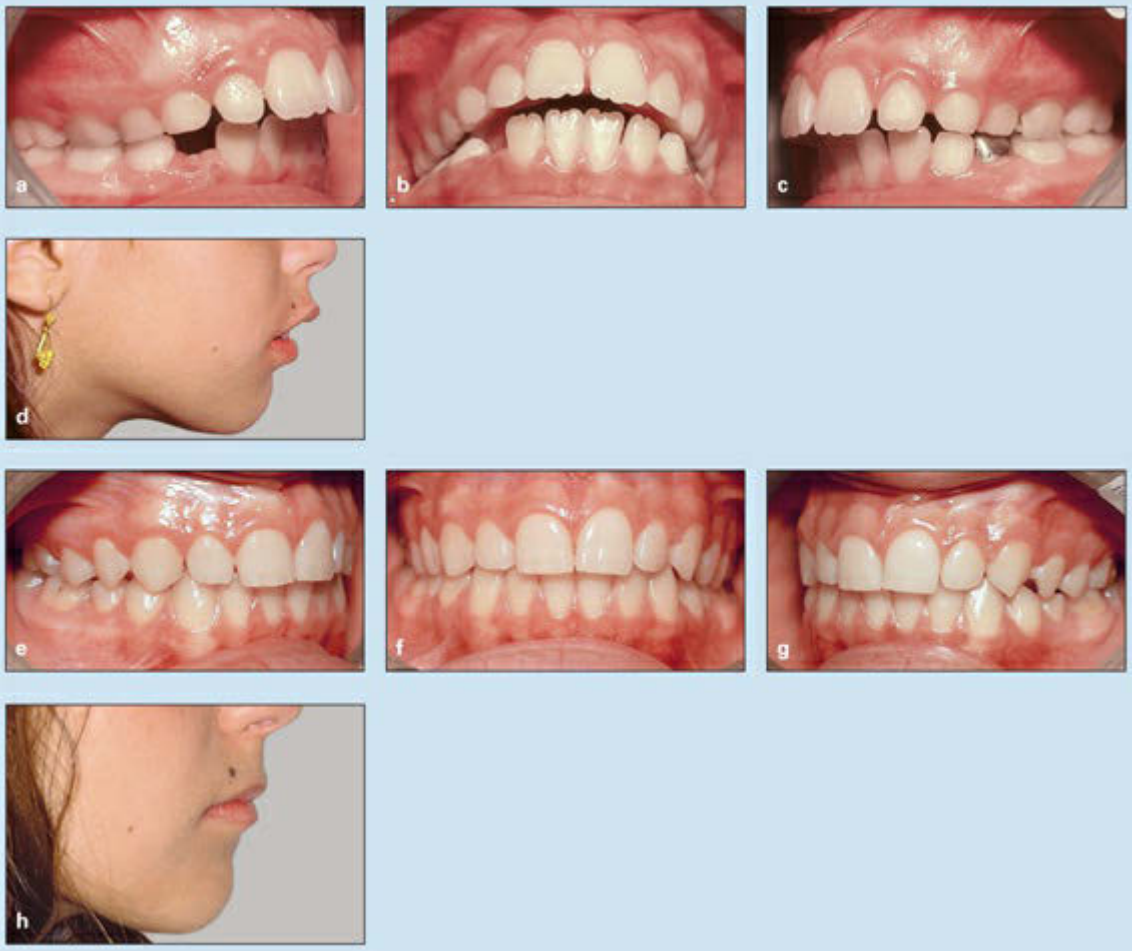
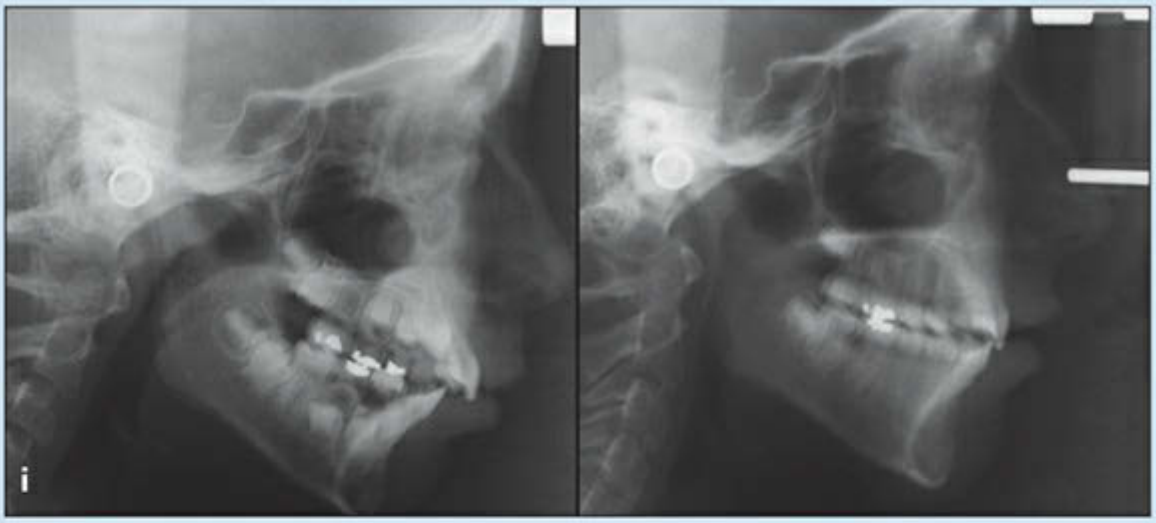


Fig 11-5 Management of minor overbite and severe overjet in a 9-year-old girl with a Class II division 1 malocclusion. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment soft tissue profile. *(e to*

g) Posttreatment occlusion. *(h)* Posttreatment soft tissue profile.



i

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	83.9	85.7
SNB (°)	78.0	70.7	73.0
SNA (°)	82.0	75.0	75.5
Maxillary depth (FH-NA) (°)	90.0	84.0	85.5
ANB (°)	2.3	4.3	2.5
FMA (MP-FH) (°)	25.5	27.8	26.9
Y-axis (SGn-SN) (°)	59.4	69.2	70.7
Interincisal angle (U1-L1) (°)	135.0	119.2	125.0
U1-FH (°)	116.2	121.1	114.6
IMPA (L1-MP) (°)	95.0	95.6	96.4
Overbite (mm)	2.5	3.6	2.0
Overjet (mm)	2.5	7.9	2.5

j

Fig 11-5 *(cont) (i)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(j)* Changes in cephalometric measurements.

Case 11-3: Two-phase interceptive treatment and observation

A 9-year-old girl presented with a Class II division 1 malocclusion, a 7.6-mm overjet, an A-B discrepancy of 7.1 degrees, impinging deep bite, and mandibular retrognathism caused by clockwise

rotation (Figs 11-6a to 11-6c).

Treatment:
This patient was treated with a two-phase active HLH procedure, including combination headgear for retraction of the maxillary first molar while controlling extrusion, and a combination lip bumper and lower holding arch (see Fig 11-3b) was used to control uprighting of the mandibular molars while preventing lip dysfunction. After Class I molar relationships were achieved, a Hawley retainer was placed during the interim phase (at first 24 hours a day and then 12 hours' use at night) until the canines and premolars had erupted (Figs 11-6d to 11-6f) . Figures 11-6g and 11-6h show the pre- and posttretment cephalometric radiographs, superimposition of tracings, and measurements.

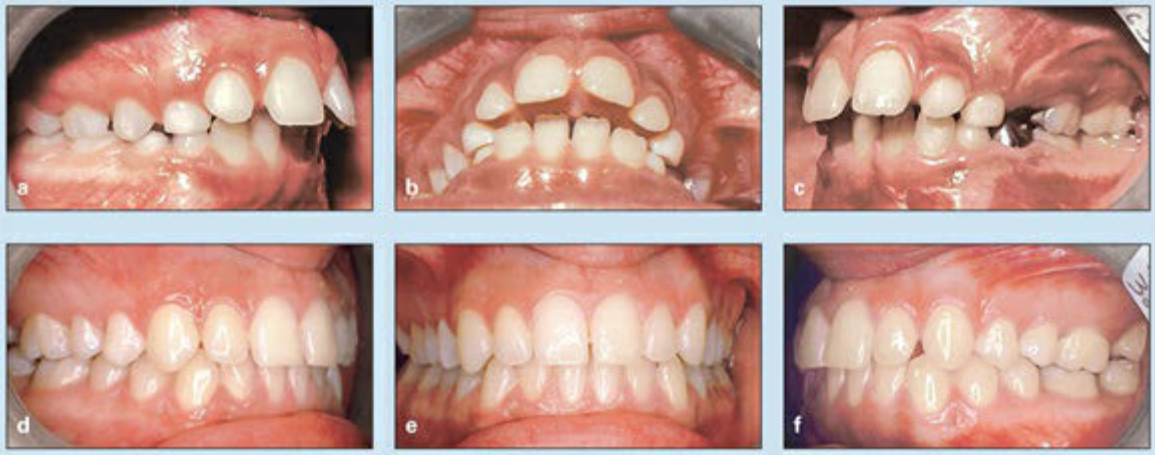
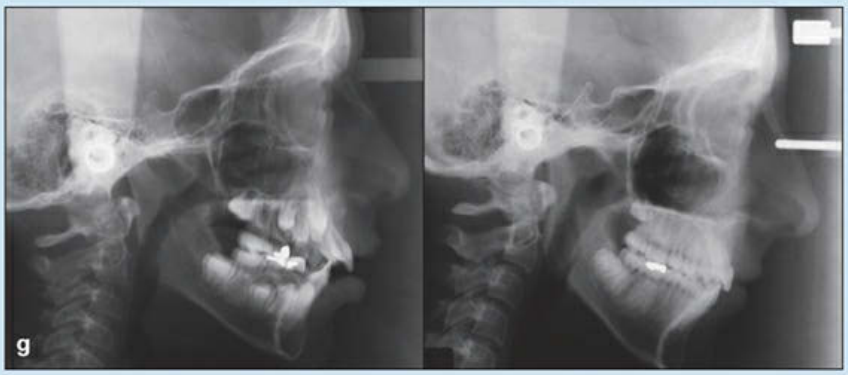


Fig 11-6 Management of severe overjet, impinging deep bite, and mandibular retrognathism in a 9-year-old girl. *(a to c)* Pretreatment occlusion. *(d to f)* Posttreatment occlusion.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	86.6	80.1
SNB (°)	78.0	71.9	71.5
SNA (°)	82.0	79.1	73.3
Maxillary depth (FH-NA) (°)	90.0	90.2	81.9
ANB (°)	2.3	7.1	1.8
FMA (MP-FH) (°)	25.9	31.6	36.1
Y-axis (SGn-SN) (°)	59.4	72.0	75.2
Interincisal angle (U1-L1) (°)	135.0	121.9	121.5
U1-FH (°)	116.2	119.1	112.4
IMPA (L1-MP) (°)	95.0	87.4	90.0
Overbite (mm)	2.5	2.2	–0.1
Overjet (mm)	2.5	8.6	2.1

h

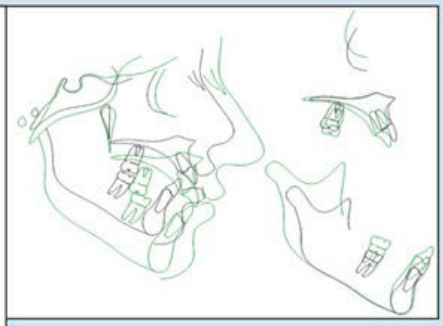


Fig 11-6 *(cont) (g)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(h)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-4: Two-phase treatment with HLH and comprehensive treatment

A 9-year, 7-month-old girl in the middle mixed dentition presented with a Class II division 1 malocclusion, an 11-mm overjet, and severe overbite that had resulted in a collapsed mandibular dentition and retrognathia (retrognathic chin). Here lips were apart, and lip dysfunction continued to worsen the irregularities (Figs 11-7a to 11-7d).

Treatment:
The treatment approach was designed as two-phase HLH treatment. In phase 1, cervical headgear was used to retract the maxillary molars and reduce overbite; an inclined maxillary Hawley appliance was used to stimulate mandibular growth by unlocking the collapsed mandible, helping forward mandibular growth; a lip bumper was used to control lip dysfunction and alleviate the lip force directed at the mandibular incisors, thereby correcting mandibular incisor retroclination, uprighting mandibular molars, and correcting the curve of Spee. Figure 11-7e illustrates the maxillary dentition and spontaneous distal movement of the buccal segments achieved by cervical headgear and good compliance. Phase 2 was

started after complete eruption of the canines and premolars. Use of the headgear was stopped, and maxillary Nance anchorage was placed. Figures 11-7f to 11-7k show the posttreatment results, which included correction of the Class II malocclusion, correction of the severe overjet (reduction from 11.0 to 1.9 mm), correction of the deep bite (4.6 to 0.8 mm), reduction in the ANB angle from 6.2 to 2.7 degrees, and a change in the retrognathic chin from 82.5 to 85.9.

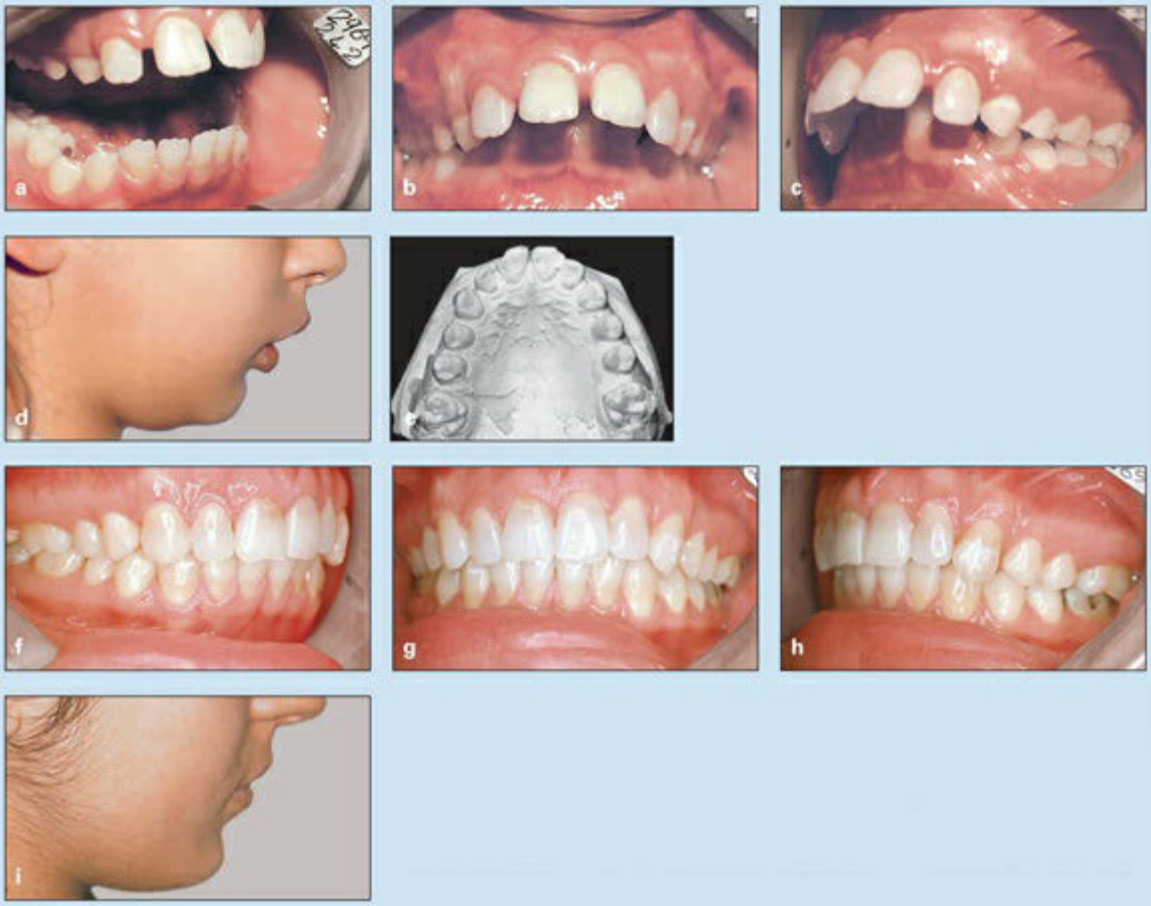
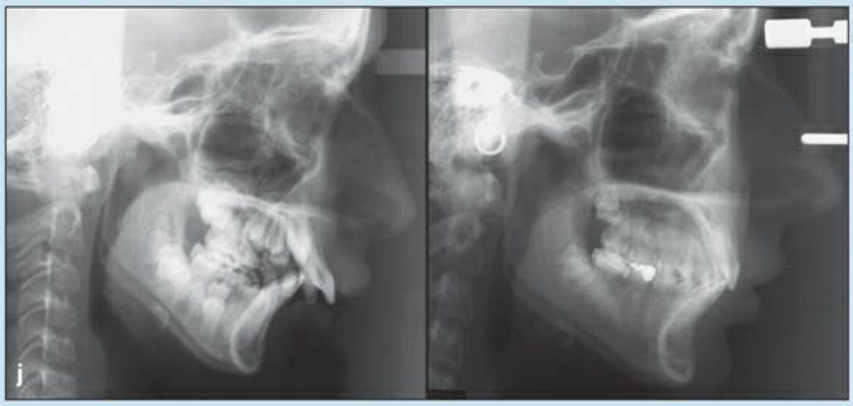


Fig 11-7 Management of collapsed mandibular dentition and retrognathia caused by severe overjet and overbite in a 9-year, 7-month-old girl. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment soft tissue profile. *(e)* Cast prepared after phase 1 treatment with the HLH protocol and prior to the start of phase 2 treatment with a Nance appliance. *(f to h)* Posttreatment occlusion. *(i)* Posttreatment soft tissue profile.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.5	86.7
SNB (°)	78.0	73.8	76.8
SNA (°)	82.0	80.0	79.5
Maxillary depth (FH-NA) (°)	90.0	87.0	89.4
ANB (°)	2.3	6.2	2.7
FMA (MP-FH) (°)	25.9	31.3	29.4
Y-axis (SGn-SN) (°)	59.4	70.0	73.3
Interincisal angle (U1-L1) (°)	135.0	118.3	128.2
U1-FH (°)	116.2	115.2	104.2
IMPA (L1-MP) (°)	95.0	93.4	94.4
Overbite (mm)	2.5	4.6	0.8
Overjet (mm)	2.5	11.6	1.9

k

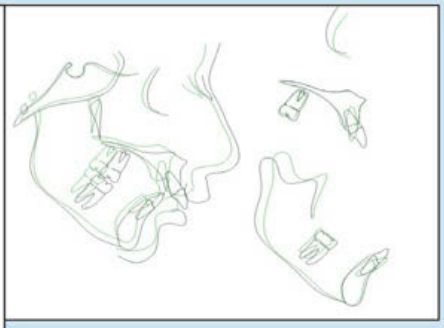


Fig 11-7 *(cont) (j)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(k)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-5: One-phase orthopedic treatment

A 10-year, 7-month-old girl in the early permanent dentition presented with a Class II division 1 malocclusion, a 12.1-mm overjet, impinging deep bite, a severe curve of Spee, incompetent and hypotonic lips, maxillomandibular dental protrusion, and a severely convex profile (Figs 11-8a to 11-8d). She exhibited some interdental spacing. Her dental age was advanced but her skeletal growth retarded.

Treatment:
Because of her skeletal age, interdental spacing, and good compliance, the treatment plan was designed as a nonextraction, one-phase HLH technique.

The patient received cervical headgear and a modified Hawley appliance with an inclined bite plane. Mandibular complete bonding was performed to correct the curve of Spee, and no lip bumper was used because of the mandibular incisor protrusion to achieve a consolidated mandibular arch against the inclined maxillary Hawley appliance during forward mandibular positioning to stimulate mandibular growth.

After good Class I molar relationships were established, complete maxillary bonding was started. Use

of the headgear was continued as anchorage. Maxillary buccal segments were retracted while the mandibular curve of Spee was corrected and the overbite was significantly decreased. The final step was removal of the maxillary inclined Hawley appliance and initiation of anterior retraction.

Figures 11-8e to 11-8k illustrate the treatment outcome.

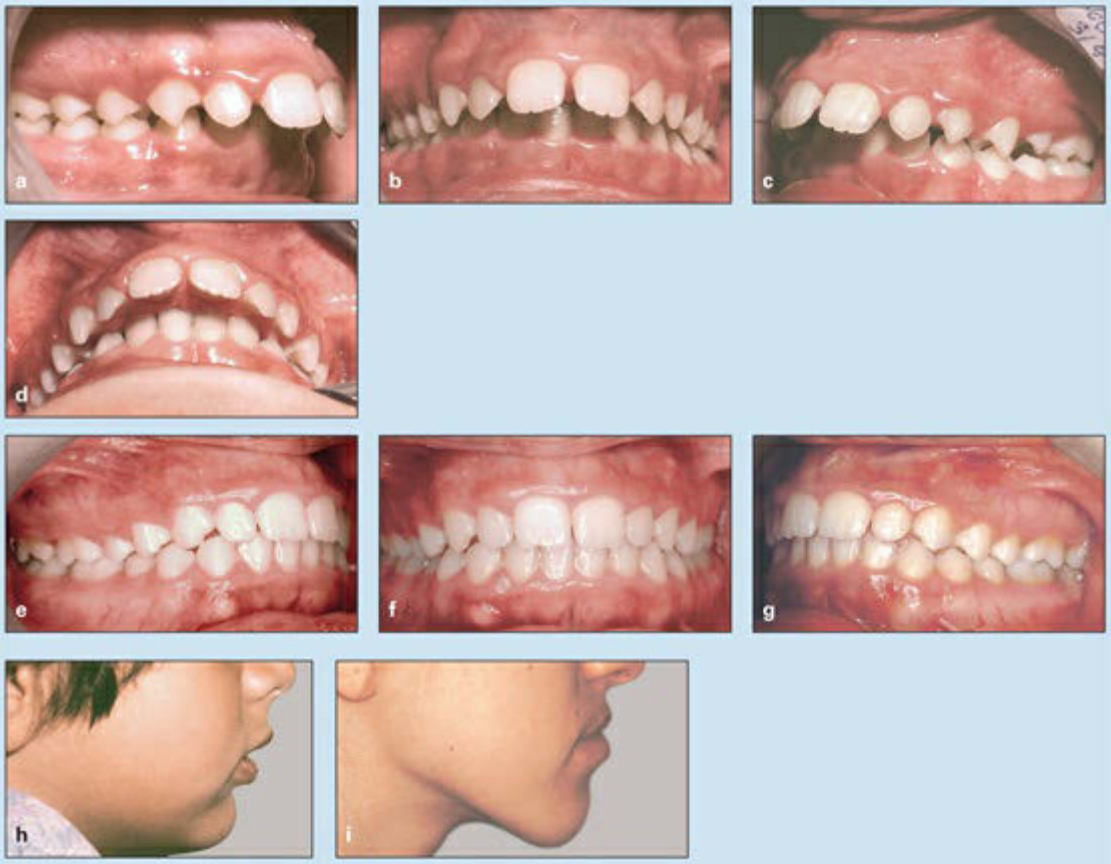
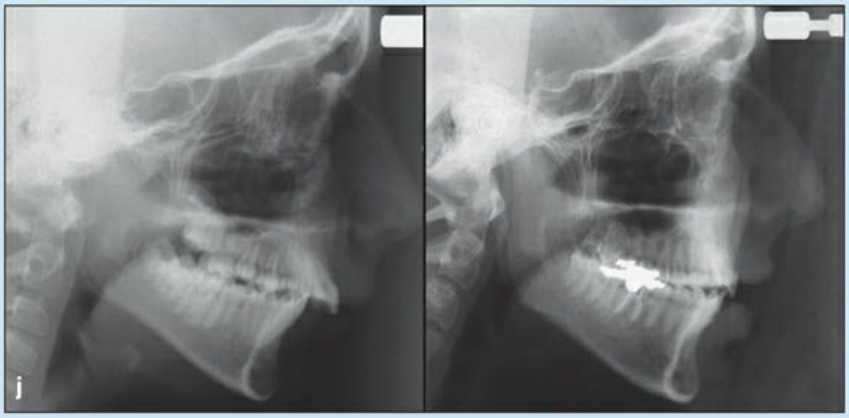
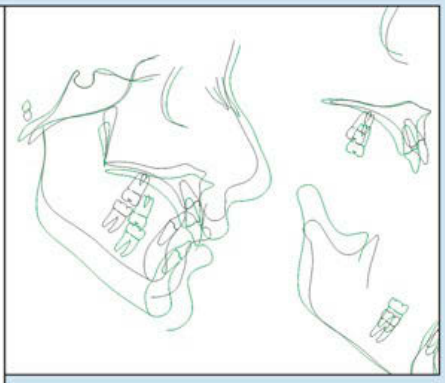


Fig 11-8 Management of severe overjet and impinging deep bite in a 10-year, 7-month-old girl. She has a severe curve of Spee, incompetent lips, and maxillomandibular dental protrusion. *(a to d)* Pretreatment occlusion. Although her dental age is advanced, her skeletal growth is retarded. *(e to g)* Posttreatment occlusion. *(h)* Pretreatment soft tissue profile. *(i)* Posttreatment soft tissue profile.



j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.4	86.9
SNB (°)	78.0	72.1	74.5
SNA (°)	82.0	79.8	77.3
Maxillary depth (FH-NA) (°)	90.0	88.2	88.2
ANB (°)	2.3	7.7	2.8
FMA (MP-FH) (°)	25.9	31.4	31.1
Y-axis (SGn-SN) (°)	59.4	71.3	73.2
Interincisal angle (U1-L1) (°)	135.0	118.3	127.2
U1-FH (°)	116.2	125.0	108.8
IMPA (L1-MP) (°)	95.0	85.3	93.0
Overbite (mm)	2.5	5.1	–0.5
Overjet (mm)	2.5	12.1	2.3



k

Fig 11-8 *(cont) (j)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs and tracings. *(k)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-6: One-phase orthopedic treatment

A 10-year-old boy in the late mixed dentition presented with a severe Class II division 1 malocclusion, a 16-mm overjet, impinging deep bite, lip dysfunction, and a 5.6-mm interlabial gap (Figs 11-9a to 11-9e). He also exhibited a deep curve of Spee and proclined maxillary incisors, causing lip incompetence and severe A-B discrepancy (ANB = 7.4 degrees). In this type of occlusion, the mandibular dentition collapses and becomes locked behind the maxillary teeth; this type of abnormality normally worsens with age.

Treatment:
The patient was treated with one-phase orthopedic treatment, which started with an anterior bite plate, cervical headgear, and a mandibular 2 × 4 bonding utility arch to upright and depress the mandibular incisors and correct the curve of Spee. After a Class I molar relationship was achieved, the maxillary incisors were bonded for interdental space closure and some retraction accompanied with intrusion to reduce overjet and overbite and control lip dysfunction.

The next stage was sequential extraction of the remaining primary molars and guidance of canine and

premolar eruption. The headgear was stopped, and insertion of Nance anchorage and complete posterior and anterior retraction with Class II elastics against the consolidated mandibular arch was initiated.

Figures 11-9f to 11-9l show the dental, dentoskeletal, and soft tissue changes achieved through treatment.

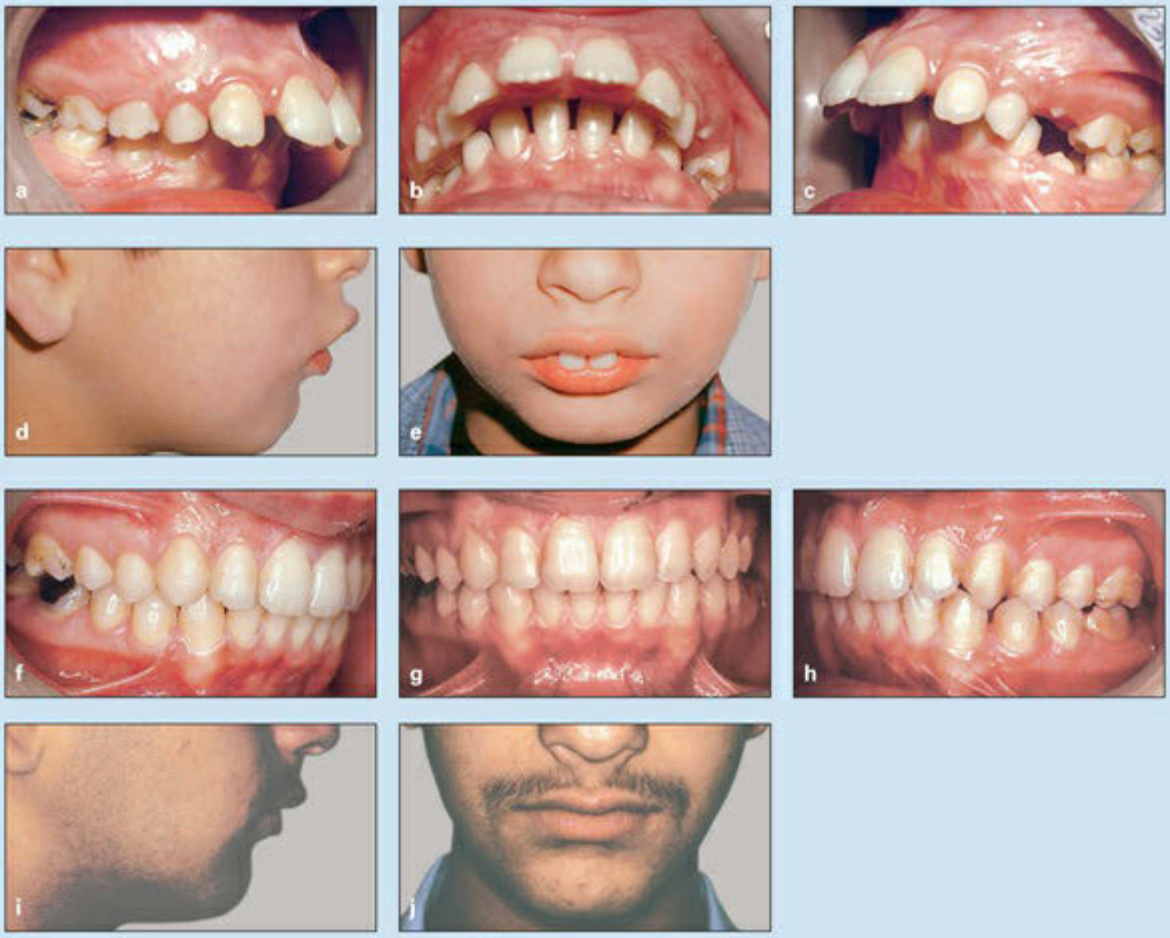
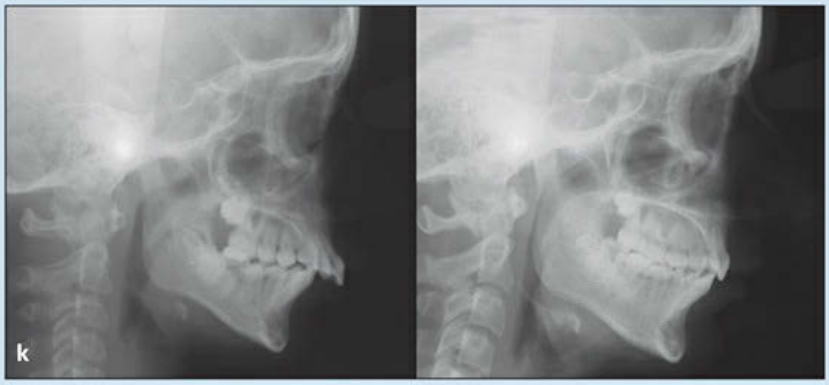
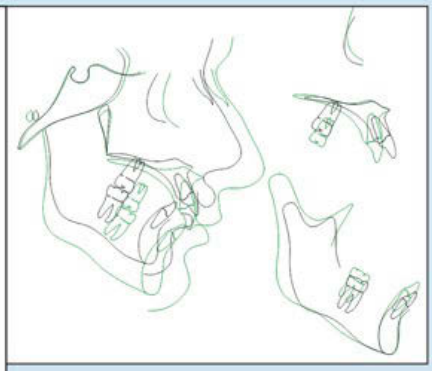


Fig 11-9 *(a to c)* Pretreatment occlusion. *(d)* Pretreatment soft tissue profile. *(e)* Pretreatment lip closure. *(f to h)* Posttreatment occlusion. *(i)* Posttreatment soft tissue profile. *(j)* Posttreatment lip closure.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.4	83.6
SNB (°)	78.0	73.0	73.1
SNA (°)	82.0	80.4	77.8
Maxillary depth (FH-NA) (°)	90.0	88.6	87.6
ANB (°)	2.3	7.4	4.7
FMA (MP-FH) (°)	25.9	24.5	25.9
Y-axis (SGn-SN) (°)	59.4	72.0	74.0
Interincisal angle (U1-L1) (°)	135.0	109.7	129.5
U1-FH (°)	116.2	121.6	101.4
IMPA (L1-MP) (°)	95.0	104.2	103.2
Overbite (mm)	2.5	3.4	1.7
Overjet (mm)	2.5	12.0	2.7



l

Fig 11-9 *(cont) (k)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(l)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-7: One-phase orthopedic treatment

A 12-year-old girl presented with a Class II division 1 malocclusion, a 13.9-mm overjet, impinging deep bite, a deep curve of Spee, and lip dysfunction (Figs 11-10a to 11-10e). Her lips were incompetent, with a 9.1-mm interlabial gap. Lip closure was very difficult and caused excessive lip strain (Figs 11-10f to 11-10h).

Treatment:
Treatment started with cervical headgear, a maxillary Hawley bite plate, and a lip bumper to control lower lip dysfunction, distalize the maxillary molars, and reduce overbite. After good molar relationships were achieved, full bonding was started to align the anterior segment and correct the mandibular curve of Spee. Then use of the bite plate and the lip bumper was stopped, but use of the headgear was continued as anchorage during anterior retraction. Figures 11-10i to 11-10o show the posttreatment results: normal overjet and overbite; significant change in profile, lips, and appearance; and boost in self-confidence.

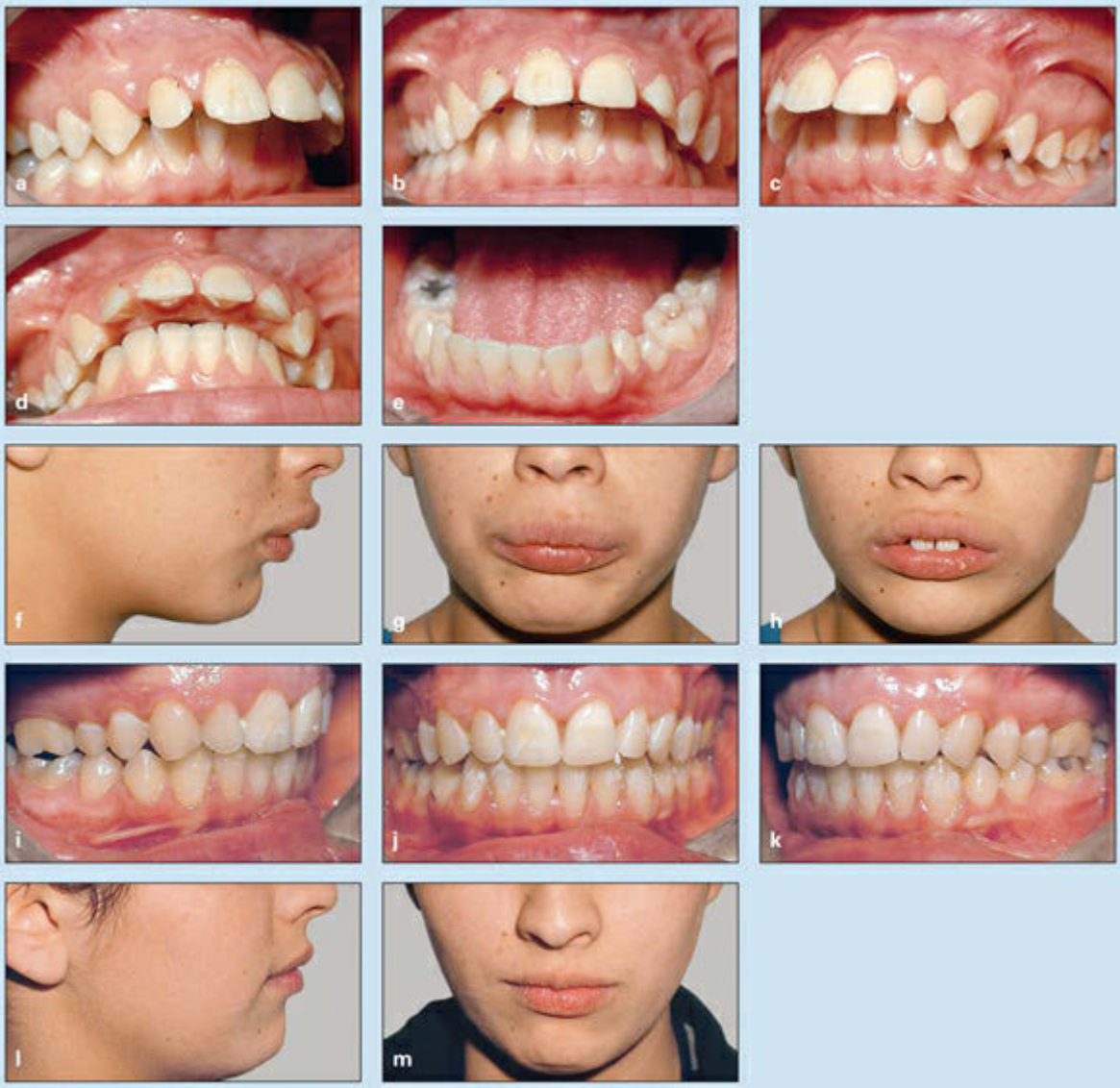
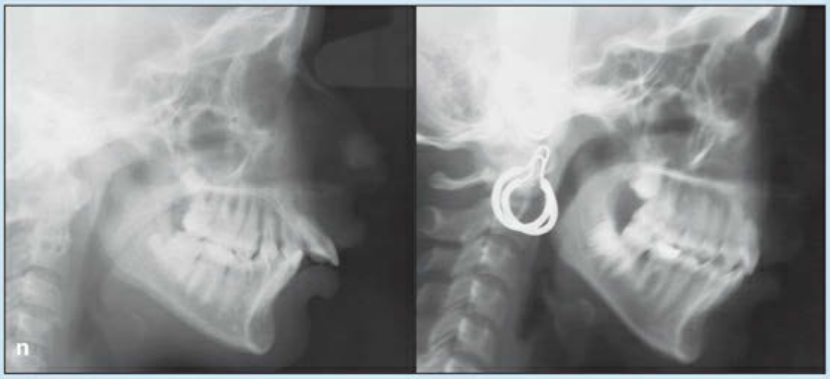


Fig 11-10 Management of severe overjet, impinging deep bite, deep curve of Spee, and lip dysfunction in a 12-year-old girl with a Class II division 1 malocclusion. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment soft tissue profile. *(g and h)* Pretreatment lip closure. *(i to k)* Posttreatment occlusion. *(l)* Posttreatment soft tissue profile. *(m)* Posttreatment lip closure.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	85.7	88.8
SNB (°)	78.0	76.3	79.4
SNA (°)	82.0	85.0	83.7
Maxillary depth (FH-NA) (°)	90.0	93.8	86.8
ANB (°)	2.3	8.7	4.3
FMA (MP-FH) (°)	25.9	36.8	38.0
Y-axis (SGn-SN) (°)	59.4	69.5	72.3
Interincisal angle (U1-L1) (°)	135.0	102.7	122.6
U1-FH (°)	116.2	132.1	110.7
IMPA (L1-MP) (°)	95.0	90.5	93.8
Overbite (mm)	2.5	1.1	1.0
Overjet (mm)	2.5	13.8	3.0

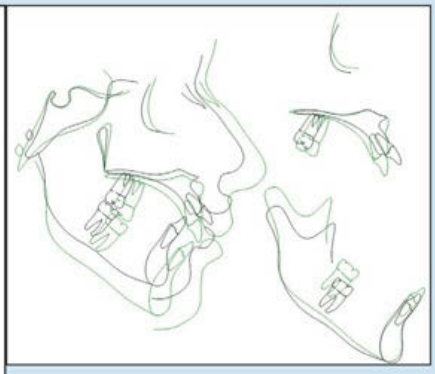


Fig 11-10 *(cont) (n)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(o)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Anterior Dental Crossbite and Class III Malocclusion

Anterior dental crossbite is a common problem in the primary or mixed dentition and merits early intervention to prevent further damage to occlusion. Treatment is effective and desirable in certain situations. Early correction of anterior crossbite not only resolves esthetic problems but also improves function and consequently prevents structural damage and adverse effects on the growth of the dentition and basal bones.

Anterior crossbites can have differing morphology, etiology, and treatment procedures. Generally there are three kinds of anterior crossbite:

1. Simple dental crossbite
2. Pseudo-Class III malocclusion (functional crossbite)

3. Skeletal Class III and potential Class III malocclusion

Precise clinical evaluation in static conditions and during functional movement and radiographic examinations (lateral cephalograms in centric occlusion and centric relation) are required for differentiation of dental, skeletal, and functional crossbites.

Differential diagnosis

Differential diagnosis of the skeletal and dental components of anterior crossbite is an important and essential part of determining the proper treatment approach and timing of treatment for these anomalies. Diagnosis consists of a complete intraoral and extraoral clinical evaluation, including assessment of the child's facial pattern and symmetry, left and right molar and canine relationships, both in centric relation and centric occlusion, and dental and facial midlines. Anterior occlusal relationship both in centric relation and centric occlusion must also be carefully evaluated. Any kind of mandibular shift must be carefully examined to determine the type of abnormality, treatment options, and prognosis.

Proper treatment planning and good results in the correction of anterior crossbite require careful evaluation of the morphology, etiology, and special characteristics for every single case. Two skeletal Class III malocclusions may look alike but have completely different characteristics that require different treatment plans. Therefore, the first step is differential diagnosis between different types of anterior crossbites, and the second step is the recognition of specific characteristics for every individual patient.

Clinical examination

The following are important points that should be evaluated in the clinical evaluation:

- Anterior and posterior dental relationships with the mandible in both centric relation and centric occlusion
- The patient's soft tissue profile in both centric relation and centric occlusion
- Any mandibular shift, by holding the mandible to the most retruded position and checking the incisor relationships
- Hereditary background, by checking for the possibility of similar traits in the

patient's parents or siblings

Cephalometric evaluation

Since 1931 and the introduction of cephalometric radiographs, several cephalometric analyses and measurements have been proposed for diagnosis and treatment planning in orthodontic treatment. However, it is not the intention of this publication to discuses the special methods or analysis to be used for diagnosis and treatment planning in early orthodontic treatment. The main purpose of this section is to emphasize and remind the reader of the importance of application of the cephalometric technique, not only in diagnosis and treatment planning but also in growth prediction in early-age treatment.

Simple Dental Crossbite

Simple anterior dental crossbite is defined as a dental malocclusion that results from lingual positioning of the maxillary anterior teeth in relation to the opposing mandibular anterior teeth. The condition is localized and involves the tipping of only a tooth or teeth and not the basal bone. In other words, simple dental crossbite is due to an abnormal labiolingual relationship between one or more maxillary and mandibular incisors within the context of a normal anteroposterior skeletal jaw relationship. Single dental crossbite usually results in no mandibular shift from rest to occlusion, exhibiting a smooth path of closure to an Angle Class I relationship. When more incisors are involved, some mandibular anterior shift may occur.

Simple dental crossbite is the most common kind of anterior crossbite seen during the primary and early mixed dentitions. Reports show a frequency of 3% to 12%. This significant variation of incidence is due to differences in reports from one ethnic group to another. For example, in a Japanese population the reported incidence was 10%, while in a US population the reported incidence was 3%.[48]

In these types of anterior crossbite, one or more of the maxillary anterior teeth occlude lingually to the mandibular incisors when the teeth are in centric occlusion. Crossbites usually do not self-correct with age. As Tausche et al[48] reported, anterior crossbites show progression in severity in the permanent dentition.

Characteristic signs

As already mentioned, patients with dental crossbite have a normal ANB angle and profile. The maxillary incisors are retroclined, and mandibular incisors are proclined, but the molars and canines show Class I relationships in both centric relation and centric occlusion.

There are two types of anterior dental crossbite:

1. Crossbite of a single or multiple incisors with lingual tipping and usually no crowding and mandibular shift.
2. Locked anterior crossbite in which one or two incisors are tipped lingually and some are labial to the mandibular incisors. Some crowding is present. No shift is present, but maxillary and mandibular incisors are locked within each other, and the anterior occlusion is usually more traumatized.

Etiology

The majority of anterior dental crossbites are caused by local dental factors. Many etiologic factors have been reported, such as:

- Congenital abnormal path of eruption (lingual eruption of maxillary anterior incisors)
- Trauma to the primary incisors that has led to displacement of the permanent tooth buds
- Direct trauma to the permanent incisors that has caused luxation and displacement
- Overretained primary teeth in the maxillary incisor region that have caused palatal eruption of the permanent incisor or overretained mandibular primary incisors that have increased the proclination of the mandibular incisors
- Labially positioned supernumerary tooth or odontoma
- Sclerotic bony or fibrous tissue barrier caused by premature loss of a primary tooth
- Rarely, a habit of biting the upper lip
- Overretained necrotic or pulpless primary tooth or root
- Crowding in the incisor region (Bolton discrepancy)
- Arch length inadequacy
- Repaired cleft lip or palate

Figure 11-11 illustrates an anterior crossbite in the middle mixed dentition caused by one overretained mandibular primary incisor that created a longer arch circumference in the anterior region of the mandibular dentition and consequently an end-to-end relationship of the incisors and crossbite.

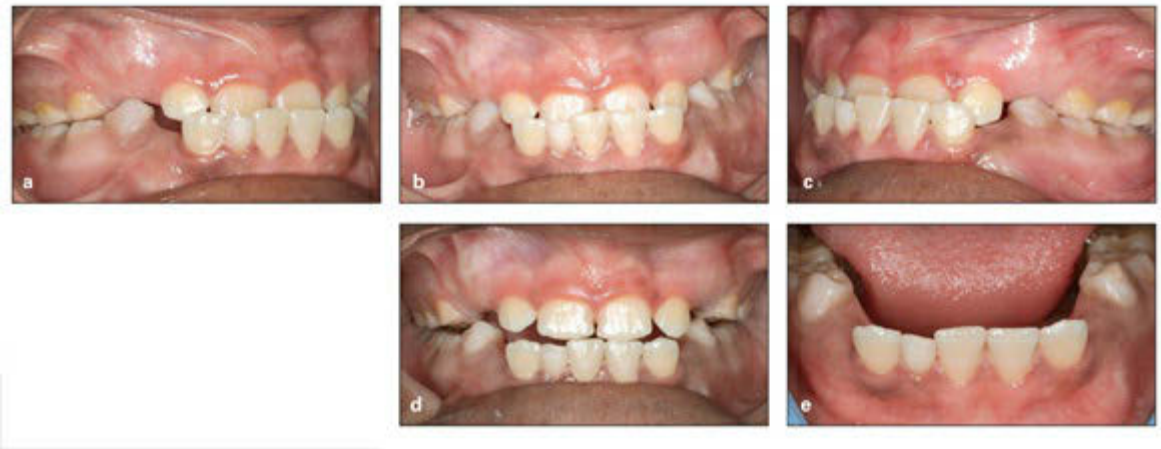


Fig 11-11 *(a to e)* Anterior crossbite resulting from overretained primary incisors that have created a longer arch circumference.

Figure 11-12 shows two overretained primary central incisors that have caused severe separation and delayed eruption of the permanent central incisors. The ultimate result was creation of a severe diastema, displacement of the permanent incisors, and development of anterior crossbite.

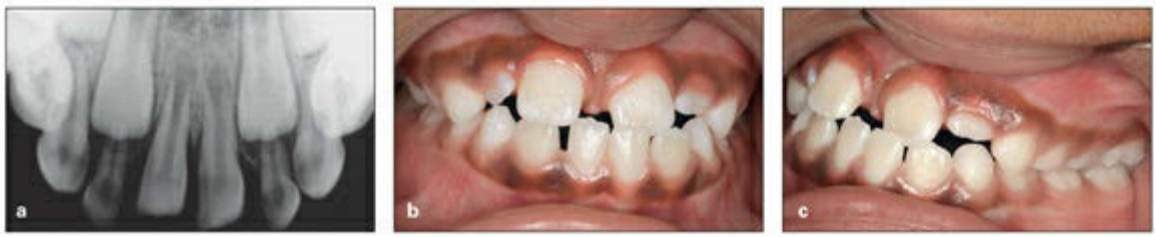


Fig 11-12 *(a)* Overretained primary incisors delaying the eruption of the permanent incisors. *(b and c)* Anterior crossbite and severe diastema resulting from the neglected, overretained primary incisors.

Figures 11-13 and 11-14 illustrate overretained primary incisors that have caused palatal eruption of the permanent incisors and crossbite.

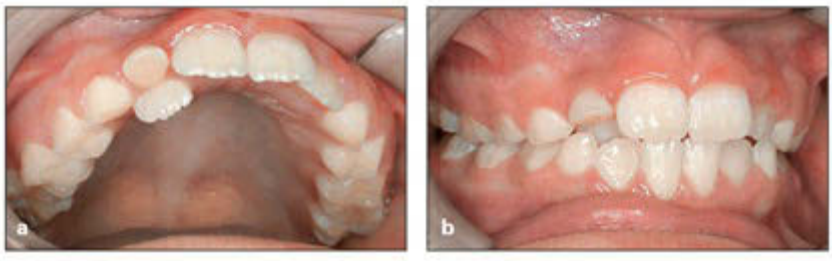


Fig 11-13 *(a and b)* Palatal eruption and crossbite of the permanent incisor caused by an overretained primary lateral incisor.

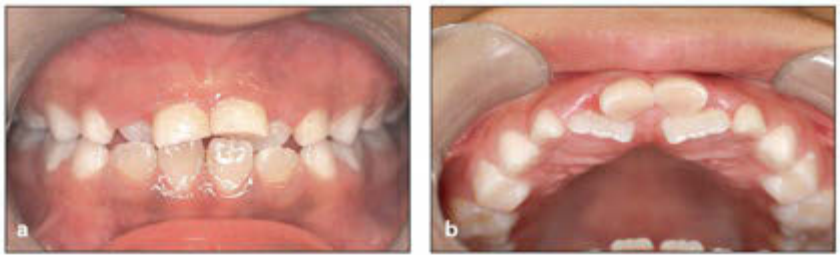


Fig 11-14 *(a and b)* Crossbite of the permanent incisors caused by overretained primary central incisors.

Figure 11-15a shows incisor crossbite that developed because both maxillary permanent canines were missing, causing diastema and displacement of the maxillary right central and lateral incisors. Figures 11-15b and 11-15c demonstrate the occlusion after orthodontic treatment.

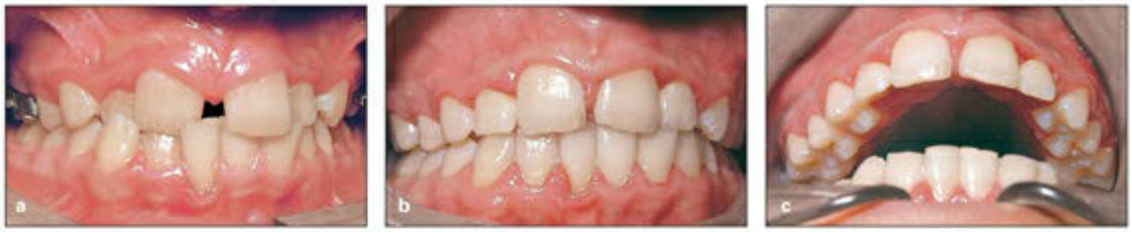


Fig 11-15 *(a)* Dental crossbite resulting from the congenital absence of the permanent canines, which has created a diastema and displaced the maxillary right central and lateral incisors. *(b and c)* Posttreatment occlusion.

Figure 11-16 demonstrates single maxillary central incisor crossbite that resulted from trauma to the primary incisor that occurred about 3 years before referral.

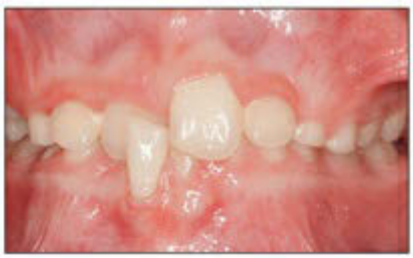


Fig 11-16 Crossbite of the maxillary central incisor caused by trauma to the primary central incisor 3 years prior to referral.

Advantages of early treatment

As already mentioned, anterior dental crossbite requires early and immediate

treatment to prevent serious problems that can result from delayed correction. Proper treatment during an early stage of the dentition is easier to achieve and less complicated and can prevent many harmful sequelae to the teeth, supporting structures, and jaw growth.

Before eruption, the maxillary permanent incisors are located behind the primary incisor roots. The maxillary permanent lateral incisors are located distal to and slightly behind the permanent central incisors; any displacement of one permanent incisor during eruption can affect the adjacent incisors; thus, early correction is recommended even for single dental crossbite. If radiographs indicate possible lingual eruption of the adjacent teeth, the finger spring of the interceptive appliance can be designed to provide eruption guidance for the next erupting incisor.

Many studies confirm the advantages of early correction of incisors crossbite.[49–52] One immediate result of early treatment of this malocclusion is increased anterior arch circumference, which corrects anterior crowding and facilitates incisor and canine eruption. The following are additional advantages:

- Improvement of dental esthetics
- Prevention of structural damage such as dental attrition, periodontal disease, and root resorption
- Prevention of dental compensation and redirection of skeletal growth modification
- Prevention of growth interferences that cause maxillary deficiency
- Improvement of the dentoalveolar relationship
- Increase in anterior arch perimeter

Consequences of delayed treatment

Postponing treatment of simple dental crossbite can have serious repercussions. Anterior dental crossbite in the primary dentition is often transferred to the permanent dentition; postponing the treatment results in prolonged and more complex treatment during the permanent dentition.[48]

Many reports have indicated that delayed correction of this anomaly can cause abnormal enamel abrasion; anterior tooth mobility and fracture; labial alveolar plate resorption and gingival recession; periodontal pathosis; reduction of anterior arch circumference, which can result in canine impaction; and temporomandibular joint disturbances.[49,53–56] In addition, dentoalveolar and even skeletal Class III malocclusion can develop due to mandibular displacement or maxillary

underdevelopment. Functional shifts cause asymmetric muscle strain and condylar displacement and consequently asymmetric mandibular growth.

Early treatment strategies

Early treatment of this abnormality is a must; the main goal of early intervention and unlocking the teeth is to stimulate well-balanced growth and occlusal development. Early treatment is also an easy and efficient procedure, and treated patients whose malocclusion is not accompanied by crowding or rotation do not need retention.

Various treatment methods have been proposed to correct anterior dental crossbite, including tongue blades, reversed stainless steel crowns, fixed acrylic resin inclined planes, bonded composite resin slopes, removable acrylic resin appliances with finger springs, and 2 × 4 bonding. Generally there are two types of appliance (fixed or removable) for correction of this anomaly. The decision regarding which appliance to use is based on the type of problem and the patient's hygiene and compliance.

Hawley appliances with anterior finger springs and occlusal coverage are appropriate removable appliances for cooperative children. Noncompliant patients are best treated with 2 × 4 bonding and occlusal composite resin to disocclude the anterior teeth. The best treatment option for patients with anterior dental crossbite, crowding, and rotation is 2 × 4 bonding.

During correction of anterior crossbite, the involved teeth must be disoccluding to prevent trauma and to facilitate jumping of the tooth or teeth in crossbite. This can be achieved by adding occlusal coverage to the removable appliance or by bonding composite resin to the molars in the 2 × 4 bonding approach.

Case 11-8: Anterior dental crossbite

A 10-year-old girl presented with a single-incisor crossbite with sufficient space. A few months' delay in early intervention caused mandibular trauma and bone recession (Figs 11-17a to 11-17d).

Treatment:
Because there was no crowding, rotation, or any displacement of teeth adjacent to the involved incisor as well as good patient compliance, she was treated with a removable Hawley appliance with occlusal coverage and a single finger spring (Figs 11-17e and 11-17f).

Ten months posttreatment, the incisors were well positioned, the permanent canines and premolars were erupting, the bone recession at the mandibular incisors had self-corrected, and no further treatment was required (Figs 11-17g and 11-17h).

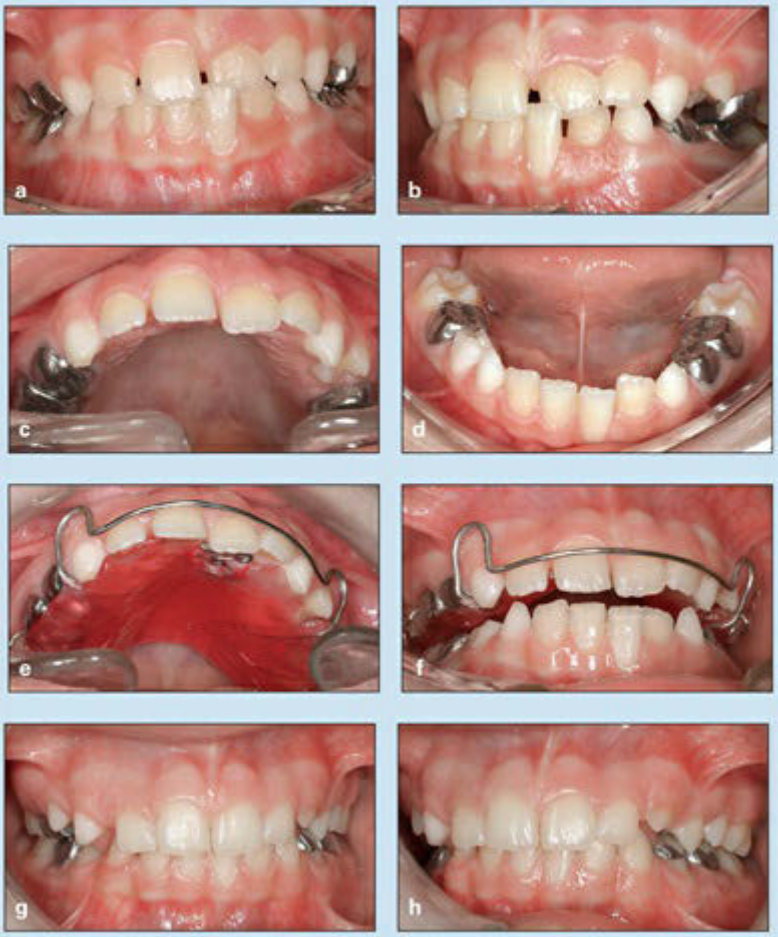


Fig 11-17 Management of crossbite of a single incisor that had sufficient space for correction. A short delay in intervention resulted in mandibular incisor crowding, trauma, and bone recession in the 10-year-old girl. *(a to d)* Pretreatment occlusion. *(e and f)* Hawley appliance with simple occlusal coverage and finger spring. *(g and h)* Posttreatment occlusion 10 months after treatment. No further treatment is required.

Case 11-9: Anterior dental crossbite

A 10-year-old girl in the middle mixed dentition presented with a Class III molar relationship on the right side because of space loss, 0- to 1-mm overbite and overjet, and three maxillary incisors in crossbite. Treatment had been delayed, causing severe crowding of the mandibular incisors and ectopic eruption of the mandibular right lateral incisor (Figs 11-18a to 11-18f).

Treatment:
Because of the severe crowding and displacement of incisors, the treatment plan incorporated fixed appliances with maxillary and mandibular 2 × 6 bonding. The first step in treatment was 2 × 4 maxillary bonding, mandibular first molar occlusal bonding to disocclude the anterior segment, and placement of 0.016-inch nickel-titanium maxillary arches (cinched back) for leveling and release of abnormal anterior contact. The second step was placement of 0.016-inch stainless steel maxillary arches with an open U-loop mesial to the molar tube (extended arch length) to procline the maxillary incisors out of crossbite. The third step was mandibular 2 × 4 bonding: first with 0.014-inch nickel-titanium archwire because of severe crowding and later with 0.016-inch nickel-titanium archwire for further leveling.

The fourth step was use of an open U-loop to place an extended-length stainless steel archwire against the mandibular molar tube to achieve minor mandibular incisor proclination in order to gain space and align the mandibular incisors. The final step was bonding the permanent canines after eruption for final anterior alignment. Figures 11-18g to 11-18k show the treatment outcome.

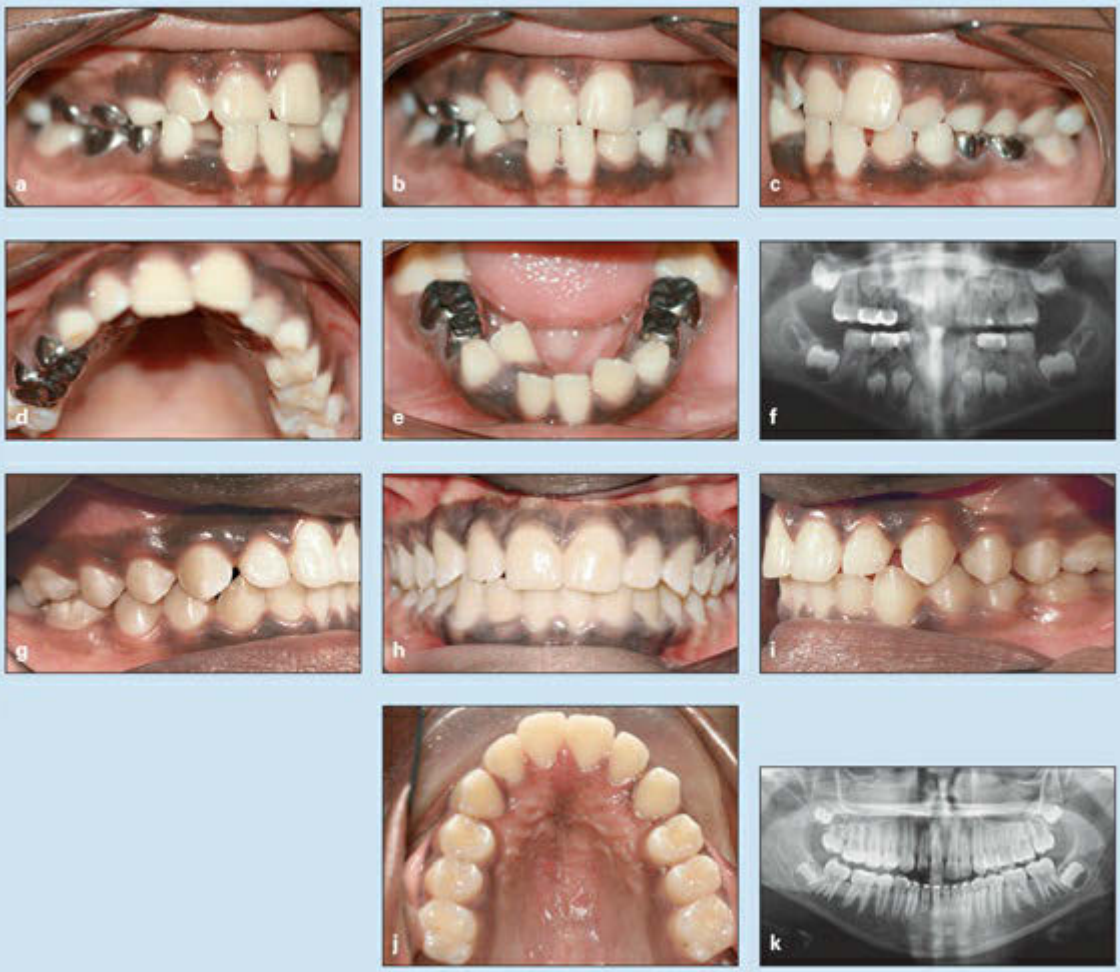


Fig 11-18 Management of incisor cross-bite in a 10-year-old girl. The locked occlusion has resulted in severe displacement and crowding of the mandibular incisors as well as ectopic eruption of the mandibular right central incisor. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g to j)* Post-treatment occlusion. *(k)* Posttreatment panoramic radiograph.

Case 11-10: Anterior dental crossbite

A 10-year, 6-month-old boy presented with Class I malocclusion, anterior locked occlusion, traumatized occlusion, and three incisors in crossbite. The crossbite had resulted in crowding of the maxillary and mandibular incisors as well as displacement and rotation of the maxillary incisors (Figs 11-19a to 11-19e).

Treatment:

The treatment plan called for maxillary and mandibular 2 × 6 bonding and placement of occlusal composite resin on the mandibular molars to disocclude the anterior segment. Treatment was started with 2 × 4 maxillary bonding of 0.016-inch nickel-titanium wire for incisor alignment. Occlusal composite resin buildup was used to disocclude the incisors. A 0.016-inch stainless steel maxillary

archwire with open U-loops was placed to procline the maxillary incisors out of crossbite (Figs 11-19f and 11-19g).

After crossbite correction was accomplished, mandibular 2 × 4 bonding of a 0.016-inch nickel-titanium arch was used to align the anterior teeth. The final step was bonding of the maxillary and mandibular permanent canines for final adjustment (Figs 11-19h to 11-19k).

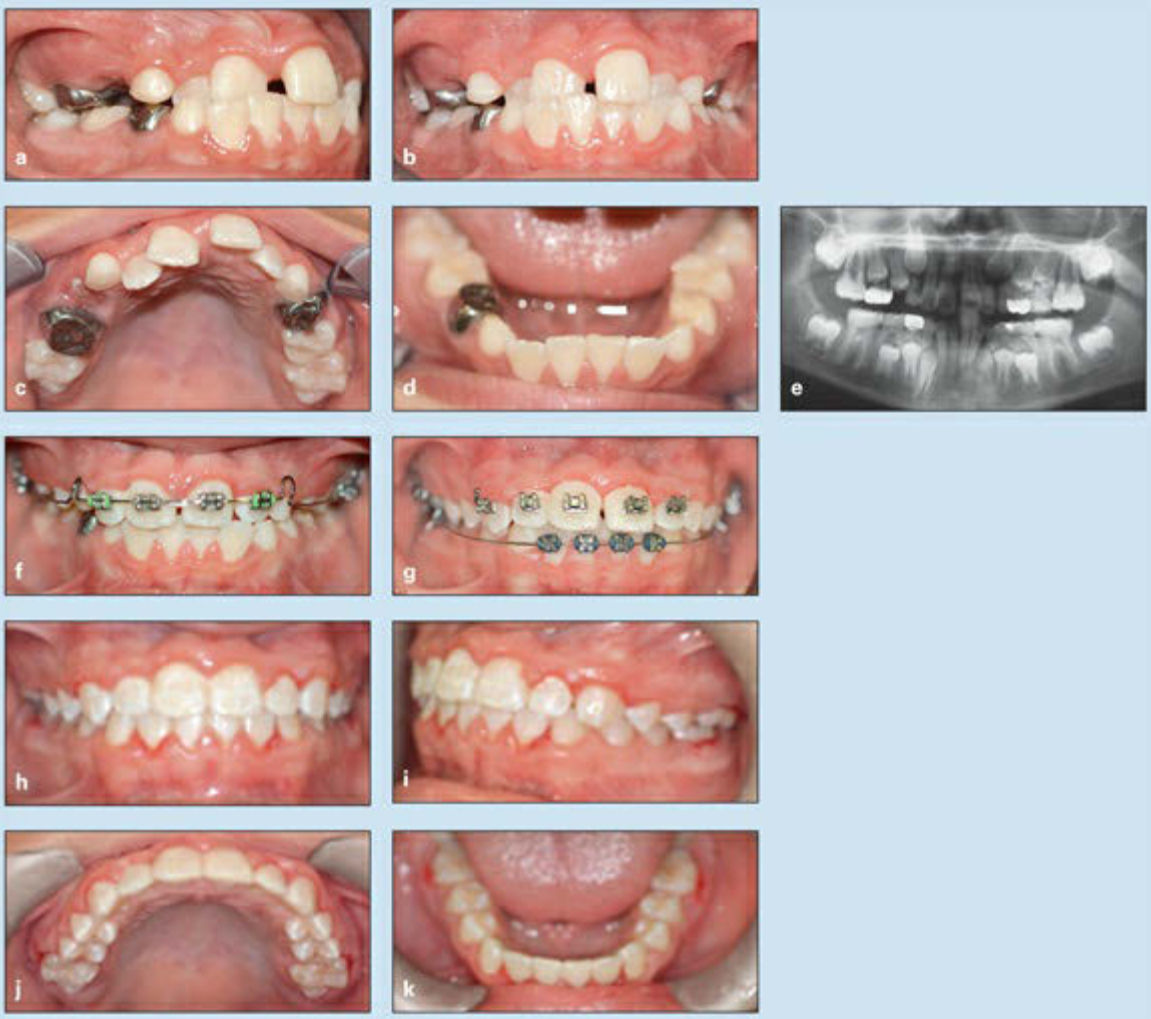


Fig 11-19 Management of anterior locked, traumatized occlusion and incisor crossbite in a 10-year, 6-month-old boy. *(a to d)* Pretreatment occlusion. *(e)* Pretreatment panoramic radiograph showing crowding of the maxillary incisors and canines. *(f and g)* Phases of mechanotherapy. *(h to k)* Posttreatment occlusion.

Pseudo–Class III Malocclusion (Functional Crossbite)

Functional crossbite is a multitooth anterior crossbite. The interferences prevent posterior occlusion, resulting in a functional shift of the mandible in an effort to avoid the anterior interference in centric relation and to achieve maximal intercuspation. Pseudo–Class III malocclusion can involve both the permanent teeth and the primary dentition. Other terms that have been used to describe this anomaly

include *pseudoprognathism*,[57,58] *postural Class III*,[50] and *functional Class III*.[49] These anomalies are usually characterized by a Class I skeletal pattern. Sometimes, especially if treatment has not been performed early, a minor maxillary deficiency or constricted palate may be present. These patients usually show a straight profile at rest position (centric relation) and a slightly concave profile at centric occlusion.

Rabie and Gu,[59] in a study designed to identify the diagnostic criteria for pseudo–Class III in comparison with Class I malocclusion in a southern Chinese population, examined 36 patients with pseudo–Class III malocclusion and 31 patients with Class I malocclusion. They found that patients with a pseudo–Class III malocclusion exhibit certain morphologic, dental, and skeletal characteristics, such as retroclined maxillary incisors, retrusive upper lip, decreased midfacial length, and increased maxillomandibular differences.

In regard to criteria for distinguishing between pseudo– Class III and skeletal Class III malocclusion, Ngan et al[52] stated that cephalometric evaluation may not be the most reliable tool in differential diagnosis. The most consistent findings seem to be the dental characteristics of Angle Class III molars and canines, retroclined mandibular incisors, and the presence of an end-to-end or anterior cross-bite occlusion.

Gu[60] attempted to clarify the dentoskeletal characteristics of pseudo–Class III malocclusion during the mixed dentition by comparing 36 patients with pseudo–Class III malocclusion (mean age: 10.7 ± 2.0 years) and 40 patients with skeletal Class III malocclusion (mean age: 9.7 ± 2.2 years). All subjects were followed after the growth spurt; cephalograms were taken in the mixed dentition for subjects with pseudo–Class III malocclusion, subjects with skeletal Class III malocclusion, and a control group of Class I subjects, and dentoskeletal characteristics were compared. Gu[60] concluded that pseudo–Class III malocclusion is characterized by decreased midfacial length, mandibular displacement, retroclined maxillary incisors, and normal vertical development.

Moyers[61] proposed that the pseudo–Class III relationship is a positional malocclusion with an acquired neuromuscular reflex and considered the hypothesis that the positional relationship in apparent Class III malocclusion may result from an early interference with the muscular reflex of mandibular closure.

Several other investigators evaluating pseudo–Class III malocclusion also attributed the retroclined maxillary incisors and proclined mandibular incisors to incisor interference.[50,57,62,63]

Characteristic signs

Evaluation of pseudo–Class III malocclusion always requires that anterior and posterior dental relationships be examined with the mandible in centric relation. The following are general signs of pseudo–Class III malocclusion that can be recognized by careful clinical and cephalometric evaluation:

- Multitooth anterior crossbite
- Normal ANB angle at rest
- Some maxillary incisor retroclination
- Minor maxillary deficiency
- Normal to concave profile (appearing normal at centric relation and slightly concave at habitual occlusion)
- Class I molar and canine relationships in centric relation
- Habitual shift from rest to occlusion

Advantages of early treatment

When multiple incisors are involved in a pseudo–Class III malocclusion and a functional shift is present, urgent intervention is needed to prevent structural damage and adverse effects on the maxilla. Unlocking the maxilla before the growth spurt normalizes maxillary growth, improving the profile and preventing structural damage. Early treatment of this kind of anterior crossbite, like anterior dental crossbite, is easy and efficient. Delayed treatment may necessitate much more comprehensive management.

Early correction of this malocclusion eliminates the mandibular shift, unlocks the maxillary incisors and the incisive bone to enhance maxillary growth for better function and esthetics, and preserves and facilitates eruption of the canines by increasing anterior arch length. Gu and Rabie[64] studied 21 consecutively treated patients who had a pseudo–Class III malocclusion and 15 untreated control subjects. They found that, on average, 4.7 mm of space was gained as a result of the treatment, whereas lack of space was evident in the untreated control subjects.

Early orthodontic intervention for pseudo–Class III malocclusion is always recommended to prevent existing problems from worsening and to prepare a better environment for occlusal development, thereby minimizing or eliminating the need

for comprehensive orthodontic treatment later.

Consequences of delayed treatment

Delayed treatment and collapse of the maxillary dentition can cause A-B discrepancy and maxillary retrusion, which may require comprehensive full bonding and face mask therapy.

Delayed treatment can cause structural damage to the involved teeth and supporting structures, crowding in the anterior region, and even canine impaction and adverse effects on jaw growth51,65 (see case 11-13).

Early treatment strategies

Depending on the severity of the problem and the patient's age and compliance, correction may be achieved with removable, functional, or fixed appliances. When maxillary protraction is needed, extraoral devices such as a face mask or chin cap might also be used.

The methods described for management of simple anterior dental crossbite (removable Hawley appliance with occlusal coverage and 2 × 4 bonding) can also be used for correction of most pseudo–Class III malocclusions. In pseudo–Class III malocclusion, multiple incisors are involved and the crossbite often is accompanied by crowding and rotation. Application of 2 × 4 bonding is preferable; the result is faster, the patient's compliance is not required, and there is the possibility of incisor alignment after crossbite correction. Patients with some maxillary deficiency resulting from delayed treatment or hereditary factors may need extraoral traction.

Removable appliances for correction of pseudo–Class III malocclusions, which usually involve multiple incisors in crossbite, have special specifications to maintain sufficient retention. The labial bow is separated from the incisors to allow labial movement of the maxillary incisors that are in crossbite. Therefore, retention must be established from the posterior segments by a long labial bow (Fig 11-20; see case 11-14), or the appliance must have a special horizontal loop on the canines, as was designed in 1977[66] (Fig 11-21).

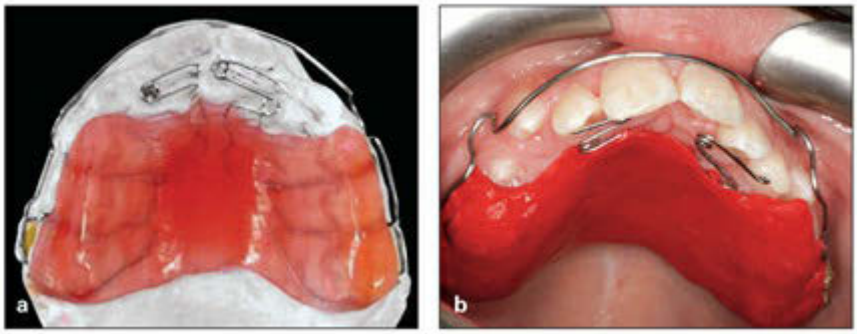


Fig 11-20 *(a and b)* Modified Hawley appliance with a long labial bow soldered to the Adams clasp. The long labial bow establishes retention in the posterior segment, which separates from the anterior segment to allow labial movement.

Fig 11-21 Removable appliance designed for crossbite correction of four incisors. It has a horizontal loop on the canines for retention.

Case 11-11: Pseudo–Class III malocclusion

A 9-year-old boy presented with pseudo–Class III malocclusion, mandibular anterior shift, and three incisors in crossbite, which were causing displacement and rotation of the maxillary incisors and crowding in the mandibular incisor region (Figs 11-22a to 11-22e).

Treatment:
Because of financial considerations and in accordance with the parent's wishes, treatment was accomplished with a removable appliance (Fig 11-22f). Posttreatment results are shown in Figs 11-22g to 11-22i.

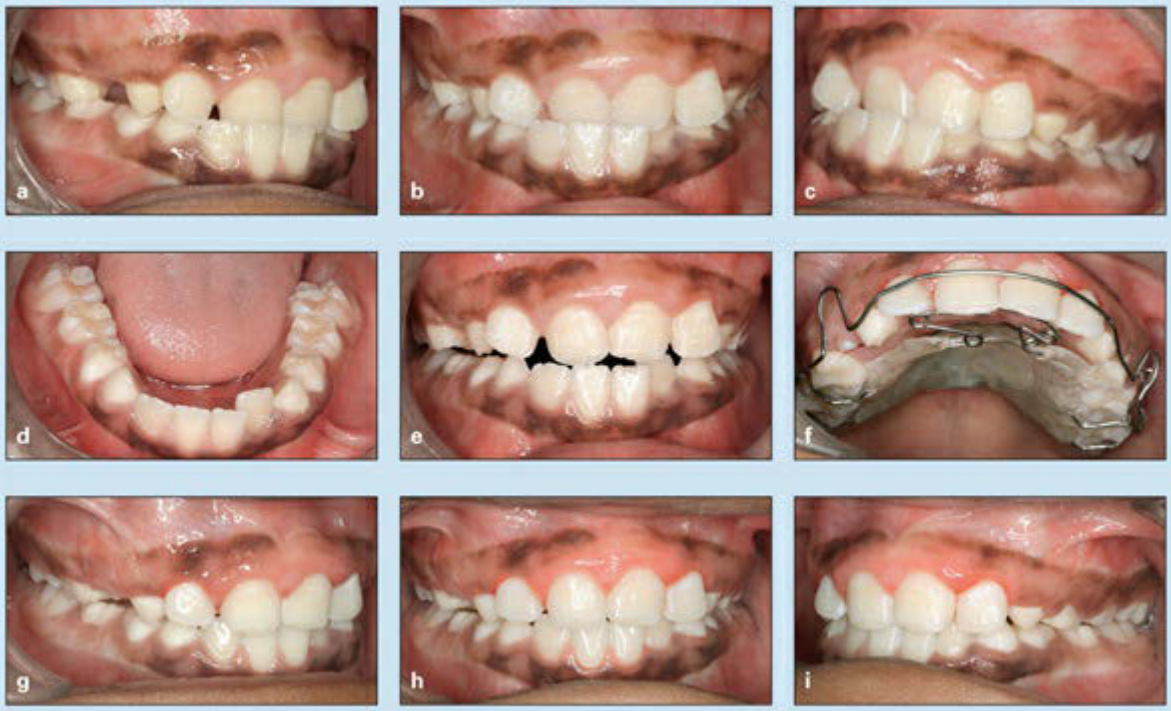


Fig 11-22 Management of a pseudo–Class III malocclusion with mandibular anterior shift. Three incisors are in crossbite. *(a to d)* Pretreatment occlusion in centric occlusion. *(e)* Pretreatment occlusion in centric relation. *(f)* Removable appliance used for treatment. *(g to i)* Posttreatment occlusion.

Case 11-12: Pseudo–Class III malocclusion

An 11-year, 7-month-old boy in the late mixed dentition presented with a Class I malocclusion, pseudo–Class III malocclusion, and a severe anterior mandibular shift (Figs 11-23a to 11-23e).

Treatment:
The treatment plan in this case was limited to 2 × 6 maxillary bonding and placement of composite resin on the occlusal surfaces of the molars to disocclude the anterior segment. No treatment was performed in the mandibular dentition. Figures 11-23f to 11-23n show the treatment outcome. Early unlocking of the incisor crossbite not only prevented structural damage and maintained sufficient space for the adjacent teeth but also allowed normal jaw growth, which improved the profile.

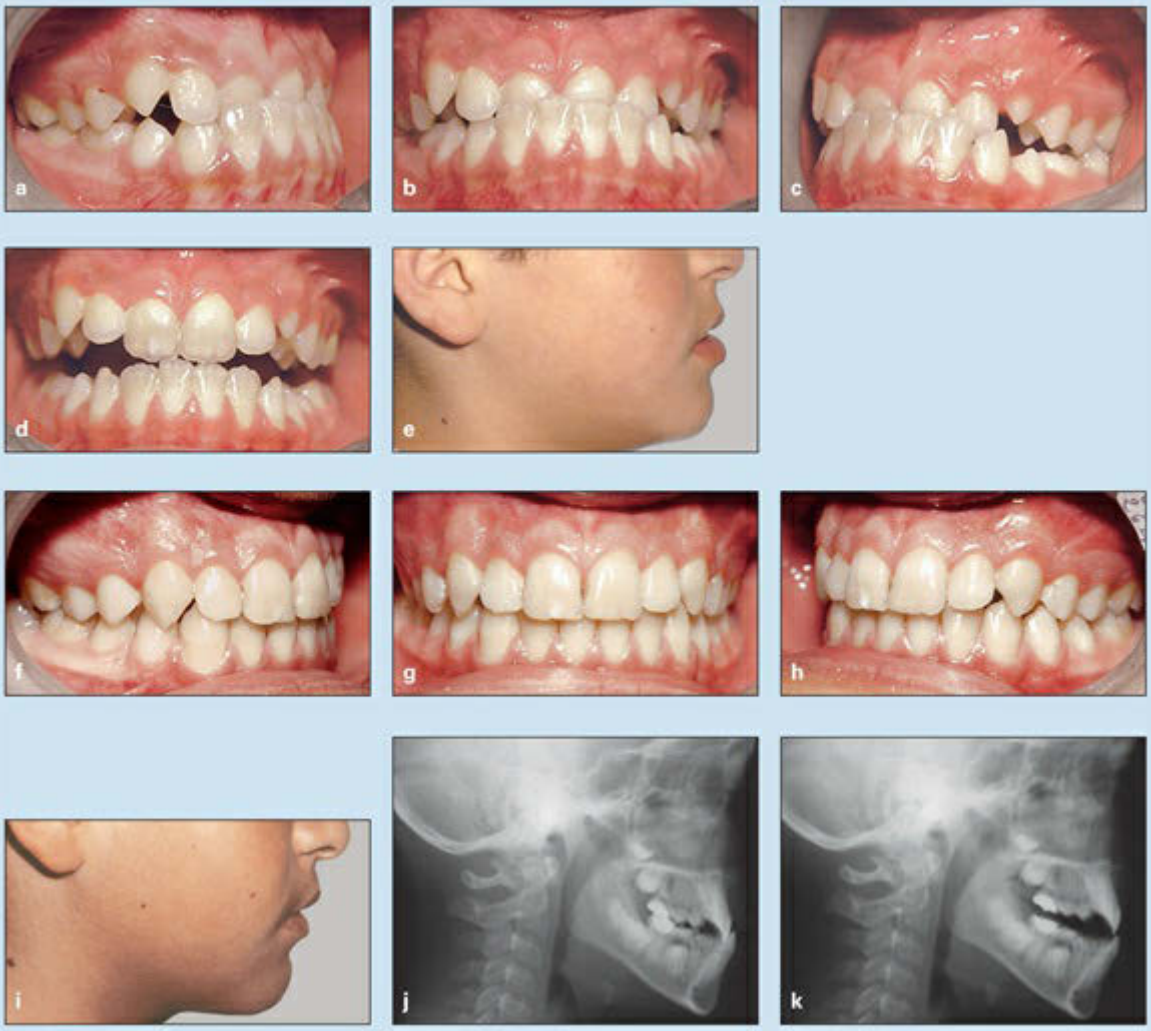
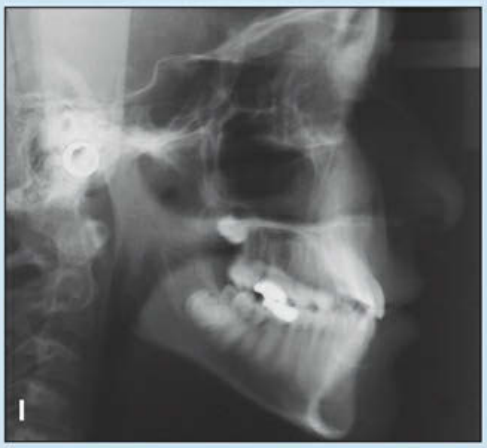
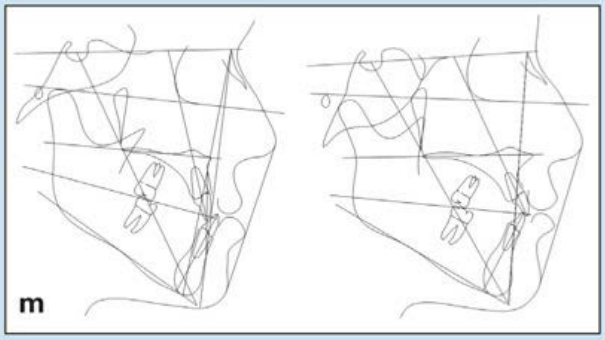


Fig 11-23 Management of a pseudo–Class III malocclusion with severe anterior shift in an 11-year, 7-month-old boy in the late mixed dentition. *(a to c)* Pretreatment occlusion in centric occlusion. *(d)* Pretreatment occlusion in centric relation. *(e)* Pretreatment soft tissue profile. *(f to h)* Posttreatment occlusion. *(i)* Posttreatment soft tissue profile. *(j)* Pretreatment cephalometric radiograph in centric occlusion. *(k)* Pretreatment cephalometric radiograph in centric relation.





n

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	88.7	88.2
SNB (°)	78.0	82.0	82.1
SNA (°)	82.0	77.8	82.0
Maxillary depth (FH-NA) (°)	90.0	85.2	88.1
ANB (°)	2.3	–4.2	-0.1
FMA (MP-FH) (°)	25.9	29.5	28.0
Y-axis (SGn-SN) (°)	59.4	65.7	64.2
Interincisal angle (U1-L1) (°)	135.0	139.1	127.9
U1-FH (°)	116.2	109.0	117.0
IMPA (L1-MP) (°)	95.0	82.4	87.1
Overbite (mm)	2.5	4.0	1.5
Overjet (mm)	2.5	4.1	1.8

Fig 11-23 *(cont) (l)* Posttreatment cephalometric radiograph. *(m)* Pretreatment *(left)* and posttreatment *(right)* tracings. *(n)* Changes in cephalometric measurements.

Case 11-13: Pseudo–Class III malocclusion

A 17-year-old girl presented with a Class I occlusion, anterior crossbite, and a mandibular shift. Her chief complaints were temporomandibular dysfunction, pain, and restricted opening. She also had severe structural damage to the maxillary and mandibular incisors and periodontium (Figs 11-24a to 11-24d). This case clearly demonstrates the consequences of delayed treatment of anterior crossbite.

Treatment:
After scaling and periodontal care were provided, orthodontic treatment was accomplished with 2 × 6 maxillary and mandibular bonding. Occlusal composite resin buildup was used for anterior disocclusion. Anterior cross-bite correction and midline alignment were accomplished successfully, but because of a lack of early treatment and some mandibular overgrowth, the mandibular incisors were also retracted with lingual root torque according to available space.

Some interdental spacing remained in the maxillary incisors as a result of some microdontia, and there was some crown damage; therefore, the maxillary anterior segment (canine to canine) was restored with metalcermic crowns (Figs 11-24e to 11-24g).

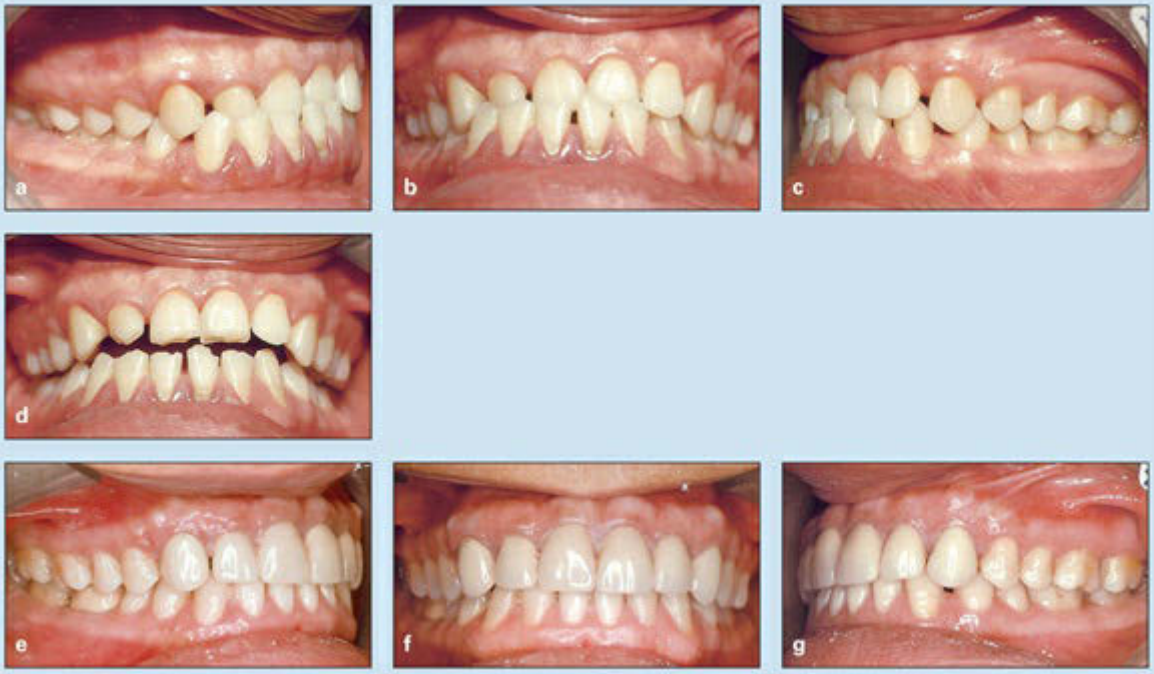


Fig 11-24 Management of anterior crossbite and mandibular shift in a 17-year-old girl. Neglect has resulted in restricted opening, temporomandibular joint pain, and structural damage to the mandibular incisors and periodontium. *(a to d)* Pretreatment occlusion. *(e to g)* Posttreatment occlusion after 2 × 6 bonding and placement of crowns on the maxillary anterior teeth.

Case 11-14: Pseudo–Class III malocclusion

An 11-year, 7-month-old girl presented with a Class I anterior pseudo–Class III malocclusion with mandibular shift, minor damage to the maxillary incisors, and minor crowding in the maxillary arch (Figs 11-25a to 11-25c).

Treatment:
Treatment was accomplished with a removable Hawley appliance that had two Adams clasps. A long labial bow, separated from the incisors to facilitate their proclination, was soldered to the Adams clasps to maintain retention from the posterior segments (see Fig 11-20). Figures 11-25d and 11-25e show the posttreatment occlusion.

Comparison of this case with previous cases, in which treatment was neglected for a period of time, clearly demonstrates the difference in complexity of treatment procedures and the damage that can occur to the teeth and supporting structures if early treatment is not provided.

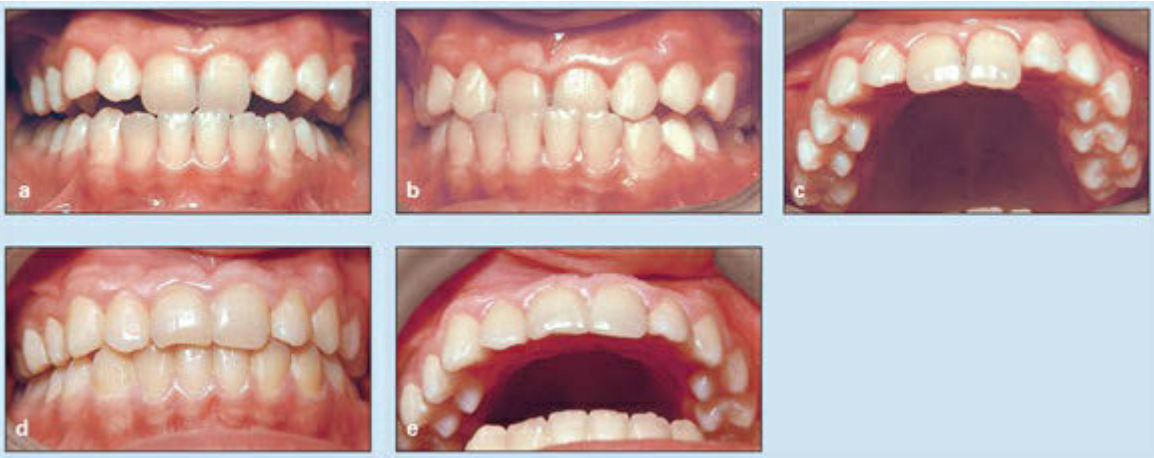


Fig 11-25 Management of a pseudo–Class III malocclusion with mandibular shift that has caused minor damage to the maxillary incisors in an 11-year, 7-month-old girl. *(a)* Pretreatment occlusion in centric occlusion. *(b)* Pretreatment occlusion in centric relation. *(c)* Retroclination of the maxillary incisors. *(d and e)* Posttreatment occlusion.

Skeletal Class III and Potential Class III Malocclusion

True Class III malocclusion, or *mesial occlusion*, is an anteroposterior dentoalveolar or skeletal malrelationship characterized by a mandibular dentition that has a more anterior position than the maxillary dentition.[67] This anomaly can be caused by mesial mandibular basal or dentoalveolar positioning, distal maxillary basal or dentoalveolar positioning, basal bone size discrepancy (either shorter maxilla or longer mandible), or a combination of any of these factors.

Skeletal Class III malocclusion can also be caused by an aberration in other bony structures, such as underdevelopment of the anterior cranial length, underdevelopment of the posterior cranial length, and divergence or convergence of some angles, such as the saddle angle, articular angle, y-axis, gonial angle, or maxillary and mandibular length. It can also be caused by variations in dental relationships without any underlying skeletal malrelationship.

Ngan et al[68] explained that patients with skeletal Class III malocclusion exhibit a concave facial profile, a retrusive nasomaxillary area, and a prominent mandibular third of the face. The lower lip is often protruded relative to the upper lip. The maxillary arch is usually narrower than the mandibular arch, and overjet and overbite can range from reduced to reverse. Therefore, occlusal relationships in patients with Class III malocclusion constitute only one part of a much larger syndrome. Proper treatment planning requires a thorough clinical assessment and cephalometric analysis.

Any of the aforementioned skeletal combinations can be, but are not always, accompanied by a mandibular shift or anterior crossbite. Therefore, the first step in recognition and treatment planning starts with accurate differential diagnosis of true Class III malocclusion from other kinds of anterior crossbite.

Developing Class III malocclusion in growing patients is one of the most challenging problems confronting clinicians. Early orthodontic intervention is recommended to encourage normal dentoalveolar and skeletal growth. Class III malocclusions are usually growth-related discrepancies; delayed intervention often develops into a more severe abnormality until growth is complete. Angle[69] reported Class III malocclusion in 3.4% of his sample of 1,000 white patients.

It can be assumed that delayed treatment of a pseudo– Class III abnormality can allow it to change to a skeletal deformity later. This is the situation the author calls *potential Class III malocclusion*. This is not true in all cases of pseudo–Class III malocclusion, but clinically the author has observed some untreated parents or relatives with more severe skeletal deformity while their children or siblings with pseudo–Class III malocclusion were treated easily.

Treatment options for this malocclusion depend on the dentoskeletal age of the patient and the morphologic characteristics of the malocclusion. Early treatment approaches include growth modification and dental camouflage. However, when growth has ceased, orthognathic surgery is the only choice. Depending on the amount of skeletal discrepancy, surgical correction may consist of mandibular setback, maxillary advancement, or a combination of mandibular and maxillary procedures.

This type of malocclusion can have genetic or environmental origins or a combination of both.

Class III malocclusions initiate and develop during the primary or early mixed dentition and are recognizable at early ages.

Dental and skeletal Class III malocclusions must be carefully differentiated. There are special situations that must be distinguished:

- Sometimes in patients with true Class III malocclusion with incisal interference, proclination of the mandibular incisors, and retroclination of the maxillary incisors, a mandibular mesial shift causes posturing of the mandible in an anterior position that can be misleading, causing a mistaken diagnosis of pseudo–Class III malocclusion. Careful clinical and cephalometric evaluations can identify true Class III malocclusion with skeletal discrepancy.
- In some patients with true Class III malocclusion with skeletal discrepancy,

especially mandibular prognathism, no mandibular shift is present, and the incisors are in and end-to-end position. This is indicative of a more severe malocclusion that may need orthognathic surgery.

- Patients with dental characteristics of Angle Class III molars and canines, retroclined mandibular incisors, and an end-to-end or anterior crossbite occlusion without a mandibular shift have true skeletal Class III malocclusion. These patients require a comprehensive orthodontic or orthosurgical approach.

Originally, it was believed that Class III malocclusions arise primarily from an overdevelopment of the mandible (mandibular prognathism). The fact that 33 of 40 members of the Hapsburg family had prognathic mandibles was considered to be a classic example of this anomaly. Now, many reports have indicated that maxillary retrusion contributes to the anomaly in up to 60% of cases.[57]

The orthopedic effect of face mask use and maxillary sutural opening in early treatment of patients with Class III malocclusion, especially those with maxillary deficiency, can correct anterior crossbite and facilitate normal maxillary growth in adaptation and synchronization with the mandible during the growth period. Late adolescent treatment and treatment when growth changes will be limited minimize these adaptations and eventually result in unsuccessful treatment.

Early treatment of Class III malocclusion with mandibular prognathism seems to be much more complicated, with minimal success and frequent relapse, when started during the pubertal growth period. Proffit[57] stated that early treatment of patients with Class III malocclusion depends on whether the problem is the result of mandibular prognathism or deficiency in maxillary growth. He also stated that restraining mandibular growth at an early stage rarely succeeds; later mandibular growth wipes out any early correction. On the other hand, face mask treatment to modify growth in children with maxillary deficiency succeeds if, and only if, the treatment is completed early.[57]

The author has found good results in early intervention during the primary dentition and correction of Class III malocclusion with mandibular prognathism if normal incisor relationships are achieved at an early age. Establishment of normal incisor relationships during the primary dentition usually allows the maxilla to continue growth stimulation and synchronize with mandibular growth, evidently with stable results (see cases 11-19 to 11-23).

Treatment for skeletal Class III malocclusion with mandibular prognathism during the early permanent dentition or even during the late mixed dentition is usually unsuccessful and has to be continued with comprehensive orthodontic and

orthognathic surgical procedures.

Other variations of skeletal Class III malocclusion accompanied by severe vertical problems are more complicated and require comprehensive treatment and possibly two-jaw surgery later. The author postpones orthodontic treatment of these patients until after the age 15 or 16 years and then follows with surgery after completion of growth.

Guidelines for pretreatment evaluation

Accurate differential diagnosis and careful pretreatment evaluations are essential for determining the proper approach to and timing of treatment. Box 11-3 presents guidelines to consider in treatment planning in order to achieve efficient correction and long-term stability.

Box 11-3	Factors influencing long-term stability of treatment in patients with Class III malocclusion
Positive factors • Convergent facial type • Anteroposterior functional shift • Symmetric condylar growth • Good growth potential • ANB of 2 degrees or less • Maxillary anterior crowding and retroclination • Good patient cooperation • Good facial esthetics • Lack of familial prognathism	***Negative factors*** • Divergent facial type • Absence of anteroposterior functional shift • Asymmetric condylar growth • Growth completed • Severe, negative ANB value • Lack of maxillary anterior crowding and proclination • Poor patient cooperation • Poor facial esthetics • Familial prognathism

Prevalence of Class III malocclusion

The prevalence of Class III malocclusion has a strong ethnic distribution; reports vary greatly depending on the different methods of classification. Nakasima et al[63,70] reported an incidence of 1% in white populations and a frequency of 10% in a Japanese population. Haynes[71] screened a white population of British females aged 11 to 22 years and reported a prevalence of 1.6%. Endo[72] reported finding reversed occlusion in 7.81% of 11-year-old Japanese girls studied. Susami et al[73] reported a prevalence of 4.24% in Japanese females between 3 and 19 years of age.

Greater frequency of reversed occlusion is not limited to Japanese individuals; the rate is also higher in other Asian populations. Chan[74] reported that the prevalence of Class III malocclusion in Chinese individuals is 9.4%, and Baik et al[75] reported a rate of 19% in Korean patients.

Box 11-4 shows the differences in characteristics of dental and skeletal crossbites.

Box 11-4	Comparison of the characteristics of dental and skeletal crossbites
Skeletal Class III malocclusion • The etiology can be genetic, environmental, or a combination of both. • The ANB angle has a negative value. • Incisors are in end-to-end relationships or exhibit negative overjet. • Molars and canines show a Class III relationship or a Class III tendency. • The malocclusion may or may not be accompanied by a mandibular shift. • The profile is straight or concave.	***Dental crossbite*** • The etiology is usually local. • The ANB angle is normal. • Only the incisors show an incorrect inclination. • Molars and canines are in Class I relationships at centric relation. • The crossbite is often accompanied by a habitual shift from rest to occlusion. • The profile is normal.

Early treatment strategies

Evaluation and specification of the exact morphology and characteristics of each patient's Class III malocclusion is the first important step in treatment.

Early interception of Class III malocclusion has been advocated for many years; Angle[69] suggested that deformities in this class begin at about the age of eruption of the permanent first molars or earlier and are always associated with enlarged tonsils. He also indicated that the skeletal aberrations worsen with time. Angle[69] stated that, when disharmony is being established, the problem usually progresses rapidly. Angle[69] was also one of the first to suggest that combined orthodontics and orthognathic surgery was the only way to correct mandibular prognathism once it has fully developed.

Salzmann[76] suggested that treatment for Class III malocclusion commence as soon as the abnormality is diagnosed. Graber et al[50] contended that because Class III malocclusions are among the most difficult to treat and surgical intervention is contemplated in some cases, it makes good sense to try early treatment with at least a chin cap to prevent worsening of the malocclusion.

Tweed[77] divided Class III malocclusions into two categories: pseudo–Class III, with a normal mandible and an underdeveloped maxilla, and skeletal Class III with a large mandible. Tweed[77] stated that these irregularities should be treated during the mixed dentition (7 to 9 years); if the conditions occur during the primary dentition, treatment can be started as early as 4 years of age. He also stated that, if treatment is not started at an early age, lingual locking of the maxillary incisors will retard maxillary growth and accelerate growth in the mandible.

Proffit[57] stated that there is some degree of maxillary deficiency in most patients with skeletal Class III malocclusion (30% to 40%) and expressed hope that improved appliance design would allow more downward and forward repositioning of the maxilla.

Björk[78] stated that circum-maxillary sutures are active sites of growth during the early juvenile period and facilitate forward maxillary growth.

General strategies for early intervention in Class III maxillary deficiencies or anterior crossbites with the potential to develop to Class III malocclusion include the following four major techniques:

1. Unlocking of the occlusion and elimination of mandibular shift
2. Incisor proclination combined with labial root torque
3. Maxillary protraction and expansion, if needed
4. Enhancement of maxillary growth

Depending on the type of problem and the dentoskeletal condition of the anomaly, different early intervention techniques are available, including palatal expansion, a face mask, a standard chin cap, a chin cap with spurs, and functional appliances.

Special appliances

Chin cap with spurs

A chin cap with spurs is an extraoral device that can be used in Class III orthopedic treatment for simultaneous control of mandibular growth and protraction of the maxilla (Fig 11-26). A rubber band connected from the spurs to the maxillary arch produces maxillary protraction (see case 11-20).

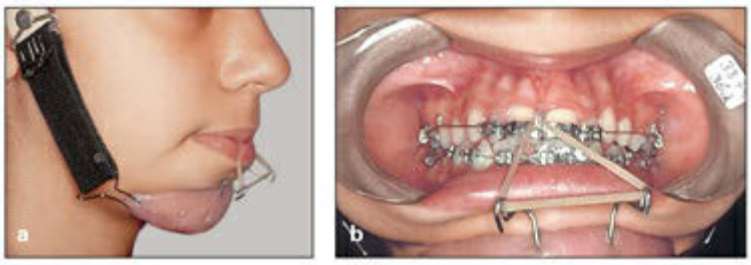


Fig 11-26 *(a and b)* Vertical chin cap with spurs. Maxillary protraction is achieved with rubber bands connected from the spurs to the maxillary arch.

Face mask–chin cap combination

The face mask–chin cap combination (FCC) is a special orthopedic device the author originally designed and constructed in 1968 and developed from that time until 1999; when practicing in Tehran, the author used this appliance with good success in many patients with skeletal Class III malocclusion. This device can be used in early treatment of all kinds of skeletal Class III malocclusion, whether they result from maxillary deficiency, mandibular prognathism, or a combination of both. With some minor modifications, the FCC can also be applied in Class III patients with a horizontal or vertical growth pattern.

Generally this appliance has two different kinds of design, type 1 and type 2. Both types have a soft head holder pad that can be adjusted to each patient's head size and shape. The advantage of the head holder is that the appliance can be firmly held to the face all night without any slipping.

The type 1 FCC has a chin cap without traction (Fig 11-27a), and the author uses it in Class III patients with maxillary deficiency and a normal mandible. The acrylic resin chin cap provides anchorage against maxillary protraction (see case 11-19).

The type 2 FCC, besides the head holder pad and acrylic resin chin cap, has a traction elastic connected to the cap on each side of the face (Fig 11-27b). These elastics can impart distal force to the mandible and therefore can be used in Class III malocclusions in which both the maxilla and the mandible are involved (see cases 11-21 and 11-23). The chin cap force can also be designed in a high-pull direction or a cervical direction, depending on the patient's mandibular growth pattern.

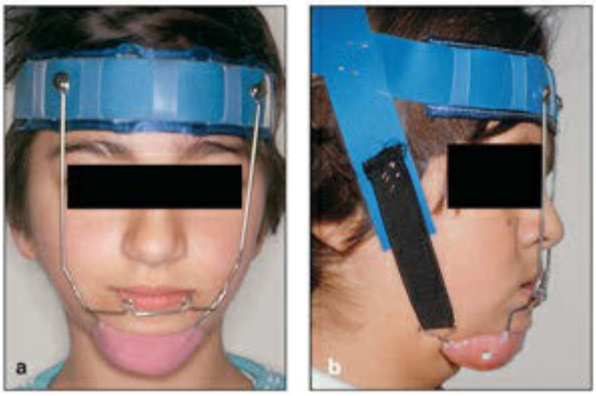


Fig 11-27 Face mask–chin cap (FCC) combination. *(a)* Type 1. *(b)* Type 2.

Case Reports

The following case reports present examples of management of various types of anterior crossbite with different characteristics and different etiologic factors; with different treatment options, such as preventive, interceptive, corrective, or combination treatment; and in patients at different dental and skeletal stages of development.

Although the major goal of this publication is to advocate early intervention for all kinds of anterior crossbites, whether dental, dentoalveolar, or skeletal, a few cases of delayed treatment are presented to illustrate the problems that can result from negligence. Therefore, case reports are presented and classified in three groups:

1. Early treatment during the primary or early mixed dentition before incisor eruption
2. Early treatment after eruption of the incisors (middle or late mixed dentition)
3. Nonsurgical treatment of the Class III permanent dentition

Early treatment during the primary or early mixed dentition before incisor eruption

Early intervention at this stage of the dentition usually consists of interceptive treatment and guidance, and the main goals are unlocking of the anterior teeth and slight proclination of the primary incisors, if needed. Correction of posterior crossbite is also another goal to achieve at this stage of treatment by either slow

dental expansion or rapid expansion, depending on the type of posterior crossbite and maxillary base conditions.

Maxillary basal protraction by extraoral devices might also be necessary in some patients with maxillary deficiency. This goal can be achieved with a face mask or chin cap with spur attached to removable or bonded expanders.

The fundamental strategy of this type of intervention is to prepare a normal environment at an early stage of the dentition to enable coordinated jaw growth and encourage proper development of the occlusion, which should continue to the next dental stages.

Case 11-15

A 6-year-old girl presented with both anterior segments and the posterior left segment in crossbite. The only permanent teeth that had erupted were the first molars. She exhibited a mandibular shift and a concave profile (Figs 11-28a to 11-28d).

Treatment:
Treatment was accomplished with only a Hawley posterior bite plate without a labial bow. The appliance included an expansion screw for posterior crossbite correction and two finger springs for anterior crossbite correction. Composite resin attachments were bonded to the buccal surfaces of the maxillary primary molars; the attachments had long C-clasps hooked over the composite resin for added retention in place of bands with tubes (Fig 11-28e). Photographs and a radiograph revealed the results of posterior and anterior crossbite correction; the mandibular permanent central incisors were erupting, and the posterior segment was settling (Figs 11-28f to 11-28j).

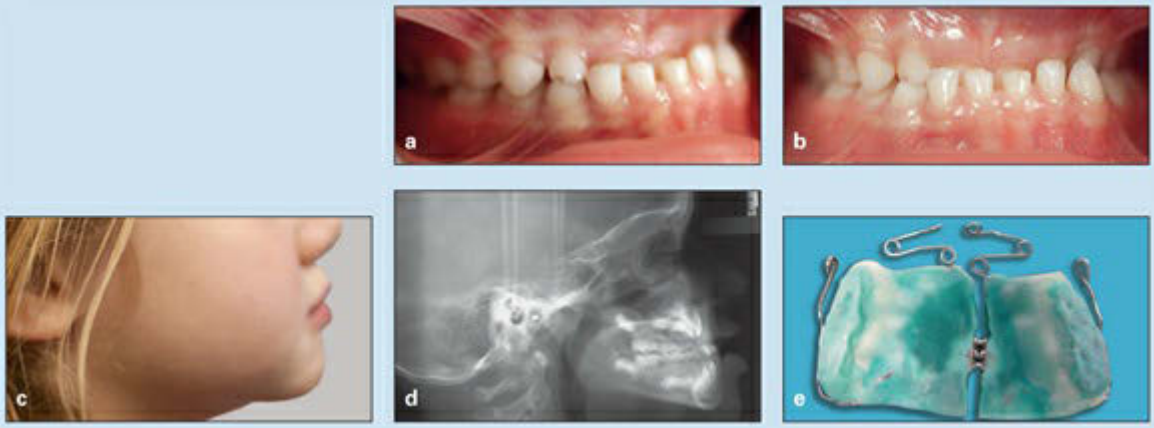


Fig 11-28 Early treatment of anterior and posterior crossbite in a 6-year-old girl. *(a and b)* Pretreatment occlusion. *(c)* Pretreatment soft tissue profile. *(d)* Pretreatment cephalometric radiograph. *(e)* Appliance used in treatment.

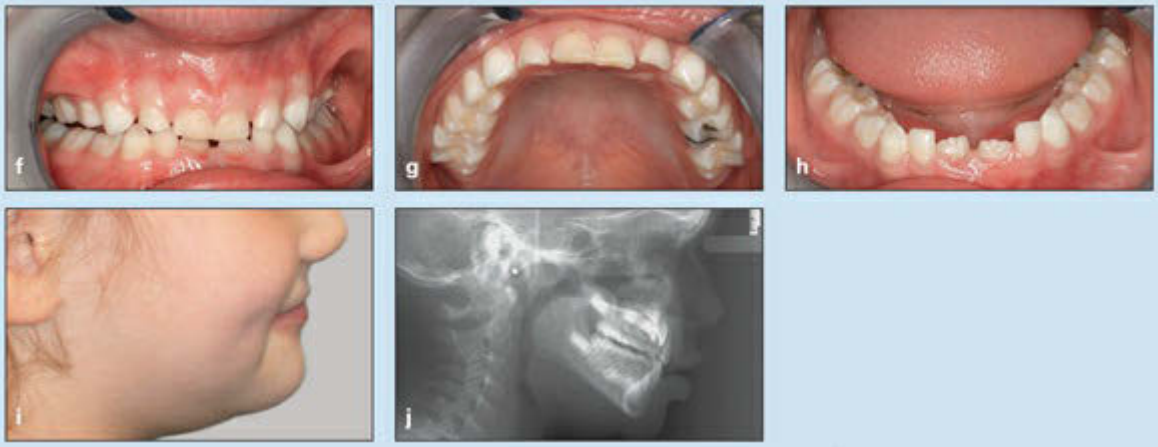


Fig 11-28 *(cont) (f to h)* Posttreatment occlusion. *(i)* Posttreatment soft tissue profile. *(j)* Posttreatment cephalometric radiograph.

Case 11-16

A 3-year, 5-month-old boy in the primary dentition presented with a Class III malocclusion that had a hereditary background. He had a mesial step at the primary second molars (slight Class III relationships at centric relation) and a mandibular mesial shift (Figs 11-29a to 11-29e).

Treatment:
During the primary dentition, even unlocking the occlusion in the area of crossbite can eliminate functional shift and guide development of normal occlusion. This boy showed good compliance, so the treatment plan included only the use of a Hawley appliance, banding of the primary second molars with a buccal tube for better retention, occlusal coverage, and a simple finger spring behind the maxillary incisors.

Figures 11-29f and 11-29g show the settling posterior segment after correction of the incisor crossbite. Figures 11-29h to 11-29k show the final occlusion, 10 months after use of the appliance was stopped.

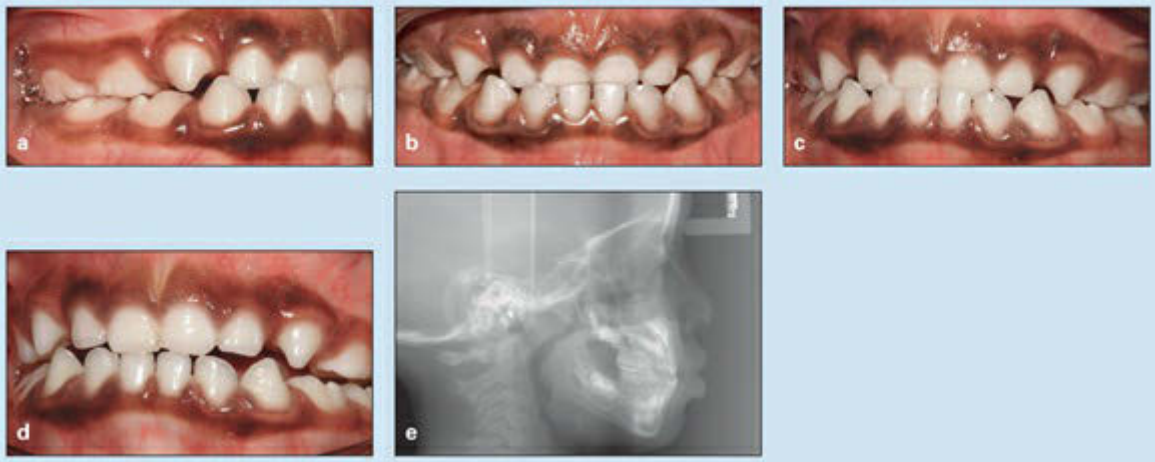


Fig 11-29 Management of a hereditary Class III malocclusion in a 3-year, 5-month-old boy in the primary dentition. He has a mesial step at the primary second molars and a mesial mandibular shift. (a to c) Pretreatment occlusion in centric occlusion. (d) Pretreatment occlusion in centric relation. (e) Pretreatment cephalometric radiograph.

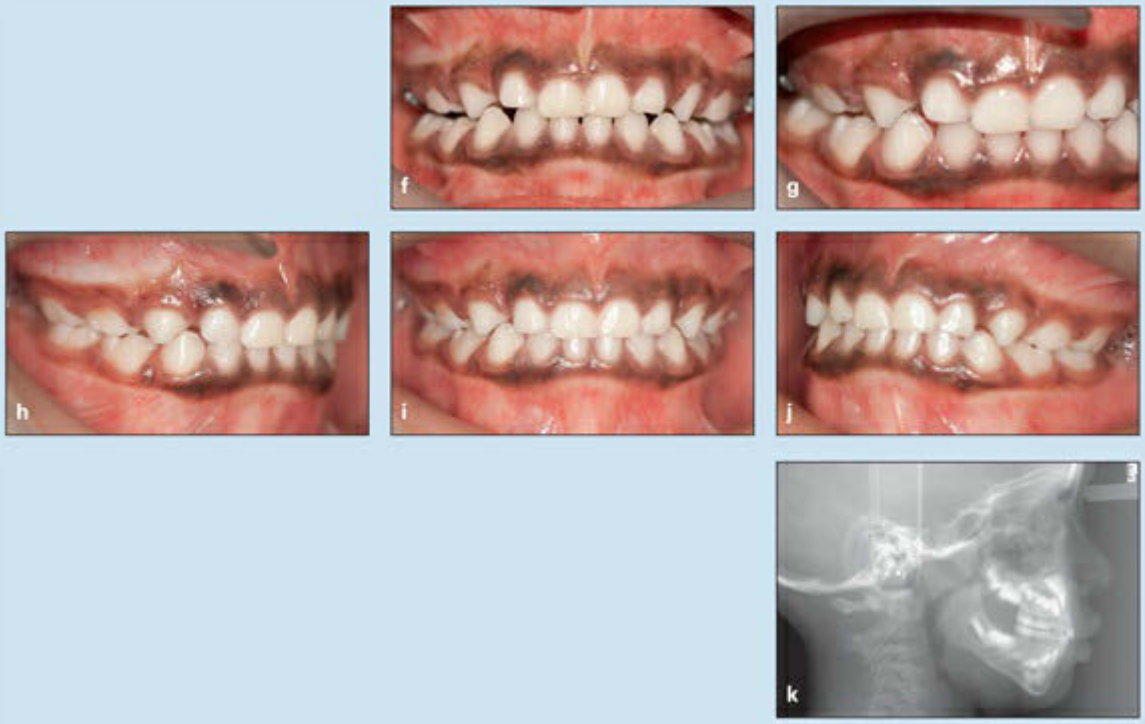


Fig 11-29 (cont) (f and g) Occlusion after correction of anterior crossbite and during settling of the posterior occlusion. (h to j) Posttreatment occlusion 10 months after cessation of the appliance. (k) Posttreatment cephalometric radiograph.

Case 11-17

A 5-year, 9-month-old boy presented with a collapsed maxillary dentition, severe reverse deep bite, and a mandibular mesial shift. Even in centric relation, the maxillary and mandibular incisors did not meet in an end-to-end position (Figs 11-30a to 11-30g). He exhibited a negative ANB angle and a concave profile. His problems had a hereditary background.

Treatment:
The treatment approach was interceptive intervention to correct the dentoskeletal malrelationships by maxillary expansion, maxillary protraction by face mask therapy, and some maxillary incisor proclination. Because the patient's compliance was excellent and the parent preferred it, treatment was performed with a removable appliance and face mask.

Figure 11-30h shows the modified Hawley appliance, which included two Adams clasps, thick posterior occlusal coverage to disocclude the anterior segment, a finger spring for slight incisor proclination, a palatal jackscrew for expansion, and two horizontal loops on the canines for connecting elastics to the face mask to achieve maxillary protraction and provide appliance retention (as discussed earlier66). Figure 11-30i shows the patient's occlusion 6 months after the start of treatment. The permanent incisors have erupted out of crossbite. During this stage, the posterior bite block was reduced gradually at each visit.

Figures 11-30j and 11-30k show different panoramic radiographs taken during treatment; they indicate that sufficient space was created for eruption of all incisors and canines. Figures 11-30l to 11-30o show the occlusion 6 months after active treatment and during the observation period.

Figures 11-30p to 11-30s were taken 15 months after active treatment and show complete eruption of the maxillary central incisors and all mandibular incisors. At this time, active treatment was stopped, and the patient was placed on a recall schedule for observation.

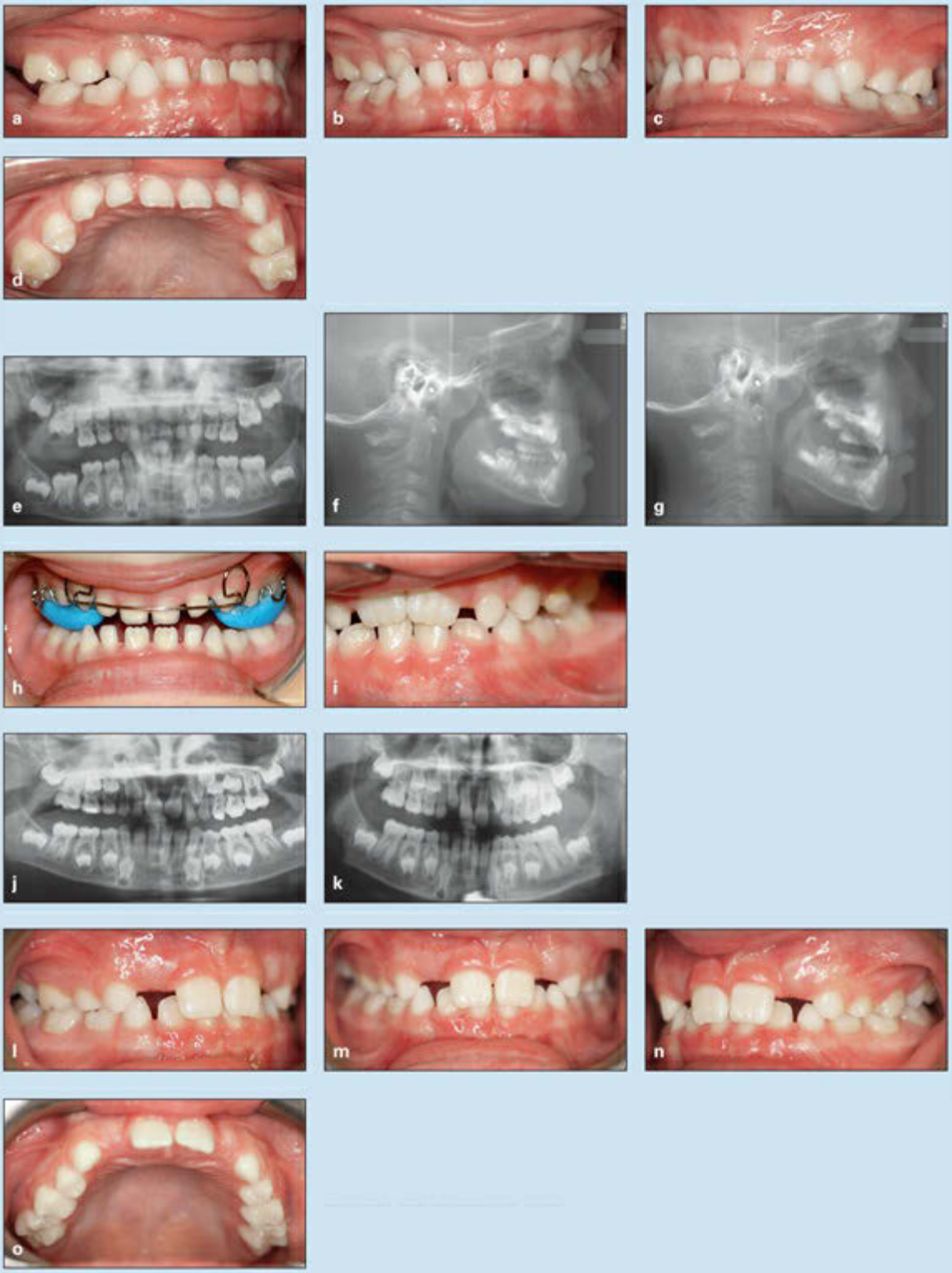


Fig 11-30 Management of collapsed maxillary dentition, severe reverse deep bite, and mesial mandibular shift in a 5-year, 4-month-old boy with a hereditary Class III malocclusion. *(a to d)* Pretreatment occlusion. *(e)* Pretreatment panoramic radiograph. *(f)* Pretreatment cephalometric radiograph in centric occlusion. *(g)* Pretreatment cephalometric radiograph in centric relation.

Even in centric relation, the maxillary and mandibular incisors do not meet end to end. *(h)* Modified Hawley appliance with thick posterior acrylic resin coverage to open the bite. *(i)* Occlusion after 6 months of treatment. The permanent incisors are erupting out of crossbite. *(j and k)* Panoramic radiographs during treatment, showing space creation. *(l to o)* Occlusion at the end of active treatment.

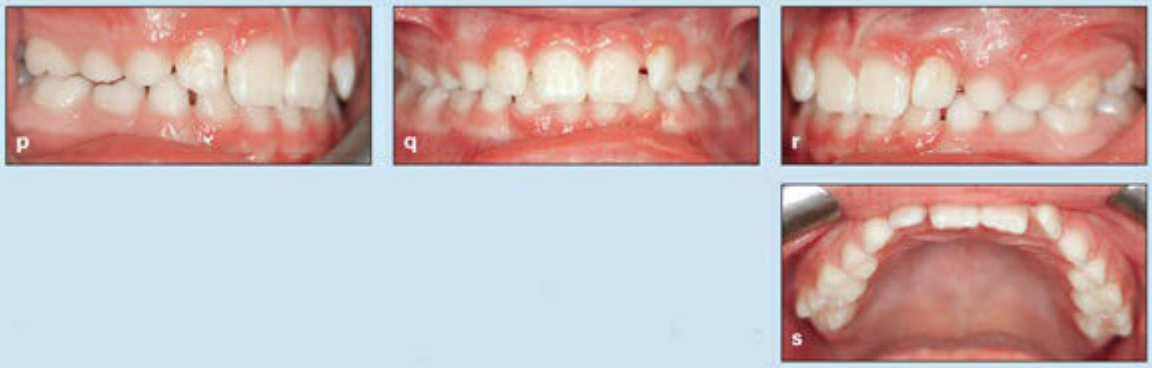


Fig 11-30 *(cont) (p to s)* Posttreatment occlusion 15 months after active treat-ment. All permanent incisors and first molars have erupted.

Case 11-18

A 6-year, 4-month-old girl presented with skeletal Class III malocclusion, posterior and anterior crossbites, and a mandibular shift (Figs 11-31a to 11-31f). Both maxillary permanent lateral incisors were missing.

Treatment:
The planned treatment was rapid palatal expansion in conjunction with maxillary protraction by face mask (Figs 11-31g and 11-31h). After anterior crossbite correction, the permanent central incisors erupted. Figure 11-31i shows a panoramic radiograph taken during the transitional dentition. Figures 11-31j to 11-31o show the patient's occlusion after phase 1 therapy and before eruption of the permanent canines. Figures 11-31p to 11-31r show the posttreatment occlusion.

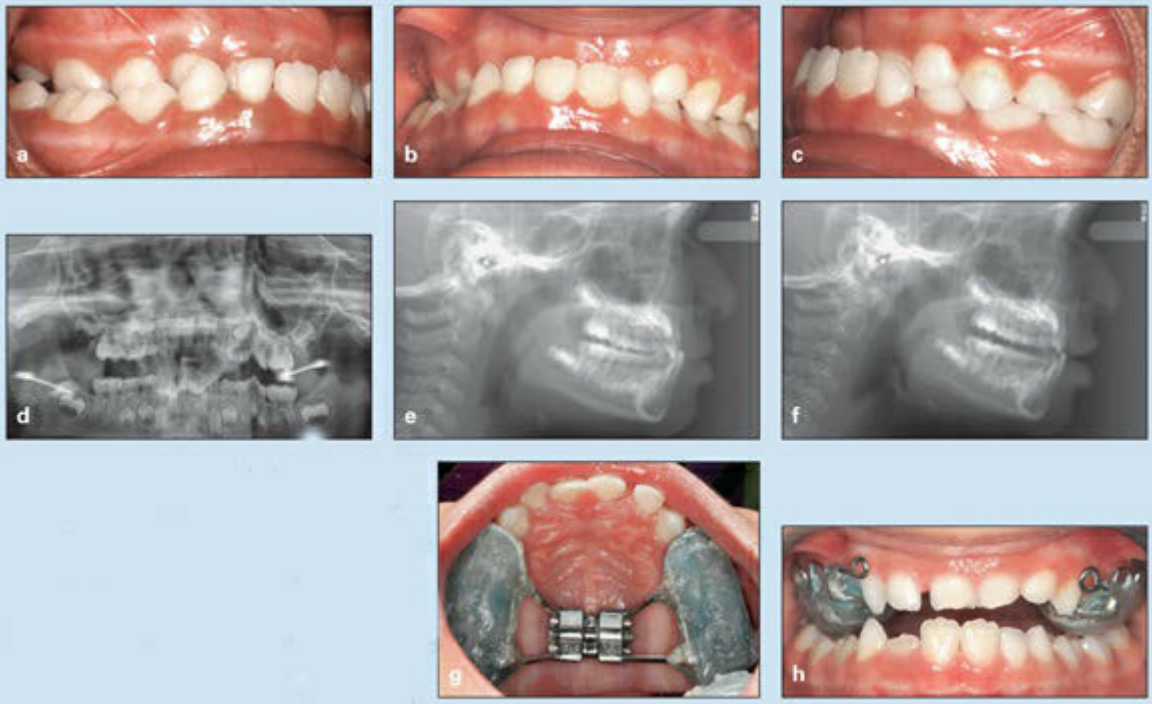


Fig 11-31 Management of skeletal Class III malocclusion, posterior and anterior crossbites, and mandibular shift in a 6-year, 4-month-old girl. Both maxillary permanent lateral incisors are congenitally missing. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e)* Pretreatment cephalometric radiograph in centric occlusion. *(f)* Pretreatment cephalometric radiograph in centric relation. *(g and h)* Bonded rapid expander with hook for mask protraction.

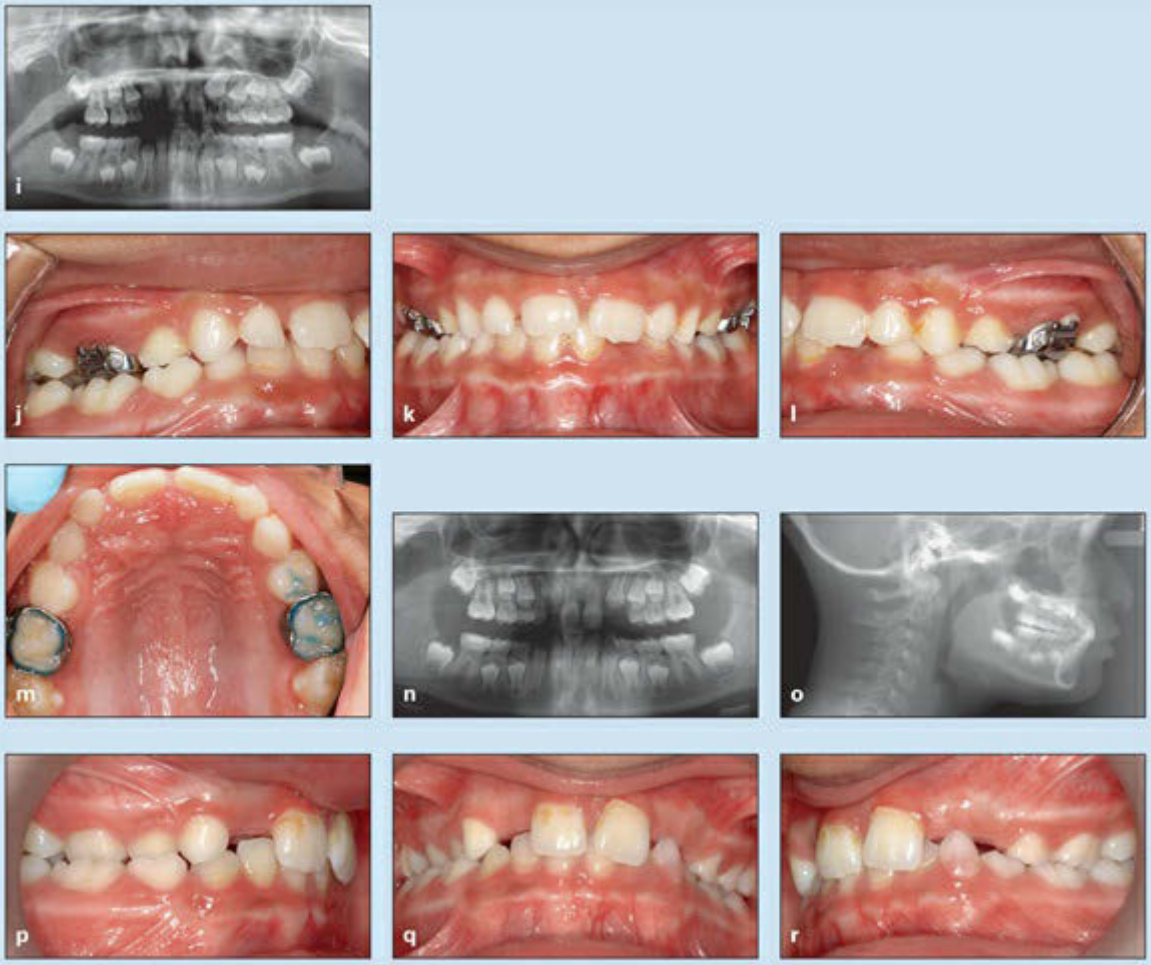


Fig 11-31 *(cont) (i)* Panoramic radiograph during the transitional dentition, before eruption of the permanent canines. *(j to m)* Occlusion after phase 1 treatment. All incisors have erupted during the observation period. *(n)* Panoramic radiograph after phase 1 treatment. *(o)* Cephalometric radiograph after phase 1 treatment. *(p to r)* Posttreatment results.

Early treatment after eruption of the incisors (middle or late mixed dentition)

As discussed earlier, anterior crossbites with mandibular shift are not only destructive to the dentition and supporting structures but also can have adverse effects on jaw growth and worsen the anteroposterior basal jaw relationships. Therefore, correction of the problem during the mixed dentition, especially the cases that have some familial background, are more complicated. Patients exhibit a greater sagittal malrelationship (A-B discrepancy) and usually need a combination of orthodontic and orthopedic treatment.

Case 11-19

A 9-year, 8-month-old girl in the middle mixed dentition presented with a maxillary deficiency (SNA = 76.5 degrees) and a maxillary arch that was collapsed within the mandibular arch (Figs 11-32a to 11-32d). There was an A-B discrepancy of –0.8 degrees as well as severe maxillary incisor crowding, rotation, and displacement. She had no mandibular shift.

Treatment:

The treatment plan included rapid maxillary expansion and maxillary protraction with a type 1 FCC (see Fig 11-27a). Treatment was started with rapid palatal expansion, occlusal coverage, and a hook to the FCC. The second step was maxillary incisor bonding and placement of a nickel-titanium arch for leveling and anterior alignment. Next, a stainless steel round arch with open loop was placed to procline the maxillary incisors out of crossbite. Finally, a rectangular arch was placed in conjunction with the type 1 FCC to achieve buccal root torque.

Figures 11-32e to 11-32h show the occlusal results. Cephalometric measurements revealed the following effects of treatment: ANB increased from –0.8 to 1.5 degrees, SNA increased from 76.5 to 82.5 degrees, and overjet increased from –1.5 to 0.8 mm (Figs 11-32i and 11-32j).

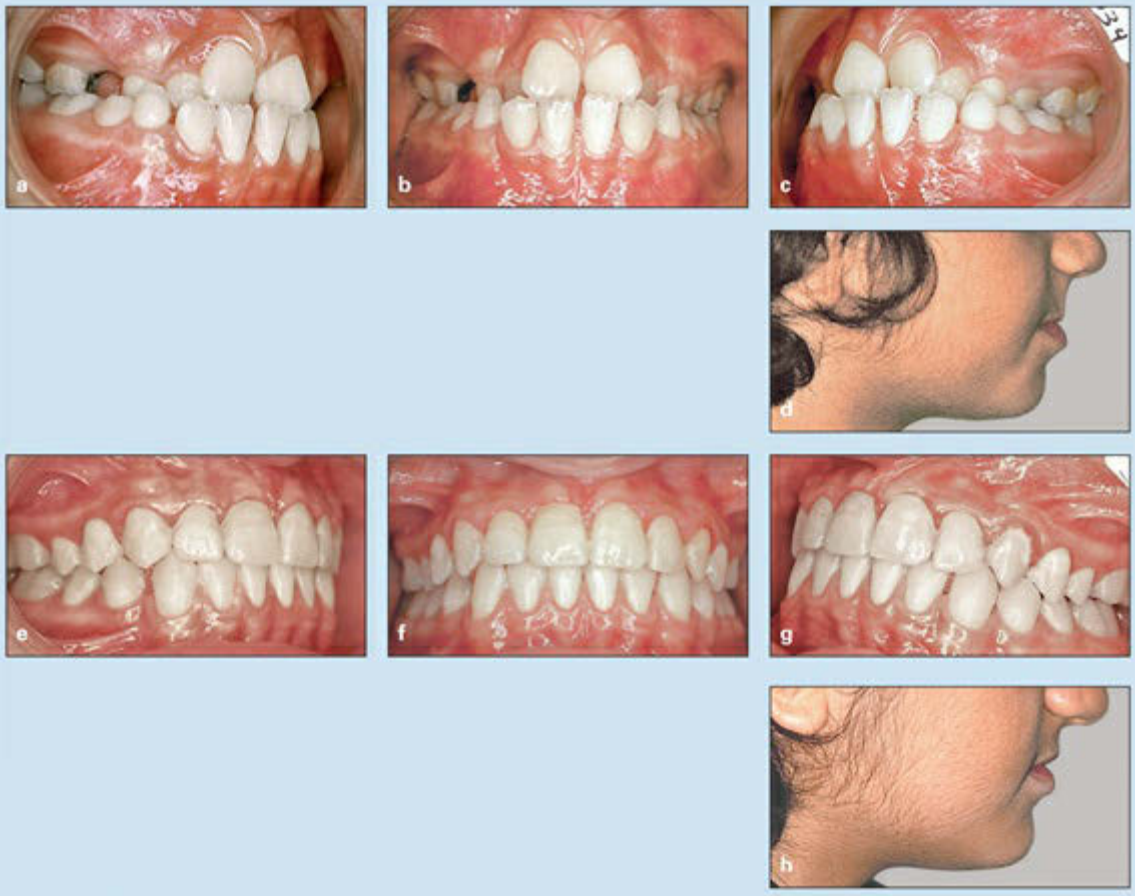
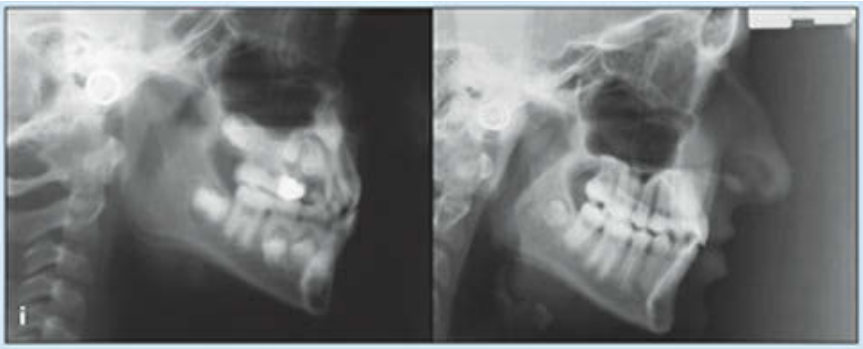


Fig 11-32 Management of maxillary arch collapse and maxillary deficiency in a 9-year, 8-month-old girl. The maxillary incisors exhibit severe crowding, rotation, and displacement. There is no mandibular shift. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment soft tissue profile. *(e to g)* Posttreatment occlusion. *(h)* Posttreatment soft tissue profile.



j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	87.8	84.2
SNB (°)	78.0	77.3	81.0
SNA (°)	82.0	76.5	82.5
Maxillary depth (FH-NA) (°)	90.0	86.9	88.4
ANB (°)	2.3	−0.8	1.5
FMA (MP-FH) (°)	25.9	24.3	25.3
Y-axis (SGn-SN) (°)	59.4	67.1	64.4
Interincisal angle (U1-L1) (°)	135.0	134.5	129.1
U1-FH (°)	116.2	106.1	112.6
IMPA (L1-MP) (°)	95.0	93.0	94.5
Overbite (mm)	2.5	−1.5	0.8
Overjet (mm)	2.5	−1.5	2.7

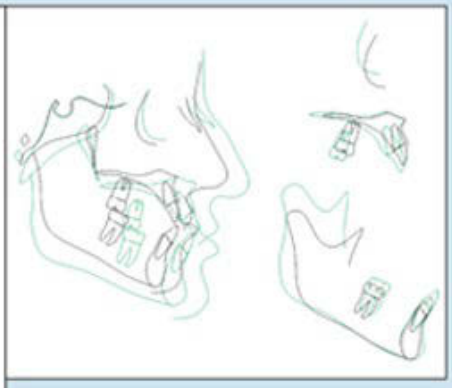


Fig 11-32 *(cont) (i)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-20

An 11-year-old boy presented with skeletal Class III malocclusion, maxillary deficiency, mandibular prognathism, a steep mandibular plane, and a mandibular shift (Figs 11-33a to 11-33d).

Treatment:
Treatment was started with rapid palatal expansion and a vertical chin cap (see Fig 11-26) that had spurs to allow the elastics to be attached from the spurs to the premolar brackets to initiate maxillary protraction (Figs 11-33e to 11-33h). After expansion and bite jumping, treatment was continued with maxillary anterior bonding. There were also elastics from spurs to the connected midline arch hook.

Figures 11-33i to 11-33l illustrate the patient's posttreatment occlusion.

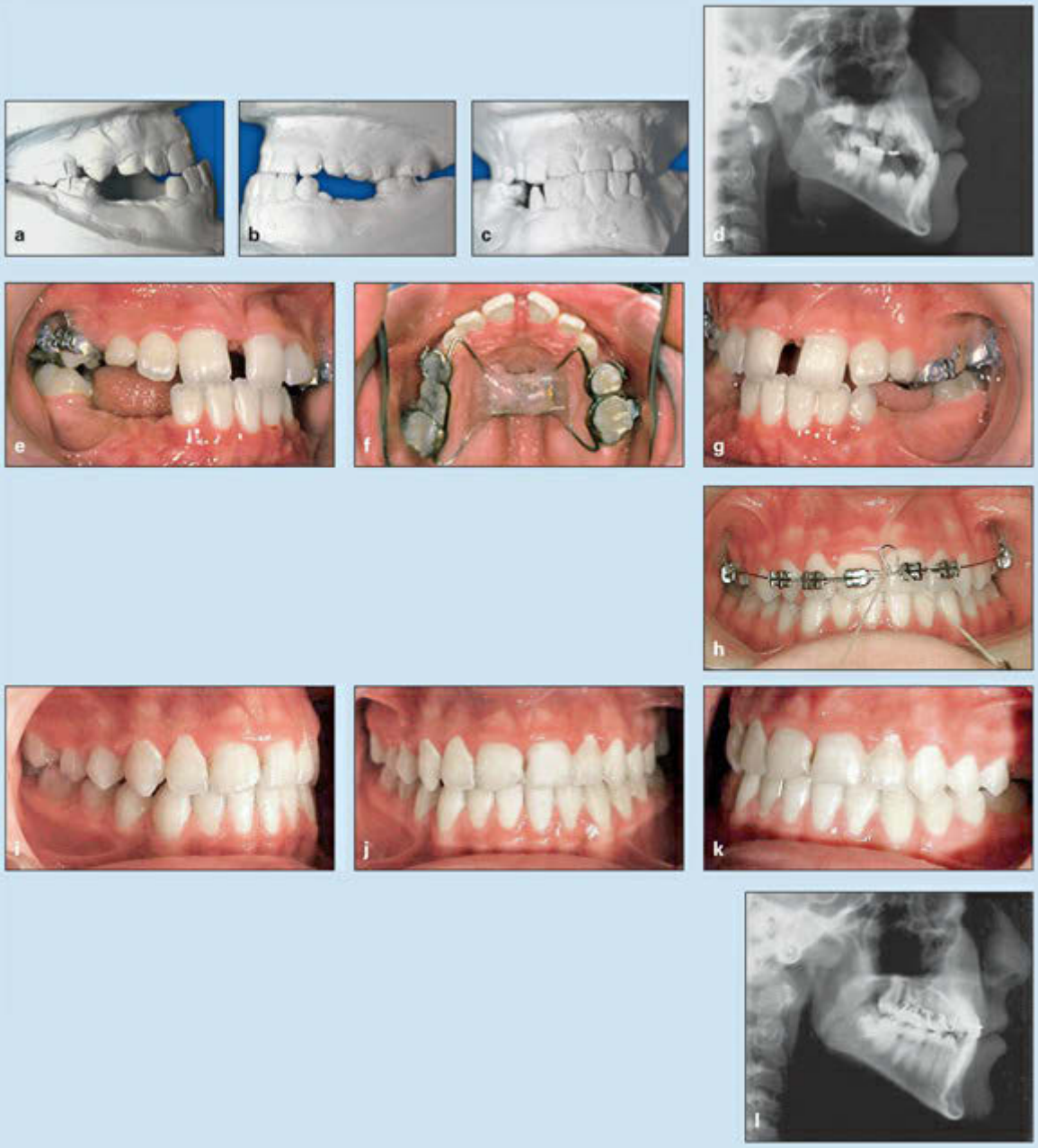


Fig 11-33 Management of skeletal Class III maxillary deficiency, mandibular prognathism, steep mandibular plane, and mandibular shift in an 11-year-old boy. *(a to c)* Casts of pretreatment occlusion. *(d)* Pretreatment cephalometric radiograph. *(e to h)* Rapid palatal expansion and maxillary protraction. *(i to k)* Posttreatment occlusion. *(l)* Posttreatment cephalometric radiograph.

Case 11-21

An 11-year, 6-month-old girl in the late mixed dentition presented with a hereditary skeletal Class III malocclusion, mandibular prognathism, slight maxillary deficiency, anterior open bite, left posterior crossbite, maxillomandibular dental protrusion, and a 1.9-mm reverse overjet (Figs 11-34a to 11-34c).

Treatment:

Because of the patient's space deficiency, dental protrusion, vertical growth tendency, and anterior open bite, treatment was designed to include extraction of four premolars combined with orthopedic treatment. As a result of the patient's dentoskeletal configuration—mandibular prognathism and mandibular incisor protrusion— two mandibular first premolars and two maxillary second premolars were removed.

First, a mandibular lingual arch was inserted to provide anchorage and prevent mandibular molar eruption. Next, a maxillary transpalatal arch was used to provide maxillary anchorage and intrude the maxillary molars. The maxillary and mandibular primary second molars were extracted first. This treatment continued later with extraction of the maxillary second premolars and mandibular first premolars to obtain greater retraction of the mandibular incisors and greater mesial movement of the maxillary molars. These changes were designed to provide counterclockwise rotation of the mandible that would help correct the open bite.

The next step was maxillary anterior bonding to procline the incisors and placement of a type 2 FCC appliance to achieve protraction of the maxilla (see Fig 11-27b).

Figures 11-34d to 11-34f show the posttreatment occlusion. Some of the results of treatment included changes in SNA from 81.0 to 87.4 degrees, ANB from –0.3 to 4.7 degrees, A-B to facial plane from 1.3 to –6.5 degrees (normal), overbite from –2.4 to 1.2 mm, and overjet from –1.9 to 2.9 mm (Figs 11-34g and 11-34h).

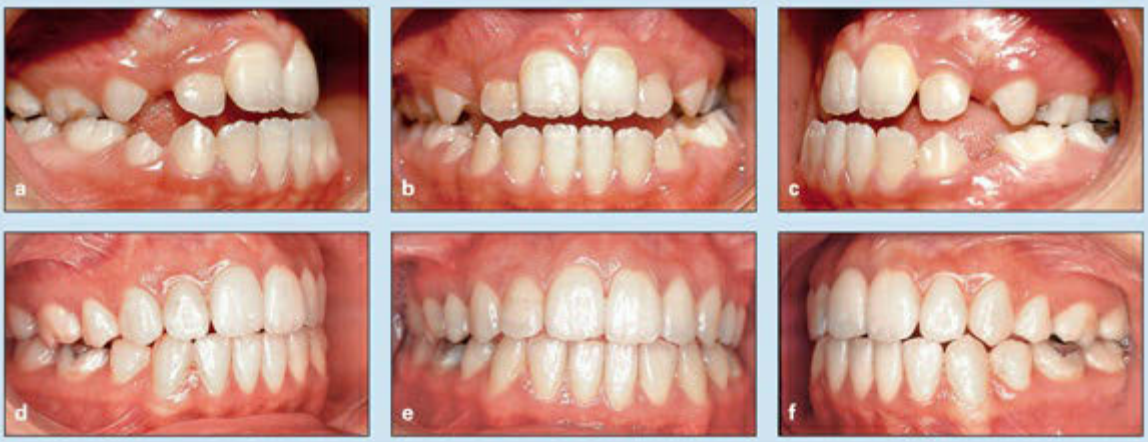
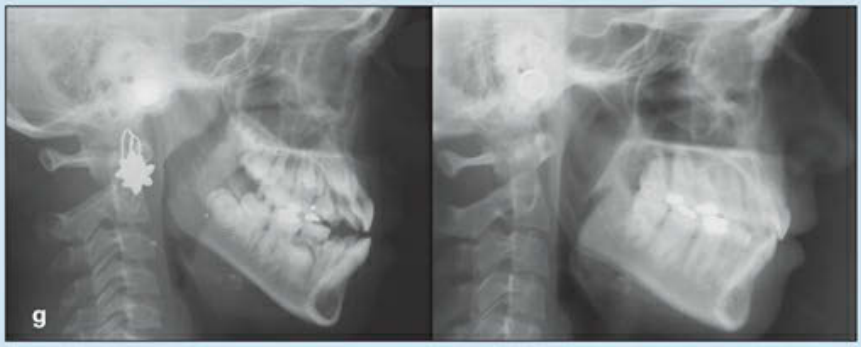


Fig 11-34 Management of skeletal Class III malocclusion, mandibular prognathism, slight maxillary deficiency, anterior open bite, left posterior crossbite, and maxillomandibular dental protrusion in an 11-year, 6-month-old girl in the late mixed dentition. *(a to c)* Pretreatment occlusion. *(d to f)* Posttreatment occlusion.



h

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	90.4	91.5
SNB (°)	78.0	81.3	82.7
SNA (°)	82.0	81.0	87.4
Maxillary depth (FH-NA) (°)	90.0	91.0	96.8
ANB (°)	2.3	−0.3	4.7
FMA (MP-FH) (°)	25.9	27.6	19.9
Y-axis (SGn-SN) (°)	59.4	65.8	64.3
Interincisal angle (U1-L1) (°)	135.0	125.4	135.4
U1-FH (°)	116.2	118.7	110.8
IMPA (L1-MP) (°)	95.0	88.2	93.9
Overbite (mm)	2.5	−2.4	1.2
Overjet (mm)	2.5	−1.9	2.9

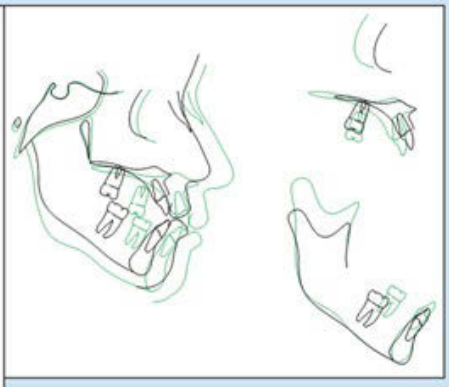


Fig 11-34 *(cont) (g)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(h)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-22

A 10-year-old girl at the beginning of the late mixed dentition presented with Class III skeletal malocclusion, maxillary deficiency, anterior and posterior crossbites (collapsed maxillary arch), and reverse deep bite (Figs 11-35a to 11-35d).

Treatment:
The treatment plan called for maxillary rapid palatal expansion and protraction with a type 1 FCC. The expander had thick occlusal acrylic resin to disocclude the anterior teeth and facilitate maxillary protraction and incisor protrusion and thereby correct the reverse overbite (Fig 11-35e). It also had hooks for connecting elastics to the face mask.

Figure 11-35f shows the occlusal situation after completion of expansion, during incisor protrusion and space closure, and before labial root torquing in conjunction with the face mask. Treatment had a number of positive effects (Figs 11-35g to 11-35l). The Landes angle changed from 83.4 to 89.9 degrees, ANB changed from –5.3 to –0.8 degrees, and overjet was increased from –3.3 to 1.6 mm.

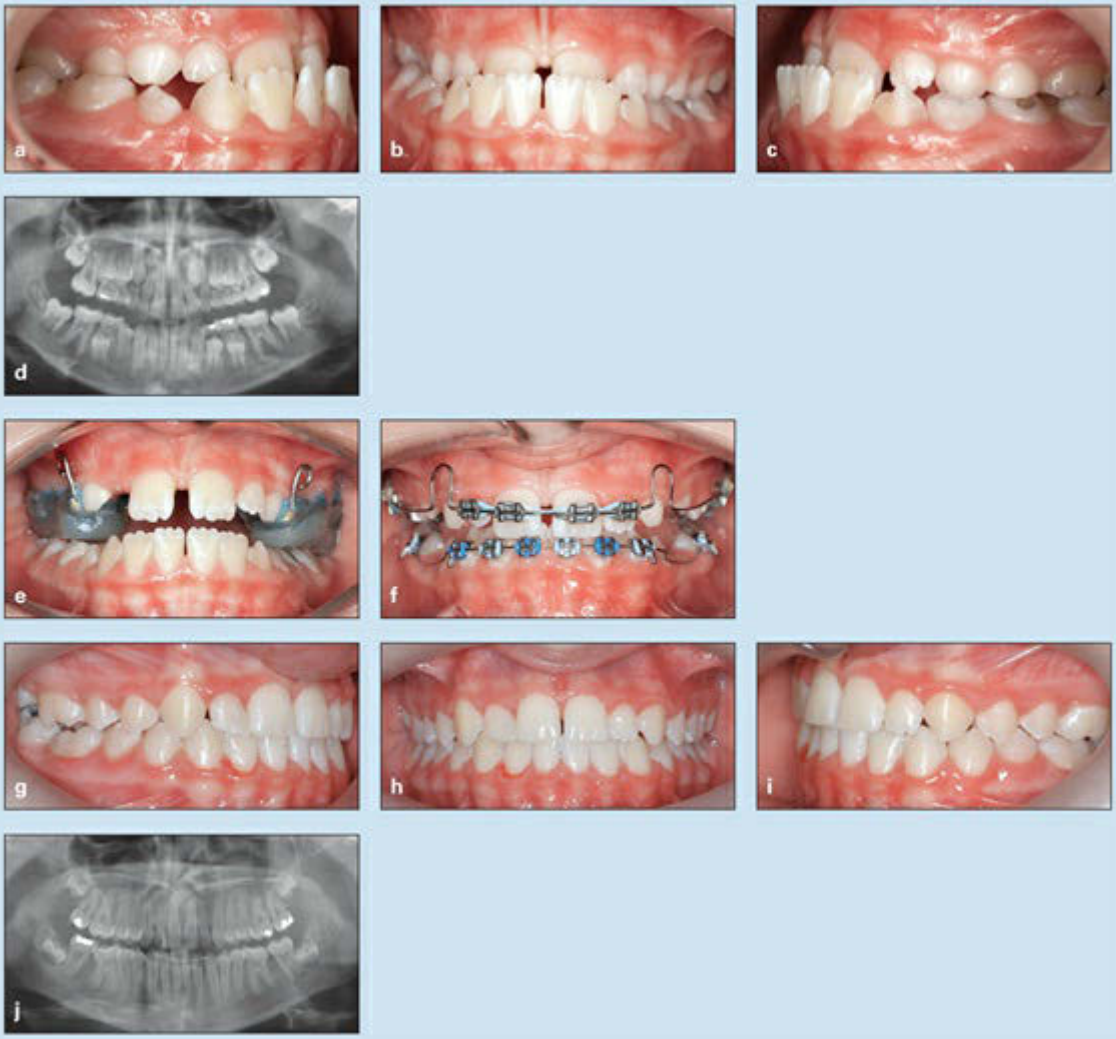
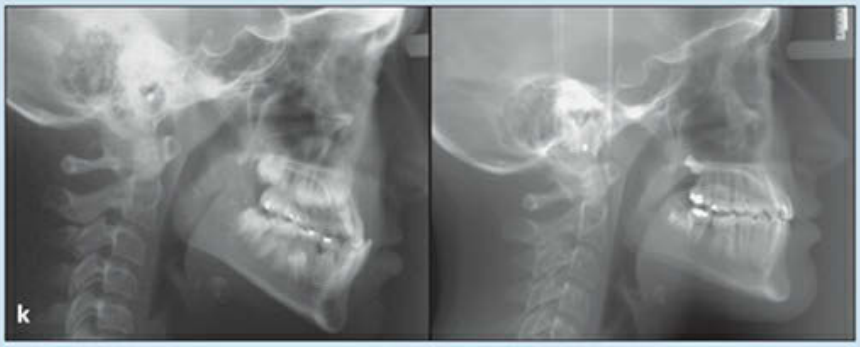
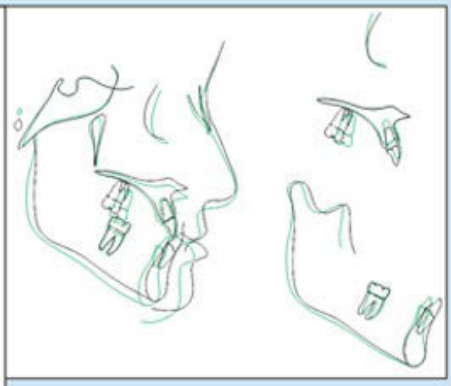


Fig 11-35 Management of skeletal Class III malocclusion, maxillary deficiency, collapsed maxillary arch, and reverse deep bite in a 10-year-old girl. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e)* Rapid palatal expander with occlusal coverage and hooks for a face mask. *(f)* Occlusion after expansion, during incisor protrusion and space closure, and before labial root torquing. *(g to i)* Posttreatment occlusion. *(j)* Posttreatment panoramic radiograph.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	88.6	92.3
SNB (°)	78.0	79.4	76.4
SNA (°)	82.0	74.1	75.5
Maxillary depth (FH-NA) (°)	90.0	83.4	89.9
ANB (°)	2.3	–5.3	–0.8
FMA (MP-FH) (°)	25.9	23.3	21.8
Y-axis (SGn-SN) (°)	59.4	67.8	69.9
Interincisal angle (U1-L1) (°)	135.0	133.5	140.7
U1-FH (°)	116.2	110.8	116.0
IMPA (L1-MP) (°)	95.0	92.4	81.6
Overbite (mm)	2.5	2.7	–0.2
Overjet (mm)	2.5	–3.3	1.6



l

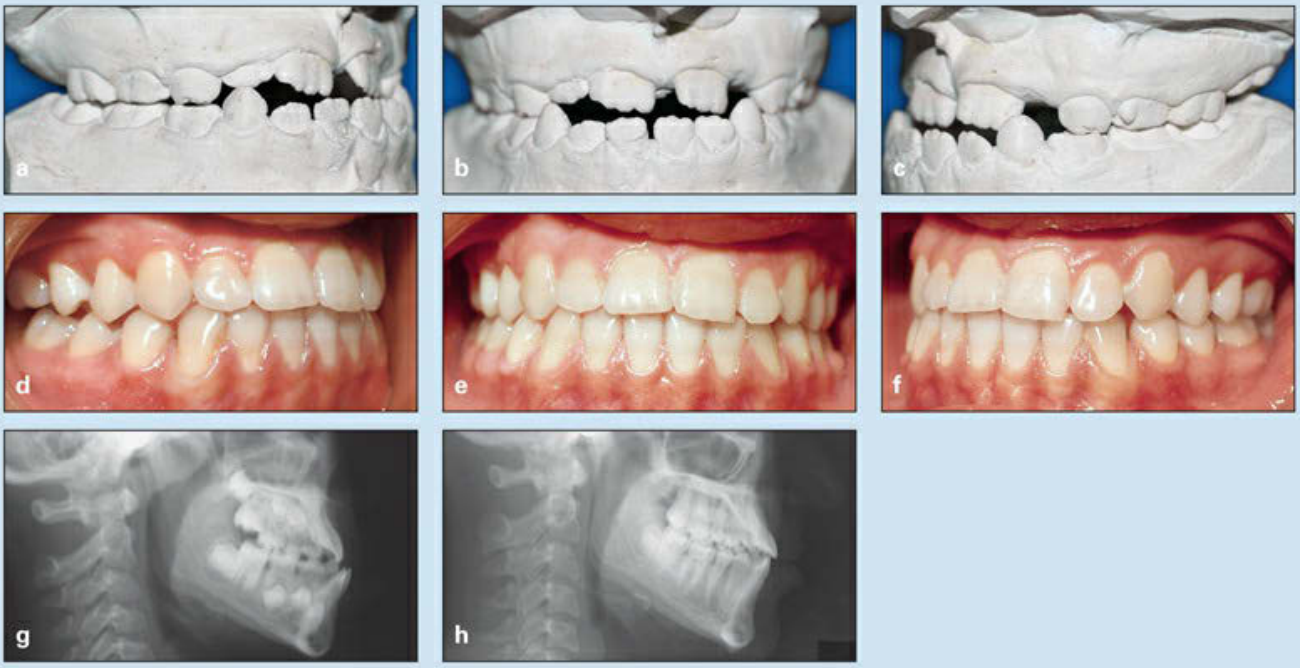
Fig 11-35 *(cont) (k)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(l)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-23

An 8-year-old girl in the early mixed dentition presented with a skeletal Class III malocclusion, maxillary deficiency, and mandibular prognathism (Figs 11-36a to 11-36c). She had an A-B discrepancy of –4.0 degrees, anterior open bite, and a 5.4-mm reverse overjet. There was no mandibular shift. The incisors and permanent first molars were partially erupted.

Treatment:
The treatment strategy was two-phase treatment. Phase 1 was orthopedic treatment to control mandibular growth and protract the maxilla. This phase was started with a lower holding arch to preserve arch length and control eruption of the mandibular molars. Maxillary orthopedic treatment was started with a type 2 FCC.

After eruption of the permanent maxillary molars and incisors, banding and anterior bonding (2 × 4) was started. Maxillary traction in a vertical direction was continued with an elastic from the maxillary arch to the type 2 FCC. Phase 2 consisted of comprehensive bonding, light Class III mechanics, and less time wearing the FCC appliance with vertical traction. Figures 11-36d to 11-36i show the treatment outcome.



i

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	88.0	89.9
SNB (°)	78.0	82.6	83.8
SNA (°)	82.0	81.5	84.7
Maxillary depth (FH-NA) (°)	90.0	87.2	90.1
ANB (°)	2.3	−1.1	1.0
FMA (MP-FH) (°)	25.9	32.6	29.3
Y-axis (SGn-SN) (°)	59.4	63.9	64.5
Interincisal angle (U1-L1) (°)	135.0	126.4	126.9
U1-FH (°)	116.2	113.1	118.1
IMPA (L1-MP) (°)	95.0	87.9	85.7
Overbite (mm)	2.5	−1.1	1.0
Overjet (mm)	2.5	−5.4	13.4

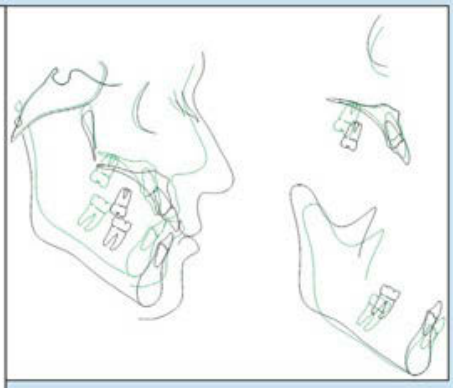


Fig 11-36 Management of skeletal Class III malocclusion, maxillary deficiency, and mandibular prognathism in an 8-year-old girl. She is in the early mixed dentition and has an anterior open bite and a 5.4-mm reverse overjet. *(a to c)* Casts of pretreatment occlusion. *(d to f)* Posttreatment occlusion. *(g)* Pretreatment cephalometric radiograph. *(h)* Posttreatment cephalometric radiograph. *(i)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Nonsurgical treatment of Class III permanent dentition

Three cases are presented to show the results of delayed correction of anterior crossbite. Treatment options available during the permanent dentition are limited to camouflage treatment, either with or without extraction, and orthogna-thic surgery, for patients with severe skeletal discrepancy.

Case 11-24

A 16-year, 5-month-old girl presented with severe anterior dentoalveolar crossbite, reverse deep bite, two-step occlusion, maxillary anterior crowding, mandibular mesial shift, and structural damage to the maxillary incisors (Figs 11-37a to 11-37e).

Treatment:
A nonextraction treatment strategy was planned. It included slight maxillary incisor proclination to eliminate crossbite and correct the overbite. A posterior bite plate with acrylic resin occlusal coverage was placed to disocclude the anterior teeth. Because of the severe reverse overbite, an occlusal composite resin attachment could not be used.

Complete maxillary and mandibular bonding was begun with a nickel-titanium archwire to align the incisors, and then stainless steel archwire with open loop was used to complete incisor proclination, level the mandibular arch, and correct the two-step occlusion. The occlusal acrylic resin was gradually reduced after crossbite correction and then use of the posterior bite plate eventually was stopped.

As mentioned earlier, dentoalveolar crossbite correction after puberty is limited to camouflage procedures or, in patients with severe skeletal discrepancy, orthognathic surgery. In this patient, the final results achieved in the dentition, including anteroposterior and buccolingual intercuspation and interdigitation, were good (Figs 11-37f to 11-37h). The dental and facial midlines were perfect, and no mandibular shift was present. However, no significant skeletal changes were achieved because of the lack of significant growth changes during corrective treatment. The major changes achieved were anteroposterior and vertical changes in occlusion accomplished through minor incisor proclination and by taking advantage of the mandibular shift. These dental changes had some positive results on the patient's soft tissue profile and upper and lower lip relationships (Figs 11-37i to 11-37l).

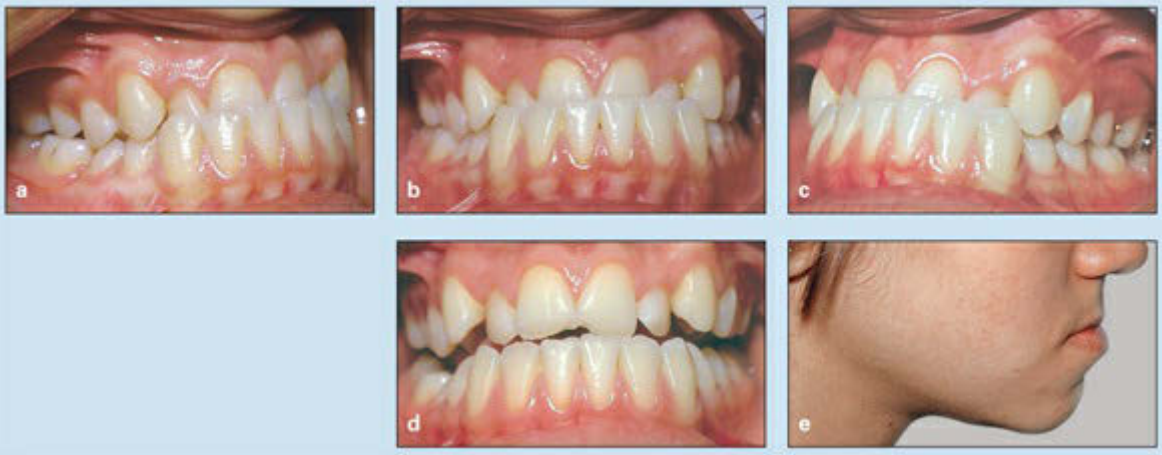
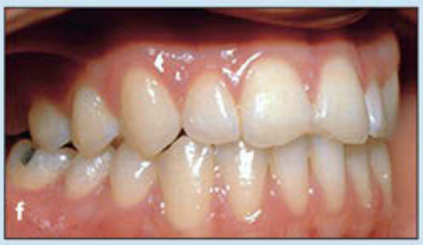
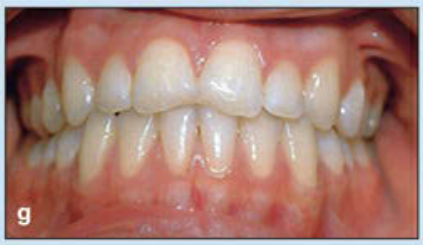
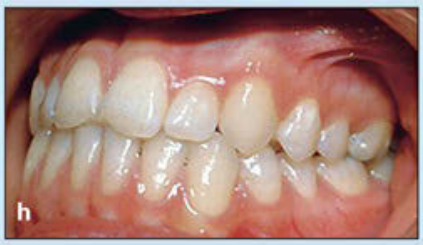
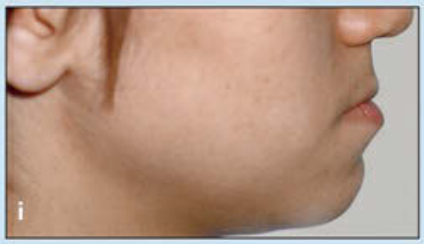
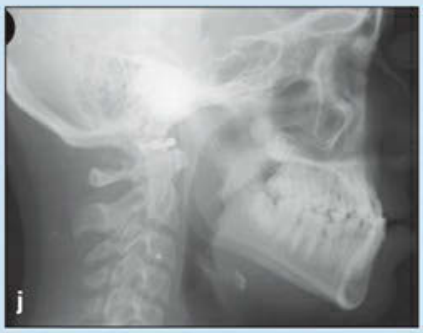
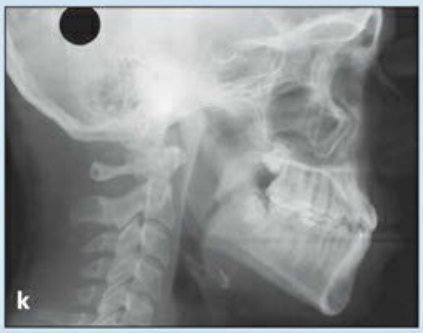


Fig 11-37 Management of severe anterior dentoalveolar crossbite and reverse deep bite in a 16-year, 5-month-old girl. She exhibited two-step occlusion, maxillary anterior crowding, and a mesial mandibular shift. *(a to c)* Pretreatment occlusion in centric occlusion. The dental problems have resulted in structural damage to the maxillary incisors. *(d)* Pretreatment occlusion in centric relation. *(e)* Pretreatment soft tissue profile.

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	95.1	91.5
SNB (°)	78.0	89.2	90.1
SNA (°)	82.0	85.2	90.2
Maxillary depth (FH-NA) (°)	90.0	92.2	91.0
ANB (°)	2.3	–4.0	0.1
FMA (MP-FH) (°)	25.9	21.9	26.8
Y-axis (SGn-SN) (°)	59.4	61.5	61.4
Interincisal angle (U1-L1) (°)	135.0	126.8	128.1
U1-FH (°)	116.2	118.4	123.5
IMPA (L1-MP) (°)	95.0	92.9	81.6
Overbite (mm)	2.5	–1.7	–9.6
Overjet (mm)	2.5	–5.4	13.4

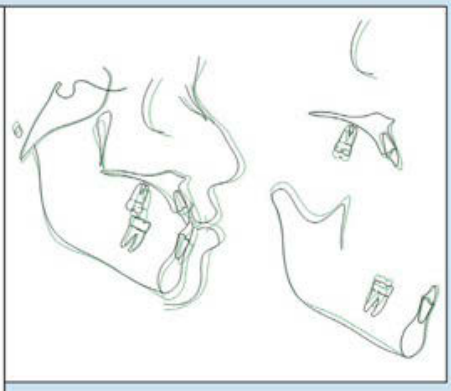


Fig 11-37 *(cont) (f to h)* Posttreatment occlusion. *(i)* Posttreatment soft tissue profile. *(j)* Pretreatment cephalometric radiograph. *(k)* Posttreatment cephalometric radiograph. *(l)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

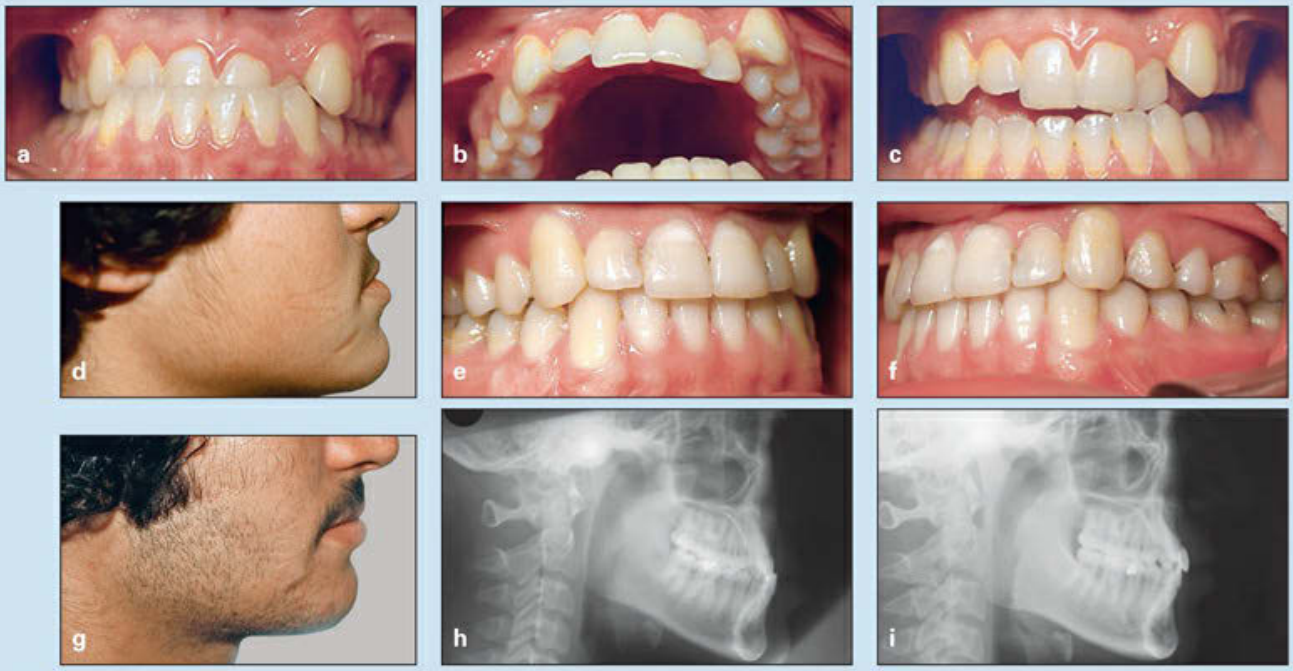
Case 11-25

A 12-year-old boy presented with a severe anterior dentoalveolar crossbite, severe maxillary anterior crowding in which the canines were out of the arch, some maxillary incisor retroclination, a mesial mandibular shift, and good mandibular dentition (Figs 11-38a to 11-38d).

Treatment:
After consideration of the maxillary incisor retroclination, mandibular shift, and good mandibular dentition, the treatment plan was designed as camouflage with a nonextraction approach that was limited to maxillary treatment. The anterior teeth were disoccluded through the use of a maxillary posterior bite block, and maxillary complete bonding was used to align and procline the maxillary incisors

to eliminate crossbite.

Unlike the patient in case 11-24, this patient did experience some positive dentoskeletal changes during treatment (Figs 11-38e to 11-38j): The Landes angle increased from 88.4 to 91.3 degrees; SNA increased from 79.9 to 83.8 degrees; ANB increased from – 2.5 to –0.5 degrees; and overjet increased from –3.3 to 1.3 mm. Presumably these positive changes could be attributed to the 12-year-old boy's jaw growth during treatment.



j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	92.0	94.3
SNB (°)	78.0	82.4	85.3
SNA (°)	82.0	79.9	84.8
Maxillary depth (FH-NA) (°)	90.0	88.4	91.3
ANB (°)	2.3	–2.5	–0.5
FMA (MP-FH) (°)	25.9	20.6	15.4
Y-axis (SGn-SN) (°)	59.4	62.9	63.6
Interincisal angle (U1-L1) (°)	135.0	145.2	133.5
U1-FH (°)	116.2	109.7	123.7
IMPA (L1-MP) (°)	95.0	87.0	88.9
Overbite (mm)	2.5	3.4	0.4
Overjet (mm)	2.5	–3.3	3.0

Fig 11-38 Management of severe anterior dentoalveolar crossbite and maxillary anterior crowding in a 12-year-old boy. The canines are out of the arch, and there is some maxillary incisor retroclination. There is a mesial mandibular shift, but the mandibular dentition is good. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment soft tissue profile. *(e and f)* Posttreatment occlusion. *(g)* Posttreatment soft tissue profile. *(h)* Pretreatment cephalometric radiograph. *(i)* Posttreatment cephalometric radiograph. *(j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 11-26

A 14-year-old girl presented with a severe dentoalveolar anterior crossbite, maxillary and mandibular crowding, minor mandibular incisor proclination, and a mesial mandibular shift (Figs 11-39a to 11-39d).

Treatment:
Because she exhibited severe maxillary and mandibular crowding and some mandibular incisor proclination, and because the incisors could not reach end-to-end contact during centric occlusion or centric relation, the treatment plan called for both camouflage and extraction of four first premolars.

Because of the age of the patient and the lack of significant jaw growth, the results of camouflage treatment were mostly dental, that is, achievement of good occlusion and dental relationship (Figs 11-39e to 11-39h). Comparison of cephalometric evaluations revealed no significant skeletal changes (Figs 11-39i and 11-39j). The major changes were an improvement in the incisor relationships and an improvement in lip relationships, which was due to mandibular incisor retraction. There was also some minor improvement in the sagittal A-B relationship from –3.2 to –1.3 degrees and an increase in overjet from –2.8 to 2.0 mm.

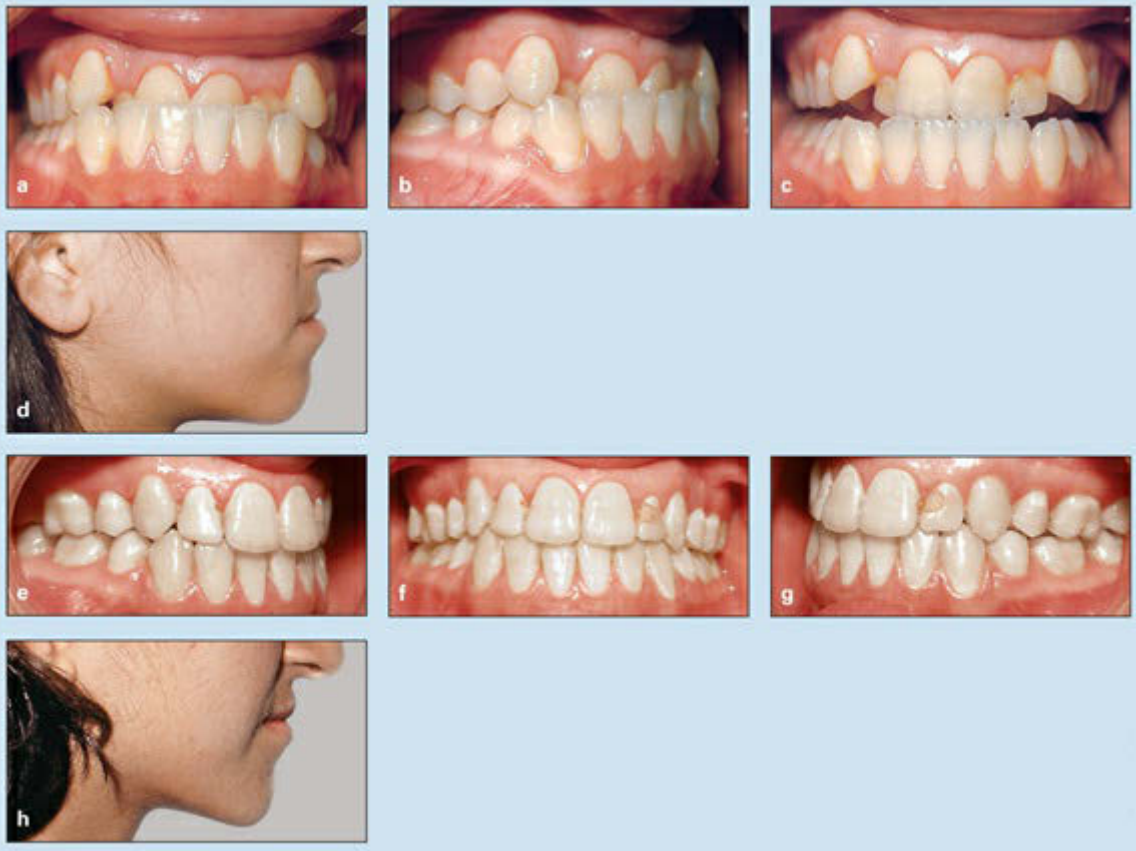
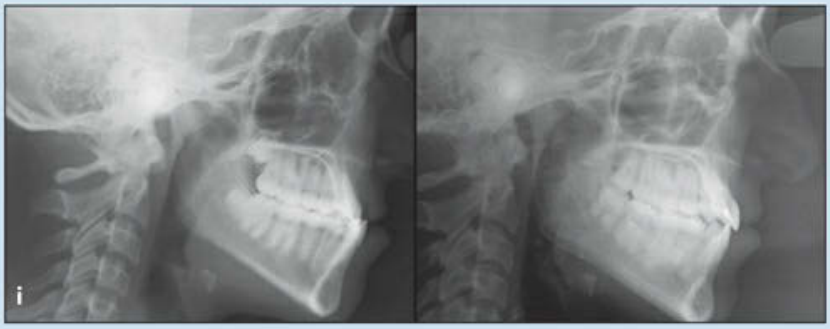
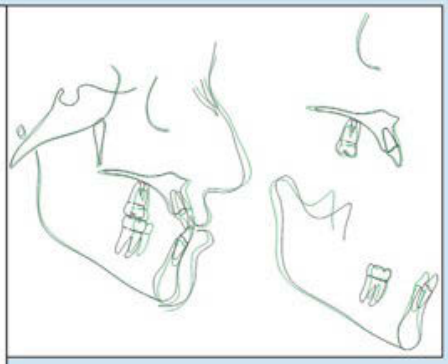


Fig 11-39 Management of dentoalveolar anterior crossbite, maxillary and mandibular crowding, and a mesial mandibular shift in a 14-year-old girl. There is minor proclination of the mandibular incisors. *(a and b)* Pretreatment occlusion in centric occlusion. *(c)* Pretreatment occlusion in centric relation. *(d)* Pretreatment soft tissue profile. *(e to g)* Posttreatment occlusion. *(h)* Posttreatment soft tissue profile.



i

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	86.0	84.8
SNB (°)	78.0	75.1	74.1
SNA (°)	82.0	71.9	72.7
Maxillary depth (FH-NA) (°)	90.0	82.9	82.0
ANB (°)	2.3	–3.2	–1.3
FMA (MP-FH) (°)	25.9	29.5	33.2
Y-axis (SGn-SN) (°)	59.4	72.0	71.4
Interincisal angle (U1-L1) (°)	135.0	130.9	135.9
U1-FH (°)	116.2	112.5	114.4
IMPA (L1-MP) (°)	95.0	87.1	76.6
Overbite (mm)	2.5	2.1	–0.5
Overjet (mm)	2.5	–2.8	2.0



j

Fig 11-39 *(cont) (i)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Summary

- Class II malocclusion accounts for about one-third of the total malocclusions in the US population.
- Long-term studies from the primary to the permanent dentition have shown that Class II abnormalities are not self-correcting and in some cases may worsen.
- Class II discrepancy has mainly been perceived as a sagittal problem; the vertical and transverse dimensions traditionally were not considered to be related to Class II discrepancy.
- Despite that perception, variations in facial height may conceal or intensify the clinical appearance of Class II malocclusion. An increased mandibular vertical dimension associated with Class II malocclusion usually rotates the

mandible downward and backward, which makes the mandible appear retruded and the profile more convex. Any mistake in treatment would worsen the situation. A decreased vertical dimension causes the mandible to rotate upward and forward, manifesting as a prominent chin point.

- Likewise, evaluation of the transverse dimension in patients with Class II malocclusion must not be overlooked. Most Class II patients with a narrow maxillary arch may show normal relationships in centric occlusion. Early intervention of transverse problems and proper expansion at earlier stages will facilitate mandibular forward growth.
- Not all Class II malocclusions are alike; numerous types of skeletal and dental malocclusions may be observed.
- Class II variations are the result of its multifactorial origins. Class II malocclusions have different etiologic factors, different morphologic characteristics, and different treatment approaches.
- Completion of thorough diagnostic procedures to enable recognition of etiologic factors and the affected anatomical structures causing the dentoskeletal malrelationships is the first step for proper treatment planning for correction of Class II malocclusion.
- Treatment strategies differ according to the specific characteristics of the anomaly as well as the skeletal age of the patient. Growth modification strategy is entirely different from treatment tactics applicable after the growth spurt has been completed. Depending on the type of Class II malocclusion and the age of the patient, four treatment options are available for Class II correction: growth modification and occlusal guidance, camouflage treatment, orthognathic surgery, or a combination of these approaches.
- Growth modification and occlusal guidance are interceptive treatments that are applied in growing children during the primary or mixed dentition. Their purpose is to eliminate or minimize the dentoalveolar and skeletal disharmonies that can interfere with the normal growth and development of occlusion.
- Deviations in skeletal growth patterns and disturbed functional matrix environments, such as mouth breathing, atypical swallowing, or other abnormal behaviors that can disrupt jaw growth, require early intervention to eliminate or modify the condition and provide the opportunity to guide dentoalveolar development.
- Several methods of Class II treatment have been used during the last decade, including functional appliances, extraoral force, several versions of the

Herbst appliance, and the Pendulum or Pendex molar distalization appliances. Theoretically, these general approaches enhance mandibular growth and move the maxillary teeth posteriorly.

- Use of headgear in combination with removable or fixed and functional appliances has also been reported by many investigators. As an extraoral device, headgear can exert force on the basal bone and dentition from different directions and produce different occlusal treatment results.
- This chapter has introduced a combination of headgear, a modified Hawley appliance, and a lip bumper—the HLH technique—as an early intervention strategy for treatment of Class II malocclusion.
- The lip bumper has many effects on the mandibular dentoalveolar structure. By controlling muscle balance, the lip bumper can induce sagittal and transverse changes in occlusion.
- When used in conjunction with headgear and a lip bumper, the Hawley appliance, with some modifications, can play several important roles in the correction of Class II malocclusion, including maxillary jaw retraction, maxillary molar retraction, mandibular growth stimulation, and overbite reduction.
- Anterior dental crossbite is a common problem during the primary or mixed dentition. This problem merits early intervention to prevent further damage to the occlusion.
- Early correction of anterior crossbite alleviates esthetic and functional problems and prevents structural damage and adverse growth effects on the dentition and basal bones.
- Generally there are three kinds of anterior crossbite: *(1)* simple dental crossbite, *(2)* functional crossbite (pseudo– Class III malocclusion), and *(3)* skeletal Class III malocclusion. Each of these crossbites has a different etiology, different characteristics, and different treatment approaches; the first step in proper treatment is early detection and differentiation of the problem.
- Simple dental crossbite is a dental malocclusion resulting from lingual positioning of the maxillary anterior teeth with or without buccal tipping of the opposing teeth. The condition is localized and involves the tipping of only a tooth or teeth and not the basal bone. Dental crossbite may or may not be accompanied by a mandibular shift.
- Functional crossbite (pseudo–Class III malocclusion) is a multitooth anterior crossbite in which interferences prevent posterior occlusion. The

response is a functional shift of the mandible in an effort to avoid the anterior interference in centric relation and to achieve maximal intercuspation. These kinds of crossbite usually are not associated with skeletal problems. However, if not treated early, maxillary anterior collapse can induce maxillary growth deficiency and mandibular overgrowth and consequently sagittal jaw discrepancy.

- True Class III malocclusions are associated with a sagittal skeletal discrepancy that results from maxillary deficiency, mandibular prognathism, or both. This abnormality can present with or without incisor crossbite. True skeletal Class III malocclusion may or may not be associated with mandibular shift.
- Dental crossbite and functional crossbites can be corrected very easily by removable or fixed mechanotherapy that changes the incisor relationships. True Class III malocclusion requires more comprehensive therapy with or without rapid palatal expansion and maxillary traction with extraoral force, such as a face mask.
- For all types of anterior crossbite, anterior bite opening is required during correction of incisor relationships. This can be accomplished through the addition of acrylic resin occlusal coverage in the posterior segments of the appliance or placement of composite resin on the molars to build up the occlusal surfaces.

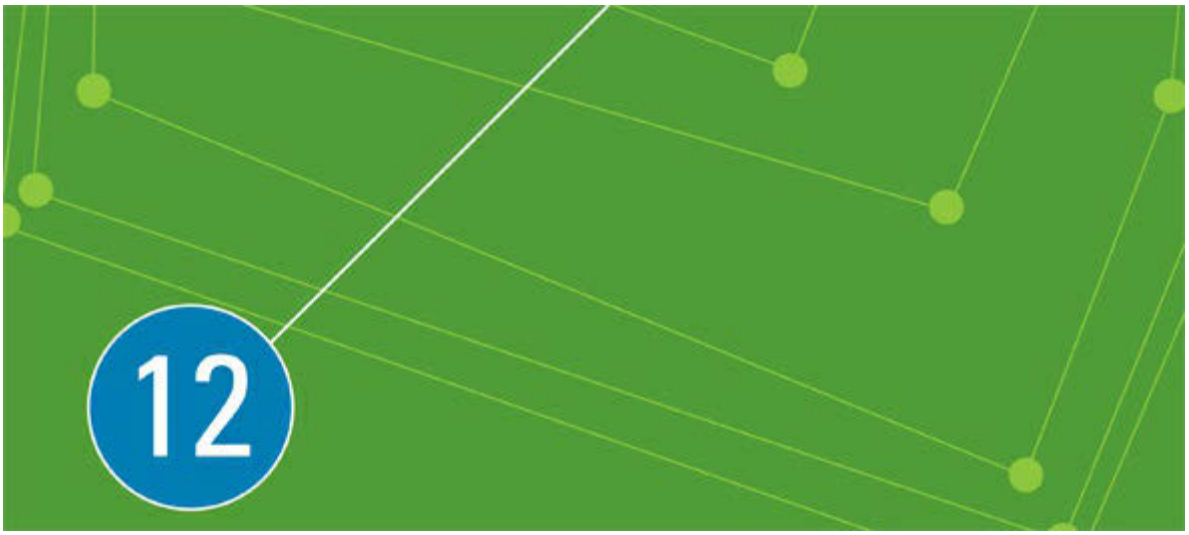
References

1. Harris EF, Johnson MG. Heritability of craniometric and occlusal variables: A longitudinal sib analysis. Am J Orthod Dentofacial Orthop 1991;99:258–268.
2. American Association of Orthodontists. Seven common questions parents have. https://www.aaomembers.org/Press/Seven-Common-Questions-Parents-Have.cfm. Accessed 10 December 2012.
3. Gugino CF, Dus I. Unlocking orthodontic malocclusions: Interplay between form and function. Semin Orthod 1998;4:246–255.
4. Proffit WR, Fields HW Jr. Contemporary Orthodontics, ed 5. St Louis: Mosby, 2012.
5. De Baets J, Schatz JP, Joho JP. Skeletal changes associated with plate-headgear therapy in the early mixed dentition. J Clin Orthod 1995;29:700–705.
6. Bishara SE. Mandibular changes in persons with untreated and treated Class II division 1 malocclusion. Am J Orthod Dentofacial Orthop 1998;113:661–673.
7. Baccetti T, Franchi L, McNamara JA Jr, Tollaro I. Early dentofacial features of Class II malocclusion: A longitudinal study from the deciduous through the mixed dentition. Am J Orthod Dentofacial Orthop 1997;111:502–509.
8. Schudy F. Vertical growth versus anteroposterior growth as related to function and treatment. Angle Orthod

1964;34:75–93.
9. Tollaro I, Baccetti T, Franchi L, Tanasescu CD. Role of posterior transverse interarch discrepancy in Class II, division 1 malocclusion during the mixed dentition phase. Am J Orthod Dentofacial Orthop 1996;110:417–422.
10. Broadbent BH Sr, Broadbent BH Jr, Golden WH. Bolton Standards of Dentofacial Developmental Growth. St Louis: Mosby, 1975.
11. You ZH, Fishman LS, Rosenblum RE, Subtelny JD. Dentoalveolar changes related to mandibular forward growth in untreated Class II persons. Am J Orthod Dentofacial Or-thop 2001;120:598–607.
12. McNamara JA Jr, Hinton RJ, Hoffman DL. Histologic analysis of temporomandibular joint adaptation to protrusive function in young adult rhesus monkeys (Macaca mulatta). Am J Orthod 1982;82:288–298.
13. Keeling SD, Wheeler TT, King GJ, et al. Anteroposterior skeletal and dental changes after early Class II treatment with Bionators and headgear. Am J Orthod Dentofacial Orthop 1998;113:40–50.
14. Tulloch JF, Phillips C, Proffit WR. Benefit of early Class II treatment: Progress report of a two-phase randomized clinical trial. Am J Orthod Dentofacial Orthop 1998;113:62–72.
15. Ghafari J, Shofer FS, Jacobsson-Hunt U, Markowitz DL, Laster LL. Headgear versus function regulator in the early treatment of Class II, division 1 malocclusion: A randomized clinical trial. Am J Orthod Dentofacial Orthop 1998;113:51–61.
16. Ricketts RM. Early treatment. 1. J Clin Orthod 1979;13:23–38.
17. Ricketts RM. Early treatment. 2. J Clin Orthod 1979;13:115–127.
18. Ricketts RM. Early treatment. 3. J Clin Orthod 1979;13:181–199.
19. Subtelny JD. Early Orthodontic Treatment. Chicago: Quintessence, 2000.
20. Bench RW, Gugino CF, Hilgers JJ. Bioprogressive therapy. 11. J Clin Orthod 1978;12:505–521.
21. Graber TM. Extraoral force—Facts and fallacies. Am J Orthod 1955;41:490–505.
22. McNamara JA Jr. Fabrication of the acrylic splint Herbst appliance. Am J Orthod Dentofacial Orthop 1988;94:10–18.
23. Harvold EP,Tomer BS, Vargervik K, Chierici G. Primate experiments on oral respiration. Am J Orthod 1981;79:359–572.
24. Woodside DG, Linder-Aronson S, Lundstrom A, McWilliam J. Mandibular and maxillary growth after changed mode of breathing. Am J Orthod Dentofacial Orthop 1991;100:1–18.
25. McNamara JA Jr. Components of Class II malocclusion in children 8–10 years of age. Angle Orthod 1981;51:177–202.
26. Carlson DS. Biological rationale for early treatment of dentofacial deformities. Am J Orthod Dentofacial Orthop 2002;121:554–558.
27. Kingsley NW. Treatise on Oral Deformities as a Branch of Mechanical Surgery. New York: Appleton & Lange, 1880.
28. Graber TM, Rakosi T, Petrovic AG. Dentofacial Orthopedics with Functional Appliances, ed 2. St Louis: Mosby, 1997.
29. Fränkel R. A functional approach to orofacial orthopaedics. Br J Orthod 1980;7:41–51.
30. Stockli PW. Jaw orthopedics—A biological problem [in German]. SSO Schweiz Monatsschr Zahnheilkd 1973;83: 727–749.
31. Clark WJ. New horizons in orthodontics and dentofacial orthopedics: Fixed Twin Blocks and TransForce lingual appliances. Int J Orthod Milwaukee 2011;22:35–40.
32. Darendeliler MA, Joho JP. Magnetic activator device II (MAD II) for correction of Class II, division 1 malocclusions. Am J Orthod Dentofacial Orthop 1993;103:223–239.
33. Seifi M, Bargrizan M, Memar-Kermani N, Vahid-Dastjerdi D. The skeletal and alveolodental effects of an innovated functional appliance (Seifi functional-phase 1). Iran J Orthod 2007;1:6–12.
34. Oppenheim A. Biologic orthodontic therapy and reality. 3. Angle Orthod 1936;6:5–38.
35. Oppenheim A. Biologic orthodontic therapy and reality. 4. Angle Orthod 1936;6:69–116.
36. Oppenheim A. Biologic orthodontic therapy and reality. 5. Angle Orthod 1936;6:153–183.
37. Kloehn SJ. Guiding alveolar growth and eruption of teeth to reduce treatment time and produce a more balanced denture and face. Angle Orthod 1947;17:10–33.

38. Kloehn SJ. Evaluation of cervical anchorage force in treatment. Angle Orthod 1961;31:91–104.
39. Kopecky GR, Fishman LS. Timing of cervical headgear treatment based on skeletal maturation. Am J Orthod Dentofacial Orthop 1993;104:162–169.
40. Baccetti T, Franchi L, Kim LH. Effect of timing on the outcomes of 1-phase nonextraction therapy of Class II malocclusion. Am J Orthod Dentofacial Orthop 2009;136:501–509.
41. De Oliveira JN Jr, Rodrigues de Almeida R, Rodrigues de Almeida M, de Oliveira JN. Dentoskeletal changes induced by the Jasper jumper and cervical headgear appliances followed by fixed orthodontic treatment. Am J Orthod Dentofacial Orthop 2007;132:54–62.
42. Ferro F, Monsurró A, Perillo L. Sagittal and vertical changes after treatment of Class II division 1 malocclusion according to the Cetlin method. Am J Orthod Dentofacial Orthop 2000;118:150–158.
43. O'Reilly MT, Nanda SK, Close J. Cervical and oblique headgear: A comparison of treatment affects. Am J Orthod Dentofacial Orthop 1993;103:504–520.
44. Gianelly AA. A strategy for nonextraction Class II treatment. Semin Orthod 1998;4:26–32.
45. Moin K, Bishara SE. Evaluation of the effect of buccal shield modification of the lip bumper on dental arch. Angle Orthod 2007;77:57–63.
46. Fishman LS. Radiographic evaluation of skeletal maturation. Angle Orthod 1982;52:88–112.
47. Poulton DR. Changes in Class II malocclusion with and without occipital headgear therapy. Angle Orthod 1959;29:234–249.
48. Tausche E, Luck O, Harzer W. Prevalence of malocclusions in the early mixed dentition and orthodontic treatment need. Eur J Orthod 2004;26:237–244.
49. Estreia F, Almerich J, Gascon F. Interceptive correction of anterior crossbite. J Clin Pediatr Dent 1991;15:157–159.
50. Graber TM, Vanarsdall RL. Orthodontics: Principles and Practice, ed 3. Philadelphia: Saunders, 2000:544–550.
51. Lee BD. Correction of crossbite. Dent Clin North Am 1978;22:647–668.
52. Ngan P, Hu AM, Fields HW Jr. Treatment of Class III problems begins with differential diagnosis of anterior cross-bites. Pediatr Dent 1997;19:386–395.
53. Hannuksela A, Laurin A, Lehmus V, Kauri R. Treatment of cross-bite in the early mixed dentition. Proc Finn Dent Soc 1988;84:175–182.
54. Jacobs SG. Teeth in crossbite: The role of removable appliances. Aust Dent J 1989;34:20–28.
55. Tobias MT, Album MM. Anterior crossbite correction on a cerebral palsy child: Report of a case. ASDC J Dent Child 1977;44:460–462.
56. Valentine F, Howitt JW. Implications of early anterior crossbite correction. ASDC J Dent Child 1970;37:420–427.
57. Proffit WR. The timing of early treatment: An overview. Am J Orthod Dentofacial Orthop 2006;129(4 suppl):47–49.
58. Jirgensone I, Liepa A, Abeltins A. Anterior crossbite correction in primary and mixed dentition with removable inclined plane (Bruckl appliance). Stomatologija 2008;10:140–144.
59. Rabie AB, GuY. Diagnostic criteria for pseudo–Class III malocclusion. Am J Orthod Dentofacial Orthop 2000;117:1–9.
60. Gu Y. The characteristics of pseudo Class III malocclusion in mixed dentition. Zhonghua Kou Qiang Yi Xue Za Zhi 2002;37:377–380.
61. Moyers RE. Handbook of Orthodontics, ed 4. Chicago: Year Book Medical, 1988.
62. Tulley WJ, Campbell AC. A Manual of Practical Orthodontics, ed 3. Bristol, England: John Wright and Sons, 1970:232–239.
63. Nakasima A, Ichinose M, Takahama Y. Hereditary factors in the craniofacial morphology of Angle's Class II and Class III malocclusions. Am J Orthod 1982;82:150–156.
64. Gu Y, Rabie AB. Dental changes and space gained as a result of early treatment of pseudo–Class III malocclusion. Aust Orthod J 2000;16:40–52.
65. Sharma PS, Brown RV. Pseudo mesiocclusion: Diagnosis and treatment. ASDC J Dent Child 1968;35:385–

392.
66. Bahreman AA. Modified Hawley appliance for better retention of cuspids. J Clin Orthod 1977;11:689.
67. Arvystas MG. The rationale for early orthodontic treatment. Am J Orthod Dentofacial Orthop 1998;113:15–18.
68. Ngan P, Hägg U, Yiu C, Merwin D, Wei SHY. Soft tissue and dentoskeletal profile changes associated with maxillary expansion and protraction headgear treatment. Am J Orthod Dentofacial Orthop 1996;109:38–49 [erratum 1996;109:459].
69. Angle EH. Treatment of Malocclusion of the Teeth, ed 7. Philadelphia: S.S. White, 1907.
70. Nakasima A, Ichinose M, Nakata S. Genetic and environmental factors in the development of so-called pseudo– and true mesiocclusions. Am J Orthod Dentofacial Orthop 1986;90:106–116.
71. Haynes S. The prevalence of malocclusion in English children aged 11-12 years. Rep Congr Eur Orthod Soc 1970:89–98.
72. Endo T. An epidemiological study of reversed occlusion. 1. Incidence of reversed occlusion in children 6 to 14 years old [in Japanese]. J Jpn Orthod Soc 1971;30:73–77.
73. Susami R, Asai Y, Hirose K, Hosoi T, Hayashi I. The prevalence of malocclusion in Japanese school children [in Japanese]. J Jpn Orthod Soc 1972;31:319–324.
74. Chan GK. Class III malocclusion in Chinese (Cantonese): Etiology and treatment. J Orthod 1974;65:152–156.
75. Baik HS, Han HK, Kim DJ, Proffit WR. Cephalometric characteristic of Korean Class III surgical patients and their relationship to plans for surgical treatment. Int J Adult Orthodont Orthognath Surg 2000;15:119–128.
76. Salzmann JA. Practice of Orthodontics, vol 1. Philadelphia: Lippincott, 1966.
77. Tweed CH. Treatment planning and therapy in mixed dentition. Am J Orthod 1963;49:881–906.
78. Björk A. Variations in the growth pattern of the human mandible; Longitudinal radiographic study by the implant method. J Dent Res 1963;42:400–411.



Management of Transverse Problems (Posterior Crossbites)

Moyers[1] defines *posterior crossbite* as a term used to indicate an abnormal buccolingual (labiolingual) relationship of the teeth. In other words, it is a deviation from ideal occlusion in the transverse plane of space in posterior segments. This anomaly can occur between one single posterior tooth or a group of teeth, unilaterally or bilaterally; it can be dental, skeletal, or a combination of both, and it can result from various combinations of transverse malrelationships between the maxillary or mandibular dental arch.

Posterior crossbites are not self-correcting (except some during the primary dentition caused by premature contacts) and in some situations worsen during later stages of the dentition; therefore, they warrant early detection and intervention. This chapter discusses the characteristics and management of posterior crossbites, especially those accompanied by mandibular shift.

Morphologic Characteristics

Posterior crossbite is one of the most prevalent malocclusions in the primary and

early mixed dentitions, with a reported incidence of 7% to 23%.[2] Posterior crossbite is usually accompanied by a mandibular shift from rest to occlusion, also known as *forced occlusion*, causing midline deviation. The most common form of posterior crossbite is a unilateral presentation with a functional shift of the mandible toward the crossbite side.[3] Posterior crossbite during the primary or early mixed dentition is usually due to a slight bilateral constriction of the maxillary arch, which, because of the functional shift to one side, looks unilateral. According to Kutin and Hawes,[4] the prevalence of functional crossbite is 8.4% during the primary dentition stage and drops to 7.2% during the mixed dentition stage. This reduction can be the result of self-grinding of premature contacts.

Reports indicate that posterior crossbites are very seldom self-correcting; delayed treatment and asymmetric muscle strain on orofacial structures can have adverse effects on the temporomandibular joint and masticatory system and cause skeletal growth changes.[5,6] Tomographic studies have shown that the asymmetric position of the condyle within the fossa in children with unilateral crossbite is restored after early treatment.[5–7]

Maxillary arch constriction, which is usually present in children with this condition, produces insufficient maxillary arch width, causing anterior crowding and sometimes canine impaction. Early treatment is advised to create an environment suitable for normalizing occlusal development.[1,4,8,9]

Etiology

Several theories concerning the etiology of posterior crossbite have been proposed. This anomaly has been associated with hereditary or congenital conditions, such as congenital developmental anomalies (cleft palate or asymmetric growth of the maxilla or the mandible), as well as some pathologic conditions, such as arthritis, acromegaly, muscular dystrophy, condylar hypoplasia or hyperplasia, and osteochondroma.[9–11] Posterior crossbite has also been attributed to local factors such as premature loss or prolonged retention of primary teeth, crowding, and abnormalities in eruption sequence.

A review of the literature revealed that most investigators[12–15] agree that the following are the most common causes of posterior crossbite:

- Digit or pacifier sucking habits

- Impaired nasal breathing
- Atypical swallowing patterns
- Low tongue position
- Combination of factors

In general, in posterior crossbite, skeletal, muscular, or dental factors or a combination of these might be involved under the influence of genetic, congenital, environmental, functional, or habitual origins.

Allen et al[16] evaluated the skeletal contribution to posterior crossbite and found that the two variables often associated with the anomaly were a smaller ratio of maxillary to mandibular intermolar dental width and a greater lower facial height. A small maxillomandibular width ratio may arise from genetic or environmental factors.

As an example of genetic factors, Fig 12-1 shows the dental occlusions of identical twin sisters, both of whom exhibited unilateral posterior crossbite, one on the left side and one on the right side.

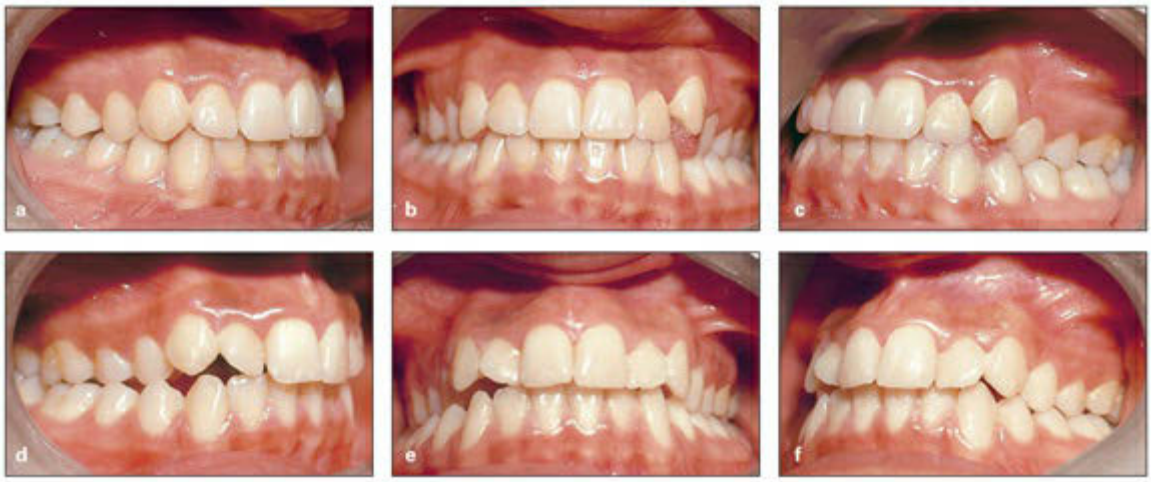


Fig 12-1 Dental occlusion of identical twin sisters. *(a to c)* One twin has posterior crossbite affecting the left side. *(d to f)* The other twin shows posterior crossbite on the right side.

Besides the hereditary and congenital causes that are rarely the cause of this anomaly, the other previously mentioned factors are the most common local causes, with an especially high prevalence during the primary and early mixed dentitions. Thus, practitioners have a responsibility to perform careful examinations and thereby ensure early recognition of these factors, which are interacting in young patients during a critical stage of occlusal development.

Many reports indicate that non-nutritive sucking habits are a prevalent cause of the

development of posterior crossbite. For example, Warren and Bishara[17] monitored the sucking habits of 372 children from birth to 4 to 5 years of age and found that the greatest dental and skeletal changes happened when the habit continued beyond 48 months. Of the children who continued thumb sucking beyond 48 months, 29% showed posterior crossbites. They also reported that children using pacifiers beyond 24 months showed a significant increase of posterior crossbite.

Many investigators have reported the effects of mouth breathing on the child's head, jaw, and tongue posture as well as occlusion. Souki et al[18] studied 401 children aged 2 to 12 years. The children's mouth breathing was confirmed by otorhinolaryngologists, who reported a higher prevalence of posterior crossbite, anterior open bite, and Class II malocclusion in mouth breathers.

Nasal obstruction and mouth breathing, which are prevalent during the primary and early mixed dentitions as a result of hypertrophied adenoids or tonsils and allergic rhinitis, are important causes of maxillary constriction and posterior crossbite. Therefore, when a child with posterior crossbite is examined, the assessment should include evaluation of nasal respiration as well.

Oulis et al[19] studied the incidence of maxillary posterior crossbite in a sample of 120 children who displayed hypertrophied adenoids with or without enlarged tonsils and underwent adenoidectomy. A lateral cephalometric radiograph was obtained for each patient to relate the presence of crossbite to the severity of upper respiratory airway obstruction. Results indicated that 47% of the children examined had developed a posterior crossbite.

In a large study of 3-year-old Scandinavian children, Ogaard et al[20] compared previous or continuing finger sucking and pacifier habits with crossbite. The study found that pacifier use was associated with increased mandibular intercanine width, decreased maxillary intercanine width, and increased prevalence of posterior crossbite. In a study of 2- to 5-year-old subjects, Adair et al[21] also reported a significantly higher prevalence of posterior crossbite in children when a pacifier was used. The results also indicated that both pacifiers and prolonged digit sucking, particularly if extended beyond age 4 years, are strongly associated with the development of posterior crossbite. In an epidemiologic study of finger sucking in 2- to 6-year-old American children, Infante[22] found a significant association with posterior crossbite.

Prevalence

The prevalence of posterior crossbite in the primary and mixed dentitions has been reported with a wide range; generally, studies have found that unilateral crossbite coupled with a lateral mandibular shift have a greater prevalence relative to other types of crossbite.

In a sample of 898 4-year-old Swedish children, Thilander and Lennartsson[9] identified crossbites in 9.6% of the children. Kutin and Hawes[4] studied 238 nursery school and 277 second grade children and found some form of posterior crossbite in 8% of 3- to 5-year-old children and 7.2% of 7- to 9-year-old subjects. The prevalence of crossbite did not differ greatly between girls and boys. Hanson et al[23] studied 227 children aged 3 to 5 years and found that the percentage of children with posterior crossbites exceeded 23%.

Consequences of Delayed Treatment

Many reports have indicated that serious defects can result from neglect or delays in treatment of posterior crossbite. Abnormal movement of the mandible resulting from mandibular shift can place a special strain on orofacial structures, causing adverse effects on the temporomandibular joint and masticatory system and asymmetric mandibular growth.

Pinto et al[6] evaluated the morphologic and positional mandibular asymmetry of young patients with unilateral functional posterior crossbite at the initiation of treatment and approximately 6 months after the retention phase. They used sonograms to assess articular joint spaces and sub-mental vertex radiographs to assess morphologic and positional asymmetry. The researchers concluded that unilateral posterior crossbites produce morphologic and positional asymmetries of the mandible in young children and that these asymmetries can be largely eliminated with early expansion therapy.

Posterior crossbites are usually accompanied with some maxillary constriction and therefore can create insufficient maxillary arch width and space deficiencies in the anterior segment. In some cases, these conditions may result in canine impaction or ectopic eruption and destruction of adjacent teeth (Fig 12-2). Early correction is advised to normalize the occlusion and prepare an environment for normal occlusal development.

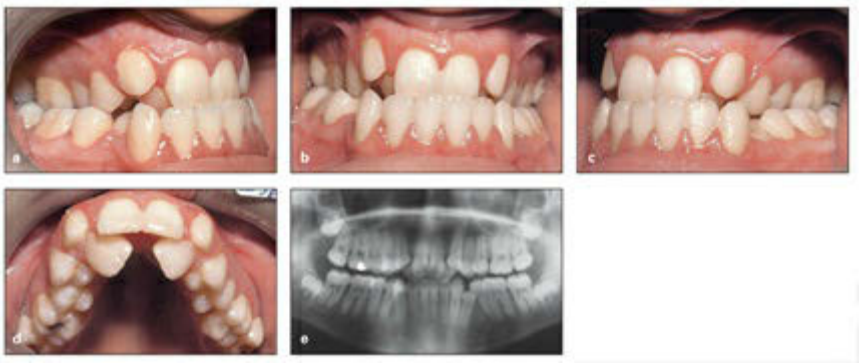


Fig 12-2 *(a to e)* Neglected posterior cross-bite, causing crowding and ectopia of the canines and destruction of the lateral incisor roots.

Primozic et al[24] assessed facial and cast asymmetry using three-dimensional laser scanning before and after 6 months of treatment in 30 children with posterior crossbite. They also examined 28 children without crossbite for comparison. The children with crossbite had statistically significantly greater asymmetry of the face, especially the mandibular third, and a significantly smaller palatal volume than did the subjects without crossbite. The researchers also found that treatment of a crossbite in the primary dentition corrected the facial asymmetry at a later age.

Kennedy and Osepchook[2] reviewed the literature regarding unilateral crossbite and concluded that unilateral crossbites with mandibular shift are not self-correcting; can cause skeletal, dental, and muscle adaptation; and have some association with temporomandibular disorders. Kennedy and Osepchook[2] also recommended early intervention for crossbite, which can be difficult to treat in adults without a combination of orthodontics and surgery.

Advantages of Early Treatment

The foundation of dental occlusion starts when the primary first molars reach occlusion. Many dentoskeletal abnormalities start and develop during the primary and early mixed dentitions and are recognizable at this age. Considering the damage to dentoskeletal structures, arch length, and mandibular growth symmetry that can arise from delayed correction of posterior crossbite, almost all investigators recommend early intervention. Correction of posterior crossbites in the permanent dentition is much more complicated and in some cases may require surgery.

The best time for correction of posterior crossbite is during the late primary or early mixed dentition; technically the expansion procedure is easy and fast and

provides good and stable results. Almost all posterior crossbites at these ages are bilateral because symmetric constriction of the maxillary arch causes a unilateral mandibular shift toward one side that looks like a unilateral crossbite. Treatment involves symmetric bilateral expansion.

Early correction of posterior crossbites is simple, efficient, and stable and has the following advantages:

- Treatment prevents adverse effects on growth and mandibular asymmetry.
- It establishes a better environment for occlusal development. • Expansion unlocks the occlusion and promotes better function.
- Expansion creates more space for the anterior dentition and especially prevents canine impaction.
- Rapid palatal expansion not only corrects posterior crossbites but also increases nasal airway capacity, which is an important benefit for nasal breathing problems that are common at these ages.
- Treatment also prevents adverse growth effects on the nasomaxillary complex.

Variations of Posterior Crossbite

Posterior crossbites have many etiologic factors and several different morphologic characteristics; thus, differential diagnosis is an essential part of treatment planning. Posterior crossbite can be dental, dentoalveolar, skeletal, or functional; each of these types can be unilateral or bilateral (Fig 12-3). Posterior crossbite can also result from other combinations of transverse malrelationships between the maxillary or mandibular dental arch. These include maxillary constriction or maxillary overexpansion, mandibular constriction or mandibular overexpansion, or a combination of these. These types of posterior transverse abnormalities are distinguished as *nonbite* or *buccal bite*, with lack of occlusal contact. Depending on the type of occlusion and number of teeth involved, different terms, including *piston bite*, *Brodie syndrome*, or *scissors bite*, have been used.

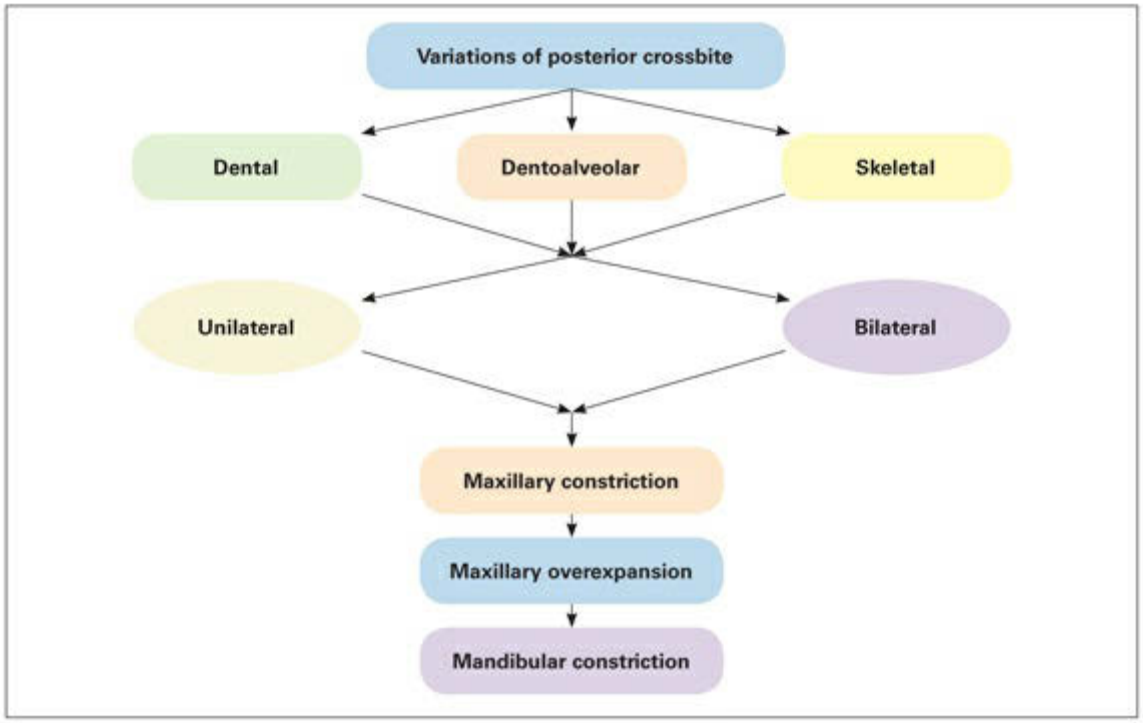


Fig 12-3 Variations of posterior crossbite.

Functional crossbite

Functional posterior crossbite is a kind of crossbite caused by an occlusal interference that forces the mandible to shift laterally to achieve maximum contact in occlusion. This is a common type of posterior crossbite observed during the primary or mixed dentition (Fig 12-4).

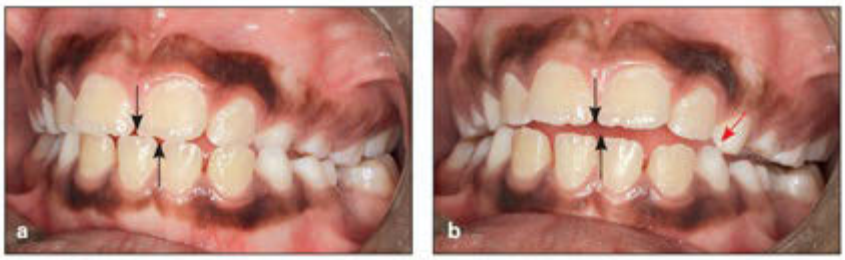


Fig 12-4 Functional shift causing unilateral posterior crossbite due to premature contact of the maxillary and mandibular primary canines. *(a)* Centric occlusion. *(arrows)* Midline deviation. *(b)* Centric relation. *(black arrows)* Coincident midlines, showing mandibular shift needed to achieve maximum contact in occlusion. *(red arrow)* Primary contact causing the shift.

Maxillary constriction is the most common cause of functional shift and unilateral posterior crossbite during the primary or early mixed dentition. Careful clinical and cast evaluations will reveal this situation and confirm symmetry of the maxillary arch; early intervention and bilateral expansion prevent functional shift and cure

unilateral crossbite (Fig 12-5; see case 12-2).

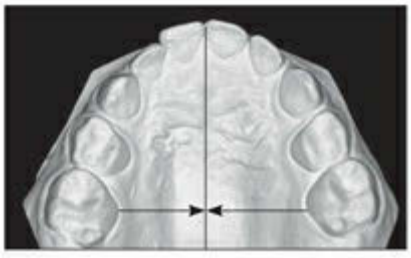


Fig 12-5 Symmetric maxillary constriction, confirmed by vertical and horizontal rules.

Buccal crossbite

Buccal crossbite, or *buccal bite*, is caused by a buccal displacement of one or multiple maxillary teeth in relation to the opposing tooth or teeth in the posterior segment of the arch. In this type of anomaly, if the affected area includes all left or right teeth, or both posterior segments, the mandibular arch is collapsed within the maxilla; this condition is also called *piston bite*, *scissors bite*, or *Brodie syndrome* (Fig 12-6).

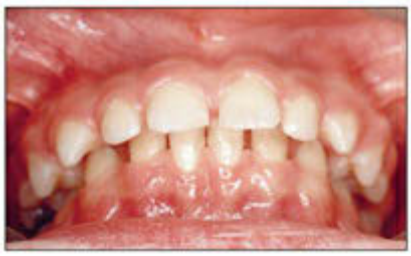


Fig 12-6 Brodie syndrome form of buccal crossbite.

Lingual crossbite

Lingual crossbite is caused by lingual displacement of one or multiple mandibular teeth in relation to the opposing tooth or teeth in the posterior segment of the arch. If the affected area includes all left or right teeth, or both posterior segments of mandibular teeth, the condition is also called *complete mandibular collapse*, *piston bite*, or *Brodie syndrome* (Fig 12-7).

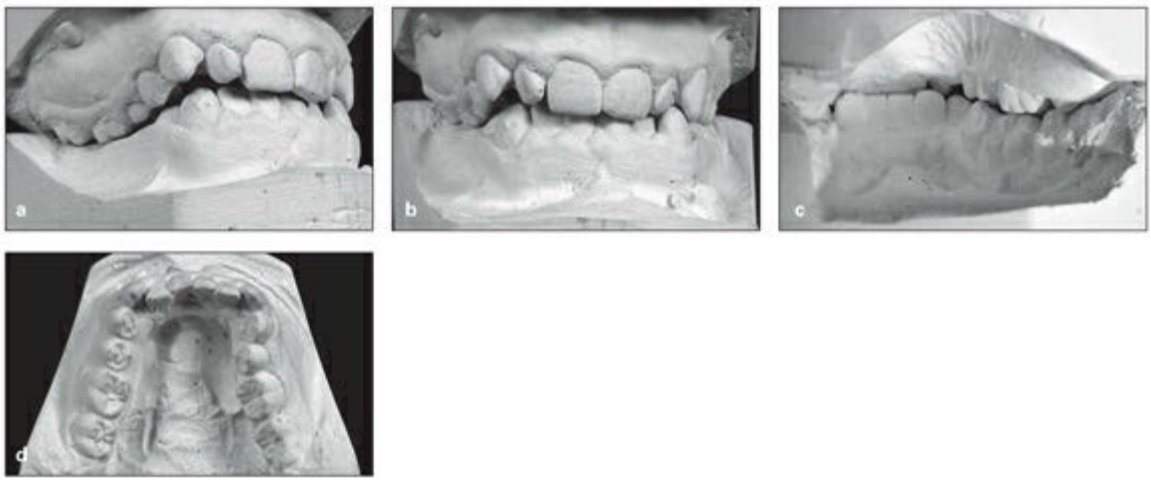


Fig 12-7 *(a to d)* Unilateral lingual crossbite resulting from the lingual position of the mandibular right segment.

Palatal crossbite

Palatal crossbite is caused by the palatal displacement of one or multiple maxillary teeth in relation to the opposing mandibular tooth or teeth, as a result of the narrower maxillary arch. If the affected area includes all left or right teeth or both posterior segments of the maxilla, it is called *complete maxillary collapse* or *scissors bite* (see Fig 12-2b).

Scissors bite

Scissors bite occurs when one or more posterior teeth are positioned completely buccally or lingually to the opposing teeth and exhibit a vertical overlap in one arch.

Brodie syndrome

One of the most complex and difficult transverse abnormalities is a nonbite malocclusion called *Brodie bite*, *Brodie syndrome*, *scissors bite*, or *nonbite occlusion*. In this abnormality, there is no contact between maxillary and mandibular posterior occlusal surfaces. This anomaly can affect just the molars or it can extend to the premolar and canine areas. Brodie syndrome occurs in 1.0% to 1.5% of the population.[25] Traditionally, this anomaly has been treated with extractions, dental arch compensations, or orthognathic surgery.

During the past 20 years, distraction osteogenesis has been shown as a viable

treatment modality to correct skeletal problems, including Brodie syndrome with transverse mandibular deficiency and mandibles that are locked within the maxilla. Mandibular expansion has been achieved with midsymphyseal osteotomy followed by gradual expansion with a special expander screw.

To correct unilateral scissors bite, Guerrero et al[25] recommended a parasymphyseal osteotomy on the ipsilateral side. Legan[26] suggested using cross-arch elastics to increase or inhibit expansion in patients with asymmetry. The patient in that report was also treated with distraction osteogenesis that included a custom-made hybrid distractor.

Nonsurgical correction of bilateral Brodie syndrome is presented in case 12-8.

Unilateral posterior crossbite may also be caused by asymmetric mandibular growth.

Differential Diagnosis

Recognition of posterior crossbites is not difficult, and a simple clinical evaluation of occlusion can easily detect the problem. However, because this anomaly can have various morphologic characteristics, careful differential diagnosis is an essential part of treatment planning. To determine what type of crossbite is present, it is necessary to answer the following questions:

- Is there a functional jaw shift on closing?
- Is the crossbite unilateral or bilateral?
- Is the problem dental, skeletal, or a combination?
- Is the problem related only to the maxilla or the mandible or is it associated with both jaws?
- Is mandibular asymmetry present?

Careful clinical and paraclinical examinations are essential to differentiate the kind of posterior crossbite that affects the patient.

Clinical examination

Orthodontic clinical examination of a child for any type of dentoskeletal abnormality includes a thorough evaluation of orofacial structures both in stasis and in function

(see chapter 3). Lack of harmony of centric occlusion and centric relation is particularly important in the diagnosis of mandibular shift problems.

True unilateral constriction results in a unilateral posterior crossbite both in centric relation and in centric occlusion, without a lateral shift. This problem is best treated with unilateral posterior expansion.

Specific areas must be considered in the clinical examination and differential diagnosis of a child with posterior crossbite:

- Facial pattern, proportion, and symmetry
- Occlusion in three dimensions
- Facial and maxillary dental midline relationships
- Maxillary and mandibular midline relationships both at rest and in centric occlusion
- Curve of Wilson
- Symmetry of the chin to the face at centric occlusion and centric relation

An asymmetric chin and lack of coincidence of the facial midline and chin can be a sign of mandibular asymmetry. This deformity can be the result of mandibular shift, condylar hyperplasia, or condylar hypoplasia. It is very important to detect this type of anomaly at an early age.

Inspection of the curve of Wilson (the horizontal plane between the mandibular right and left occlusal surfaces) can be helpful in determining mandibular asymmetry and left and right ramal heights.

Differentiating between hypertrophy and hypotrophy of condylar growth is essential in diagnosis and treatment planning; each anomaly has special characteristics.

Condylar hypertrophy

Condylar hypertrophy of one side of the mandible has the following characteristics (Fig 12-8):

- Chin shift to the opposite side
- Lower level of occlusal surface of the affected side (due to longer ramus)
- Class III tendency on the affected side

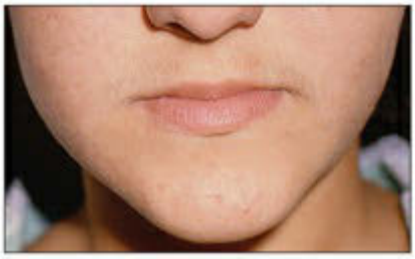


Fig 12-8 Facial asymmetry resulting from hypertrophy of the right condyle.

Condylar hypotrophy

Condylar hypotrophy of one side has the following characteristics:

- Chin shift to the affected side
- Higher level of occlusal surface of the affected side (due to shorter ramus)
- Class II tendency on the affected side (due to hypotrophic growth of the affected side)

Paraclinical evaluations

Clinical inspections are not complete without a thorough cast evaluation and radiographic assessment (lateral, posteroanterior, and oblique cephalometric radiographs). Lack of harmony of centric occlusion and centric relation is an important sign in the diagnosis of mandibular shift that can be detected by comparison of posteroanterior cephalometric radiographs. Assessment of lateral and posteroanterior cephalometric radiographs can be helpful for identifying skeletal problems, skeletal asymmetry in skeletal structures, and the transverse situation of dental arches, including situations such as buccal bite or facial asymmetry.

Careful evaluation of the cast through direct observation and measurements with compass and symmetrograph can reveal the following information:

- Type of occlusion
- Maxillary and mandibular arch forms
- Maxillary and mandibular arch widths
- Any asymmetry in the dental arches

For more detail, see the section on evaluation of study casts in chapter 3.

Early Treatment Strategies

Early correction of posterior crossbite, especially in patients who exhibit a mandibular shift, is recommended by almost all investigators. The best age for correction of posterior crossbites is the late primary dentition or early mixed dentition. The best treatment option depends on the answers to the following questions:

- Is the problem unilateral or bilateral?
- Is the problem dental or skeletal?
- Is the maxillary or mandibular arch involved or are both arches involved?
- Is the problem the result of overexpansion or constriction of the arch?
- Is the treatment of choice slow expansion or rapid expansion?

After careful clarification of the problem, the clinician can determine the mechanical design and type of appliance that should be applied. A variety of fixed and removable expanders with different kinds of functions, such as rapid expansion or slow expansion, are available.

Another important consideration before correction of posterior crossbites is to determine the patient's vertical status (vertical or horizontal growth pattern); posterior expansion usually causes some extrusion of the posterior tooth segment, bite opening, and mandibular clockwise rotation. To overcome this problem, the appliance must be designed with posterior occlusal coverage to prevent posterior extrusion. In patients with deep bite or a horizontal growth pattern, palatal expansion not only can correct the posterior crossbite problem but also can reduce the overbite. In contrast, in patients with open bite or open bite tendency, posterior expansion without control of the vertical dimension, such as occlusal coverage, can worsen the vertical problem.

Appliance therapy

Depending on the type of problem, the age of the patient, and the patient's compliance, the appliances selected for treatment of posterior crossbite problems can be fixed or removable, rapid or slow expanders.

Fixed expanders

Fixed expanders available for rapid or slow expansion include the W-arch, quad helix, Haas, and hyrax expanders, among others.

W-arch. The W-arch, or Porter, appliance is a fixed expander; it is efficient for the correction of posterior crossbites and is ideal for bilateral expansion. It includes two bands for the permanent first molars or any distal teeth in the arch (primary or permanent) and a 0.036- or 0.040-inch stainless steel archwire soldered to the bands. The arch should be free of tissue to prevent tissue impingement (Figs 12-9 and 12-10).

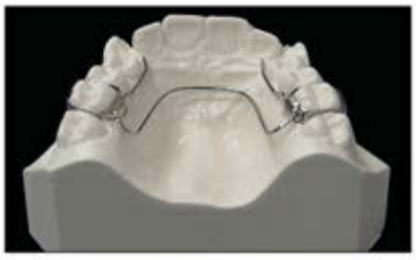


Fig 12-9 W-arch expander. (Courtesy of Great Lakes Orthodontics.)

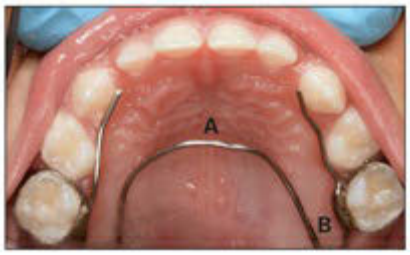


Fig 12-10 W-arch fixed palatal expander in position. A—Activation in the center of the palatal bow expands the molars; B—activation in the U-loop, close to the bands, expands the buccal segments.

The W-arch can be activated in the center of the palatal bow for expansion of the molars or on the U-loop close to the bands to activate the arms for expansion of buccal segments. Depending on the type of crossbite, the appliance can be activated unilaterally or bilaterally. It should be activated about 3 to 4 mm before fixation. The appliance is activated approximately every 4 or 5 weeks until the cross-bite has been corrected and then remains in the mouth, inactive, for 4 to 6 months to provide retention. The W-arch requires little patient cooperation and therefore has better patient compliance.

When used at an early age during the primary dentition, the appliance seems to provide efficient palatal expansion or accelerate the rate of normal expansion of the midpalatal suture. In children with posterior crossbite associated with thumb sucking, the W-arch can simultaneously function as a habit controller. The W-arch is

a suitable expander in 3- to 5-year-old children, especially in situations where activation of a fixed expander, such as the Haas appliance, is difficult for the parent.

Quad helix expander. The quad helix is also a fixed expander; it is fabricated from 0.036-inch stainless steel wires soldered to molar bands.The quad helix is more flexible than the W-arch; because four helixes are incorporated in the arch, the range and springiness of the appliance are increased (Fig 12-11).The wire should contact the teeth involved in the crossbite and should be slightly separated from the palate to prevent any soft tissue impingement. The distal helixes are about 2 to 3 mm distal to the molar bands. Activation of the anterior helixes produces posterior expansion, while activation of the distal helixes produces buccal segment expansion.

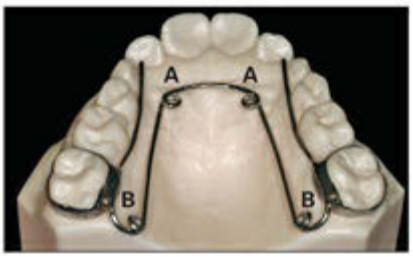


Fig 12-11 Quad helix expander. A—Activation of the anterior helixes produces posterior expansion; B—activation of the posterior helixes expands the buccal segments.

Reactivation can be performed to some extent intraorally by bird beak pliers, but it is difficult to activate the appliance in the desired direction. It is recommended that the appliance be removed for correct activation every 6 to 8 weeks.

The quad helix is used to correct bilateral maxillary constriction in the primary dentition. However, through some modifications to the appliance and the elimination of one extension arm, the quad helix can also work unilaterally. In situations of severe unilateral deformity, a cross elastic from the mandibular opposing tooth can be added to quad helix therapy, especially if teeth in both arches contribute to the problem.

In some instances, by extension of the arms to include the canine, lateral incisors, and central incisors, anterior crossbites can be also corrected.

The combination of a posterior crossbite and a finger-sucking habit is the best indication for this appliance because the anterior helix can effectively serve as a reminder to the patient to stop the finger sucking habit.

To achieve tongue control, a tongue crib can be added to the anterior bow (Fig 12-12).

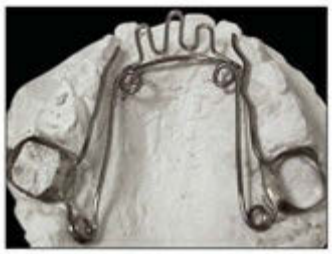


Fig 12-12 Combination quad helix and tongue guard.

Arnold expander. The Arnold expander is a fixed expander consisting of two molar bands on the permanent first molars with two soldered bars (0.040-inch stainless steel wire) touching the primary molars or premolars, a 0.040- or 0.045-inch tube soldered to one side, and a wire soldered to the other side with push coil to expand the anterior segment (Fig 12-13). This appliance has good results for anterior expansion.

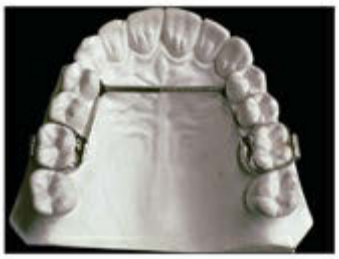


Fig 12-13 Arnold expander.

Haas expander. The Haas expander is also a fixed expander but is attached to both the left and right buccal segments of the arch as anchorage and has a screw embedded in the palatal acrylic resin. The screw can be activated once or twice a day; this rapid force, dual-side anchorage, and expansion force will open the palatal suture (Fig 12-14).

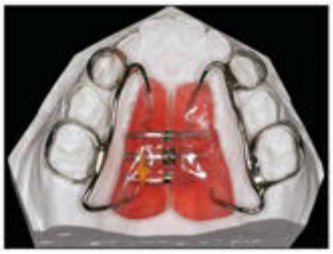


Fig 12-14 Haas rapid palatal expander.

Rapid palatal expansion is a specific type of expansion that can increase the palatal width skeletally. Rapid palatal expansion has several indications in orthodontic and orthopedic treatments, such as skeletal crossbite and constricted

maxilla, maxillary protraction, and maxillary deficiency. This type of expander has been used during the primary or early mixed dentition with good results.

Hyrax expander. The Hyrax expander is also a type of fixed rapid expander, but the screw is not embedded in acrylic resin and is separated from the palate. Thus, the Hyrax is more hygienic than the Haas expander (Fig 12-15).

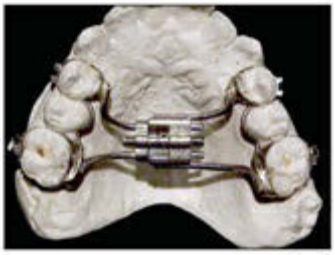


Fig 12-15 Hyrax rapid palatal expander.

Rapid expander with occlusal coverage. It has been established that expansion of posterior segments of the arch reduces anterior overbite; therefore, transverse expansion must be applied cautiously in patients with open bite tendency and vertical problems. One tactic to avoid this outcome is occlusal coverage of the posterior segment.

Figure 12-16 shows an example of this type of expander, which the author has used in the past. It is a combination of the Haas appliance and bite block acrylic resin that covers the occlusal surfaces and is cemented to the buccal segments.

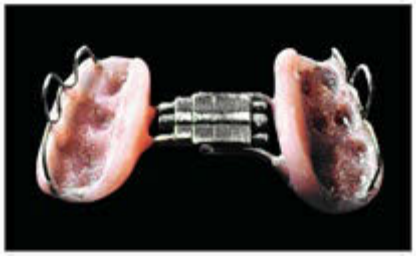


Fig 12-16 Rapid palatal expander with occlusal coverage.

Bonded expander. The bonded expander was introduced by McNamara.[15] This appliance is a combination of Hyrax expander and two acrylic resin parts covering the buccal segments (Fig 12-17).

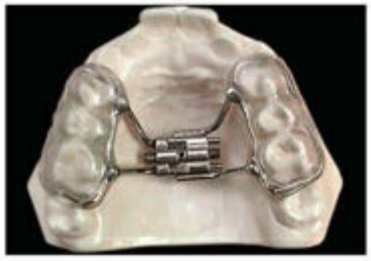


Fig 12-17 Bonded rapid palatal expander.

Removable expanders

Removable expanders are also available for palatal and mandibular expansion. Removable expanders are a slow type of expander and usually are applied for dental or dentoalveolar expansion, not palatal expansion (Fig 12-18). In slow expansion, the screw is opened every 3 to 5 days, in contrast to rapid expanders, which are opened once or twice a day.

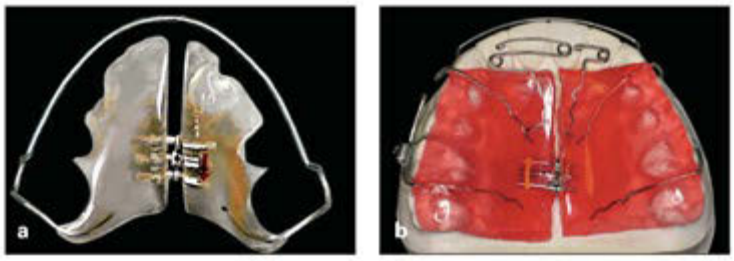


Fig 12-18 Removable Hawley slow expanders with jackscrew. *(a)* Without occlusal coverage. *(b)* With occlusal coverage.

Most posterior crossbites in the primary or mixed dentition that are associated with mandibular shift are caused by bilateral constriction of the maxillary arch. Hence, the removable expander is constructed with the screw on the center of the appliance. In true unilateral constriction, the jackscrew is placed asymmetrically, and the appliance is sectioned asymmetrically. This has the effect of putting more teeth against fewer teeth and results in asymmetric movement.

The success of this type of expander depends on good construction and design and on the patient's cooperation.

Case Reports

As discussed earlier, posterior crossbites have many variations in morphology, etiology, and possible treatment tactics. Because these types of anomaly form and

develop during the primary and early mixed dentitions, the earliest intervention not only is easier to accomplish but also provides more stable results and prevents many consequences of delayed correction. Delays in treatment can result in structural damage to the dentition and supporting tissue, temporomandibular joint dysfunction, and adverse effects on jaw growth.

The following cases present early intervention treatments carried out at different stages of the dentition with different tactics for different kinds of crossbite.

Case 12-1

A 7-year-old boy presented with a posterior left-side crossbite and mandibular shift. The first etiologic factor was diagnosed as a prematurity present between the maxillary and mandibular primary left canines (Figs 12-19a and 12-19b).

Treatment:
Treatment was started with occlusal equilibration and grinding of the buccal surface of the mandibular primary canine and palatal surface of the maxillary primary canine on the left side. The result was an immediate change in tooth relationships, mandibular shift, and dental midlines (Figs 12-19c and 12-19d).

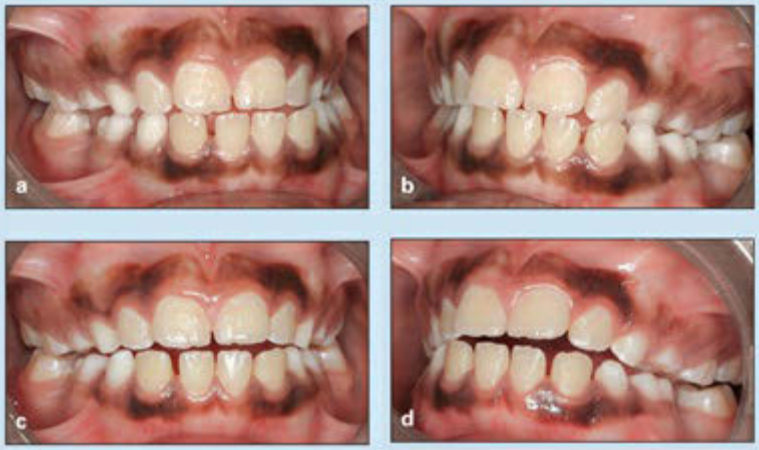


Fig 12-19 Management of posterior crossbite and mandibular shift in a 7-year-old boy. The primary etiologic factor was premature contact between the maxillary and mandibular canines on the left side. *(a and b)* Pretreatment occlusion. *(c and d)* Immediate improvement in relationships after occlusal equilibration and selective grinding.

Note: When unilateral posterior crossbite and mandibular shift are the result of premature contact and the problem is not corrected at an early age, the situation may cause a strong habitual shift. In this situation, because the child's habit is sustained, the equilibration technique alone cannot solve the problem. In addition to occlusal equilibration, slight bite opening with composite resin or acrylic resin occlusal coverage, with gradual reduction of the material, can help. Otherwise, an expansion procedure might be the best option.

Case 12-2

A 5-year-old boy in the primary dentition presented with right-side complete posterior crossbite and mandibular shift. In centric occlusion, the mandibular midline shifted to the right. He had a Class I occlusion on the left side and Class II malocclusion on the right side (Figs 12-20a to 12-20c).

The study cast revealed symmetric maxillary and mandibular arches, and clinical evaluation at centric relation indicated slight bilateral constriction of the maxillary arch.

Treatment:

Treatment was initiated with a fixed Haas expander with four bands on the primary second molars and primary canines. The appliance was expanded once a day by the patient's mother, and expansion was completed after 2 weeks. Figures 12-20d to 12-20f illustrate the appliance and changes that took place during activation. The patient experienced correction of crossbite and developed some spacing between the incisors that was not present before.

The appliance was kept in place for 3 months to provide retention. As a result of treatment, the crossbite was corrected, there was no more shift, midlines were coincident, and spacing remained to facilitate transition of the anterior teeth (Figs 12-20g and 12-20h).

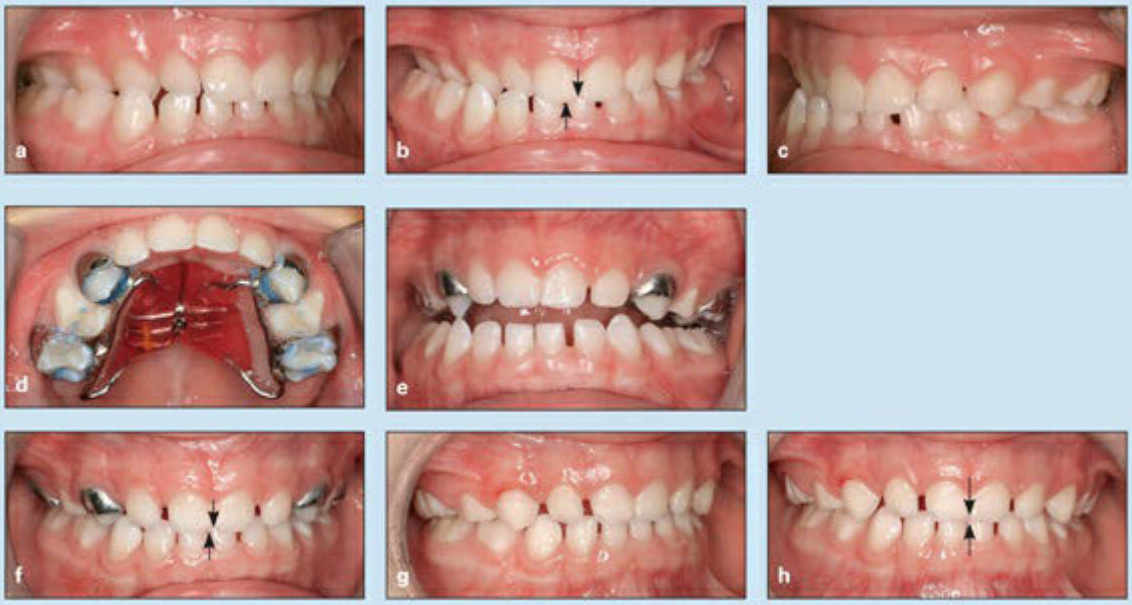


Fig 12-20 Management of complete posterior crossbite of the right side and mandibular shift in a 5-year-old boy. *(a to c)* Pretreatment occlusion. *(arrows)* Midline deviation. *(d and e)* Occlusion during use of the Haas rapid palatal expander. *(f)* Occlusion 2 weeks after completion of expansion; the midline deviation is almost corrected *(arrows)*, and interincisal spacing has developed. *(g and h)* Posttreatment occlusion 3 months after expansion. *(arrows)* Corrected dental midlines.

Case 12-3

A 10-year-old girl in the middle mixed dentition presented with a complete anterior and left posterior cross-bite. She exhibited a severe mandibular shift. The mandibular dentition was good, but there was some bony recession of mandibular central incisors because of the traumatic occlusion. The maxillary

incisors show minor crowding and retroclination (Figs 12-21a to 12-21c).

Careful cast assessment and clinical evaluation of the patient's occlusion at centric relation clarified that retroclination of three maxillary incisors (both central incisors and the left lateral incisor) caused a severe mandibular shift to the left and created the complete left posterior crossbite and anterior crossbite in centric occlusion. Assessment of the patient's occlusion in centric relation indicated that there was no maxillary arch constriction (Figs 12-21d to 12-21g).

Treatment:

Because of the maxillary incisor retroclination and minor crowding, treatment was limited to proclination of the incisor and correction of the anterior crossbite.

Treatment involved 2 × 4 maxillary bonding and composite resin on the occlusal surface of the mandibular molars to disocclude the incisors (Figs 12-21h to 12-21j).

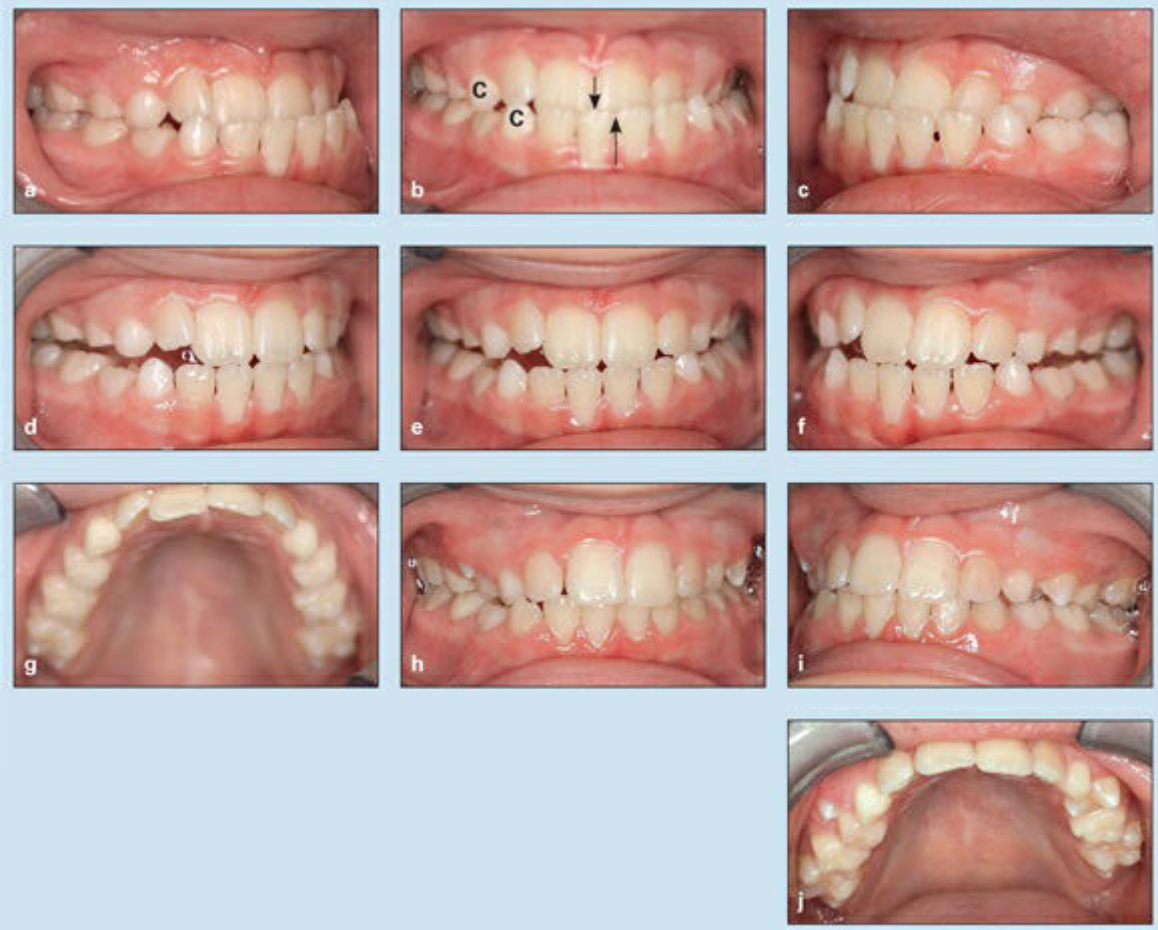


Fig 12-21 Management of complete anterior and left posterior crossbite in a 10-year-old girl in the mixed dentition. There was a severe mandibular shift. *(a to c)* Pretreatment occlusion in centric occlusion. C—primary canines; *(arrows)* midline deviation. *(d to f)* Pretreatment occlusion in centric relation. *(g)* Retroclination of both central incisors and the left lateral incisor. *(h to j)* Posttreatment occlusion after proclination of the maxillary incisors.

Case 12-4

A 5-year, 6-month-old girl in the primary dentition presented with left posterior crossbite and mandibular

shift. The midlines were not coincident in centric occlusion (Figs 12-22a and 12-22b).

Treatment:
A jackscrew slow expander with occlusal coverage was used (Fig 12-22c). When the expander was in place and the patient occluded on the acrylic resin, there was no midline shift in centric occlusion (Figs 12-22d and 12-22e).

After 6 weeks of active treatment (opening the appliance every 3 days), the patient lost the appliance, but the problem was completely corrected (no crossbite and no shift). A 4-week trial without the appliance was decided upon. Figures 12-22f and 12-22g show the occlusion after 6 months without the appliance. Figures 12-22h and 12-22i show the occlusion 1 year later, after all permanent first molars, mandibular incisors, and maxillary central incisors had erupted. Figures 12-22j to 12-22l shows the occlusion 3 years after treatment.

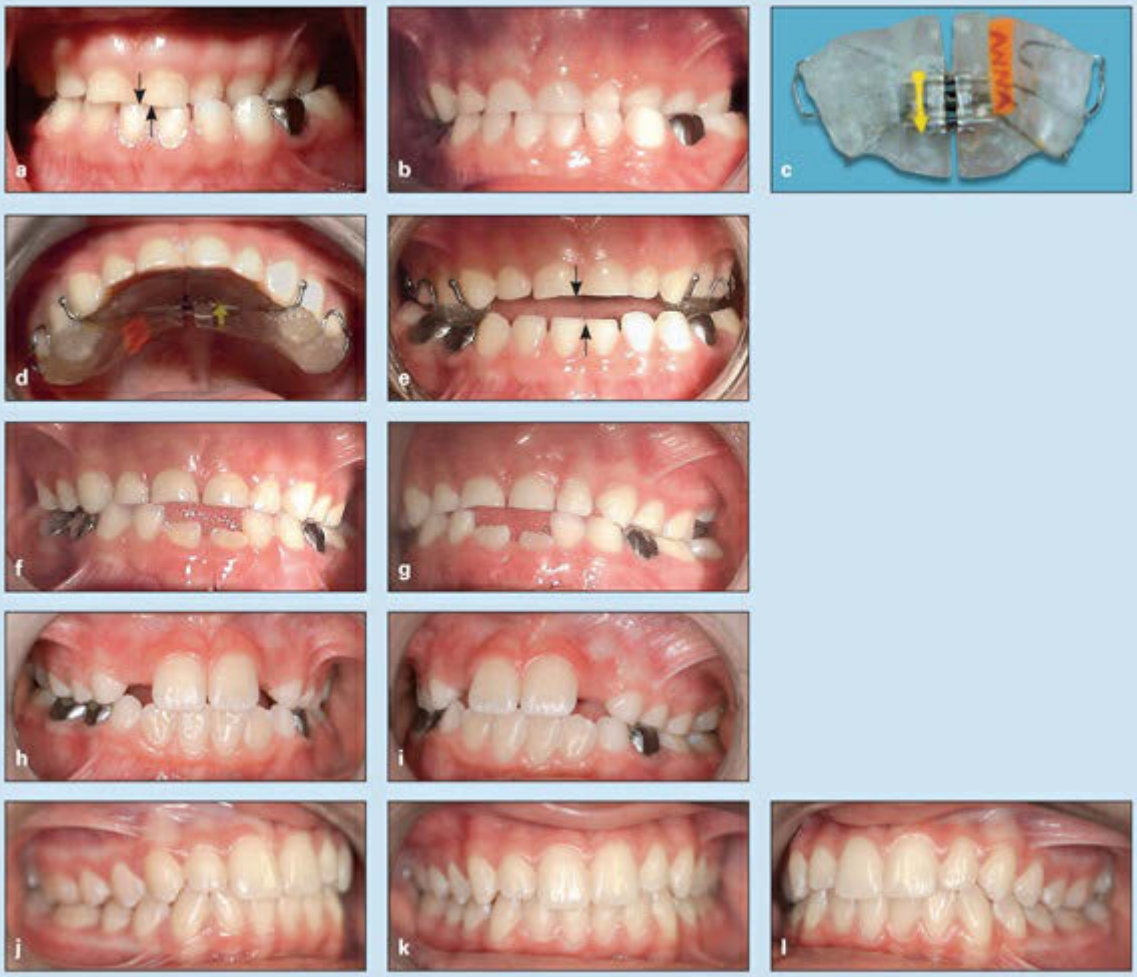


Fig 12-22 Management of posterior left crossbite and mandibular crossbite in a 5-year, 6-month-old girl. *(a and b)* Pretreatment occlusion. *(arrows)* Midlines in centric occlusion. *(c)* Jackscrew slow expander with occlusal coverage. *(d and e)* Centric occlusion with expander in place; there is no midline shift *(arrows)*. *(f and g)* Posttreatment occlusion 6 months after active treatment. *(h and i)* Posttreatment occlusion 1 year later. *(j to l)* Posttreatment occlusion 3 years after treatment.

Case 12-5

A 7-year, 4-month-old boy presented with left-side posterior crossbite, mandibular shift, and space deficiency in the maxillary anterior segment (Figs 12-23a to 12-23d).

Treatment:
The treatment modality in this case was a Haas rapid palatal expander, on which the screw was opened once a day. The expander was designed with only two bands on the permanent first molars and a jackscrew embedded in acrylic resin, together with two 0.032-inch stainless steel bars adapted to the primary molars and canines and bonded to the palatal side of these teeth with composite resin.

Figures 12-23e to 12-23g show the occlusion 3 months after completion of expansion and at the time of removal of the expander. The results of expansion were correction of the crossbite, coincident midlines, elimination of the shift, and creation of space for the anterior segment. All incisors had erupted.

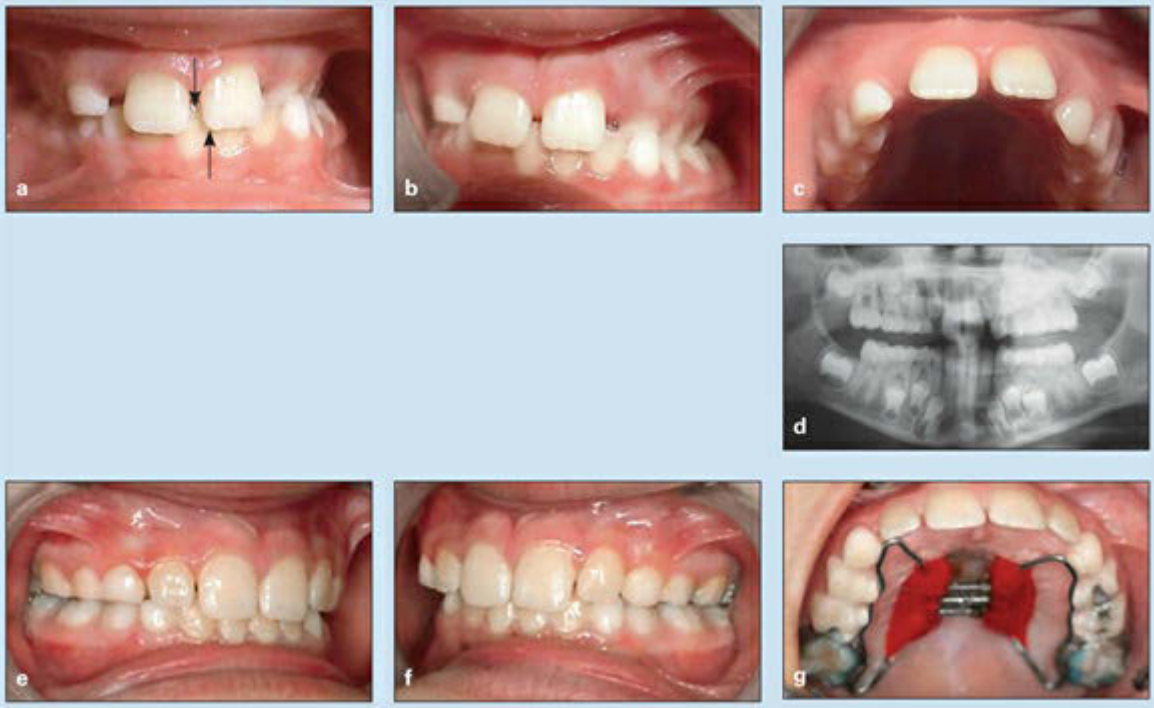


Fig 12-23 Management of posterior left crossbite, mandibular shift, and space deficiency in a 7-year, 4-month-old boy. *(a and b)* Pretreatment occlusion in centric occlusion. *(arrows)* Midline deviation. *(c)* Pretreatment view revealing space deficiency in the maxillary anterior region. *(d)* Pretreatment panoramic radiograph. *(e to g)* Occlusion at the time of expander removal, 3 months after completion of expansion.

Case 12-6

A 5-year, 8-month-old boy in the primary dentition presented with posterior crossbite on the left side, mandibular shift, and a mandibular midline that was shifted 4 mm to the left (Figs 12-24a to 12-24c). He had class I spacing according to the Baume classification.[27]

Treatment:
Because of the patient's medical conditions (attention deficit–hyperactivity disorder, asthma, and poor oral hygiene), the treatment plan included use of a W-arch expander. The appliance consisted of two molar bands on the maxillary primary second molars and a W-shaped 0.036-inch stainless steel wire

adapted in such a way as to have more activation on the left side (Figs 12-24d and 12-24e).

Figures 12-24f to 12-24h illustrate the occlusion 3 months after removal of the expander and after placement of crowns and restorations.

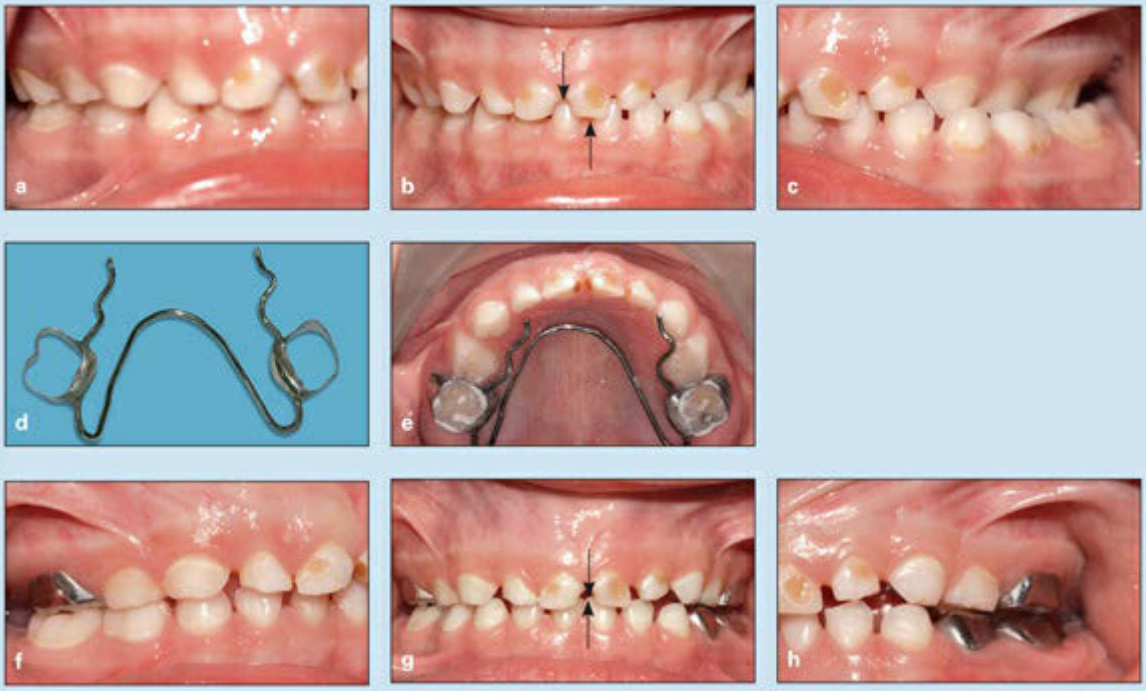


Fig 12-24 Management of posterior left crossbite, mandibular shift, and a 4-mm midline deviation in a 5-year, 8-month-old boy with class I Baume spacing. *(a to c)* Pretreatment occlusion. *(arrows)* Midline deviation. *(d)* W-arch appliance used for expansion. *(e)* Appliance in place. *(f to h)* Posttreatment occlusion 3 months after removal of the appliance and after placement of restorations and crowns. *(arrows)* Corrected midlines.

Case 12-7

A 4-year, 9-month-old girl presented with a left-side posterior crossbite and mandibular shift (Figs 12-25a to 12-25c).

Treatment:

The appliance used for this patient was a W-arch expander with 0.036-inch stainless steel wire (Fig 12-25d). The appliance was expanded 8 mm when placed and activated only once, 4 weeks after insertion.

After completion of expansion and slight overcorrection, the appliance was left in place for 2 months for retention. Figures 12-25e and 12-25f show the results 3 months after the end of retention.

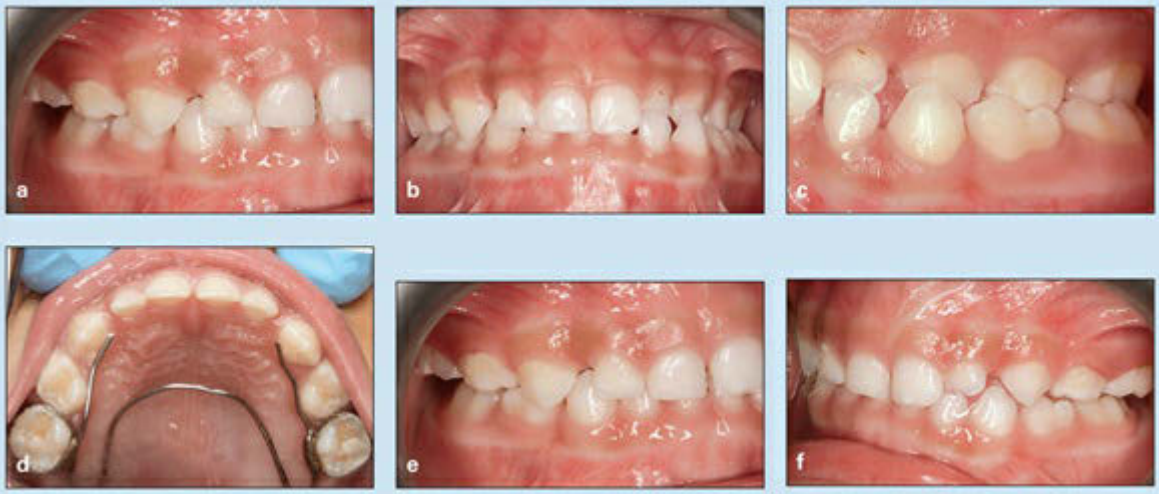


Fig 12-25 Management of posterior left crossbite and mandibular shift in a 4-year, 9-month-old girl. *(a to c)* Pretreatment occlusion. *(d)* W-arch expander in place. *(e and f)* Occlusion 3 months after the end of retention.

Case 12-8

A 14-year, 6-month-old boy presented with Class II division 1 bilateral complete buccal bite (Brodie syndrome). The patient's family had limited financial resources, and their chief concern was the fact that the boy could not chew properly. Besides the transverse problem, this patient had some other anomalies that made the situation more complicated. These included a Class II sagittal problem with severe overjet and 90% impinging overbite; missing maxillary and mandibular permanent left first molars; microdontia, which was causing severe interdental spacing; and poor interdigitation and intercuspation (Figs 12-26a to 12-26f).

The result of all these anomalies was a collapsed mandibular arch with spacing within the maxillary arch that made the occlusion and dentofacial component not only unesthetic but also dysfunctional.

Treatment:

Because of the family's financial situation, no surgical, prosthetic, or implant restoration could be considered as part of the treatment plan. Therefore, the problems had to be solved orthodontically. The treatment was completed in a number of steps:

1. Maxillary anterior bite plane to disocclude the locked mandibular arch within the maxilla
2. Mandibular heavy semiremovable lingual arch to expand the constricted mandibular molars gradually
3. Maxillary constricting cervical headgear to constrict the maxillary molars and distalize them for Class II correction (Fig 12-26g)
4. Full maxillary and mandibular bonding as soon as molar occlusal contact was established
5. Retraction of maxillary buccal segments and continuing headgear use as anchorage
6. Consolidation of maxillary buccal segments between molars and premolar retraction
7. After retraction of maxillary molars and premolars, heavy class II mechanics to retract the maxillary canines and mesialize the posterior mandibular segment while transverse relationships were controlled
8. Maxillary incisor retraction and intrusion to correct the deep bite and severe overjet

Figures 12-26h to 12-26j illustrate the posttreatment occlusion after closure of the maxillary and mandibular arches. All the mechanical effort was focused on narrowing the maxillary arch while simultaneously widening the mandibular arch, especially in the molar areas.

Sagittal and transverse changes in the dentition were accomplished in this treatment. The pretreatment width at the maxillary first premolars was 35 mm; after treatment, the width was 30 mm (Fig 12-26k). The maxillary intermolar width was 40 mm pretreatment and 35 mm after treatment (Fig 12-26l). The width at the mandibular first premolars changed from 30 to 29 mm (Fig 12-26m). The pretreatment mandibular intermolar width was 35 mm; it remained the same after treatment, despite the severe mesial movement that was done because of the missing first molar and significant interdental spacing (Fig 12-26n).

Significant dentoskeletal changes were achieved through this treatment (Figs 12-26o to 12-26q):

- Correction of severe bilateral buccal bite
- Correction of Class II malocclusion
- Closure of the severe interdental spacing caused by missing teeth and microdontia
- Significant change in lower facial height (correction of mandibular collapse) from 53.7% to 60.5%
- Change in point A–nasion–point B angle from 4.2 to 3.6 degrees
- Correction of maxillomandibular dental protrusion (increase in interincisal angle from 111.4 to 136.0 degrees)

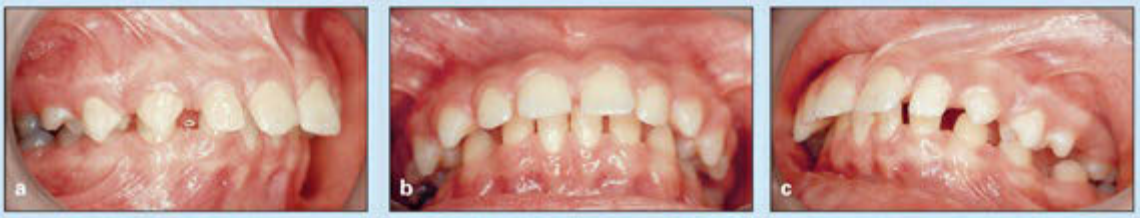


Fig 12-26 Management of bilateral complete buccal bite (Brodie syndrome) in a 14-year, 6-month-old boy with Class II division 1 malocclusion. *(a to c)* Pretreatment occlusion.

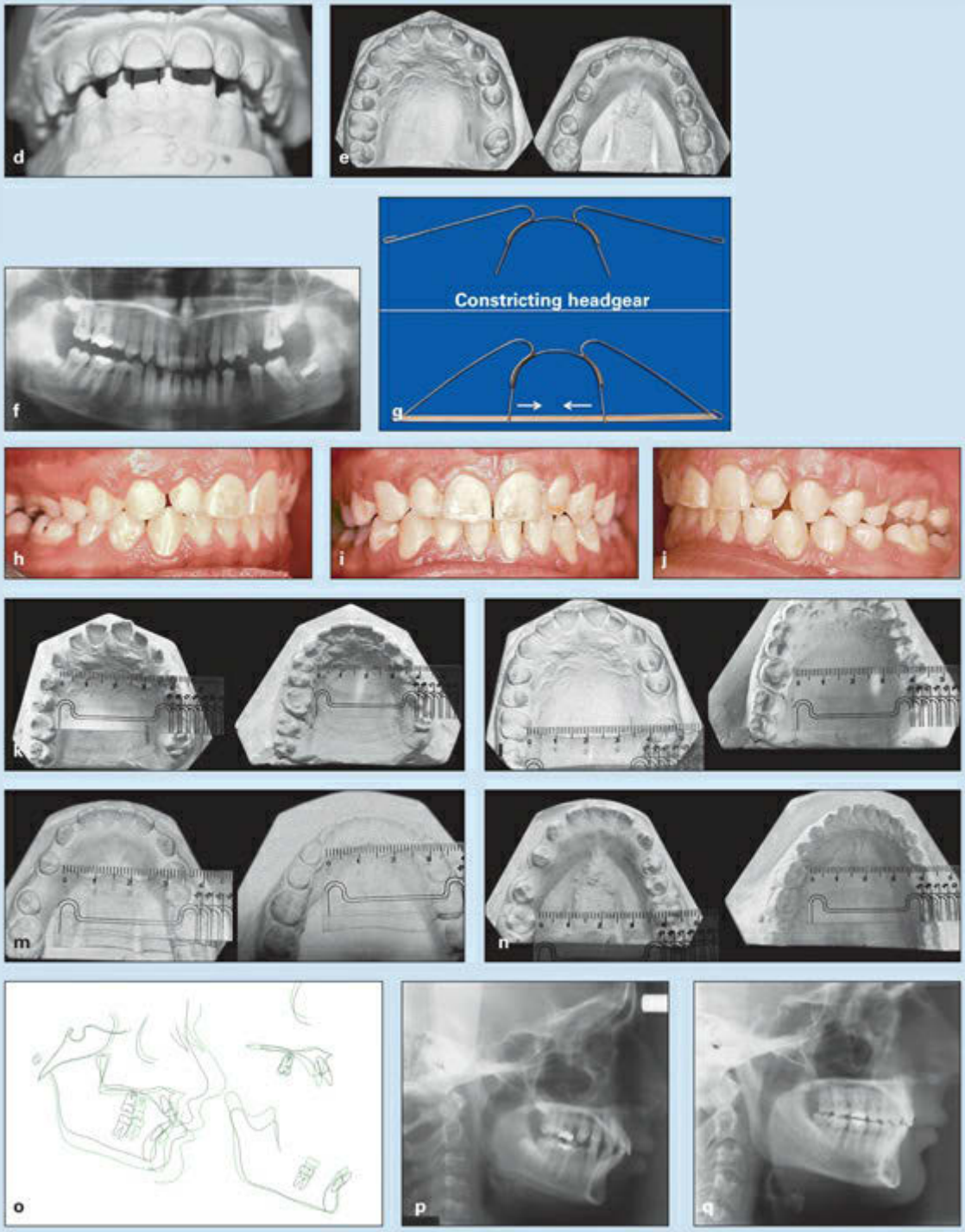


Fig 12-26 *(cont) (d and e)* Pretreatment casts. *(f)* Pretreatment panoramic radiograph. *(g)* Design of the facebow used for constriction and distalization of the maxillary molars. *(arrows)* Direction of movement. *(h to j)* Posttreatment occlusion. *(k)* Changes in maxillary premolar width. *(l)* Changes in maxillary molar width. *(m)* Changes in mandibular premolar width. *(n)* No changes in mandibular molar width. *(o)* Superimposition of pretreatment *(black)* and posttreatment *(green)* tracings. *(p)* Pretreatment cephalometric radiograph. *(q)* Posttreatment cephalometric radiograph.

Case 12-9

A 13-year-old boy presented with a Class II division 1 malocclusion, unilateral buccal bite on the left side, severe overjet (16.5 mm), and impinging deep bite. Study casts indicated that the buccal bite was due to asymmetry in both the maxillary and mandibular left posterior segments, that is, buccoversion of the maxillary left segment and linguoversion of the mandibular left segment (Figs 12-27a to 12-27d).

Treatment:

Treatment was started with a maxillary anterior bite plate to open the locked posterior left segment. A mandibular lingual arch was placed for expansion of the mandibular left segment, and constricting headgear was used for distalization of the maxillary left and right molars and constriction of the maxillary left segment. Treatment followed the same steps described in case 12-8, including sagittal changes of the posterior segments to Class I molar, premolar, and canine relationships and finishing with maxillary anterior retraction.

Because the transverse problems of this patient were unilateral, all of the mechanical effort was focused on constricting only the maxillary left segment and expanding only the mandibular left segment. This goal was achieved with placement of continuous cross-arch elastics on the left side.

Significant changes were achieved through this treatment procedure (Figs 12-27e to 12-27j). These changes included correction of the unilateral buccal bite; correction of the Class II malocclusion and reduction of overjet, from 16.5 to 3.5 mm; correction of deep bite; change in the point A–nasion–point B angle, from 12.3 to 4.5 degrees; and significant changes in the angle of convexity and the angle of point A–point B to the facial plane.

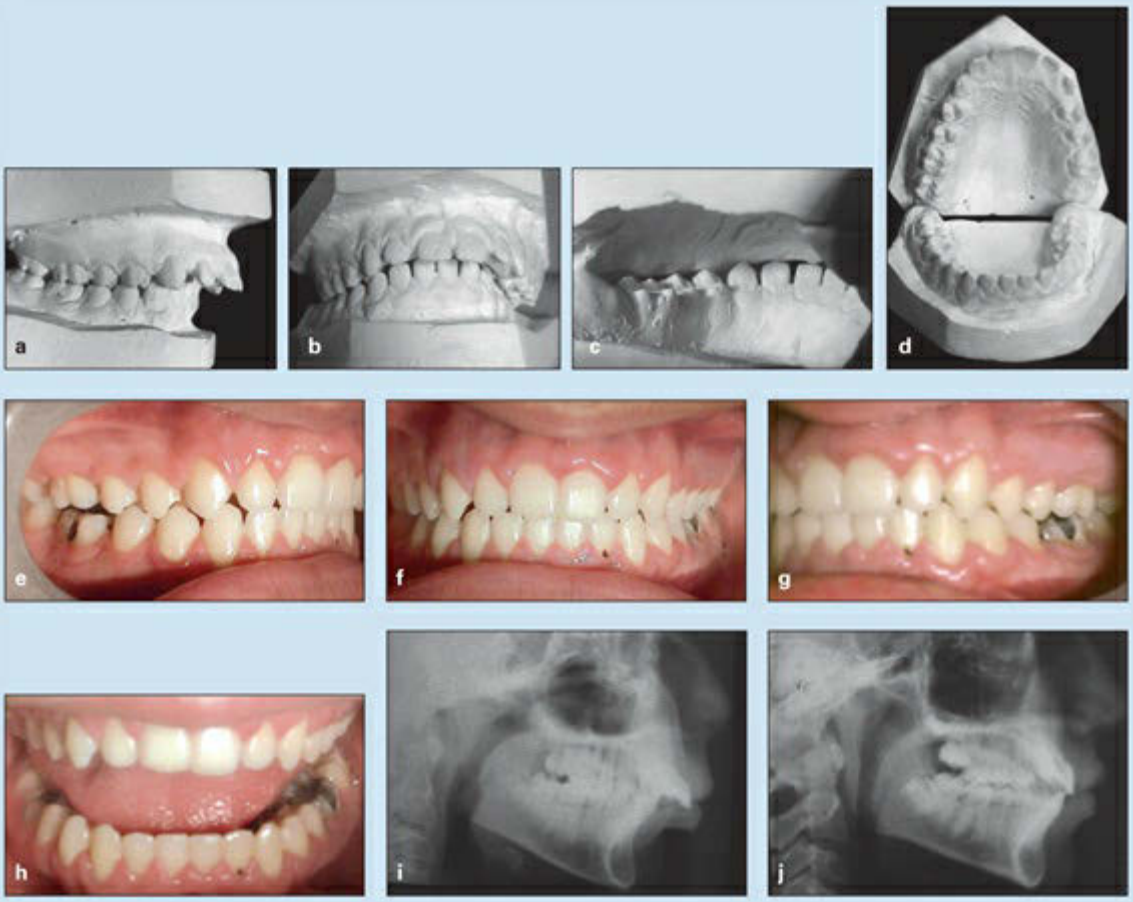


Fig 12-27 Management of unilateral buccal bite on the left side, severe overjet, and impinging deep bite in a 13-year-old boy with a Class II division 1 malocclusion. *(a to d)* Pretreatment casts revealing overexpansion of maxillary left segment and constriction of the mandibular left segment. *(e to h)* Posttreatment occlusion. *(i)* Pretreatment cephalometric radiograph. *(j)* Posttreatment

cephalometric radiograph.

Summary

- Posterior crossbite is one of the most prevalent malocclusions found during the primary and early mixed dentitions. The most common form of posterior crossbite is a unilateral crossbite with a functional shift of the mandible toward the crossbite side.
- Posterior crossbites are seldom self-correcting; delayed treatment and asymmetric muscle strain in the orofacial structures can have adverse effects on the temporomandibular joint, the masticatory system, and skeletal growth changes.
- Maxillary arch constriction, which is usually present in patients with this condition, creates insufficient maxillary arch width and results in anterior crowding and sometimes canine impaction.
- Early treatment is advised to create an environment that facilitates normal occlusal development.
- The most common causes of posterior crossbite are digit or pacifier sucking habits, impaired nasal breathing, atypical swallowing patterns, and low tongue position. Therefore, evaluation during the primary and mixed dentitions is recommended. These facts emphasize the important responsibility that practitioners have in careful examination for and early recognition of these factors, which are interacting in young patients during a critical stage of occlusal development.
- Almost all investigators studying these anomalies recommend early intervention and correction of posterior cross-bites. Correction of posterior crossbite affecting the permanent dentition is much more complicated and in some cases may require surgery.
- The best time for correction of posterior crossbite is during the late primary or early mixed dentition. The expansion procedure is easy and fast and provides good, stable results.
- Posterior crossbite has many etiologic factors and several different morphologic characteristics; thus, differential diagnosis is an essential part of treatment planning.
- Posterior crossbite can be dental, dentoalveolar, skeletal, or functional; each of these types can be unilateral or bilateral. Posterior crossbite can also

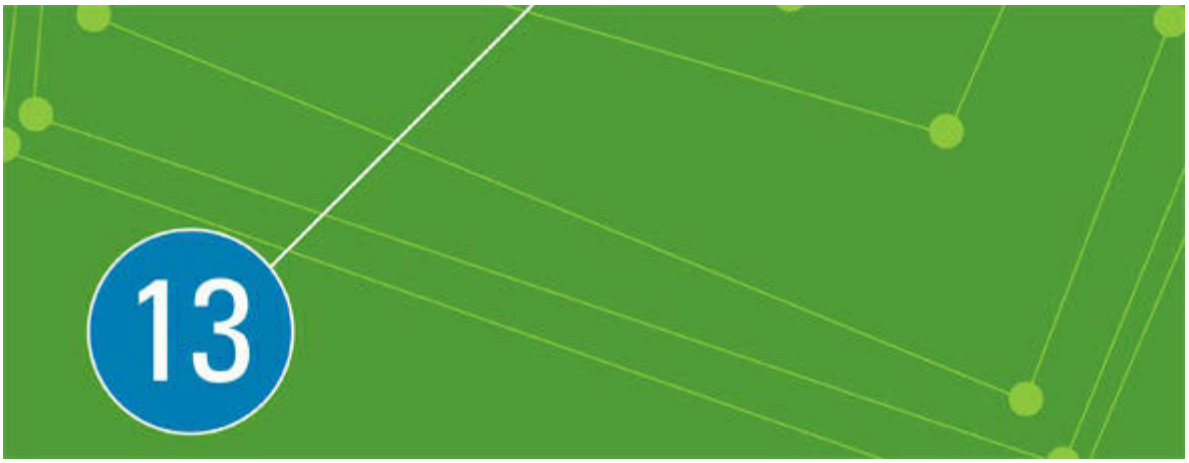
result from other combinations of transverse malrelationship between the maxillary or mandibular dental arch, such as buccal bite, also called *Brodie syndrome*, scissors bite, and palatal bite.

- Recognition of posterior crossbite is not difficult, and a simple clinical evaluation of occlusion, study casts, and frontal cephalometric radiographs can enable the practitioner to easily identify the type of problem.
- Depending on the type of posterior crossbite, many treatment options incorporating fixed and removable appliances that offer slow or rapid palatal expansion are available.

References

1. Moyers R. Handbook of Orthodontics, ed 4. Chicago: Year Book Medical, 1988.
2. Kennedy DB, Osepchook M. Unilateral posterior cross-bite with mandibular shift: A review. J Can Dent Assoc 2005;71:569–573.
3. Harper DL. A case report of a Brodie bite. Am J Orthod Dentofacial Orthop 1995;108:201–206.
4. Kutin G, Hawes RR. Posterior cross-bites in the deciduous and mixed dentitions. Am J Orthod 1969;56:491–504.
5. Hesse KL, Artun J, Joondeph DR, Kennedy DB. Changes in condylar position and occlusion associated with maxillary expansion for correction of functional unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 1997;111: 410–418.
6. Pinto AS, Buschang PH, Throckmorton GS, Chen P. Morphological and positional asymmetries of young children with functional unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 2001;120:513–520.
7. Myers DR, Barenie JT, Bell RA, Williamson EH. Condylar position in children with functional posterior cross-bite: Before and after crossbite correction. Pediatr Dent 1980;2:190–194.
8. Egermark-Ericksson I, Carlsson GE, Magnusson T, Thilander B. A longitudinal study on malocclusion in relation to signs and symptoms of cranio-mandibular disorders in children and adolescents. Eur J Orthod 1990;12:399–407.
9. Thilander B, Lennartsson B. A study of children with unilateral posterior crossbite, treated and untreated, in the deciduous dentition—Occlusal and skeletal characteristics of significance in predicting the long-term outcome. J Orofacial Orthop 2002;63:371–383.
10. Schröder U, Schröder I. Early treatment of unilateral posterior crossbite in children with bilaterally contracted maxillae. Eur J Orthod 1984;6:65–69.
11. Proffit WR, Fields HW Jr. Contemporary Orthodontics, ed 5. St Louis: Mosby, 2012.
12. Subtelny JD. Oral respiration: Facial maldevelopment and corrective dentofacial orthopedics. Angle Orthod 1980;50:147–164.
13. Kurol J, Berglund L. Longitudinal study and cost-benefit analysis of the effect of early treatment of posterior cross-bites in the primary dentition. Eur J Orthod 1992;14:173–179.
14. Linder-Aronson S. Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. Acta Otolaryngol Suppl 1970;265:1–132.
15. McNamara JA. Early intervention in the transverse dimension: Is it worth the effort? Am J Orthod Dentofacial Or-thop 2002;121:572–574.
16. Allen D, Rebellato J, Sheats R, Ceron AM. Skeletal and dental contributions to posterior crossbites. Angle

Orthod 2003;73:515–524.
17. Warren JJ, Bishara SE. Duration of nutritive and nonnutritive sucking behaviors and their effects on the dental arches in the primary dentition. Am J Orthod Dentofacial Orthop 2002;121:347–356.
18. Souki BQ, Pimenta GB, Souki MQ, Franco LP, Becker HM, Pinto JA. Prevalence of malocclusion among mouth breathing children: Do expectations meet reality? Int J Pediatr Otorhinolaryngol 2009;73:767–773.
19. Oulis CJ, Vadiakas GP, Ekonomides J, Dratsa J. The effect of hypertrophic adenoids and tonsils on the development of posterior crossbite and oral habits. J Clin Pediatr Dent 1994;18:197–201.
20. Ogaard B, Larsson E, Lindsten R. The effect of sucking habits, cohort, sex, intercanine widths, and breast or bottle feeding on posterior crossbite in Norwegian and Swedish 3-year-old children. Am J Orthod Dentofacial Orthop 1994;106:161–166.
21. Adair SM, Milano M, Lorenzo I, Russell C. Effects of current and former pacifier use on the dentition of 24- to 59-month old children. Pediatr Dent 1995;17:437–444.
22. Infante PF. An epidemiologic study of finger habits in preschool children as related to malocclusion, socioeconomic status, race, sex, and size of community. ASDC J Dent Child 1976;43:33–38.
23. Hanson M, Barnard L, Case J. Tongue thrust in preschool children. 2. Dental occlusal patterns. Am J Orthod 1970;57:15–22.
24. Primozic J, Richmond S, Kau CH, Zhurov A, Ovsenik M. Three-dimensional evaluation of early crossbite correction: A longitudinal study. Eur J Orthod 2013;35:7–13.
25. Guerrero CA, Bell WH, Contasti GI, Rodriguez AM. Mandibular widening by intraoral distraction osteogenesis. Br J Oral Maxillofac Surg 1997;35:383–392.
26. Legan HL. Orthodontic planning and biomechanics for transverse distraction osteogenesis. Semin Orthod 2001;7:160–168.
27. Baume LG. Physiological tooth migration and its significance for the development of occlusion. 1. The biogenetic course of the deciduous dentition. J Dent Res 1950;29:123–132.



Management of Vertical Problems (Open Bites and Deep Bites)

This chapter discusses common vertical problems that initiate and develop during the primary or early mixed dentition: *(1)* open bites associated with persistent oral habits and other dysfunctions and *(2)* deep bites (dental deep bite and skeletal deep bite). These anomalies are not self-corrected and in some situations worsen in later stages of the dentition; therefore, early detection and intervention are important.

Open Bite

Treatment of skeletal open bite, or hyperdivergent phenotype malocclusion, and anterior open bite is one of the common difficulties facing orthodontists. They are especially problematic during the permanent dentition and are one of the most challenging malocclusions to treat.

The intent of this book is to emphasize the advantages of early-age intervention for all malocclusions, including open bites that initiate and develop during the primary and mixed dentitions. Open bite is a multifactorial phenomenon; recognition of the etiology plays an important role in diagnosis and treatment of this anomaly. The

problem can be diagnosed clinically and cephalometrically; however, diagnosis should be viewed in the context of both skeletal and dental structures.

Hereditary factors influence the direction, form, and rate of skeletal growth; they can produce abnormal growth patterns and incorrect jaw posture, resulting in skeletal open bite. Environmental factors during the primary and mixed dentitions can also play an important role in developing open bites. Therefore, different kinds of open bite with different morphologic and etiologic characteristics must be accurately classified for precise treatment planning. This requires experience and training.

Early recognition of the cause and proper intervention can prevent the development of abnormalities or at least reduce the severity of the problem later. At the present time, it is not possible to eliminate or prevent the influence of genetic factors on dentoskeletal structure; however, with accurate examination and recognition of the effect, practitioners might be able to prevent, modify, or at least reduce the severity of the problem. Because environmental factors are mostly recognizable, recognition of and intervention in such influences at an early stage of formation can prevent development of open bite or at least guide the abnormality toward normality. The following are the major common local or environmental factors that play a role in development of open bite at early age:

- Digit sucking
- Abnormal tongue size, posture, and function
- Abnormal neuromuscular function
- Enlarged lymphatic tissue causing mouth breathing and postural alteration that induces dental and skeletal modifications

The etiology and mechanism of the factors causing open bite are discussed in detail in chapter 6. The current chapter presents the differential diagnosis, treatment strategies, and techniques for early correction of open bite during the primary and mixed dentitions.

Pediatric dentists and general practitioners who see children at an early age have greater responsibility for the detection of and intervention in these developing anomalies. Differential diagnosis and treatment of open bite malocclusion are sometimes challenging. However, successful identification of the etiology through careful clinical and cephalometric evaluation as well as some general history of the problem, improve the chances of treatment success. A brief review of the problem and its etiologic factors will be provided here, for the purpose of recognition and

proper intervention.

Depending on the etiologic factors and the complexity of the abnormality, treatment for open bite ranges from observation to simple habit control to complex surgical procedures.

Etiology

Components of vertical problems must be diagnosed and classified under two contexts: skeletal structures and dental structures. Skeletal open bite usually has a genetic background and interacts with environmental causes. It can develop to a more complex dentoskeletal open bite and in some cases, especially if not resolved by the end of the mixed dentition years, will require complicated orthodontic and orthopedic treatment, with or without surgical intervention.

The first step toward early treatment is recognition of the problem, and the best way to recognize the problem is to understand and differentiate between the characteristics of different types of open bite. No single factor can be considered the cause of open bite. The most common local causes of this anomaly are thumb and finger sucking, tongue problems (such as abnormal function, size, position, and posture), mouth breathing as a result of enlarged lymphatic tissue or other airway obstruction, and lip dysfunction.

Finger sucking

Depending on the type of sucking and the position of the fingers, sucking can affect the occlusion by different mechanisms. Finger force directed to the anterior teeth and the incisive bone can cause dental and dentoalveolar protrusion and open bite, prevent eruption of anterior teeth, and lead to underdevelopment of the alveolar process; it can also cause posterior crossbite. A prolonged habit and absence of contact in the posterior teeth can also cause overeruption of the posterior teeth and even clockwise rotation of the mandible (Fig 13-1).

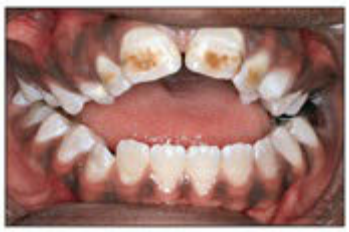


Fig 13-1 Dentoskeletal open bite and posterior crossbite caused by thumb sucking.

Abnormal tongue force

The tongue can cause anterior open bite through different mechanisms, such as tongue size, tongue position, tongue posture, and abnormal tongue function. Depending on the size of the tongue, macroglossia can have different destructive effects on the occlusion, such as interdental spacing, Class III malocclusion, and open bite. Severe enlargement of the tongue can also cause functional difficulties in speaking, eating, swallowing, and sleeping. This condition can be seen in individuals with certain inherited or congenital disorders, such as neonatal hypothyroidism, Down syndrome, and acromegaly.

Abnormal position of the tongue is another condition that can cause dentoskeletal deformity. This situation is not the result of the size of the tongue; rather the tongue is positioned more anteriorly as a subconscious reaction to large tonsils or because of mouth breathing in response to nasal obstruction or enlarged adenoid tissue (Fig 13-2).

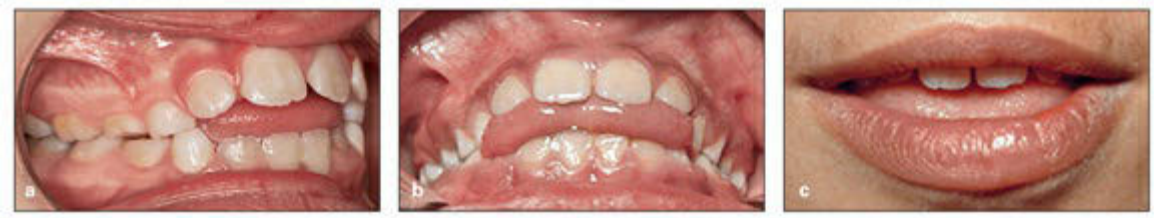


Fig 13-2 *(a to c)* Dentoskeletal open bite caused by abnormal size, position, or function of the tongue.

Oral respiration

During mouth breathing, no matter what the cause, the mouth is open, the mandible is postured inferiorly, and the tongue is protruded between the anterior teeth and resting against the oral floor. This situation causes postural alteration and induces dental and skeletal modifications similar to those caused by thumb sucking.

Mouth breathing can also cause excessive eruption of the posterior teeth and maxillary constriction, change head posture, cause an increase in the vertical dimension of the face, and result in development of anterior dentoskeletal open bite.

Genetic factors

Genetic factors, either alone or in combination with local factors, can disrupt the normal skeletal growth pattern through different mechanisms, such as disturbance of the normal size, shape, and proportion of different parts of skeletal structures.

Development of skeletal abnormalities causes several skeletal malocclusions, including open bite, which mostly require orthosurgical management.[1]

Tongue thrust is an abnormal tongue function that usually develops secondary to thumb sucking and then maintains or increases the open bite that was produced by the finger, even after the finger-sucking habit has stopped. During correction of open bite and application of different treatment mechanics, abnormal tongue function must be controlled.

Morphologic characteristics

For practical purposes and simplification of differential diagnosis, the open bite syndrome can be classified into two types: dental open bite and skeletal open bite.

Dental open bite

Simple dental open bite is considered an open bite without skeletal abnormalities, with the following characteristics:

- Normal craniofacial pattern
- Normal skeletal jaw relationships
- Previous history of finger sucking
- Proclined incisors
- Anterior tongue thrust
- Undererupted incisors
- Normal or slightly excessive molar height
- Normal inclination of posterior dentition
- Divergent occlusal plane
- No gummy smile
- No vertical excess or posterior tipping of the maxilla

Prolonged continuation of environmental factors and failure to initiate early treatment of simple dental open bite can produce more complicated effects, such as molar overeruption, mandibular clockwise rotation, and subsequently skeletal defects.

Skeletal open bite

True skeletal open bite abnormalities are usually caused by genetics, prolonged environmental factors, or a combination of both. They consist of many unfavorable growth patterns and skeletal disharmonies that must be distinguished by careful clinical and cephalometric evaluation before treatment planning.

The following are the important structural defects that can be detected by careful clinical and paraclinical evaluation of patients with skeletal open bite or vertical divergent face:

- Steep mandibular plane
- Retrognathic mandible
- Obtuse gonial angle
- Short ramal height
- Short anterior cranial base
- Short posterior cranial base
- Obtuse y-axis angle
- Obtuse saddle angle
- Obtuse articular angle
- Obtuse occlusal plane–mandibular plane angle
- Obtuse mandibular plane to palatal plane angle
- Counterclockwise rotation of the palate
- Increased lower facial height
- Decreased upper facial height
- Decreased posterior facial height
- Steep anterior cranial base

Differential diagnosis

Simple dental open bite is a common abnormality that occurs during the primary or early mixed dentition, caused by environmental problems such as abnormal habits, the type of nutrition, or enlargement of lymphatic tissues. Untreated open bite not only increases in severity over time but also can cause other dentoskeletal damage that can be more complicated to manage at a later age. These consequences are not

confined to undereruption of anterior teeth; open bite can cause overeruption of the maxillary or mandibular molars or both, and in severe cases it can cause skeletal deformities such as clockwise rotation of the mandible or counterclockwise rotation of the maxilla.

For early intervention in simple anterior dental open bite, the first step before mechanotherapy is recognition of the type of open bite and the structure involved. Therefore, besides the clinical and paraclinical evaluations that always must be performed for assessment of all malocclusions, special attention to cephalometric analysis of the vertical dimension is important in diagnosis of open bite malocclusions. For differentiation of dental open bite and to verify whether the open bite is due to anterior undereruption or posterior overeruption, the author recommends a simple cephalometric evaluation of the occlusal plane relative to mandibular plane–palatal plane angle. In normal occlusion, the mandibular plane–palatal plane angle is about 30 degrees, and the occlusal plane bisects this angle almost equally. In patients with open bite, variations in this configuration indicate the cause of the open bite (Fig 13-3).

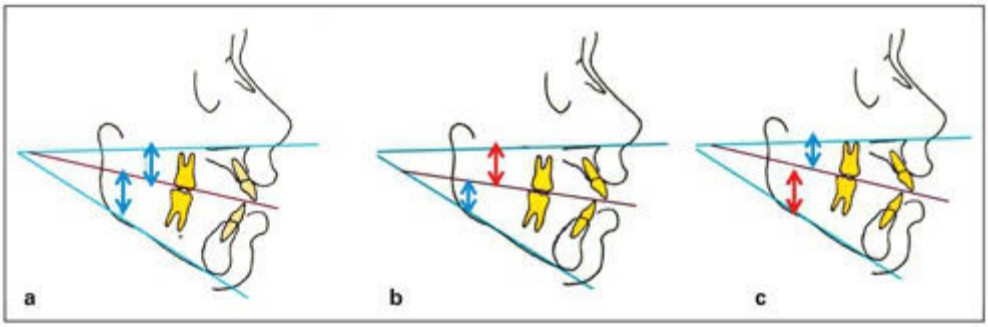


Fig 13-3 Cephalometric evaluation of mandibular plane– palatal plane angle to determine the cause of open bite. In normal occlusion, this measurement is about 30 degrees, and the occlusal plane bisects this angle almost equally. *(a)* Normal molar position with undereruption of the incisors. *(b)* Overeruption of the maxillary molars. *(c)* Overeruption of the mandibular molars.

Treatment strategies during the permanent dentition

As already discussed, early detection and interception of the developing anomaly at its early stage of development (primary or mixed dentition) is easier and provides more stable results. However, for the purposes of comparison, the following is a brief review of open bite correction in adolescents and adults.

Sustained open bite malocclusion during the late mixed dentition or permanent dentition is not self-correcting, and orthodontic treatment is almost always challenging; this is one of the most difficult malocclusions to treat orthodontically.

Treatment approaches in this period depend on the age of the patient and the severity of the problem.

Habit control and lip seal

Habit control, lip seal, and swallowing exercises can have a positive effect in camouflage treatment, even during the permanent dentition. These methods facilitate orthodontic tooth movement against the tongue force, which can be achieved by a device such as a tongue guard.

Control of vertical growth

Control of vertical dental eruption and posterior dentoalveolar development during camouflage treatment can also be helpful in correction of the permanent dentition. This is achieved by use of a lower holding arch and a transpalatal arch.

Orthodontic camouflage

Orthodontic camouflage consists of only orthodontic procedures, with special tactics depending on the morphologic and anatomical structures involved in this malocclusion. These approaches include posterior intrusion, anterior extrusion, or a combination of both. This type of treatment can be combined with tooth extraction if appropriate.

Many appliances for correction of anterior open bite in the permanent dentition have been proposed, including a bite block; an extrusion or intrusion arch; a multiloop edgewise archwire[2]; functional appliances[3]; and extraoral forces such as high-pull headgear, vertical chin cup (which can have some positive effect in the early permanent dentition), titanium screw anchorage,[4] titanium miniplates fixed bilaterally to the zygomatic buttress area,[5] and a magnetic active vertical corrector device.[6]

Orthognathic surgery

True skeletal open bites cannot be treated by orthodontic camouflage alone and require a combination of orthodontics and surgical treatment,[1] such as surgical impaction of the maxillary posterior segment, which is considered to be the most effective treatment option for correction of skeletal open bites in adult patients.

Early Treatment Strategies for Open Bite

Vertical growth of the face, as shown by Enlow,[7] is the last dimension of the dentofacial structure to be completed during early postnatal growth and the transitional dentition. As Carlson[8] pointed out, only 50% of growth in the midface and the mandible is complete by the age of 8 to 10 years. Therefore, early treatment and control of vertical growth during this period is more efficient.

Harris and Johnson,[9] in a longitudinal sibling analysis, concluded that most occlusal variability is acquired, not inherited.

As mentioned earlier, the most common local causes of anterior open bites are thumb and finger sucking, mouth breathing in response to enlarged lymphatic tissue or other airway obstruction, lip dysfunction, and tongue problems (such as abnormal function, size, position, and posture). Anterior open bites caused by local factors are often accompanied by a downward rotation of the mandible, excessive eruption of the posterior teeth, or undereruption of the anterior teeth. The main strategy of early intervention is to overcome these abnormalities by eliminating or preventing the cause, as well as to correct or guide the affected sites, through the following tactics:

- Elimination or control of the etiologic factors that are disturbing incisor eruption and alveolar growth (such as finger sucking, tongue thrust, and lip dysfunction)
- Prevention or correction of maxillary and mandibular molar overeruption
- Prevention, control, or guidance of clockwise rotation of the mandible
- Incisor retraction to "close the drawbridge" between separated maxillary and mandibular incisors
- Achievement of an increase in the posterior facial height–anterior facial height ratio

Different fixed and removable appliances have been used to prevent and control the force of finger sucking and abnormal tongue thrusting in children (see chapter 6). The purpose of this discussion is to clarify the mechanotherapy and tactics that can be efficient in early treatment of anterior open bite.

Thumb or finger sucking is perhaps the most common type of non-nutritive oral habit present in children. Depending on the severity of the problem and type of finger sucking, this habit can cause severe dentoskeletal defects, including open bite (ranging from a simple dental open bite to a severe skeletal open bite), posterior crossbite, overeruption of molars, and mandibular rotation. Thus, treatment would

be more efficient if started at an earlier age.

Placement of the tongue between the incisors has been sought as a causative factor for anterior open bite for some years. Subtelny[10] stated that abnormal tongue function between incisors is a type of adaptation of the tongue to the open bite area formed by previous finger sucking. Proffit[11] showed that individuals who place the tongue tip forward when they swallow usually do not have more tongue force against the teeth than do those who keep the tongue tip back; in fact, tongue pressure may be lower.

Swallowing is not a learned behavior. Forward tongue placement is a physiologic, subconscious function to fill the gap present between incisors during swallowing. The lips are brought together, and the tongue is placed between the separated anterior teeth to form an anterior seal to prevent food or liquids from escaping. Correction of anterior open bite is more complicated and disturbing when persistent tongue activity is present. Resolving the problem is easier if it is corrected at an earlier age; correction of open bite with persistent tongue thrust is much more difficult in the permanent dentition.

Depending on the etiologic factors and the complexity of the abnormality, early orthodontic treatment for open bite ranges from observation to simple habit control to complex surgical procedures.

Classification of open bite

To simplify the treatment planning process for anterior open bite correction during the primary or early mixed dentition, the author classifies open bite into three types of abnormality: type I, type II, and type III.

Type I simple dental open bite

Simple dental open bite is an anterior open bite in which some environmental factor has affected anterior tooth eruption and/or vertical growth of the alveolar process; as a result, a gap has developed between the anterior teeth. The size of the gap depends on the severity of the cause. In response to the open bite, abnormal tongue function has developed subconsciously to close the gap during swallowing, and therefore continuous tongue force maintains or worsens the gap. In type I open bite, no skeletal discrepancy or molar overeruption is involved; only the incisors or the dentoalveolar process is underdeveloped.

Type II combined dental open bite

In type II anterior open bite, an abnormality was initially caused by some environmental factor plus some minor skeletal disharmony or molar overeruption. In these situations, sustained local factors or hereditary background can cause further deformity to the dentition or basal bone, such as molar overeruption or slight mandibular rotation; therefore, signs of vertical problems are also present.

Type III dentoskeletal open bite

Type III open bite is an anterior open bite in which hereditary or congenital defects with or without local factors have changed the growth pattern of the child and therefore have changed the morphology and foundation of the dentoskeletal occlusion. This type of open bite is usually combined with some type of divergence of dentoskeletal structures and can be clearly recognized by cephalometric evaluation. Early detection and proper intervention can solve the problem or at least reduce the severity of the anomaly. Treatment during the permanent dentition is much more complicated and occasionally requires orthognathic surgery.

Treatment considerations

Early intervention in open bite differs depending on the stage of dentition and the type of open bite. Early treatment strategy for open bite malocclusion can be divided into three periods of dental development: primary dentition, mixed dentition, and permanent dentition. The tactics for treatment of the permanent dentition were discussed briefly earlier in the chapter. The following sections focus on early orthodontic intervention during the primary and mixed dentition periods.

Management during the primary dentition

Generally, active orthodontic treatment of open bite during the primary dentition is not recommended, because most cases at this stage of the dentition involve local factors such as pacifier or finger sucking or mouth breathing, which can be self-corrected after etiologic factors are eliminated. However, periodic observation by the dentist or the awareness of the parents can be helpful.

If abnormal habits are persistent and severe enough to affect normal jaw growth, for example by preventing anterior dentoalveolar growth or causing mandibular rotation, the patient and the parents should be advised that these severe habits can be controlled with a simple appliance such as a fixed tongue guard.

Management during the mixed dentition

After careful clinical and paraclinical evaluations of the problem are completed, treatment during the mixed dentition stage can be designed in one or two phases with or without orthognathic surgery. The pretreatment assessments should carefully verify the following:

- Type of open bite (skeletal, dental, or combination)
- Etiologic factors (whether the cause is still active)
- Morphology of the malocclusion and affected structures
- Skeletal age of the patient
- Cooperation of the patient and parents

The best age for early intervention is during the early mixed dentition.

Tactics of Early Intervention During the Mixed Dentition

The main strategy of early intervention is to eliminate or prevent the cause of the abnormality behind the open bite by controlling or guiding the affected sites.

Depending on the stage of the dentition, etiologic factors, characteristics, and the morphology of the open bite, the following six tactics may be applied.

1. Habit control

Control of finger or tongue force as an early intervention is the first and most important tactic in treatment of this anomaly. This tactic will facilitate anterior tooth eruption, alveolar growth, and open bite correction. Several kinds of fixed or removable appliances are available for this purpose. This tactic is very helpful in treatment of type I open bite, and if applied at the proper time such appliances can correct the problem without the need for further mechanotherapy (see cases 13-1 to 13-3).

As Rakosi[3] and Huang et al[12] have shown, the tongue crib has been used widely and successfully to stop thumb sucking and the abnormal tongue forces that produce and maintain anterior open bite.

2. Lip seal and swallowing exercises

To establish normal neuromuscular function and maintain stability of the result, lip seal and tongue exercises have been recommended.[3,13] The patient is instructed to keep the lips together at all times and swallow without thrusting the tip of the tongue toward the maxillary or mandibular incisors. These exercises can be performed before appliance therapy and during treatment and then continued during retention.

3. Growth modification

The third tactic for open bite correction during the mixed dentition involves correction or guidance of the affected sites by controlling vertical growth, overeruption of the posterior teeth, and dentoalveolar development. This goal can be achieved by controlling the eruption or intrusion of the posterior segments. Overeruption of molars can occur in patients with prolonged finger sucking while the posterior teeth are not in contact.

Different devices have been proposed for this purpose: simple bite block, spring-loaded bite block, functional appliances, titanium screw anchorage, magnetic active vertical corrector, anterior extrusion arch. Two appliances that the author has designed and applied with excellent results— the tongue crib–transpalatal arch (TC-TPA) and the step-up or step-down anterior arch— are introduced here.

Tongue crib–transpalatal arch

The TC-TPA is a combination of a modified transpalatal arch and tongue crib (Fig 13-4). This appliance is made with 0.036- or 0.040-inch stainless steel wire and consists of two parts (the transpalatal arch and the tongue crib) in a one-unit appliance (see case 9). The transpalatal bar should be separated from the palate by at least 1 mm. The omega loop is located distally to help molar intrusion during swallowing. The tongue crib or tongue guard is designed as a vertical loop (see Fig 13-4a) or as a bow with vertical pieces of wire soldered to the arch (see Fig 13-4b).

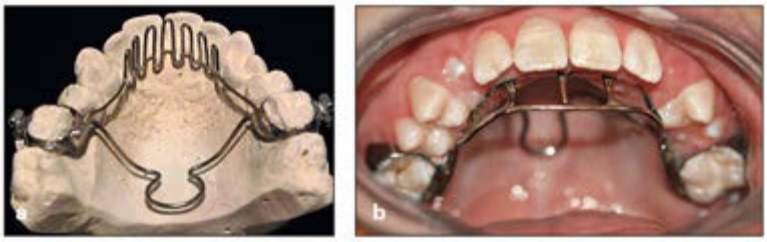


Fig 13-4 *(a and b)* TC-TPA appliance. This appliance can control tongue force, prevent molar eruption, and intrude the molars.

When the treatment plan for anterior open bite calls for intrusion of the maxillary molars, it is necessary to prevent overeruption of the mandibular molars at the same time; the results of intrusion of maxillary molars alone may not be effective. The best tactic for this purpose is insertion of a mandibular lingual arch at the same time.

Step-type anterior arch

In patients with a vertical growth pattern, early intervention can be followed with treatment of the permanent dentition by application of special archwires that the author has designed, the step-up and step-down anterior arches. Figure 13-5 shows a step-down anterior arch for the maxillary arch (see case 13-9).

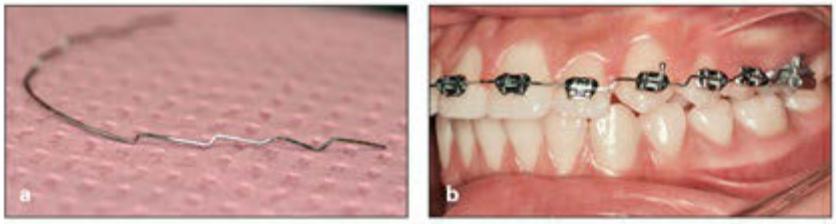


Fig 13-5 *(a and b)* Step-down anterior arch to intrude the posterior teeth and extrude the anterior teeth.

In severe open bites involving the maxilla, the mandible, and the dentition, two step-type arches can be applied at the same time: one step-down arch in the maxillary dentition and one step-up arch in the mandibular dentition. When skeletal open bite is combined with underdeveloped anterior dentoalveolar structure, vertical elastics can be added to step-down arches if vertical loops are incorporated in the maxillary and mandibular anterior segments.

The step-down and step-up arches can also be used in the treatment of deep bite problems by changing the direction of the steps opposite to what is used in open bite cases (see Deep Bite section later in chapter).

4. Closing the drawbridge

Another tactic suitable for elimination of some anterior open bites is retraction of proclined incisors. This method can be applied in patients with incisor spacing or after premolar extraction (Fig 13-6). This approach has been termed *closing the drawbridge* by Subtelny and Sakuda.[14]

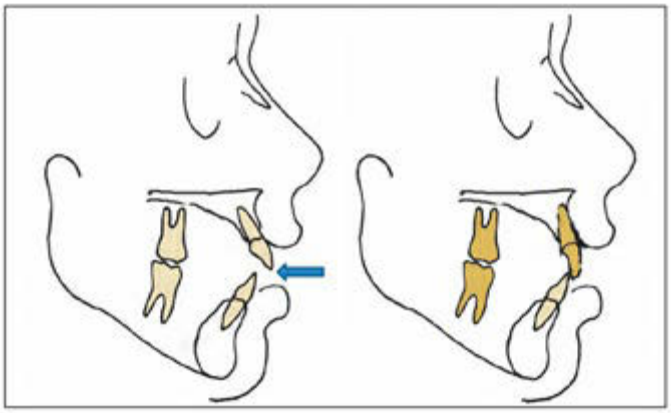


Fig 13-6 Retraction of incisors followed by gap closure (closing the drawbridge).

5. Increase of posterior facial height– anterior facial height ratio

Increasing the ratio of the posterior facial height to the anterior facial height is another tactic for correction of skeletal anterior open bite. Combinations of bite block and anterior vertical elastics or combinations of bite block with vertical chin cap or high-pull headgear have been used for this purpose by many investigators.[15,16]

6. Combined mechanotherapy with selective extraction

For some morphologic situations, selective extraction can be used along with mechanotherapy. These approaches include extraction of premolars to close the drawbridge in cases of anterior protrusion (see case 13-8) and extraction of permanent molars to close the bite by inducing counterclockwise rotation of the mandible (see case 13-10).

Case Reports

The following case reports show the different types of open bite: type I simple dental open bite (cases 13-1 to 13-3), type II combined dental open bite (case 13-4), and type III dentoskeletal open bite (cases 13-5 to 13-10).

Case 13-1: Type I open bite

A 14-year-old boy presented with an anterior open bite. He had a history of thumb sucking that was stopped by age 9 years, but a severe tongue thrust habit continued (Figs 13-7a to 13-7d).

Clinical, cast, and cephalometric evaluations indicated that he had a simple dentoalveolar open bite caused by persistent tongue thrust. No skeletal discrepancy or molar overeruption was evident.

Treatment:
The treatment plan called only for the application of a fixed tongue guard and tongue exercises (Fig 13-7e). This treatment was very effective and terminated after 9 months. The corrected occlusion was maintained with a Hawley retainer. Figures 13-7f to 13-7h show the posttreatment results.

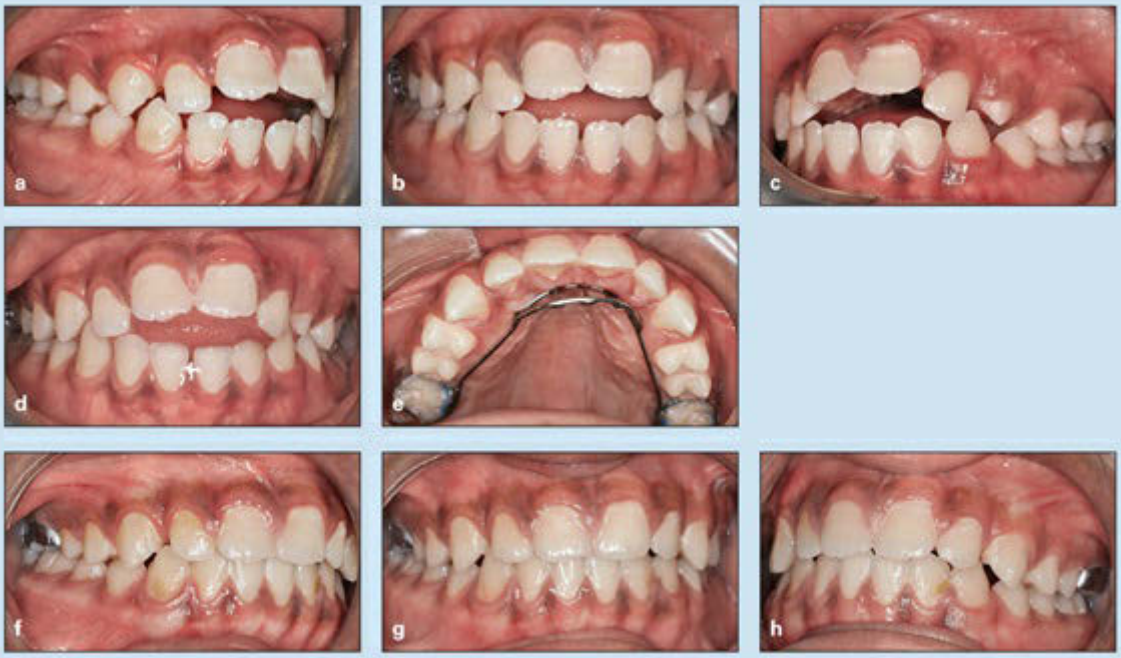


Fig 13-7 Management of simple anterior open bite in a 14-year-old boy with a history of thumb sucking and severe tongue thrusting. *(a to c)* Pretreatment occlusion. *(d)* Activity of the tongue during deglutition. *(e)* Fixed tongue guard, the only appliance used. *(f to h)* Posttreatment occlusion.

Case 13-2: Type I open bite

A 12-year, 4-month-old girl in the late mixed dentition presented with a severe anterior dental open bite (3 to 5 mm) extending from the left primary second molar to the right primary second molar (Figs 13-8a and 13-8b). She had a Class I dentoskeletal pattern and no skeletal abnormality or divergence. According to the parents, no finger sucking was involved, but the patient had a prolonged history of

pacifier use.

Treatment:

The problem was diagnosed as a dentoalveolar anterior open bite, and the treatment plan was simply to control tongue forces and instruct the patient in tongue exercises. Because the patient showed good compliance, the only appliance used was a removable tongue guard (Figs 13-8c and 13-8d). Figures 13-8e to 13-8g show the patient's posttreatment occlusion.

Significant dentoskeletal changes that were achieved were anterior tooth eruption, slight retroclination of the incisors, change from open bite of –3.5 mm to overbite of 1.9 mm, reduction in overjet from 4.0 to 2.3 mm, and change in point A–nasion–point B (ANB) angle from 4.5 to 2.6 degrees (Figs 13-8h to 13-8j).

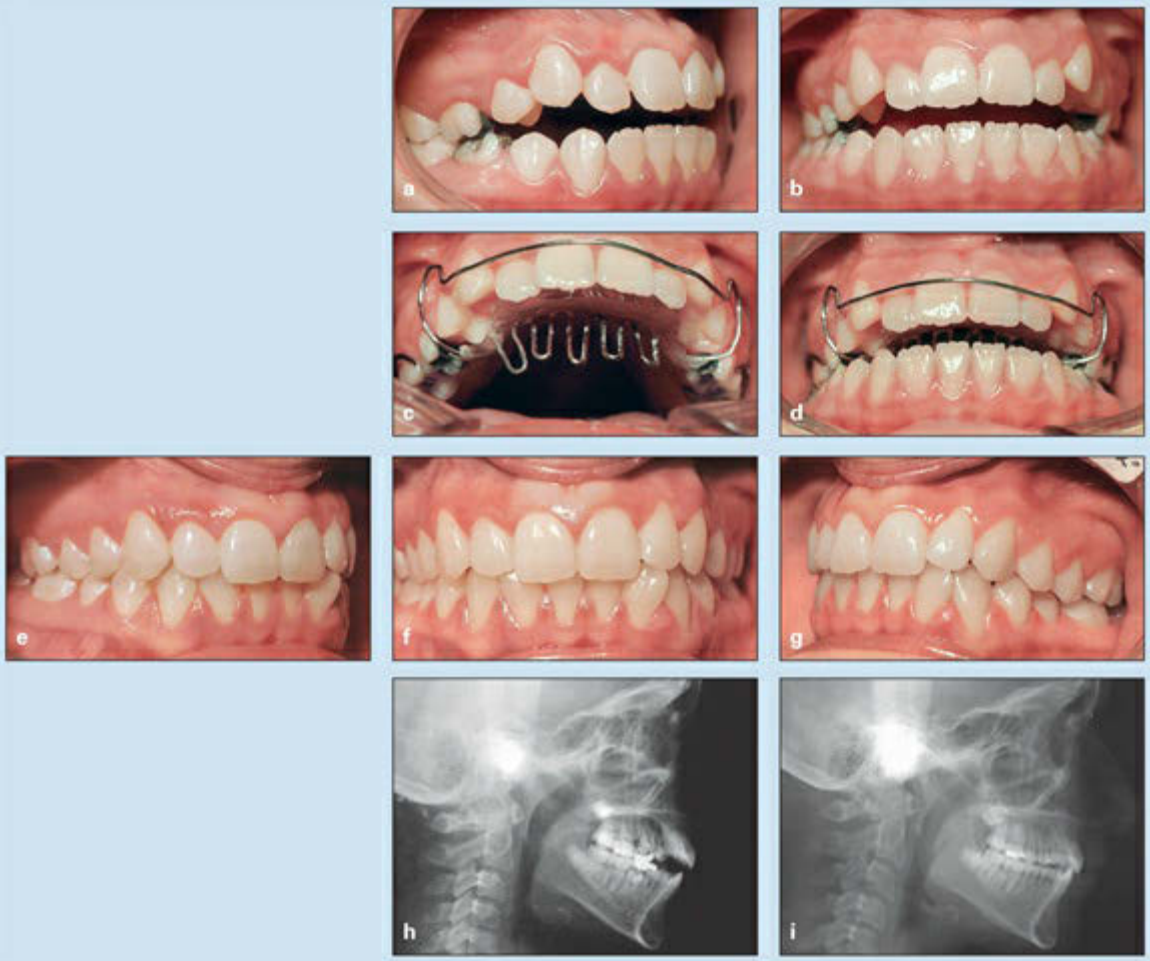


Fig 13-8 Management of severe anterior dental open bite extending from the maxillary left to maxillary right primary second molars in a 12-year, 4-month-old girl in the late mixed dentition. There is no skeletal abnormality. *(a and b)* Pretreatment occlusion. *(c and d)* Removable tongue guard, the only appliance used. *(e to g)* Posttreatment occlusion. *(h)* Pretreatment cephalometric radiograph. *(i)* Posttreatment cephalometric radiograph.

j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	86.9	90.0
Maxillary depth (FH-NA) (°)	90.0	90.6	90.9
ANB (°)	2.3	4.5	2.6
FMA (MP-FH) (°)	25.9	30.6	24.2
Y-axis (SGn-SN) (°)	59.4	70.9	66.9
Interlabial gap (mm)	0.0	5.5	1.5
Upper face height (N-AGS) (mm)	50.0	51.6	49.8
Lower face height (ANS-Gn) (mm)	65.0	69.6	69.7
Mx base–occ plane (PP-OP) (°)	10.0	7.4	3.4
Interincisal angle (U1-L1) (°)	135.0	129.6	138.9
IMPA (L1-MP) (°)	95.0	88.8	87.2
Overbite (mm)	2.5	–3.5	1.9
Overjet (mm)	2.5	4.0	2.3

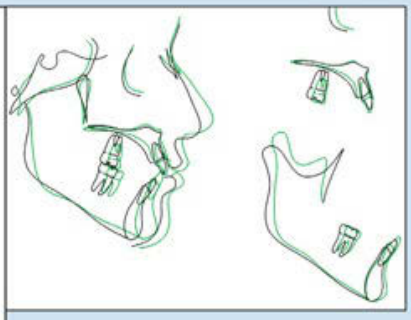


Fig 13-8 *(cont) (j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 13-3: Type I open bite

A 9-year-old boy with thumb-sucking and tongue-thrusting habits presented with an anterior dental open bite, slight maxillary constriction, and a mandibular shift (Figs 13-9a to 13-9d). Clinical and paraclinical evaluations indicated that he had a type I simple dental open bite that originally was caused by thumb sucking and later was combined with tongue thrusting.

Treatment:
Because the slight maxillary constriction on the right side was causing mandibular shift, treatment was designed as a slight unilateral maxillary arch expansion with simultaneous control of tongue force. The appliance used was a quad helix unilateral expander with a tongue guard soldered to the anterior bow (Fig 13-9e). Figures 13-9f to 13-9h show the results after phase 1 treatment and correction of anterior open bite and posterior crossbite.

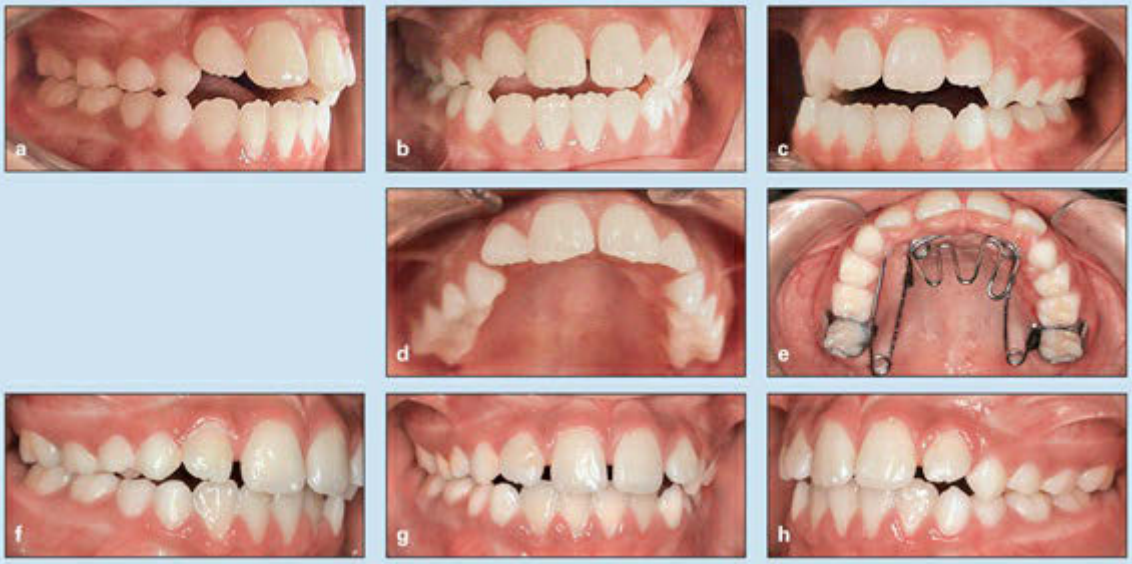
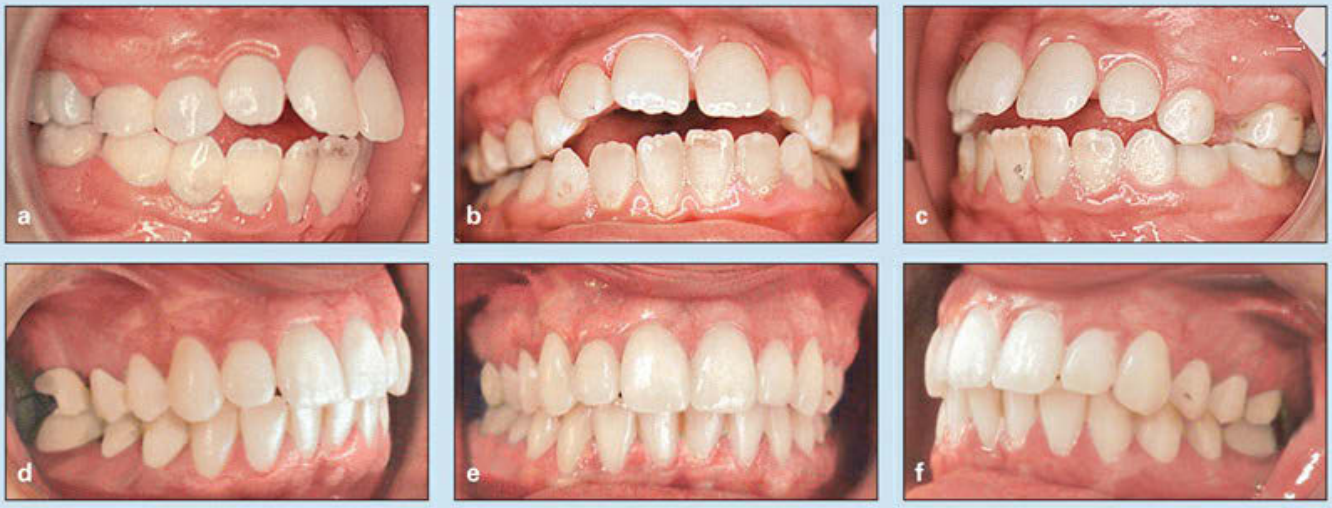


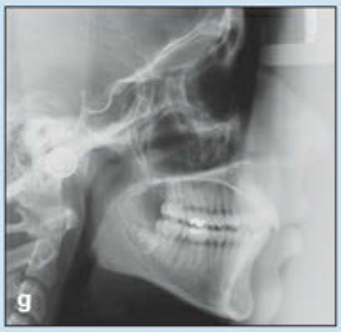
Fig 13-9 Management of anterior dental open bite, slight maxillary constriction, and mandibular shift in a 9-year-old boy with thumb-sucking and tongue-thrusting habits. *(a to d)* Pretreatment occlusion. *(e)* Unilateral quad helix expander with a tongue guard. *(f to h)* Occlusion after completion of phase 1 treatment and correction of anterior open bite and posterior crossbite.

Case 13-4: Type II open bite

A 9-year, 10-month-old girl presented with Class I occlusion, an anterior dentoalveolar open bite (type II combined open bite), and maxillomandibular dental protrusion (Figs 13-10a to 13-10c). She exhibited a tongue thrust habit and had a history of previous thumb sucking. Cephalometric analyses indicated that she had a slight vertical growth pattern (Frankfort plane–mandibular incisor angle [FMA], 30.6 degrees; y-axis, 73.4 degrees; lower facial height, 68.7 mm.

Treatment:
Based on the morphologic conditions, the treatment strategy was to control tongue force and to alleviate the vertical growth tendency by intrusion of the maxillary molars and prevention of extrusion of the mandibular molars, which would in turn correct the open bite and induce counterclockwise rotation of the mandible. This patient was given a maxillary TC-TPA to control the tongue force and simultaneously intrude the maxillary molars. A lower holding arch was used to prevent mandibular molar extrusion. Figures 13-10d to 13-10h show the results of treatment.





h

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	83.4	85.4
Maxillary depth (FH-NA) (°)	90.0	89.7	91.2
ANB (°)	2.3	6.8	5.9
FMA (MP-FH) (°)	25.9	30.6	34.2
Y-axis (SGn-SN) (°)	59.4	73.4	76.3
Interlabial gap (mm)	0.0	2.4	1.7
Upper face height (N-AGS) (mm)	50.0	45.9	51.1
Lower face height (ANS-Gn) (mm)	65.0	68.7	70.6
Mx base–occ plane (PP-OP) (°)	10.0	13.5	6.8
Interincisal angle (U1-L1) (°)	135.0	105.2	122.5
IMPA (L1-MP) (°)	95.0	100.3	92.7
Overbite (mm)	2.5	–2.6	0.6
Overjet (mm)	2.5	4.9	2.6

Fig 13-10 Management of Class I anterior dentoalveolar open bite with maxillomandibular dental protrusion and tongue thrusting in a 9-year, 10-month-old girl. *(a to c)* Pretreatment occlusion. *(d to f)* Posttreatment occlusion. *(g)* Posttreatment cephalometric radiograph. *(h)* Changes in cephalometric measurements.

Case 13-5: Type III open bite

An 11-year-old girl presented with Class I occlusion, a 3.5-mm anterior open bite, a tongue thrust, and a vertical growth pattern (Figs 13-11a to 13-11c). She had no history of finger sucking.

Treatment:
Treatment was designed to have two phases. Phase 1 was control of tongue forces and interceptive treatment (growth modification) to control the vertical growth pattern. Mechanotherapy at this stage was limited to a TC-TPA for maxillary molar intrusion and tongue control (Fig 13-11d) and a lower holding arch to control mandibular molar eruption (Fig 13-11e). Figures 13-11f and 13-11g show the

change in occlusion after 8 months of interceptive treatment, which clearly resulted in molar intrusion (no contact in the molar area) and significant improvement of the anterior open bite. Significant cephalometric dentoskeletal changes were achieved in this phase of treatment (Figs 13-11h to 13-11j). These changes can be summarized as intrusion of maxillary molars, prevention of mandibular molar eruption, mandibular counterclockwise rotation, control of the increased lower facial height, and correction of anterior dentoskeletal open bite. Phase 2 will be carried out after complete eruption of the permanent dentition (excluding third molars).

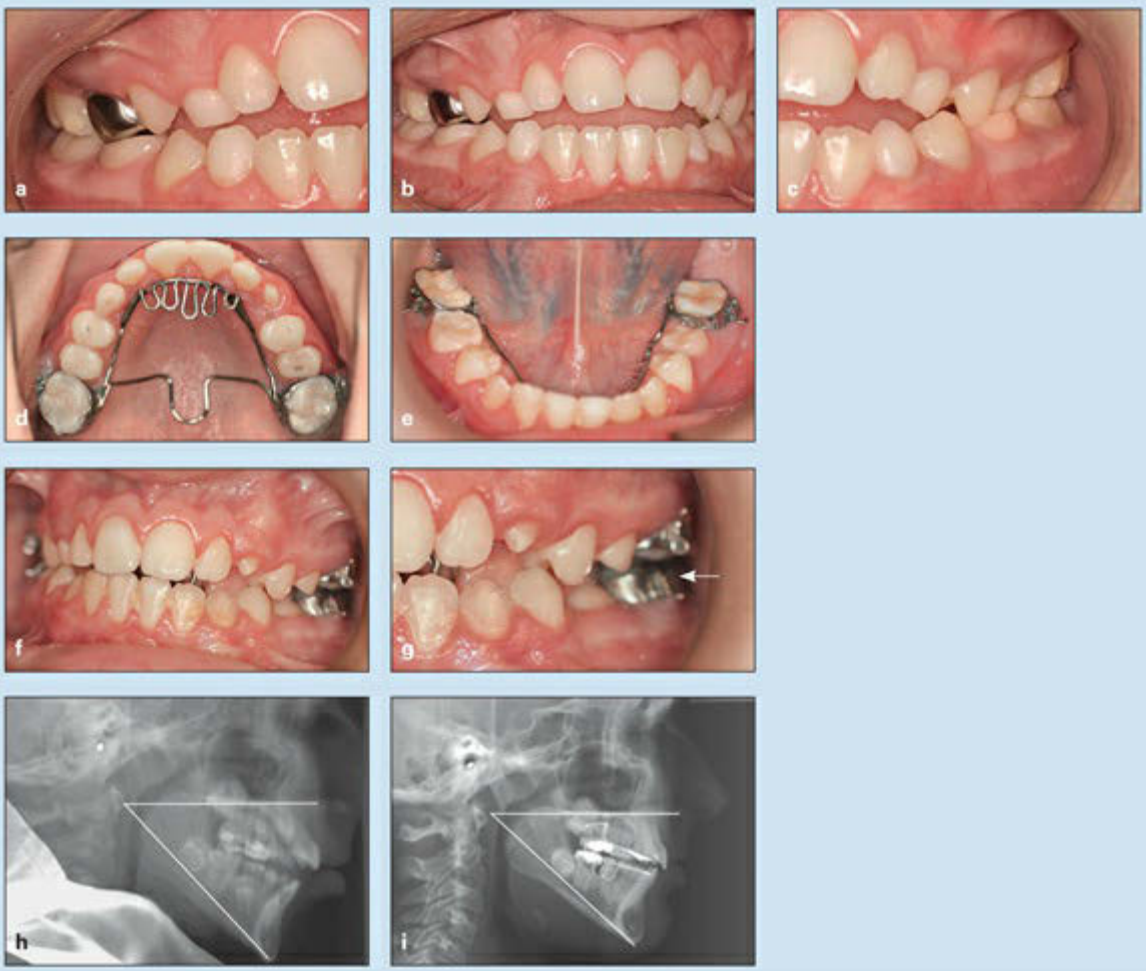


Fig 13-11 Management of Class I malocclusion and type III anterior open bite and tongue thrust in an 11-year-old girl. She has a vertical growth pattern and no history of finger sucking. *(a to c)* Pretreatment occlusion. *(d)* Combined TC-TPA for maxillary molar intrusion and tongue control. *(e)* Lower holding arch to control mandibular molar eruption. *(f and g)* Posttreatment occlusion with only 8 months of interceptive treatment. *(arrow)* Molar separation achieved by intrusion. *(h)* Pretreatment cephalometric radiograph. *(i)* Posttreatment cephalometric radiograph.

j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.2	84.6
Maxillary depth (FH-NA)	90.0	81.9	84.7
ANB (°)	2.3	1.3	1.4
FMA (MP-FH) (°)	25.9	36.9	32.1
Y-axis (SGn-SN) (°)	59.4	68.6	67.1
Interlabial gap (mm)	0.0	1.0	1.5
Upper face height (N-AGS) (mm)	50.0	34.8	42.2
Lower face height (ANS-Gn) (mm)	65.0	55.3	53.5
Mx base–occ plane (PP-OP) (°)	10.0	16.7	12.8
Interincisal angle (U1-L1) (°)	135.0	116.9	122.0
IMPA (L1-MP) (°)	95.0	84.0	94.7
Overbite (mm)	2.5	–4.8	–0.7
Overjet (mm)	2.5	4.3	2.4

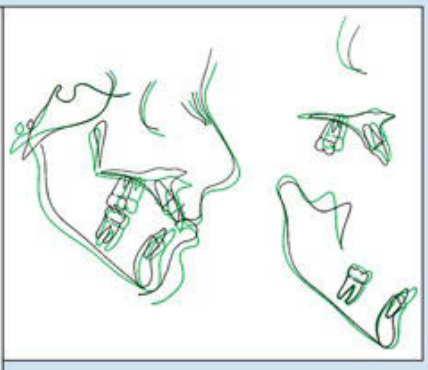


Fig 13-11 *(cont)* *(j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 13-6: Type III open bite

A 9-year, 10-month-old boy presented with a Class II division 1 malocclusion and a severe anterior dentoskeletal open bite caused by a severe vertical growth pattern and divergent skeletal structure combined with a severe finger-sucking habit (two fingers) and consequently a severe tongue thrust. Clinical and paraclinical evaluations indicated the following problems: Class II malocclusion; dentoskeletal open bite; steep mandibular plane angle; divergent facial pattern (long-face syndrome); undereruption of the anterior teeth and overeruption of the posterior teeth, resulting in mandibular rotation; and space deficiency (Figs 13-12a to 13-12f).

Treatment:

The treatment plan was divided into two phases. Phase 1 included the following appliances: a TC-TPA to control the finger habit and tongue force and intrude the maxillary molars; a mandibular bite block to prevent further eruption and to intrude the posterior mandibular segments; and maxillary high-pull headgear to intrude and distalize the maxillary molars to obtain Class I relationships (Fig 13-12g and 13-12h).

Phase 2 consisted of complete maxillary bonding for leveling and correction of the rotation and application of a step-down arch for final correction of the open bite. The results of treatment are shown in Figs 13-12i to 13-12m.

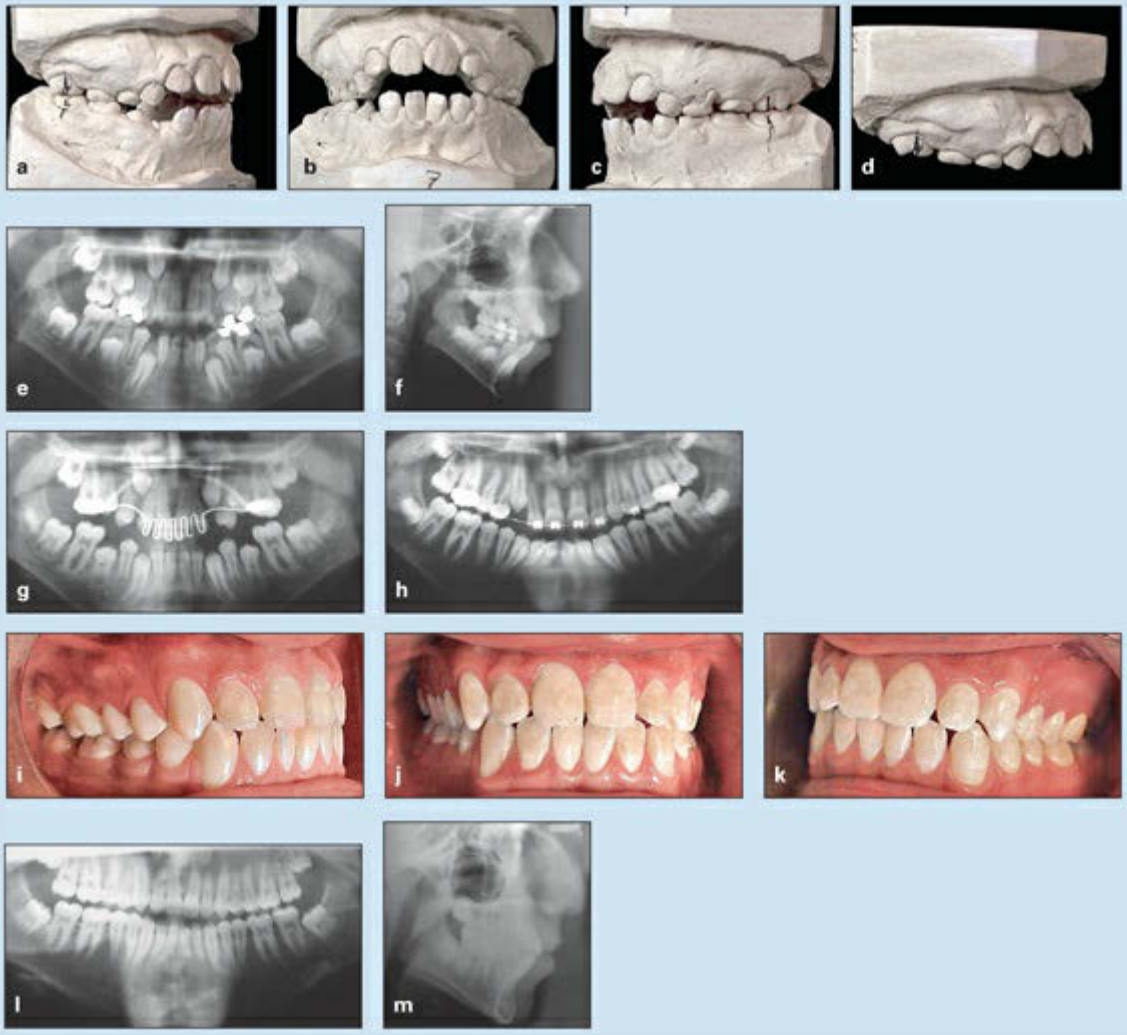


Fig 13-12 Management of severe dentoskeletal anterior open bite in 9-year, 10-month-old boy with a Class II division 1 malocclusion. The open bite is caused by a severe vertical growth pattern, divergent skeletal structure, and severe finger sucking and tongue thrusting. *(a to d)* Casts of the pretreatment occlusion. *(e)* Pretreatment panoramic radiograph. *(f)* Pretreatment cephalometric radiograph. *(g and h)* Panoramic radiographs taken during treatment. *(i to k)* Posttreatment occlusion. *(l)* Post-treatment panoramic radiograph. *(m)* Posttreatment cephalometric radiograph.

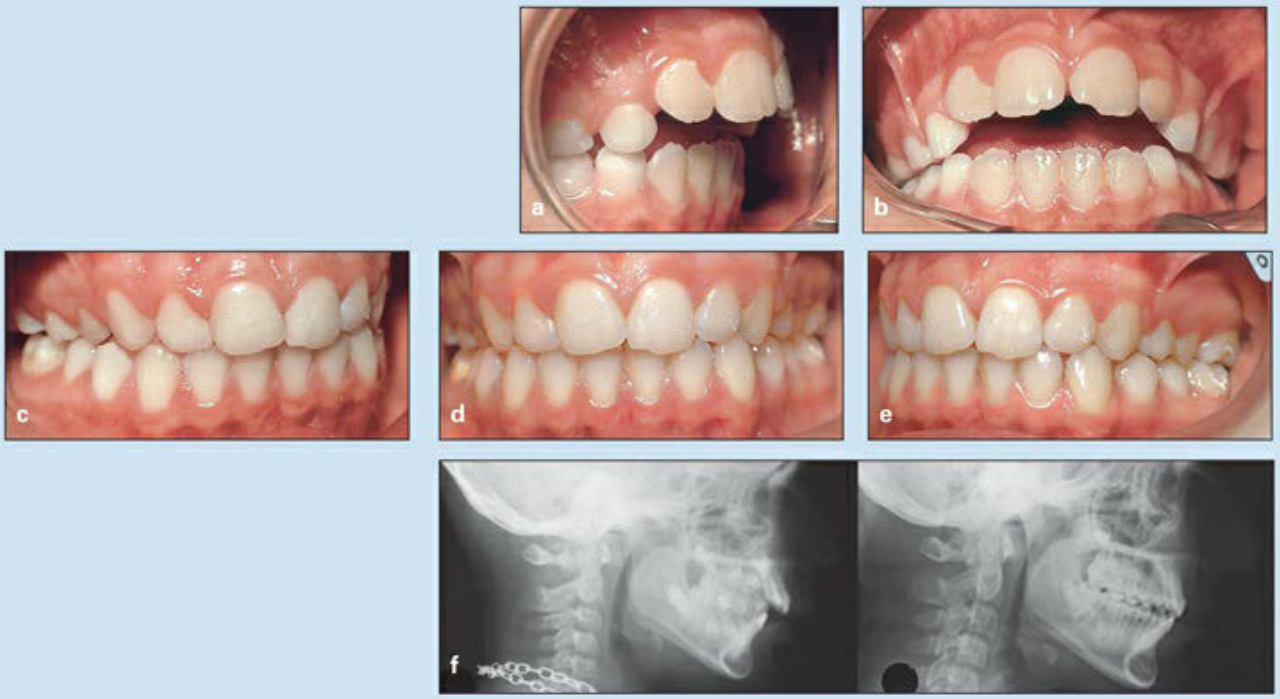
Case 13-7: Type III open bite

A 7-year, 6-month-old girl in the middle mixed dentition presented with a Class II division 1 malocclusion, severe overjet (10.7 mm), and open bite (4.0 mm). She had a severe thumb-sucking habit that was combined with tongue thrusting (Figs 13-13a and 13-13b). Cephalometric evaluation indicated slight mandibular plane steepness, an increased y-axis, increased lower facial height, and a high A-B discrepancy.

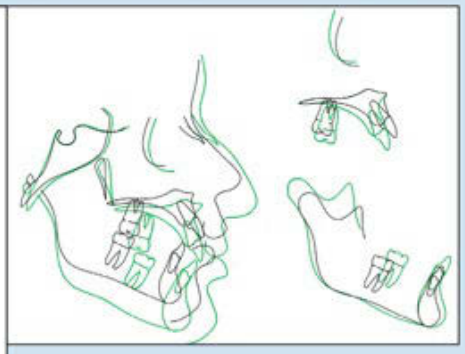
Treatment:
Treatment started with placement of a TC-TPA appliance and high-pull headgear to control the habit and abnormal tongue forces and to intrude and distalize the maxillary molars for normal molar

relationships and bite closure. A heavy lingual lower holding arch was applied to preserve E space and prevent molar eruption. Figures 13-13c to 13-13e show the posttreatment occlusion.

Cephalometric evaluations indicated that treatment resulted in the following changes: correction of the Class II malocclusion, reduction in overjet from 10.7 to 2.6 mm, change in overbite from –4.0 to 1.1 mm, and reduction in ANB from 8.4 to 3.2 degrees (Figs 13-13f and 13-13g).



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	85.8	85.2
Maxillary depth (FH-NA) (°)	90.0	92.3	87.0
ANB (°)	2.3	8.4	3.2
FMA (MP-FH) (°)	25.9	25.9	28.7
Y-axis (SGn-SN) (°)	59.4	66.7	68.9
Interlabial gap (mm)	0.0	4.8	1.5
Upper face height (N-AGS) (mm)	50.0	44.6	49.8
Lower face height (ANS-Gn) (mm)	65.0	66.8	74.2
Mx base–occ plane (PP-OP) (°)	10.0	11.9	11.9
Interincisal angle (U1-L1) (°)	135.0	121.7	127.9
IMPA (L1-MP) (°)	95.0	92.3	97.6
Overbite (mm)	2.5	–4.0	1.1
Overjet (mm)	2.5	10.7	2.6



g

Fig 13-13 Management of a Class II division 1 malocclusion, severe overjet, and severe open bite in a 7-year, 6-month-old girl with severe thumb-sucking and tongue-thrusting habits. *(a and b)* Pretreatment occlusion. *(c to e)* Posttreatment occlusion. *(f)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(g)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 13-8: Type III open bite

A 9-year, 8-month-old boy presented with severe skeletal open bite. As a result of the severe clockwise rotation of the mandible, early loss of the primary molars, and tipping of the permanent molars, the only occlusal contact between the maxillary and mandibular dentitions was between the distal cusps of the permanent first molars (Figs 13-14a to 13-14d). The patient had a long lower facial height and divergence, a convex profile, and severely procumbent lips due to maxillomandibular dental protrusion. There was space deficiency in both the maxillary and mandibular dentitions; cephalometric analysis showed that the open bite was 5.4 mm and the overjet was 0.9 mm.

Treatment:

The treatment plan was based on extraction of both maxillary first premolars and both mandibular permanent first molars to achieve the following goals: closure of the bite, counterclockwise rotation of the mandible, creation of space for the dentition, and retraction of both the maxillary and mandibular anterior segments.

Treatment procedures were started with insertion of a TC-TPA to serve as anchorage, to control tongue force, and to prevent overeruption of the maxillary molars and intrude them. The second stage of mechanotherapy, started after maxillary canine eruption, was complete bonding for leveling and canine retraction. The next step was mandibular bonding, after permanent second molar eruption was completed, for leveling and mandibular anterior retraction. The final stage was maxillary incisor retraction and coordination of both arches. Treatment achieved open bite correction, with the overbite changing from –5.4 to 1.2 mm and the overjet changing from 0.9 to 2.0 mm (Figs 13-14e to 13-14j). Incisor proclination and consequently lip protrusion were normal, and the patient's profile improved.

Despite the good dental relationships and interdigitation and intercuspation, the extreme vertical growth pattern continued, indicating that severe dentoskeletal deformity would have developed if early treatment had not been initiated.

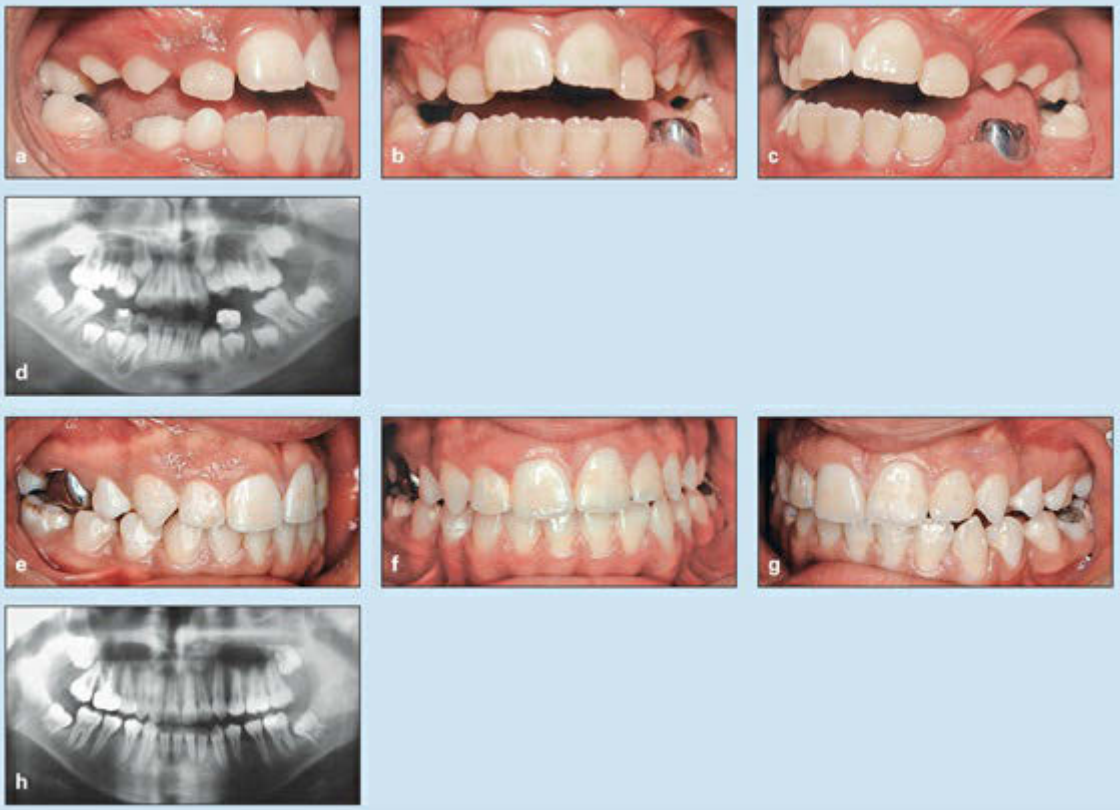
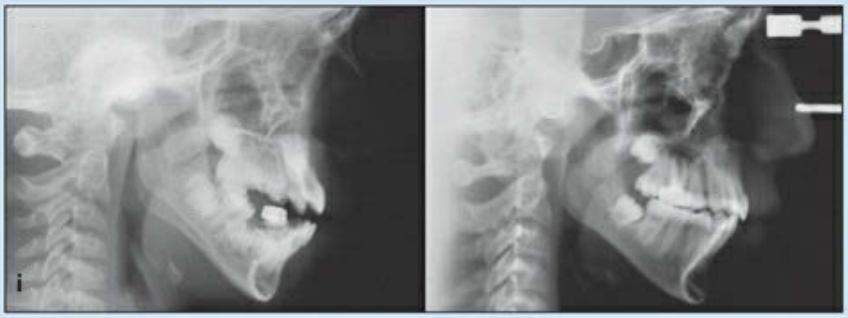
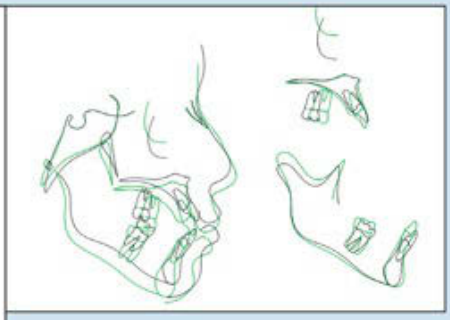


Fig 13-14 Management of severe skeletal open bite in a 9-year, 8-month-old boy. There is severe clockwise rotation of the mandible. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph. *(e to g)* Posttreatment occlusion. *(h)* Post-treatment panoramic radiograph.



i

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	84.7	76.7
Maxillary depth (FH-NA) (°)	90.0	85.4	80.1
ANB (°)	2.3	0.2	3.7
FMA (MP-FH) (°)	25.9	31.1	40.6
Y-axis (SGn-SN) (°)	59.4	69.6	73.1
Interlabial gap (mm)	0.0	1.8	3.5
Upper face height (N-AGS) (mm)	50.0	48.3	53.6
Lower face height (ANS-Gn) (mm)	60.0	69.2	73.0
Mx base–occ plane (PP-OP) (°)	10.0	6.4	7.6
Interincisal angle (U1-L1) (°)	135.0	110.7	128.0
IMPA (L1-MP) (°)	95.0	93.7	87.7
Overbite (mm)	2.5	–5.4	1.2
Overjet (mm)	2.5	0.9	2.0



j

Fig 13-14 *(cont) (i)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(j)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Case 13-9: Type III open bite

A 9-year, 6-month-old girl presented with a Class I occlusion and an anterior open bite resulting from maxillary molar overeruption, a severe tongue thrust, and a vertical growth pattern (Figs 13-15a to 13-15c). She had no history of finger sucking.

Treatment:
The treatment strategy was based on interceptive treatment to control the abnormal tongue force and modify the vertical growth tendency. The treatment procedure was begun with insertion of a TC-TPA (see Fig 13-4b) and initiation of tongue exercises to control abnormal tongue function, intrude the maxillary molars, and induce mandibular counterclockwise rotation (Figs 13-15d to 13-15f). This stage was followed by maxillary full bonding, and treatment continued with a step-down maxillary arch (see Fig 13-5a) to reinforce maxillary molar intrusion and provide gradual extrusion of the maxillary dentition anteriorly (Figs 13-15g to 13-15i). Figures 13-15j to 13-15l show the final results of treatment.

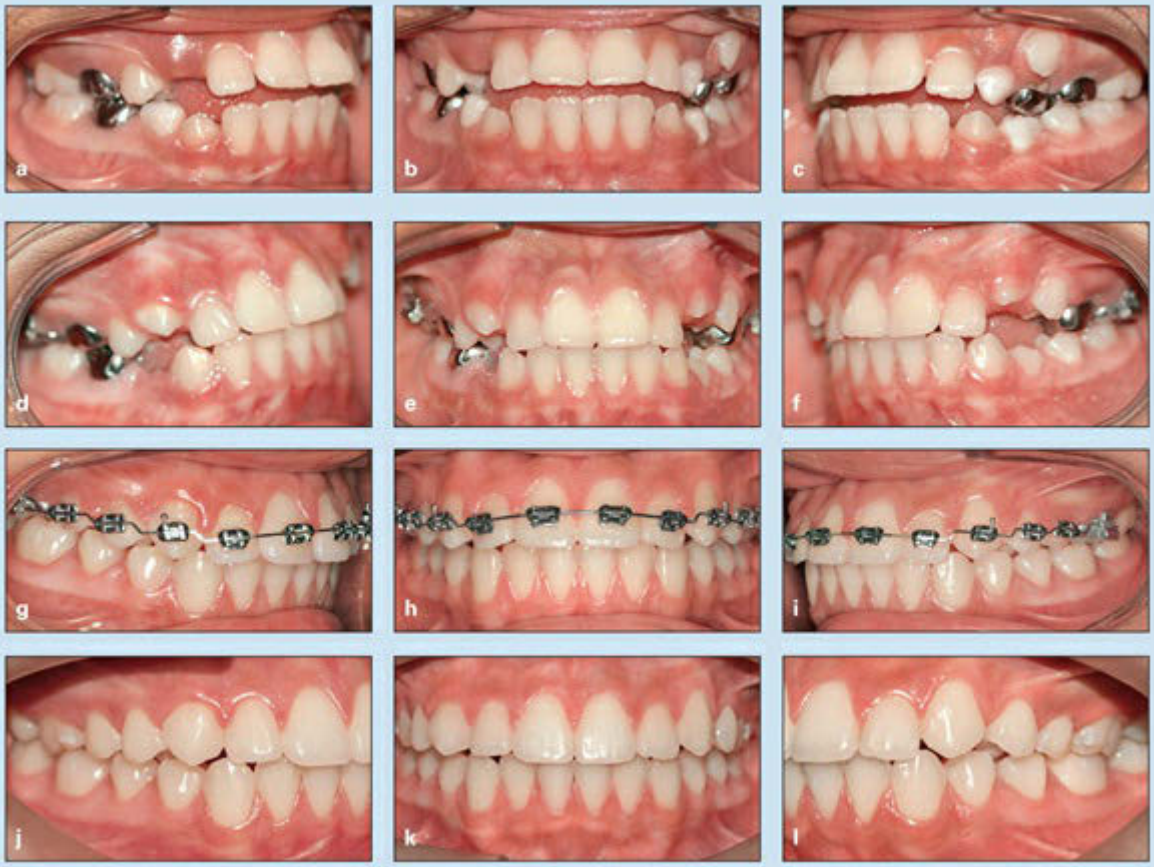


Fig 13-15 Management of Class I malocclusion with type III anterior open bite in a 9-year, 6-month-old girl. The open bite is caused by overeruption of the maxillary molars, severe tongue thrusting, and a vertical growth pattern. *(a to c)* Pretreatment occlusion. *(d to f)* Progress after 7 months' use of a TC-TPA appliance and tongue exercises. *(g to i)* Final phase of treatment, with maxillary full bonding and step-down archwire. *(j to l)* Posttreatment occlusion.

Case 13-10: Type III open bite

An 11-year, 6-month-old girl presented with Class III dentoskeletal problems, a long-face pattern, and anterior teeth in an end-to-end relationship (Fig 13-16a to 13-16c). The pretreatment panoramic radiograph revealed the severe mesial position of the mandibular dentition relative to the maxillary dentition, especially at the molars (Fig 13-16d).

Clinical and cephalometric analyses revealed a long mandibular base, severe divergence of the lower face, a long lower facial height (67.6 mm), an FMA of 35.6 degrees, a y-axis of 70.1 degrees, a negative sagittal basal relationship (ANB angle, –2.7), normal maxillary and mandibular incisor inclinations, and a normal interincisal angle.

Treatment:
The treatment strategy was mandibular counterclockwise rotation and slight retraction of the mandibular incisors to correct the incisor relationships and reduce the vertical problem. To accomplish these goals, treatment included the following procedures:

- Extraction of the mandibular permanent first molars
- Placement of a maxillary transpalatal arch similar to the transpalatal arch used in the TC-TPA

method, to intrude the maxillary molars and serve as anchorage until maxillary canine eruption

- Banding of the mandibular second molars and complete bonding
- Placement of a mandibular posterior bite block to intrude the posterior segment, thereby helping in counterclockwise rotation of the mandible, and to provide anchorage during premolar retraction.
- Complete banding of the maxillary arch for leveling and then placement of a step-down arch to increase the overbite anteriorly after canine alignment.
- Removal of the mandibular bite block and insertion of a lower holding arch to prevent mandibular molar extrusion after establishment of Class I relationships between the maxillary first and mandibular second molars
- Retraction of the mandibular anterior segment and coordination of both the maxillary and mandibular dentitions

Figures 13-16e to 13-16j show the posttreatment results.

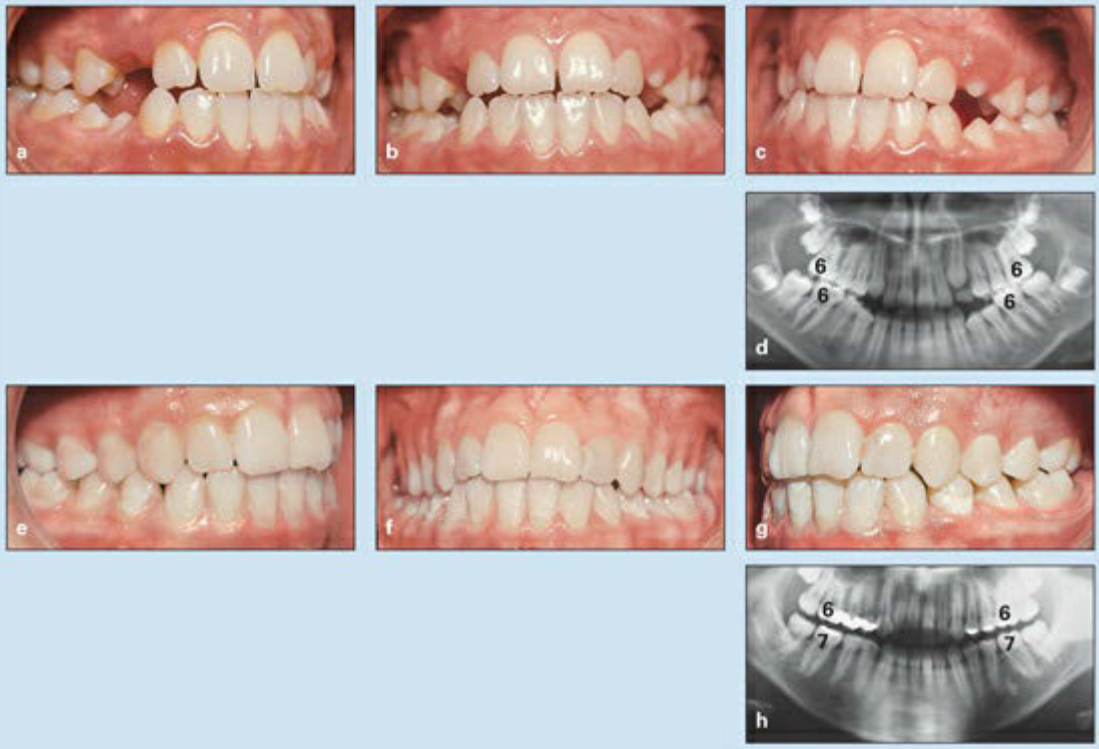
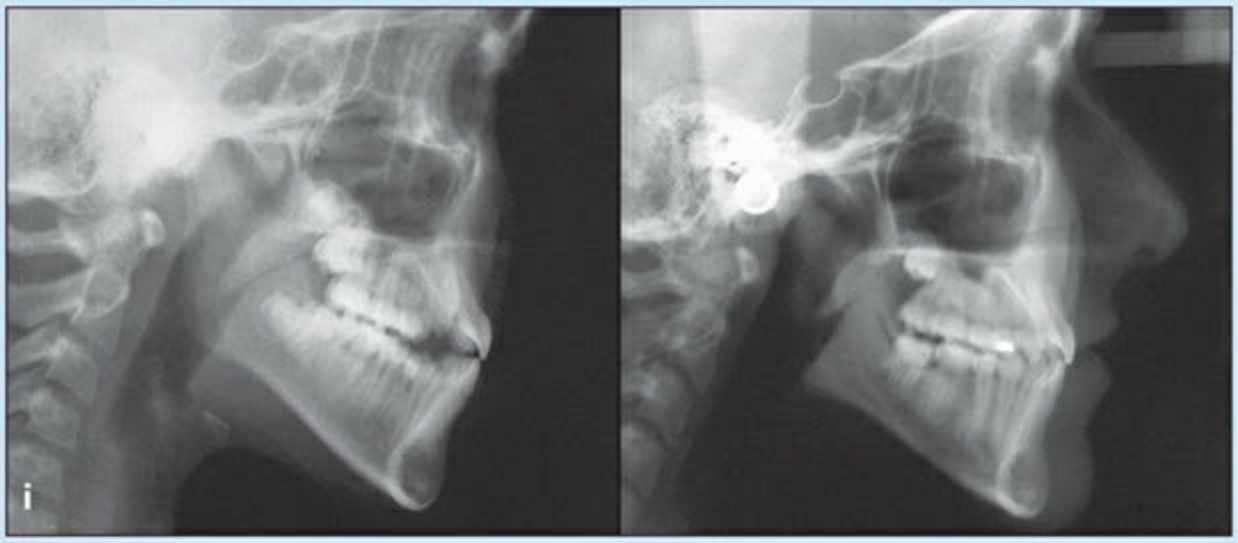


Fig 13-16 Management of a Class III dentoskeletal malocclusion in an 11-year, 6-month-old girl with a long-face skeletal pattern and end-to-end anterior tooth relationships. *(a to c)* Pretreatment occlusion. *(d)* Pretreatment panoramic radiograph showing the severe forward position of the mandibular molars and entire mandibular dentition. 6—permanent first molars. *(e to g)* Posttreatment occlusion. *(h)* Posttreatment panoramic radiograph. 6—permanent first molars; 7—permanent second molars.



j

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	84.6	85.9
Maxillary depth (FH-NA) (°)	90.0	82.1	86.5
ANB (°)	2.3	–2.7	1.8
FMA (MP-FH) (°)	25.9	35.6	33.2
Y-axis (SGn-SN) (°)	59.4	70.1	69.9
Interlabial gap (mm)	0.0	5.2	1.8
Upper face height (N-AGS) (mm)	50.0	49.7	49.7
Lower face height (ANS-Gn) (mm)	65.0	67.6	73.1
Mx base–occ plane (PP-OP) (°)	10.0	12.6	10.1
Interincisal angle (U1-L1) (°)	135.0	129.6	139.0
IMPA (L1-MP) (°)	95.0	84.5	77.9
Overbite (mm)	2.5	–0.8	0.2
Overjet (mm)	2.5	0.0	2.1

Fig 13-16 *(cont) (i)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(j)* Changes in cephalometric measurements.

Deep Bite

Excessive anterior overlapping of incisors, or *deep bite*, is a common malocclusion

that poses a significant problem for orthodontists because treatment is difficult, and the strong relapse tendency means that results lack long-term stability.

Strang[17] defined *overbite* as "the overlapping of the upper anterior teeth over the lower teeth in the vertical plane." Deep bites are not always alike; deep bite may be associated with a Class I, a Class II division 2, or a Class II division 1 malocclusion, and reverse overbite may be associated with a Class III malocclusion; each type has a different skeletal morphology, different etiology, and different treatment strategies.

Normal occlusion is defined as the overlapping of maxillary incisors over mandibular incisors with a range of 2 to 3 mm. However, because the crown lengths of maxillary and mandibular incisors vary significantly between individuals, percentage is a more accurate and descriptive evaluation of the amount of overbite. Thus, more appropriately, around 5% to 30% of overlap is accepted as normal overbite. According to Nanda,[18] a range of 25% to 40% may be considered normal, provided that no functional problems exist during various movements of the temporomandibular joint. Neff[19] expressed overbite as a percentage of the mandibular incisor covered by the corresponding maxillary incisor; he considered a 20% overbite to be ideal.

Etiology

Excessive overlapping of incisors is not the whole problem in a patient with excessive overbite; rather, it is a part of the total malocclusion. Deep bites are not all alike; they can involve a simple overlapping of incisors, dentoalveolar overlap, skeletal overclosure, or a combination of these. Each of these can develop through different etiologic factors, have different morphologic characteristics, and require different treatment options.

Strang[17] suggested that the cause of developing incisor overbite is either supereruption of the incisors, infraeruption of the posterior teeth, or a combination. Diamond[20] believed that excessive overbite is due to a lack of growth in the vertical height of the mandibular ramus. In cephalometric studies, Wylie[21] concluded that the growth of the mandibular ramus has no direct relationship on the development of overbite.

Development

In a survey of 52 series of casts taken before and after eruption of the permanent incisors, Baume[22] assessed the development of overbite during the transitional dentition. He found that change in the amount of overbite depends on three factors: *(1)* the degree of overbite in the primary dentition, *(2)* the forward growth of the mandible relative to the maxilla, and *(3)* the relative position of the mandibular incisor edges to the maxillary incisor edges; he explained the effects of these factors as follows.[22]

Degree of overbite. The degree of overbite during the primary dentition is a determining factor in the formation of overbite during the permanent dentition. A slight overbite affecting the primary teeth is followed by increased overbite of the permanent teeth; deep bite during the primary dentition has an unfavorable prognosis for the permanent dentition.

Amount of mandibular forward growth. The degree of overbite depends primarily upon the amount of mandibular forward growth. If the mandible grows at the same rate as the maxilla, incisor overlap will develop normally. If the mandible grows more slowly than the maxilla, the overbite will increase; if the mandible grows more quickly, the amount of overbite will decrease.

Baume[22] also explained that these growth changes take place during three different periods: *(1)* during eruption of the primary canines, which determines the amount of overbite of the primary incisors; *(2)* during eruption of the permanent incisors, which regulates the permanent incisor overlap; and *(3)* during eruption of the permanent canines and premolars, which defines the degree of permanent overbite.

Relative position of the mandibular incisors. Baume[22] stated that the position of the edge of the mandibular incisors guides the eruption of the maxillary incisors; the more the mandibular incisors remain lingual to the maxillary incisors, the greater the overlap by the maxillary incisors.

Flemming[23] showed that the extent of vertical overbite varies with age. At 5 to 6 years of age, the percentage of overbite varies between 36.5% and 39.2%; the overbite usually increases from 9 to 12 years of age and then decreases from 12 years to adulthood. Therefore, in any type of sagittal or transverse occlusal relationship, there may be normal or abnormal overlapping of the incisors in the vertical dimension.

Generally, the cause of excessive overbite is complex and may be genetic, environmental, or a combination. The morphology of different deep bites can also

demonstrate variations of different structures. For the purposes of differentiation, the etiology, morphologic characteristics, and treatment of deep bite can be reviewed under two classifications:

1. Dental deep bite
2. Skeletal deep bite (overclosure or closed bite)

Dental deep bite

Dental deep bites can develop because of supraocclusion (overeruption) of the incisors, infraocclusion (undereruption) of the posterior teeth, or a combination of both factors.

Dental deep bites are primarily caused by environmental factors and therefore can be assumed to be acquired. The following environmental or local conditions can result in dental deep bite:

- Diminished posterior dental height due to loss of posterior teeth and mesial tipping of the posterior segment
- Early loss of primary molars and lingual tipping of permanent molars, causing scissors bite (buccal bite) and collapse of the mandibular arch
- Early loss of the primary canines, lingual collapse, and overeruption of the mandibular anterior teeth
- Abrasion of molar teeth or molar cusps
- Bilateral undereruption of primary teeth due to ankylosis
- Periodontal disease and pathologic mesial migration of the posterior teeth that worsens the existing anterior deep bite
- Hypodontia or microdontia in one arch relative to the opposing arch
- Buccal bite and mandibular collapse
- Perioral neuromuscular imbalance
- Imbalance of the posterior vertical chain of orofacial muscles
- Abnormal oral habits such as lateral tongue thrust, bruxism, clenching, lip dysfunction

Neuromuscular balance around the dentition is an important factor in the development of normal occlusion. It is well known that equilibrium between the buccinator, mentalis, and orbicularis oris muscles and the tongue is required for

normal occlusal development.

The posterior vertical chains of muscles (masseter, internal pterygoid, and temporal muscles) is another neuromuscular mechanism that can influence the vertical dimension of occlusion. The posterior vertical chains of muscles are strong and attached anteriorly to the mandible. During function, these muscles stretch in a nearly straight vertical direction. The molars are directly under the impact of masticatory forces during function. When the posterior vertical chain of muscles is strong and anteriorly positioned, a greater depressive action is transmitted to the dentition, preventing vertical growth of the mandible and the alveolar process and eruption of the posterior teeth and consequently resulting in excessive anterior bite. When these chains of muscles are positioned slightly posteriorly and the direction of force is posterior, there is a potential for downward and backward rotation of the mandible and incisor open bite tendencies.

Abnormal oral habits can sometimes influence the vertical dimension. For example, lateral tongue thrust can affect posterior tooth eruption, disturb posterior vertical height, and consequently increase anterior bite. Bruxism and clenching can affect posterior height by grinding the occlusal surfaces of posterior teeth. In addition, the continuous abnormal vertical forces can affect posterior vertical growth and molar eruption.

Another abnormal habit that can increase incisor overlap is lip dysfunction. Abnormal lower lip function can result from hypertonicity of the lower lip muscles (mentalis and orbicularis oris muscles) or develop as an abnormal function of the lower lip when overjet is increased and the lower lip is displaced behind the maxillary incisors (Fig 13-17).

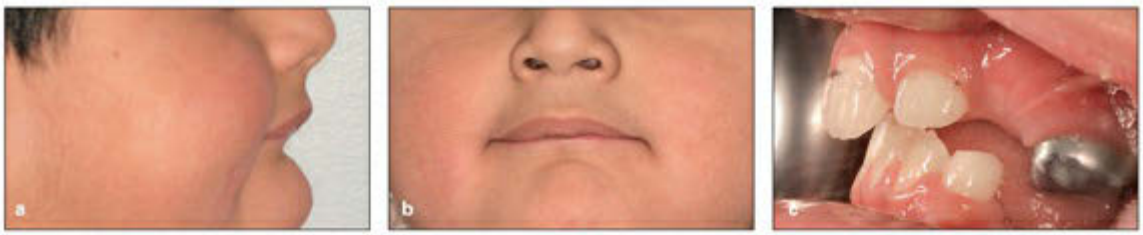


Fig 13-17 *(a to c)* Hypertonic lower lip muscles (mentalis and orbicularis oris), causing lingual tipping of the mandibular incisors and increased overbite and overjet.

During swallowing, lip dysfunction not only increases overjet but also can cause retroclination and overeruption of the mandibular incisors and consequently a two-step occlusion resulting in incisor deep bite.

Skeletal deep bite (closed bite)

Skeletal deep bite (closed bite) or complex deep bite is a dentoskeletal malrelationship of dentoskeletal structures in the vertical dimension. In other words, it is an overlapping of incisors combined with a malrelationship of vertical basal bone. This can be a malrelationship of alveolar bones and/or underlying mandibular or maxillary bones or a malrelation-ship caused by an overgrowth or undergrowth of one or both alveolar segments. Complex deep bite is frequently found in patients with Class II division 2 malocclusion and occasionally in individuals with Class III malocclusion.

This anomaly is usually the result of specific hereditary characteristics of the dental, skeletal, and soft tissue profiles; therefore, management is more complicated and difficult, and the chance of relapse is high if the condition is treated late.

Morphologic characteristics

Dental deep bite

Dental deep bites or simple deep bites are confined to the teeth and alveolar processes. Therefore, the problem lies mainly within the dentition. This abnormality develops because of excessive overlapping of incisors (incisor overeruption), molar undereruption, or a combination of both.

Excessive incisor overlap can be due to extrusion of incisors with normal inclination, such as in Class II division 1 deep bite; or it can be the result of incisor extrusion combined with retroclination of incisors that exaggerates the situation, such as in Class II division 2 malocclusion. Patients with dental deep bite usually have a normal vertical skeletal relationship of the basal bones, normal lower facial height, normal profile, normal upper lip–lower lip relationships, and normal lip-chin and lip-nose relationships.

Other variables also control the depth of the bite:

- Length of the incisor crowns
- Long axis of the incisors, which can accentuate the depth by more retroclination
- Width of the maxillary dental arch (a broader maxillary arch increases anterior deep bite)

Skeletal deep bite

Dentoskeletal characteristics. The skeletal morphology dominates the dental features in skeletal deep bite. Patients with skeletal deep bite show the following dentoskeletal characteristics:

- Reduced lower facial height (brachycephalic)
- Excessive freeway space
- Increased ramus height and width
- Large coronoid process
- Two-step occlusion (incisors and canines at higher levels relative to the premolars and molars)
- Strong temporalis and masseter muscles
- Small gonial angle
- Increased ramus height and posterior cranial base causing counterclockwise rotation of the mandible
- Short and broad mandibular symphysis
- Deep nasion relative to frontal boss
- Small cranial base angle
- Convergent face (four facial planes of the face are horizontal)
- Straight or concave profile

Soft tissue and profile characteristics. Patients with skeletal deep bite exhibit the following soft tissue characteristics:

- Competent lips
- Thin lips with excessive height
- Curled appearance of lips
- Deep mentolabial sulcus

Relapse

Excessive overbite in adolescents and adults is challenging and difficult to treat, and there is a high tendency for relapse. Canut and Arias[24] assessed the study casts of 30 subjects with Class II division 2 malocclusion pretreatment, at the end of treatment, and postretention after a period of at least 3 years. The researchers found that molar

relationships were stable, overcorrection of overbite showed some relapse (10% of patients showed maxillary and 30% mandibular irregularity of anterior teeth after retention), and mandibular arch length and width showed a decrease.

Wasilewsky[25] studied treated cases of deep bite 10 years or more after retention and observed 22.5% correction at the time of retention and 44.9% relapse. The following factors were most commonly associated with relapse:

- Maxillary molar extrusion
- Forward positioning of the mandibular molars (as part of treatment)
- Inadequate mandibular posterior dentoalveolar height
- Divergent facial pattern

Maximum relapse was seen in patients with a horizontal mandibular plane, and the minimum relapse occurred in patients with some mandibular steepness. Patients who demonstrated forward positioning of the mandible with growth and vertical growth in the posterior aspect showed long-term stability.[25]

Differential diagnosis

Dental deep bite and skeletal deep bite share some characteristics and differ in others:

- Both dental deep bite and skeletal deep bite cause incisor overlap of different severity.
- Patients with dental deep bite usually have a normal vertical basal bone relationship, while patients with skeletal deep bite have a convergent facial pattern with shorter lower facial height.
- Both kinds of deep bite are associated with a relatively broad facial pattern (brachycephalic pattern), but this characteristic is more severe in closed bite because of the short facial height.
- The soft tissue profile is flat in both patients with skeletal deep bite and those with dental deep bite, but there is greater concavity in patients with the skeletal type because the chin is more prognathic.
- Lips are competent, thin, and curled and show excess height but are more inverted in patients who have closed bite.
- The mentolabial sulcus is deeper in individuals with skeletal deep bite.

- Freeway space is greater in closed bite.
- Closed bite mostly is associated with two-step occlusion, while dental deep bite is associated with a deep curve of Spee.

The occlusal plane provides specific cephalometric differentiation between incisor overeruption and molar undereruption in all kinds of deep bite. The author has used this variable for several years for differentiation of the etiology of both deep bite and open bite. In normal occlusion, the occlusal plane bisects the angle between the mandibular and palatal planes almost equally. In normal occlusion, the palatal plane–occlusal plane angle is slightly smaller than the mandibular plane–occlusal plane angle (see Fig 13-3). In deep bite, if the palatal plane–occlusal plane angle is larger than the mandibular plane–occlusal plane angle, the deep bite is due to overeruption of the maxillary incisors or intrusion of the maxillary molars.

If the palatal plane–occlusal plane is smaller, the deep bite is due to overeruption of the mandibular incisors or intrusion of the mandibular molars. This comparison helps to determine whether maxillary or mandibular molars must be extruded, maxillary or mandibular incisors must be intruded, or both.

Factors influencing the extent of overbite

Other factors that can influence the extent of overbite have been reported. Among the most important are cuspal height, ramus length, and interincisal angle.

Cuspal height

Popovich[26] studied the natural dentition and found that, in patients with normal occlusion, a strong positive correlation existed between cuspal height and the degree of vertical overbite.

Ramus length

Diamond[20] argued that ramus length is the primary factor in the vertical development of dental height. He indicated that retardation of the growth in ramus length inhibited eruption of the posterior teeth but did not interfere with eruption of the anterior teeth, thus resulting in an excessive degree of overbite.

Interincisal angle

The degree of overbite also depends on the degree of angulation of the incisors. Overbite decreases as the interincisal angle decreases. Popovich[26] confirmed this finding by showing a significant positive correlation between the interincisal angle and the degree of vertical overbite.

Treatment of Deep Bite

Consequences of delayed treatment

Failure to correct deep bite problems, especially in the permanent dentition, contributes to a variety of pathologic conditions affecting the masticatory apparatus. For example, a common problem associated with deep bite is difficulty in proper restoration. The general practitioner may refer a patient for orthodontic treatment of excessive deep bite, which usually displays a significant loss of vertical dimension.

Neglected deep bite conditions can interfere with the normal closure pattern of the mandible and lead to periodontal disease, malfunction of the temporomandibular joint, and labial migration of the maxillary incisors.

Interference with the normal closure pattern of the mandible

Interference with the normal closure pattern of the mandible is sometimes seen in deep bite patients. Alexander et al[27] investigated the effects of deep bite malocclusion on masticatory function to determine changes that may occur with orthodontic correction. The study evaluated typical chewing patterns of five subjects with deep bite. Results indicated that deep bite malocclusions did not create a major disturbance in chewing. Some differences between preoperative and postoperative conditions were noted, however. Prior to treatment, three subjects showed a nearly vertical chewing cycle that avoided tooth contact at the border path. Following treatment, all subjects showed a greater coincidence between the closing chewing path and lateral border movement.

Periodontal disease

Many reports indicate that there is a relationship between periodontal pathosis and dental malocclusion. Deep bites in particular are often accompanied by periodontal pathosis, especially in the presence of poor oral hygiene.

Wragg et al[28] reported that deep traumatic overbite can be associated with periodontal lesions. They found an association between deep overbite and unusual periodontal lesions showing on radiographs as circumscribed radiolucencies from the alveolar crest and sometimes close to the apex.

Bollen[29] conducted a systematic review on the effects of a malocclusion on periodontal health and suggested that subjects with a malocclusion have worse periodontal health than subjects without a malocclusion.

Nasry and Barclay[30] reported an association between deep overbite, especially in patients with traumatic overbite, and poor hygiene. They noted that an unusual periodontal lesion often appeared on radiographs as a circumscribed radiolucency and bone destruction, remote from the alveolar crest and sometimes close to the apex. The infection appeared to track labially, creating a tunnel through the bone adjacent to the root surface.

Malfunction of the temporomandibular joint

Temporomandibular joint syndromes with joint pain, tinnitus, clicking, vertigo, and sometimes loss of hearing have been reported in patients with severe deep bite. Sonnesen and Svensson[31] compared two groups of adult patients, 30 with a deep bite and 30 with neutral occlusion. Examinations for temporomandibular dysfunction and bite force were performed, lateral cephalometric radiographs were obtained, and occlusal registrations were completed. Deep bite patients more frequently reported nocturnal and diurnal clenching, an uncomfortable bite, jaw stiffness, and "ringing" in the ears than did the controls. Headache, muscle disorders, disc displacement, and other joint disorders occurred significantly more often in the deep bite group than in the control group.

Labial migration of the maxillary incisors

Labial migration of the maxillary incisors is due to impingement of the mandibular incisors on the palatal tissue or periodontal involvement and bone loss at the maxillary incisors as a result of deep bite and vertical loss.

Advantages of early treatment

Early treatment of deep bite malocclusion has the following advantages:

- Dentoalveolar growth and posterior facial height increase are best achieved through early intervention.
- Long-term stability can be achieved.
- Treatment is readily established.
- Growth potential can produce better skeletal relationships.
- Changes in the dentoskeletal relationships and environment can bring more positive muscular adaptation.
- Less mass tooth movement and comprehensive mechanics are needed.

Treatment considerations

Deep bite correction can be managed into two stages of dentition: during different stages of the mixed dentition or during the permanent dentition (adolescence and adult). In other words, deep bite problems can be managed at early ages as a preventive or interceptive procedure or late during the permanent dentition as a corrective procedure.

Proper management of deep bite problems, like any other malocclusion, requires thorough evaluation and examination to determine the type of problem, including the etiology and morphology. Before treatment planning is started, some important points must be considered for each patient:

- The patient's age, especially skeletal maturation
- Whether the problem is hereditary, environmental, or a combination
- Whether the structures involved are dental, dentoalveolar, skeletal, or a combination
- Whether freeway space is normal, excessive, or short (the greater the freeway space, the better the result)
- The patient's profile (convexity and concavity, lip condition, and chin and nose relationships)
- The facial vertical proportion
- Incisor inclination and relationship to the lips and profile
- Amount of incisor exposure during a smile

Treatment strategies during the permanent dentition

It is widely accepted that correction of deep bite before the patient's skeletal growth is completed is easier to accomplish and provides more stable results. A detailed discussion of deep bite treatment in adults is beyond the scope of this book. However, for the purposes of comparison, the basic strategy and the most common treatment options applied in adult treatment will be briefly reviewed.

By the time the complete permanent dentition has erupted, the age for fast facial growth changes has passed. Therefore, all orthodontic treatment during this stage of dentition is confined to the teeth or movements of the teeth and the alveolar process that have a strong relapse tendency. Bell et al[32] stated that conventional orthodontic correction of Class II deep bite deformity accompanied by decreased mandibular anterior facial height can be mechanically difficult, inefficient, and, in many instances, impossible. Orthodontic treatment alone in either adults or adolescents with such deformities can neither increase mandibular anterior facial height sufficiently to achieve ideal facial proportions nor achieve long-term occlusal stability. To achieve efficient and stable treatment results for this deformity in adults, various surgical techniques in combination with orthodontic treatment have been presented.

Orthodontic treatment mechanics of all deep bite cases in adults or adolescents are generally based on four general tactics:

1. Intrusion of maxillary or mandibular incisors or both
2. Extrusion of maxillary or mandibular posterior teeth or both
3. A combination of intrusion and extrusion of the anterior and posterior segments
4. Combined surgical and orthodontic intervention in patients with severe skeletal overclosure

Intrusion of the incisors

One common strategy of deep bite correction in adult and adolescent patients is intrusion of the maxillary or mandibular incisors. Whether maxillary or mandibular incisors or both must be intruded depends on the results of clinical, cast, and radiographic evaluations.

However, there are also other important points to be considered in this determination:

- Interlabial gap (normally 2 to 4 mm)
- High smile line

- Alveolar problems
- Growth pattern (vertical or horizontal)
- Presence of adequate freeway space or interocclusal clearance

Intrusion of the maxillary incisors is generally indicated in patients with vertical maxillary excess, a large interlabial gap, and a high smile line. The primary goal of the appliance is to achieve "true incisor intrusion."

A variety of mechanical techniques have been applied for incisor intrusion, including intrusion arches, utility arches, three-piece intrusion arches or segmental arch mechanics, and miniscrews.

Extrusion of the posterior teeth

Extrusion of the posterior teeth in adults is another type of treatment for correction of deep bite. Incorrect treatment planning for extrusion of posterior teeth may worsen the esthetics, for example, by increasing the interlabial gap or causing functional and stability problems.

Considering the high tendency for relapse in adult patients, a long retention time with a Hawley anterior bite plate is recommended. The following methods can be used to accomplish extrusion of posterior segments: archwires with a reverse curve of Spee, maxillomandibular vertical elastics (box elastics), combination of anterior bite plate and vertical elastics, and mini-implants.

Early Treatment Strategies for Deep Bite

Considering the complications and difficulty associated with adult treatment of excessive deep bite and the high tendency for relapse, early orthodontic treatment is highly recommended.

Deep bite malocclusion occurs in the primary dentition but is rarely treated at this age. Horizontal growth patterns and deep bite malocclusions have specific facial and dental characteristics that can be recognized in their primary stage of development.

The foundation of occlusion is established during the primary and mixed dentitions. Environmental causes that affect occlusion are also interacting during the primary and mixed dentitions. These environmental causes are mostly detectable,

and in many situations the anomalies that are developing can be controlled. Proper intervention can play an important role in intercepting and guiding abnormality toward normality.

Dental deep bite

As already explained, there are some local causes that can either initiate or aggravate the problem. For the sake of simplicity, the strategies and techniques of early intervention of excessive dental deep bite are discussed as a combination of the three basic parts of the issue together: cause, effect, and remedy. The following are local factors that can initiate or aggravate dental deep bite.

Mesial migration of the posterior teeth

Mesial migration of the posterior teeth after early loss of primary or permanent teeth is a common local cause of early development of anterior deep bite. When posterior teeth shift mesially, incisor overlap increases. This process is clearly evident as it is happening and can be prevented or corrected if the mesialized teeth are distalized to their normal position and maintained in place until eruption of the adjacent dentition.

The author recommends using an anterior bite plate at the same time to disocclude the posterior segments. This facilitates distalization of the posterior segments and will help overbite correction.

Abnormal habits

The presence of abnormal habits such as lateral tongue thrust, bruxism, and clenching during occlusal development can disturb normal posterior tooth eruption and vertical alveolar growth and create anterior deep bite. Early recognition and habit control can facilitate normal eruption of the posterior segment, alveolar growth, and opening of the anterior bite.

Lower lip dysfunction

Lower lip dysfunction can result from hypertonicity of the lower lip or from overjet that traps the lower lip behind the maxillary incisors and in front of the mandibular incisors during swallowing. Any type of lip dysfunction during swallowing can increase overjet, cause retraction and overeruption of mandibular incisors, and

increase the curve of Spee, consequently increasing anterior overbite.

The best type of intervention at this age is control of lip dysfunction by application of a lip bumper to separate the lip force from the mandibular incisors and distalize and upright the molars (correcting the curve of Spee and reducing deep bite). Others tactics to control deep bite at this stage of the dentition include *(1)* a maxillary Hawley bite plate to disocclude posterior segments and facilitate overeruption of the posterior segment, consequently reducing overbite, and *(2)* maxillary and mandibular 2 ×4 bonding and application of a utility arch for anterior intrusion.

Early loss of the primary canines

Early loss of the primary canines also causes mandibular incisor retroclination and extrusion, which increases the curve of Spee and anterior deep bite. Treatment involves immediate insertion of a lower holding arch to maintain the dental arch. In situations where arch length has already been lost and overbite has increased, the same option described for management of lip dysfunction can be applied.

Skeletal deep bite

Deep overbite with a horizontal growth pattern has specific facial and dental characteristics and a heredity background. It may manifest itself only as an excessive incisor overlap or as severe overclosure of the jaws, convergent facial pattern, and short anterior facial height, especially short lower facial height; the latter pattern is mostly seen in patients with Class II division 2 malocclusion.

Early intervention during the early and middle mixed dentitions is the best option to guide the abnormality toward normality. The early treatment strategy for this type of anomaly depends on the severity and type of deep bite as well as sagittal skeletal relationships, which vary in different stages of dentition. In general, early deep bite treatment during the mixed dentition in growing children is based on the following strategies:

- Intrusion of the maxillary or mandibular incisors or both
- Extrusion of the maxillary or mandibular posterior segments or both
- Growth modification
- Any combination of the above

To determine whether intrusion, extrusion, or both of these tactics must be applied, or whether growth modification is the proper strategy to overcome skeletal malrelationships, thorough clinical and paraclinical evaluations are an important first step. After clarification and clear recognition of the type of malocclusion, and before initiation of the treatment, other important points must be considered in treatment planning:

- Interlabial gap (normal gap: 2 to 4 mm)
- High smile line
- Alveolar problems
- Facial skeletal pattern and form
- Mandibular form and direction of growth
- Presence of adequate freeway space or interocclusal clearance
- Intraoral and extraoral musculature balance
- The patient's age

Management during the primary dentition

Anterior deep bites are fairly common during the primary dentition but are rarely treated at this stage. Treatment of deep bite during the primary dentition is indicated only when a severe impinging deep bite is present, especially if excessive grinding is associated with the deep bite. The presence of excessive bite during the primary dentition indicates the presence of a skeletal aberration. If the deep bite is not impinging, intervention must be postponed until after permanent incisor eruption.

Management during the early mixed dentition

Normally, overbite slightly increases after eruption of the permanent incisors, and overbite is reduced after eruption of the posterior segment. To take advantage of posterior eruption and alveolar growth, if there is some incisor overlap, incisor inclination is normal, and skeletal disharmony is not present, early intervention can be postponed until the canines and premolars begin to erupt. If the overbite is severe or impinging during the early mixed dentition and evidence of posterior undereruption or a horizontal growth pattern is present, application of a removable bite plate is recommended at this age.

A removable bite plate appliance can reduce overbite for patients who have less

than normal eruption of the posterior teeth or in whom overbite is associated with reduced facial height. The anterior acrylic resin part is made in such a way that the patient's mandibular incisors occlude with the plastic plane lingual to the maxillary incisors and the posterior teeth are disoccluded about 1.0 to 1.5 mm. This encourages posterior tooth eruption and alveolar growth. This appliance must be worn full time. At periodic recalls, if the posterior teeth are reaching the occlusion again, the anterior acrylic resin must be increased to disocclude the posterior segment until normal anterior overlap of incisors is achieved.

For patients who exhibit poor cooperation, the anterior bite plate can be made as a fixed appliance. Two bands with a palatal bow similar to that of the Nance appliance are placed on the maxillary first molars, but the acrylic resin button is behind the maxillary incisors (Fig 13-18).

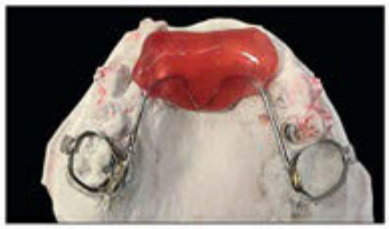


Fig 13-18 Fixed anterior bite plate for disoccluding the posterior segment, facilitating posterior eruption, and reducing anterior overbite.

In either situation, use of the appliance must be continued as a retainer until eruption of the premolars is complete, with full-time use at first and gradually reducing to half-time use.

Management during the middle mixed dentition

Correction of deep bite during the middle mixed dentition can also be managed with the same procedure as recommended during the early mixed dentition, if undereruption of the posterior segment is the cause or if short facial height is present. If incisor intrusion is required, application of a utility arch with 2 × 4 bonding in the maxilla, mandible, or both is the best option at this stage of dentition. If extrusion of the posterior segment is also desired in addition to incisor intrusion, utility arches can be combined with an anterior bite plate. This enhances the correction of deep bite by separating the posterior segments while the utility arches are working both anteriorly and posteriorly.

Utility arches used during the mixed dentition are a type of archwire engaged only to the incisors and molars. They have a bent-down step bypassing the buccal

segment and a V-bend at the end in the buccal tube. Utility arches produce an intrusive force on the anterior teeth and a reciprocal extrusive force on the molars; they can also be used for protraction or even retraction of incisors. Molar extrusion is part of the treatment plan in many cases of incisor deep bite; however, if molar extrusion is not indicated, both extrusion and rotation of molars can be prevented by application of a transpalatal arch or lower holding arch.

The utility arch must be constructed in such a way to avoid impinging on the gingival or buccal soft tissues. Use of soft elastic tubing to cover the areas between the lateral incisor bracket and the molar tube can help this problem.

If incisor deep bite is combined with a Class II malocclusion, application of extraoral traction (headgear) or a functional appliance and growth modification is recommended (see chapter 11).

Management during the late mixed dentition

Comprehensive orthodontic treatment is usually required to treat cases of deep bite during late stages of the mixed dentition. Depending on the type of malocclusion and skeletal variation, treatment can be easy or complicated. The treatment becomes more complicated if there is, in addition to the deep bite, excessive overjet, reverse overjet, crowding in either anterior region, or excessive alveolar bone loss.

One advantage of deep bite correction during the late mixed dentition is the existence of rapid growth changes during the growth spurt that takes place at these ages. These changes can be utilized very effectively for management of closed bite conditions where growth modification is a part of the strategy.

Correction of the curve of Spee or two-step occlusion is another part of deep bite correction during the late mixed dentition or early permanent dentition. This is accomplished by leveling of the teeth to elevate the posterior teeth and depress the anterior segments.

Different appliances used for treatment during this stage of the dentition are the intrusion arch, utility arch, three-piece intrusive arch or segmented arch mechanics, mini-screw, and posterior vertical elastics (box elastics).

Management of combined problems

Anterior deep bite can be associated with other sagittal problems such as Class II or Class III malocclusion. Treatment of these types of abnormalities can be combined with deep bite correction. Depending on the type of sagittal deformity, correction

can be achieved by the use of headgear, face mask, or other functional appliances (see chapter 11).

Appliances

Several types of mechanics are available for overbite reduction, including bite plates, headgear, lip bumpers, lateral box elastics, different types of intrusion archwires, mini-implants, or step-type arches.

Use of step-up and step-down arches was explained earlier in the discussion on open bite correction. With the same mechanism, the step-down arch can also be used for correction of deep bite. In open bite correction, the step-type arch is bent up anteriorly in the mandibular arch (step-up arch), and the step-down arch is applied in the maxillary arch; in deep bite, the designs are reversed.

Each of these devices can reduce overbite; the best results and greatest stability are achieved when the type of malocclusion is identified correctly and proper mechanics are applied. For example, both deep curve of Spee and two-step occlusion can produce deep bite malocclusion, but the mechanical strategy for each of these two anomalies is entirely different. Regardless of the mechanics, however, the best results for treatment of deep bite can be achieved through early-age intervention.

Case 13-11

A 10-year-old boy presented with Class I occlusion and 100% anterior overlap (impinging deep bite). There was no crowding or any other complication (Figs 13-19a to 13-19e).

Treatment:
Because of the patient's age and the stage of dentition, that is, the time of canine and premolar transition, the treatment strategy was to accelerate tooth eruption and alveolar vertical growth in the posterior segment. Therefore, the best treatment option was application of a Hawley appliance with an anterior acrylic resin plane to disocclude the posterior segment (about 1.0 to 1.5 mm). All mobile primary molars were also removed to accelerate eruption. Every 4 weeks, if the posterior segments were reaching occlusal contact, the acrylic resin plane was built up with new material (Figs 13-19f and 13-19g). Figures 13-19h to 13-19k show the resulting overbite correction. The canines and premolars were erupting, and no further treatment would be required.

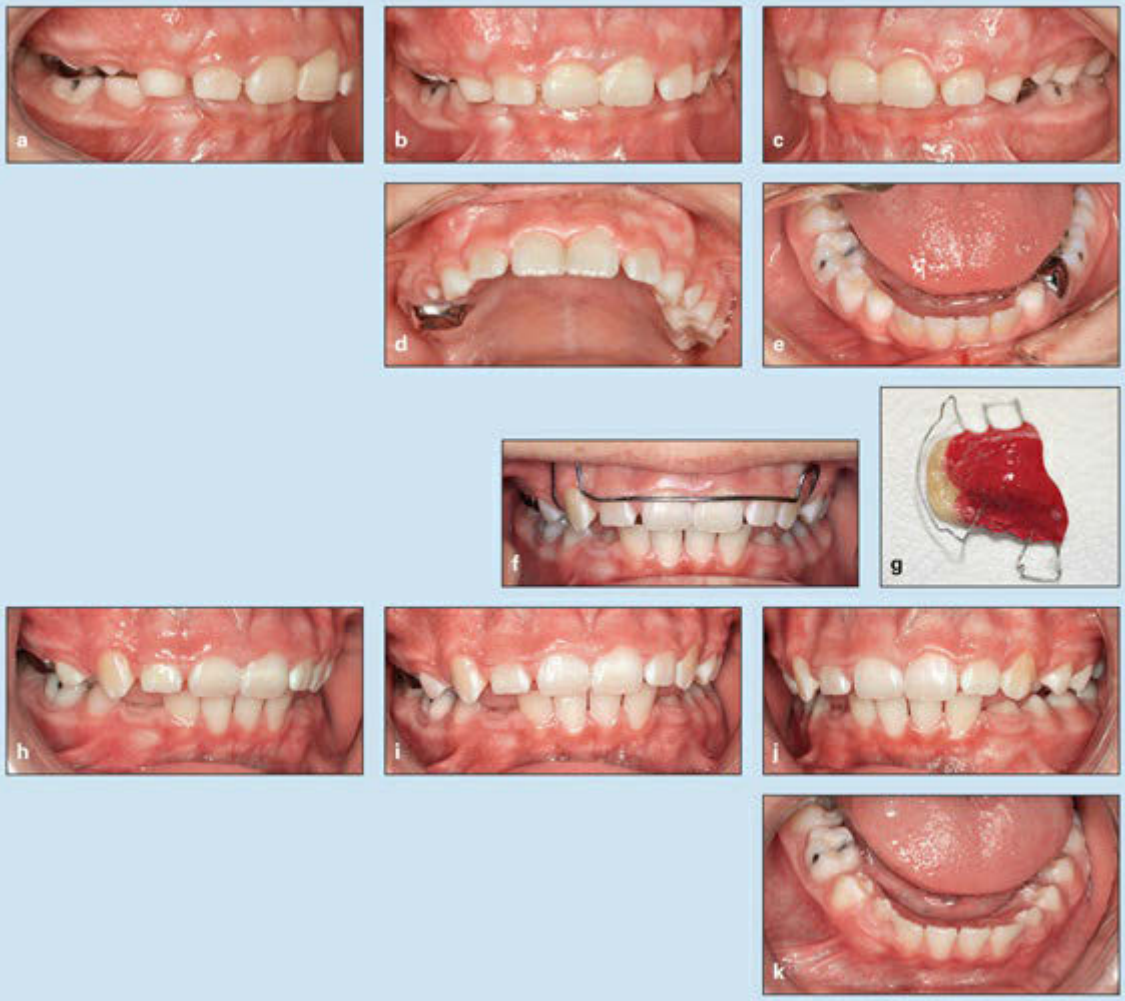


Fig 13-19 Management of 100% anterior overlap (impinging deep bite) in a 10-year-old boy. He has a Class I occlusion and no crowding or other complications. *(a to e)* Pretreatment occlusion. *(f and g)* Hawley anterior bite plate, the only appliance used. *(h to k)* Posttreatment occlusion. The canines and premolars are erupting.

Case 13-12

A 10-year, 6-month-old girl presented with a Class I malocclusion, maxillary right lateral incisor crossbite, mandibular incisor crowding, and lingual displacement of the mandibular permanent right canine (Figs 13-20a to 13-20e). Early loss of the primary canines caused a midline shift, deep bite, anterior crowding, and crossbite. Earlier correction of the crossbite and control of the deep bite might have eliminated the anterior locked occlusion, permitted mandibular anterior tooth alignment, and prevented mandibular incisor crowding.

Treatment:
Treatment in the maxillary arch involved placement of a modified Hawley appliance with posterior occlusal coverage and a Z-spring to correct the lateral incisor crossbite (Fig 13-20f). Occlusal acrylics must be removed immediately after crossbite correction, and acrylic buildup must be added to the anterior part of the Hawley appliance instead of a spring to disocclude the posterior segment while the overbite is reduced. Correction of mandibular incisor crowding was achieved by insertion of a lower

holding arch with a lingual soldered spring to align the incisors. The lower holding arch had no incisor brackets to interfere with the maxillary incisors (Fig 13-20g).

Figures 13-20h to 13-20k show the final stage of active treatment, and Fig 13-20l shows the lower holding arch used as a retainer.

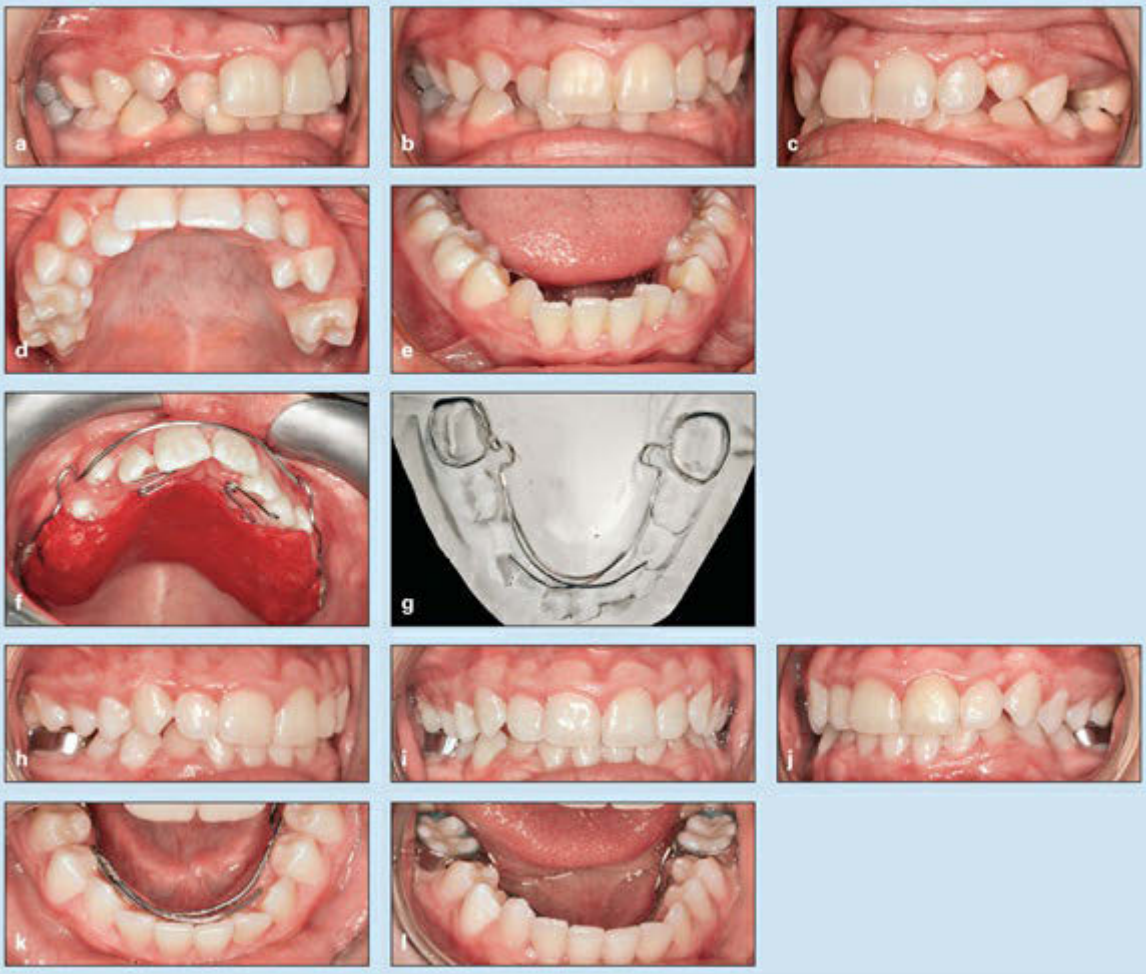


Fig 13-20 Management of right lateral incisor crossbite, mandibular incisor crowding, and lingual displacement of the mandibular permanent right canine in a 10-year, 6-month-old girl. The problems are caused by early loss of the primary canines. *(a to e)* Pretreatment occlusion. *(f)* Modified Hawley appliance with occlusal coverage and a spring to correct the lateral incisor crossbite. *(g)* Lower holding arch with a spring to unravel the crowded mandibular incisors. *(h to j)* Final phase of active treatment. *(k)* Lower holding arch used in active treatment. *(l)* Lower holding arch used as a retainer.

Case 13-13

A 12-year-old boy in the early permanent dentition presented with a Class II division 1 malocclusion, a horizontal growth pattern, short lower facial height, and deep bite. He had a two-step occlusion; that is, the mandibular anterior teeth from canine to canine were at a higher occlusal level than the posterior segments (Figs 13-21a to 13-21d).

Treatment:

Because of the patient's growth potential, the treatment strategy involved interceptive Class II treatment with the headgear, lip bumper, and modified Hawley appliance (HLH) technique (see chapter 11). The maxillary Hawley appliance was modified with an inclined surface and a lip bumper.

Treatment was continued with mandibular complete bonding to correct the two-step occlusion by intrusion of the anterior segment and eruption of the posterior segment, first with a mandibular double show loop and then with a step-down anterior arch. A maxillary bite plate was used to disocclude the posterior segments.

The next step was maxillary complete bonding and anterior retraction and continuation of the cervical headgear. Figures 13-21e to 13-21g show posttreatment views of the occlusion. Cephalometric evaluations revealed that treatment had the following results (Figs 13-21h and 13-21i): Overbite was reduced from 7.8 to 1.1 mm, overjet was reduced from 11.8 mm to 2.7 mm, ANB was reduced from 4.8 to 2.5 degrees, FMA was increased from 17.6 to 20.6 degrees, upper facial height increased from 56.8 to 58.8 mm, and lower facial height increased from 54.2 to 56.8.

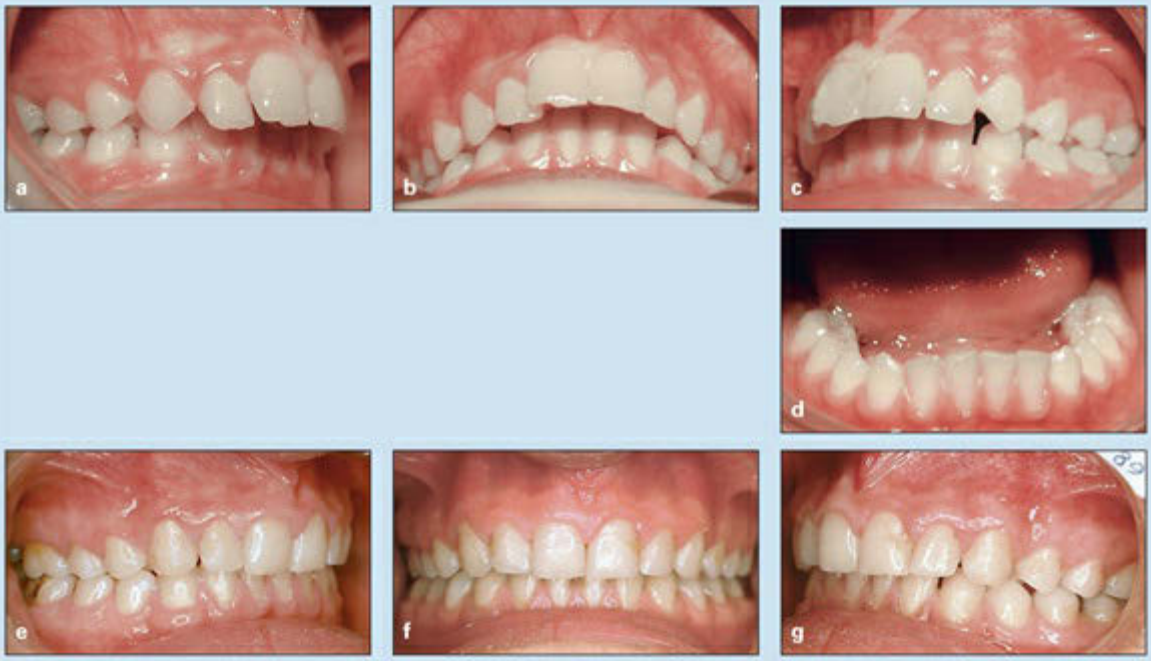
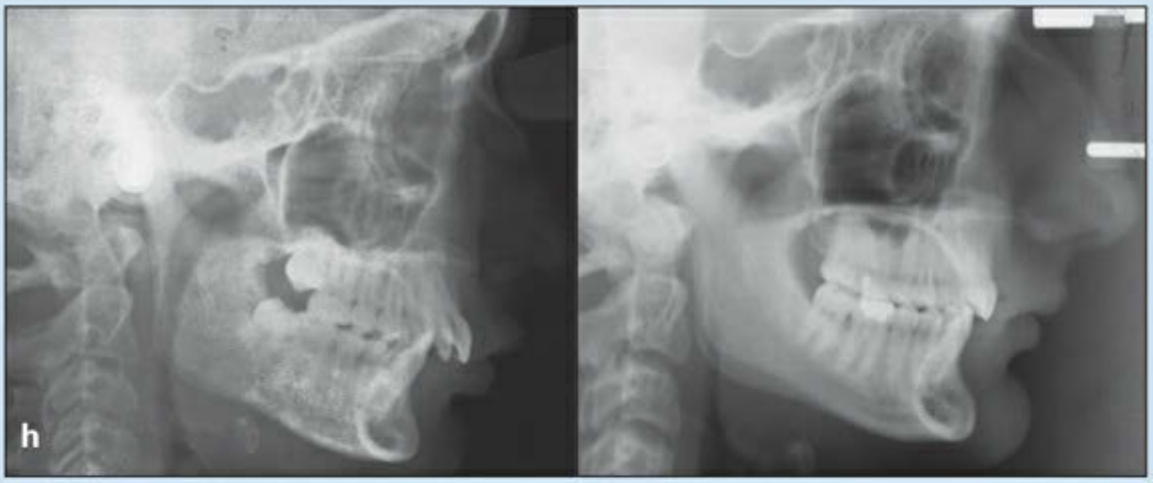


Fig 13-21 Management of deep bite and two-step occlusion in a 12-year-old boy with a Class II division 1 malocclusion. He has a horizontal growth pattern and short lower facial height. *(a to d)* Pretreatment occlusion. *(e to g)* Posttreatment occlusion.



i

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	86.8	85.6
Maxillary depth (FH-NA) (°)	90.0	87.2	88.7
ANB (°)	2.3	4.8	2.5
FMA (MP-FH) (°)	25.9	17.6	20.6
Y-axis (SGn-SN) (°)	59.4	62.9	65.3
Interlabial gap (mm)	0.0	1.4	1.4
Upper face height (N-AGS) (mm)	50.0	56.8	58.8
Lower face height (ANS-Gn) (mm)	60.0	54.5	57.8
Mx base–occ plane (PP-OP) (°)	10.0	7.5	8.7
Interincisal angle (U1-L1) (°)	135.0	125.8	140.7
IMPA (L1-MP) (°)	95.0	101.1	98.1
Overbite (mm)	2.5	7.8	1.1
Overjet (mm)	2.5	11.8	2.7

Fig 13-21 *(cont) (h)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(i)* Changes in cephalometric measurements.

Case 13-14

An 11-year-old girl in the late mixed dentition presented with a Class I malocclusion, deep bite, and severe maxillary and mandibular space deficiency (Figs 13-22a to 13-22f).

Treatment:

Treatment was planned as a serial extraction approach, despite the increased overbite, which was controlled mechanically. Extraction treatment was chosen for several reasons: *(1)* the severe space deficiency in both jaws (space needed: 10 mm in the maxillary arch, 12 mm in the mandibular arch), *(2)* panoramic radiographic evaluation and clues that confirmed that this was a hereditary type of crowding (palisading maxillary molars, mandibular molar crowding, and the possibility of second molar impaction), and *(3)* clinical signs of mandibular incisor compression and the effect of that finding on bone support.

Treatment procedures included the following steps:

1. Placement of a maxillary Hawley bite plate as anchorage and to control deep bite
2. Insertion of a lower holding arch as anchorage
3. Extraction of all remaining primary molars and maxillary first premolars
4. Extraction of the mandibular first premolars
5. Removal of the Hawley appliance and complete bonding to finalize treatment

Figures 13-22g to 13-22j show the results of treatment.

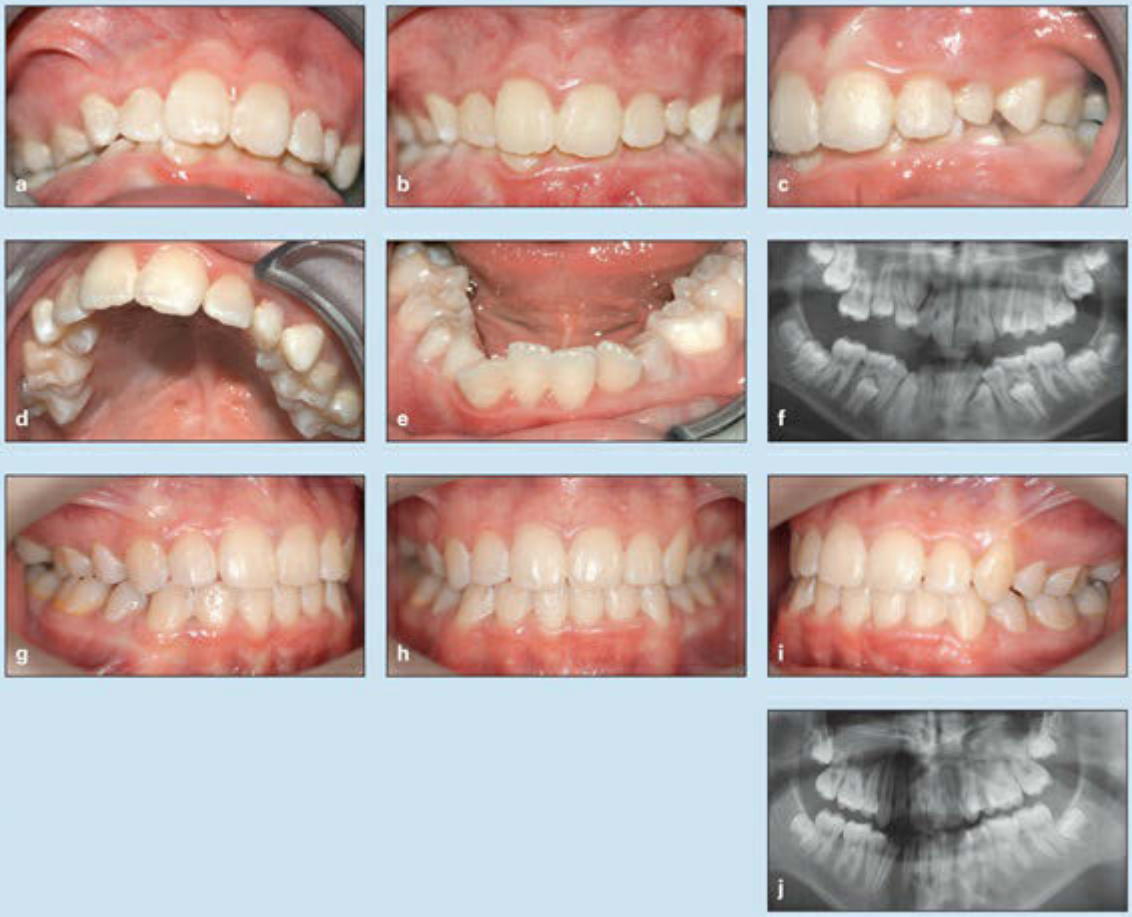


Fig 13-22 Management of deep bite and severe maxillary and mandibular space deficiency in an 11-year-old girl in the late mixed dentition. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g to i)* Posttreatment occlusion. *(j)* Posttreatment panoramic radiograph.

Case 13-15

A 10-year, 8-month-old girl in the late mixed dentition presented with a Class II division 2 malocclusion and deep overbite. She exhibited maxillary moderate space deficiency and slight retroclination of the mandibular incisors (Figs 13-23a to 13-23g).

Treatment:

Because of the patient's skeletal age and growth potential, the treatment plan was designed as interceptive treatment of the Class II malocclusion, with intraoral and extraoral appliances to encourage growth modification and provide space for the dentition. Treatment was started with maxillary cervical headgear and a maxillary Hawley anterior bite plate to reduce overbite, retract the maxillary molars, and modify maxillary growth.

Mandibular treatment was started with 2 × 4 bonding to achieve slight protrusion of the incisors. This was followed by some intrusion with a utility arch and sequential extraction of primary teeth to achieve guidance of eruption and overbite control.

After full Class I molar relationships were established and normal overbite was achieved, the maxillary Hawley appliance was removed. Treatment continued with full bonding for leveling and anterior retraction (Figs 13-23h to 13-23n).

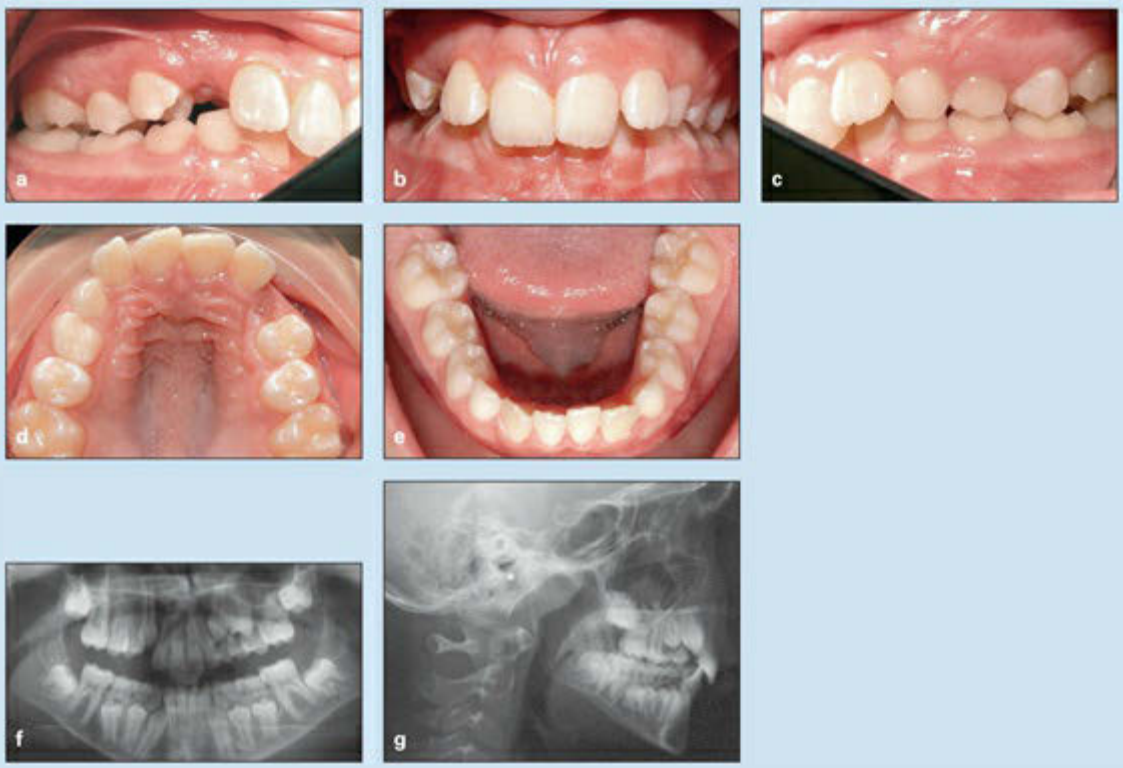


Fig 13-23 Management of a Class II division 2 malocclusion and deep overbite in a 10-year, 8-month-old girl. She exhibits moderate maxillary space deficiency and slight retroclination of the mandibular incisors. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph. *(g)* Pretreatment cephalometric radiograph.

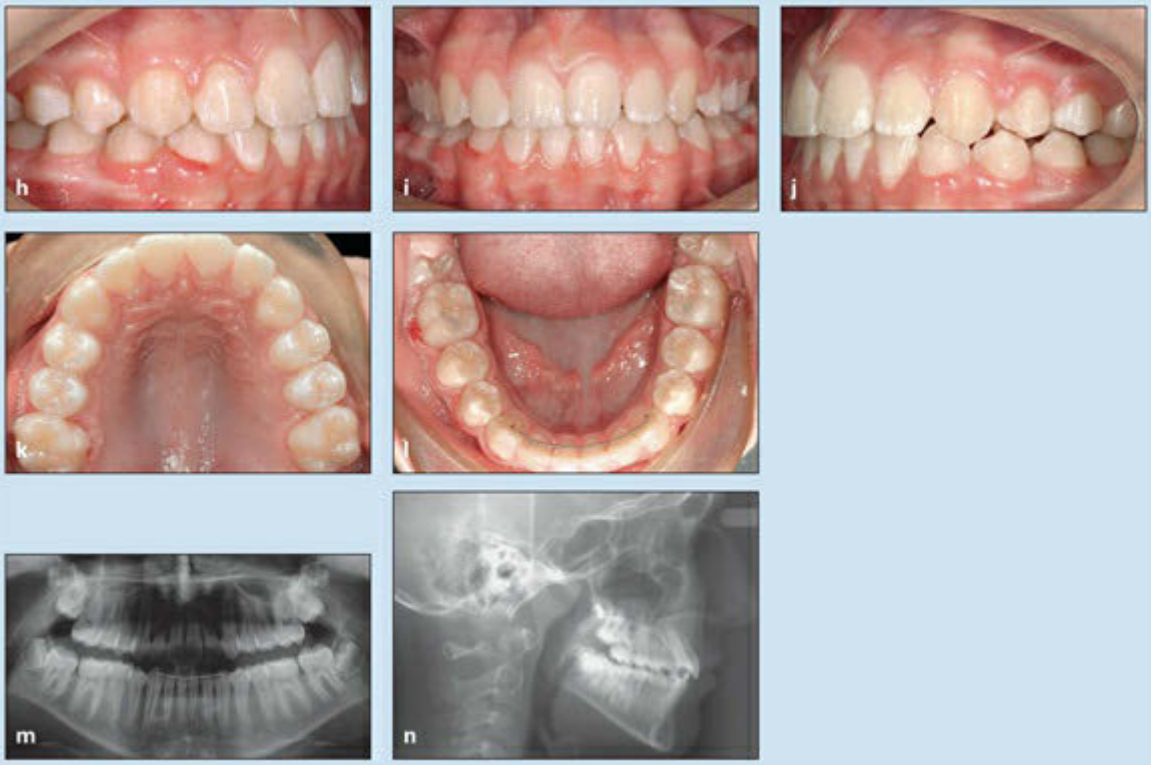


Fig 13-23 *(cont) (h to l)* Posttreatment occlusion, achieved without extractions. *(m)* Posttreatment panoramic radiograph. *(n)* Posttreatment cephalometric radiograph.

Case 13-16

A 14-year-old boy in the late mixed dentition presented with a Class I malocclusion, upright maxillary and mandibular incisors, and severe deep bite (110%) that had caused mandibular incisor collapse. The collapse contributed to severe mandibular crowding and ectopic eruption of the mandibular right lateral incisor (Figs 13-24a to 13-24f).

Treatment:

Because of the patient's severe deep bite and retrusion of the maxillary and mandibular incisors, the treatment strategy was to correct incisor retroclination and open the bite to resolve the mandibular incisor collapse and regain some space for the mandibular incisors.

First, the maxillary molars were banded, and a removable bite plate with no labial bow was inserted to disocclude the posterior segments and thereby stimulate mandibular molar eruption. This was followed by maxillary 2 × 4 bonding; nickel-titanium archwire was used for leveling, and then stainless steel round wire with an open loop was used for incisor proclination. A maxillary utility arch was used for maxillary incisor intrusion and molar uprighting. After some bite opening was achieved, three mandibular incisors and the primary first molars were bonded to achieve mandibular incisor alignment and protrusion and to regain space for the ectopic lateral incisors. Concomitant with eruption of the permanent teeth, complete bonding and the final stage of treatment was continued. Figures 13-24g to 13-24i show some of the stages of active treatment.

Significant changes were achieved in the dentoskeletal structures (Figs 13-24j to 13-24q). Changes included deep bite correction and improvement of incisor inclination and therefore elimination of the severe crowding. The vertical growth pattern continued, and facial height increased after treatment.

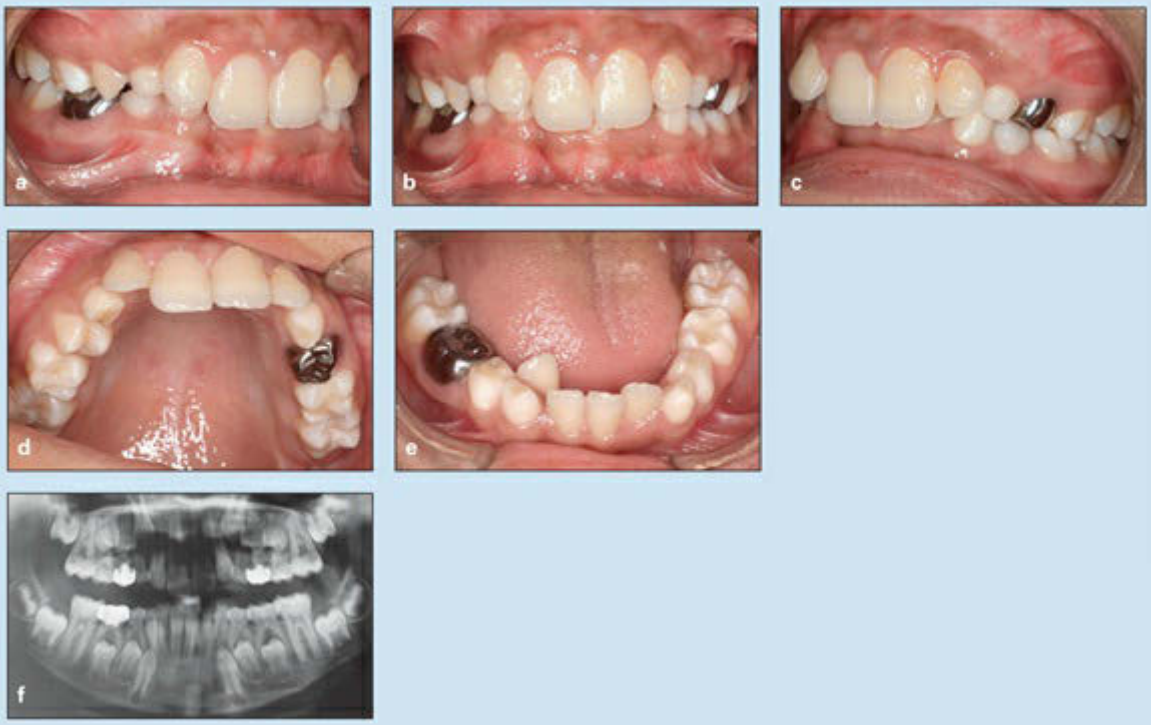
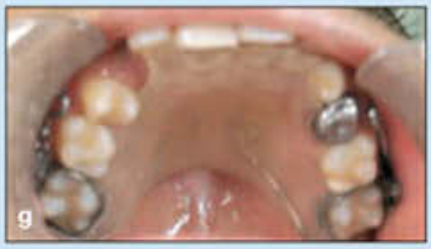
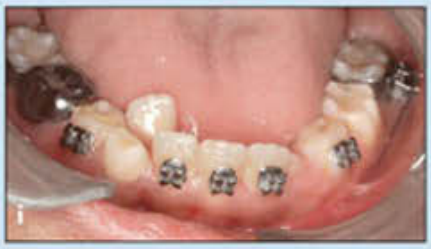
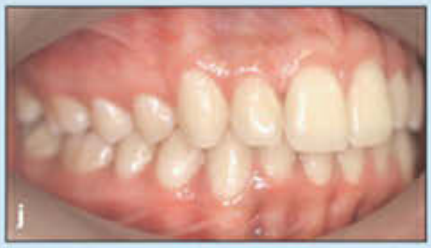
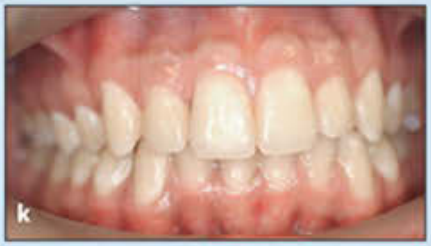


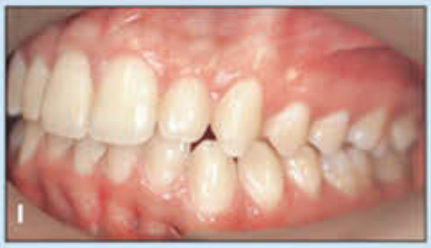
Fig 13-24 Management of severe deep bite (110%) and maxillary and mandibular retrusion that have caused mandibular incisor collapse in a 14-year-old boy in the late mixed dentition. The collapse has contributed to severe mandibular crowding and ectopic eruption of the mandibular right lateral incisor. *(a to e)* Pretreatment occlusion. *(f)* Pretreatment panoramic radiograph.

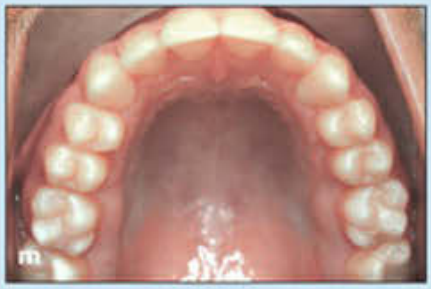


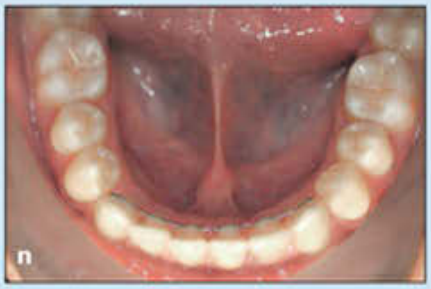


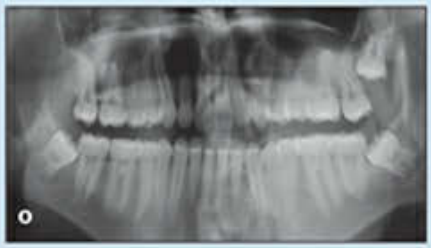


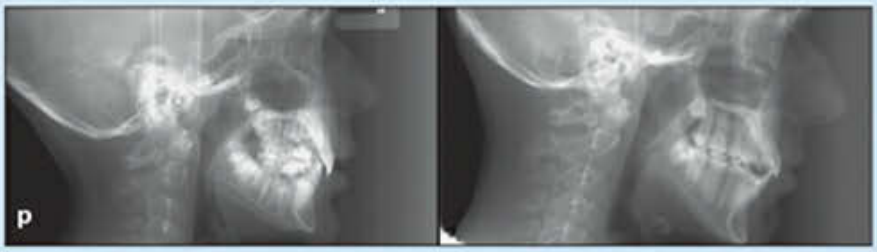












q

Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	81.0	85.6
Maxillary depth (FH-NA) (°)	90.0	85.1	86.0
ANB (°)	2.3	5.6	1.7
FMA (MP-FH) (°)	25.9	33.7	33.9
Y-axis (SGn-SN) (°)	59.4	75.9	76.2
Interlabial gap (mm)	2.0	6.7	2.3
Upper face height (N-AGS) (mm)	50.0	48.9	48.9
Lower face height (ANS-Gn) (mm)	60.0	63.9	73.7
Mx base–occ plane (PP-OP) (°)	10.0	10.2	8.7
Interincisal angle (U1-L1) (°)	135.0	154.6	126.8
IMPA (L1-MP) (°)	95.0	86.1	89.7
Overbite (mm)	2.5	6.9	1.3
Overjet (mm)	2.5	2.9	2.6

Fig 13-24 *(cont) (g to i)* Phases of active treatment. *(j to n)* Posttreatment occlusion. No extractions have been performed. *(o)* Posttreatment panoramic radiograph. *(p)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs. *(q)* Changes in cephalometric measurements.

Summary

- Open bite is a multifactorial phenomenon, initiating and developing during the primary and mixed dentitions. Recognition of the etiology plays an important role in the diagnosis and treatment of this anomaly. The problem can be diagnosed clinically and cephalometrically; however, diagnosis should be viewed in the context of both skeletal and dental structures.
- Hereditary factors influence the direction of skeletal growth by changing form and size, causing abnormal growth pattern and incorrect jaw posture, and initiating skeletal open bite.
- Environmental factors during the primary and mixed dentitions can also play an important role in the development of open bite. Therefore, different kinds of open bite with different morphologic and etiologic characteristics must be accurately classified for precise treatment planning.
- The major common local or environmental factors that play a role in developing open bite at an early age are digit sucking; abnormal tongue size, posture, and function; abnormal neuromuscular function; and enlarged lymphatic tissues that force mouth breathing and postural alterations that induce dental and skeletal modifications.
- Pediatric dentists and general practitioners who see children at an early age have great responsibility in the detection of and intervention in these developing anomalies.
- Tongue thrust is an abnormal tongue function that usually develops secondary to thumb sucking and then maintains or increases the open bite that was produced by the finger, even after the finger habit has stopped. During correction of open bite, abnormal tongue function must also be controlled.
- Simple dental open bite is considered an open bite without skeletal abnormalities. Patients with dental open bite have the following characteristics: normal craniofacial pattern, normal skeletal jaw relationships, previous history of finger sucking, proclined incisors, anterior tongue thrust, undererupted incisors, normal or slightly excessive molar

height, normal inclination of the posterior dentition, divergent occlusal plane, lack of a gummy smile, and no vertical excess or posterior tipping of the maxilla.

- Skeletal open bites are usually caused by genetics, prolonged environmental factors, or a combination of both. Patients with skeletal open bite exhibit unfavorable growth patterns and skeletal disharmonies: steep mandibular plane, retrognathic mandible, obtuse gonial angle, short ramal height, short anterior cranial base, short posterior cranial base, obtuse y-axis angle, obtuse saddle angle, obtuse articular angle, obtuse occlusal plane–mandibular plane angle, obtuse mandibular plane–palatal plane angle, counterclockwise rotation of the palate, increased lower facial height, decreased upper facial height, decreased posterior facial height, and steep anterior cranial base.
- Open bite management during the primary dentition is usually limited to observation or control of habits. In patients with a persistent habit, in addition to habit control, control of the vertical dimension is recommended. This can be accomplished, for example, by application of a lower holding arch and transpalatal arch.
- Open bite management during the mixed dentition can be designed to include one or two phases.
- The best timing for early intervention is during the early mixed dentition. Depending on the etiologic factors and morphology of the anomaly, the following tactics can be applied: habit control, lip seal and swallowing exercises, growth modification, closing the drawbridge, and increasing the posterior facial height–anterior facial height ratio.
- Excessive anterior overlap of incisors, or *deep bite*, is a common malocclusion that presents significant problems for orthodontists if treatment is delayed until the patient is an adolescent or adult. This is due to difficulty in treatment procedures, lack of long-term stability, and strong relapse tendency.
- Not all deep bites are alike; deep bite can be associated with a Class I, Class II division 2, or Class II division 1 malocclusion, and each type has different skeletal morphology, different etiology, and different treatment strategies. Deep bite can manifest as a simple incisor overlap, a dentoalveolar overlap, skeletal overclosure, or a combination of both.
- When a patient with excessive overbite is examined, it is important to remember that excessive overlapping of incisors is not the whole problem; rather it is a part of the total malocclusion.

- Dental deep bites can develop because of supraocclusion (overeruption) of incisors, infraocclusion (undereruption) of the posterior teeth, or a combination of both. Dental deep bites are confined to the teeth and alveolar processes. Therefore, the problem lies mainly within the dentition and is caused by the environment.
- Patients with dental deep bite usually have a normal vertical skeletal relationship of the basal bones, normal lower facial height, normal profile, and normal relationships of the lips to each other and to the chin and nose.
- Skeletal deep bite (closed bite), or *complex deep bite*, is a dentoskeletal malrelationship of the dentoskeletal structures in the vertical dimension. In other words, it is a combination of an overlapping of incisors and a vertical basal bone malrelationship, a malrelationship of the alveolar bones, and/or malrelationship of the underlying mandibular or maxillary bones. The skeletal malrelationship of the basal bones may be partially the result of counterclockwise rotation of the mandible or clockwise rotation of the maxilla.
- The major etiologic factors of skeletal deep bite are usually hereditary influences on the dental, skeletal, and soft tissue profiles; therefore, this type of anomaly has more complicated and difficult management and has a high tendency for relapse if treated late.
- Dentoskeletal and soft tissue characteristics of skeletal deep bite include reduced lower facial height, excessive freeway space, increased ramus height and width, two-step occlusion (incisors and canines at higher occlusal levels relative to the premolar and molars), strong temporalis and masseter muscles, a small gonial angle, increased ramus height and posterior cranial base causing counterclockwise rotation of the mandible, a small cranial base angle, a convergent face (four facial planes of the face are horizontal), and a straight or concave profile.
- Deep bite problems can be managed at early ages as a preventive or interceptive procedure or late during the permanent dentition as a corrective procedure. Early treatment is easier to achieve and produces more stable results.
- Before treatment planning, the following points must be considered: the patient's age, especially skeletal maturation; the influence of genetics and/or environment; the structures involved (dental, dentoalveolar, skeletal, or combination); the available freeway space (normal, excessive, or short); the patient's profile; the patient's facial vertical proportions; the incisor

inclination and their relation to the lips and profile; and the amount of incisor exposure during a smile.

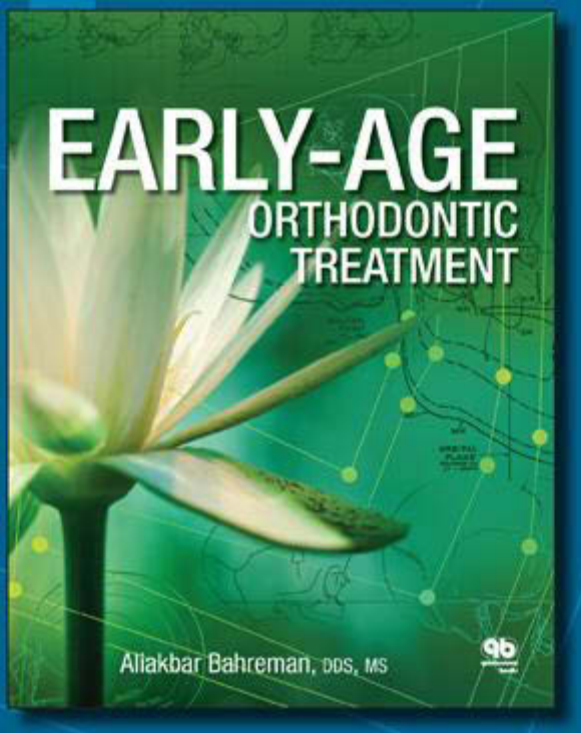
- The general treatment strategy in adults with deep bites includes intrusion of the maxillary or mandibular incisors or both, extrusion of the maxillary or mandibular posterior teeth or both, a combination of intrusion and extrusion of the anterior and posterior segments, or combined surgical and orthodontic intervention in patients with severe skeletal overclosure.
- Deep bite malocclusion occurs in the primary dentition but is rarely treated at this age. Children with horizontal growth patterns and deep bite malocclusions have specific facial and dental characteristics that can be recognized in their primary stage of development.
- Environmental causes that affect occlusion interact mostly during the primary and mixed dentitions. These are mostly detectable, and in many situations the anomalies that are developing can be controlled. Common environmental causes that can aggravate the problem are mesial migration of the posterior teeth, the presence of abnormal habits, lower lip dysfunction, early loss of the primary canine, and a horizontal growth pattern.
- There are several general strategies for early deep bite treatment during the mixed dentition and in growing children: intrusion of the maxillary or mandibular incisors or both, extrusion of the maxillary or mandibular posterior segment or both, growth modification, or any combination of these strategies.

References

1. Beane RA. Non surgical management of the anterior open bite: A review of the options. Semin Orthod 1999;5:275– 283.
2. Kim YH. Anterior openbite and its treatment with multiloop edgewise archwire. Angle Orthod 1987;57:290–321.
3. Rakosi T. The open bite malocclusion. In: Graber TM, Ra-kosi T, Petrovic AG (eds). Dentofacial Orthopedics with Functional Appliances, ed 2. St Louis: Mosby Year Book, 1997:481–508.
4. Kuroda S, Katayama A, Takano-Yamamoto T. Severe anterior open-bite case treated using titanium screw anchorage. Angle Orthod 2004;74:558–567.
5. Seres L, Kocsis A. Closure of severe skeletal anterior open bite with zygomatic anchorage. J Craniofac Surg 2009;20:478–482.
6. Barbre RE, Sinclair PM. A cephalometric evaluation of anterior openbite correction with the magnetic active vertical corrector. Angle Orthod 1991;61:93–102.
7. Enlow DH. Handbook of Facial Growth, ed 2. Philadelphia: Saunders, 1982.
8. Carlson DS. Biological rationale for early treatment of dentofacial deformities. Am J Orthod Dentofacial Orthop 2002;121:554–558.

9. Harris EF, Johnson MG. Heritability of craniometric and occlusal variables: A longitudinal sib analysis. Am J Orthod Dentofacial Orthop 1991;99:258–268.
10. Subtelny JD. Examination of current philosophies associated with swallowing behavior. Am J Orthod 1965;51:161– 182.
11. Proffit WR. Lingual pressure patterns in the transition from tongue thrust to adult swallowing. Arch Oral Biol 1972;17:555–563.
12. Huang GJ, Justus R, Kennedy DB, Kokich VG. Stability of anterior openbite treated with crib therapy. Angle Orthod 1990;60:17–24.
13. Van der Linden FPGM. Vertical dimension. In: McNamara JA, Brudon WL (eds). Orthodontics and Dentofacial Orthopedics. Ann Arbor, MI: Needham Press, 2001:111–148.
14. Subtelny JD, Sakuda M. Open-bite: Diagnosis and treatment. Am J Orthod 1964;50:337–358.
15. English JD. Early treatment of skeletal open bite malocclusions. Am J Orthod Dentofacial Orthop 2002;121:563–265.
16. Erverdi N, Keles A, Nanda R. The use of skeletal anchorage in open bite treatment: A cephalometric evaluation. Angle Orthod 2004;74:381–390.
17. Strang RHW. A Textbook of Orthodontia, ed 3. Philadelphia: Lea & Febiger, 1950.
18. Nanda R. The differential diagnosis and treatment of excessive overbite. Dent Clin North Am 1981;25:69–84.
19. Neff C. Tailored occlusion with the anterior coefficient. Am J Orthod 1949;35:309–313.
20. Diamond M. The development of the dental height. Am J Orthod 1944;30:589–605.
21. Wylie WL. The assessment of anteroposterior dysplasia. Angle Orthod 1947;17:97–109.
22. Baume LJ. Physiological tooth migration and its significance for the development of the occlusion. J Dent Res 1950;29:440–447.
23. Flemming HB. An investigation of the vertical overbite during the eruption of the permanent dentition. Angle Orthod 1961;31:53–62.
24. Canut JA, Arias S. A long-term evaluation of treated Class II division 2 malocclusions: A retrospective study model analysis. Eur J Orthod 1999;21:377–386.
25. Wasilewsky H. Three Dimensional Evaluation of Overbite Relapse [thesis]. Rochester, NY: University of Rochester, 1985.
26. Popovich F. Cephalometric evaluation of vertical overbite in young adult. J Can Dent Assoc 1955;21:209–222.
27. Alexander TA, Gibbs CH, Thompson WJ. Investigation of chewing patterns in deep-bite malocclusions before and after orthodontic treatment. Am J Orthod 1984;85:21–27.
28. Wragg PF, Jenkins WM, Watson IB, Stirrups DR. The deep overbite: Prevention of trauma. Br Dent J 1990;168:365– 367.
29. Bollen AM. Effects of malocclusions and orthodontics on periodontal health: Evidence from a systematic review. J Dent Educ 2008;72:912–918.
30. Nasry HA, Barclay SC. Periodontal lesions associated with deep traumatic overbite. Br Dent J 2006;200:557–561.
31. Sonnesen L, Svensson P. Assessment of pain sensitivity in patients with deep bite and sex- and age-matched controls. J Orofac Pain 2011;25:15–24.
32. Bell WH, Jacobs JD, Legan HL. Treatment of Class II deep bite by orthodontic and surgical means. Am J Orthod 1984;85:1–20.

BONUS CASES



Chapter 6

An 8-year-old boy with a long-term, chronic thumb-sucking habit developed a callus on his thumb (Figs 1A to 1D). He exhibited a Class II division 1 malocclusion, an 8- to 10-mm open bite, contact only on the primary second and permanent first molars, and an overjet of about 17 mm.

Treatment:

The patient was a very cooperative and determined boy, and after consultation with his parents, a removable appliance was planned for the first phase of treatment (Fig 1E). The patient's level of compliance was excellent, and the habit was stopped after 2 months. The use of the appliance was continued for 12 months: He wore it 24 hours a day for 6 months and at night only for 6 months.

The habit was completely discontinued, and the open bite and overjet were reduced significantly, but the patient stopped treatment and returned 2 years later. Figures 1F and 1G show the patient's occlusion during the permanent dentition. He had a Class II division 1 occlusion, 9-mm overjet, 2- to 3-mm open bite, acceptable mandibular dentition, and severe maxillary anterior crowding with the canines out of the arch.

In accordance with the parents' wishes and the patient's age, the second phase of treatment was limited to the maxillary arch and extraction of two first premolars. Figures 1H and 1I show the results of phase 2 treatment.

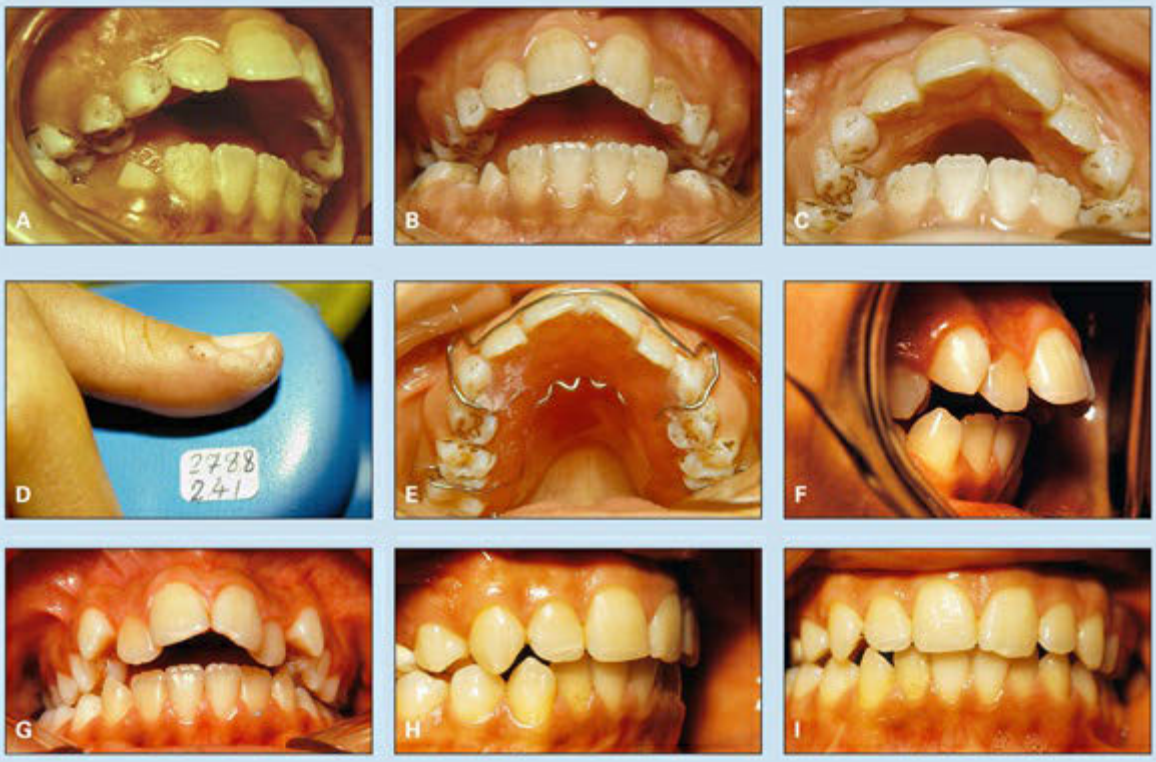


Fig 1 Treatment of an 8-year-old boy with a severe, long-term thumb-sucking habit. He has a Class II division 1 malocclusion, an 8- to 10-mm open bite, and a 17-mm overjet. Only the primary second and permanent first molars make contact. *(A to C)* Pretreatment occlusion. *(D)* Calluses developed as a result of the sucking habit. *(E)* Removable habit breaker. *(F and G)* Occlusion of the permanent dentition 30 months after habit control. *(H and I)* Posttreatment occlusion after phase 2 therapy.

Chapter 7

An 18-year-old man presented with oligodontia. He was missing 10 teeth, including third molars, and five remaining submerged primary molars had arrested the growth of the alveolar process. The extreme spacing and tipping of all dentition, including distal tipping of the anterior teeth, had resulted in a bizarre occlusion (Fig 1A to 1F). Orthodontic problems included an anterior deep bite, severe open bite affecting the buccal segments (no bite), interdental spacing, mesial tipping of all molars, and distal tipping of all remaining permanent teeth. The patient's major complaint was a chewing problem; because of financial problems he did not consider esthetics to be an issue.

This is a clear example of the necessity for general practitioners to understand the importance of early detection of and intervention in ankylosed primary molars as well as hypodontia. Timely extraction of the ankylosed teeth and proper space maintenance, instead of placement of amalgam restorations, would have restored the integrity of the occlusion of this patient earlier and more easily.

Treatment:

After extraction of all primary ankylosed molars, the treatment plan for this young man included orthodontic treatment first and then prosthodontic restoration. The comprehensive orthodontic approach included leveling, correction of rotation, uprighting, space closure, anterior retraction, torque control, and overbite reduction.

The prosthetic approach was limited to fixed partial dentures because implants were not common at that time. Considering the patient's age and skeletal maturation, it was recommended to restore the occlusion toward some bite opening. Figures 1G to 1J show the final occlusion after prosthodontic restoration.

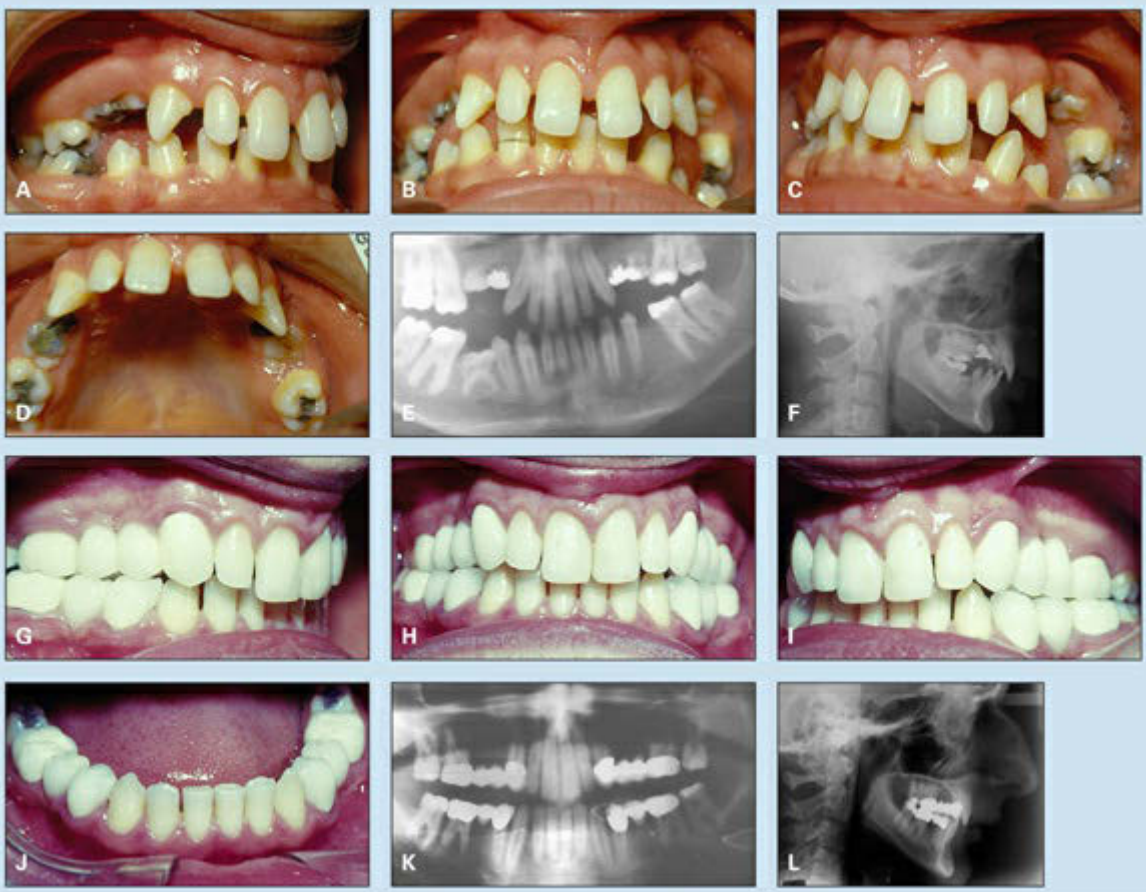


Fig 1 Treatment of an 18-year-old man with oligodontia. He is missing 10 teeth, including third molars. Some submerged primary molars remain. *(A to D)* Pretreatment occlusion. *(E)* Pretreatment panoramic radiograph. *(F)* Pretreatment cephalometric radiograph. *(G to J)* Posttreatment occlusion, after prosthetic restoration. *(K)* Posttreatment panoramic radiograph. *(L)* Posttreatment cephalometric radiograph.

Chapter 8

A 9-year, 7-month-old boy in the early mixed dentition had a Class I malocclusion and two mesiodentes preventing eruption of the maxillary right central and lateral incisors (Fig 1A). Damage to the central incisor crown and space loss had resulted from the neglected supernumeraries.

Figure 1B, taken after removal of the mesiodentes, clearly demonstrates the space closure in the anterior segment that has resulted from the failure to maintain the necessary space for the unerupted central incisor.

Figure 1C was taken during the initial stage of orthodontic treatment for space opening. The lateral incisor had erupted, but the central incisor was still highly positioned. The central incisor was exposed for traction attachment. The crown damage and pulp exposure were restored first with hydroxyapatite (Figs 1D to 1G) and later, after completion of orthodontic movement, with a composite resin restoration (Figs 1H to 1K).

Undiagnosed or neglected mesiodentes that delay eruption of the central incisors can result in the mesial movement of erupted lateral incisors and space loss even before removal of the mesiodentes. Therefore, an important consideration in management of hyperdontia is maintaining the space between erupted teeth after removal of the supernumerary. Space maintenance for unerupted teeth is especially a factor when unerupted teeth are located deeply, and, even after extraction of the supernumerary tooth, their eruption is delayed and slow.

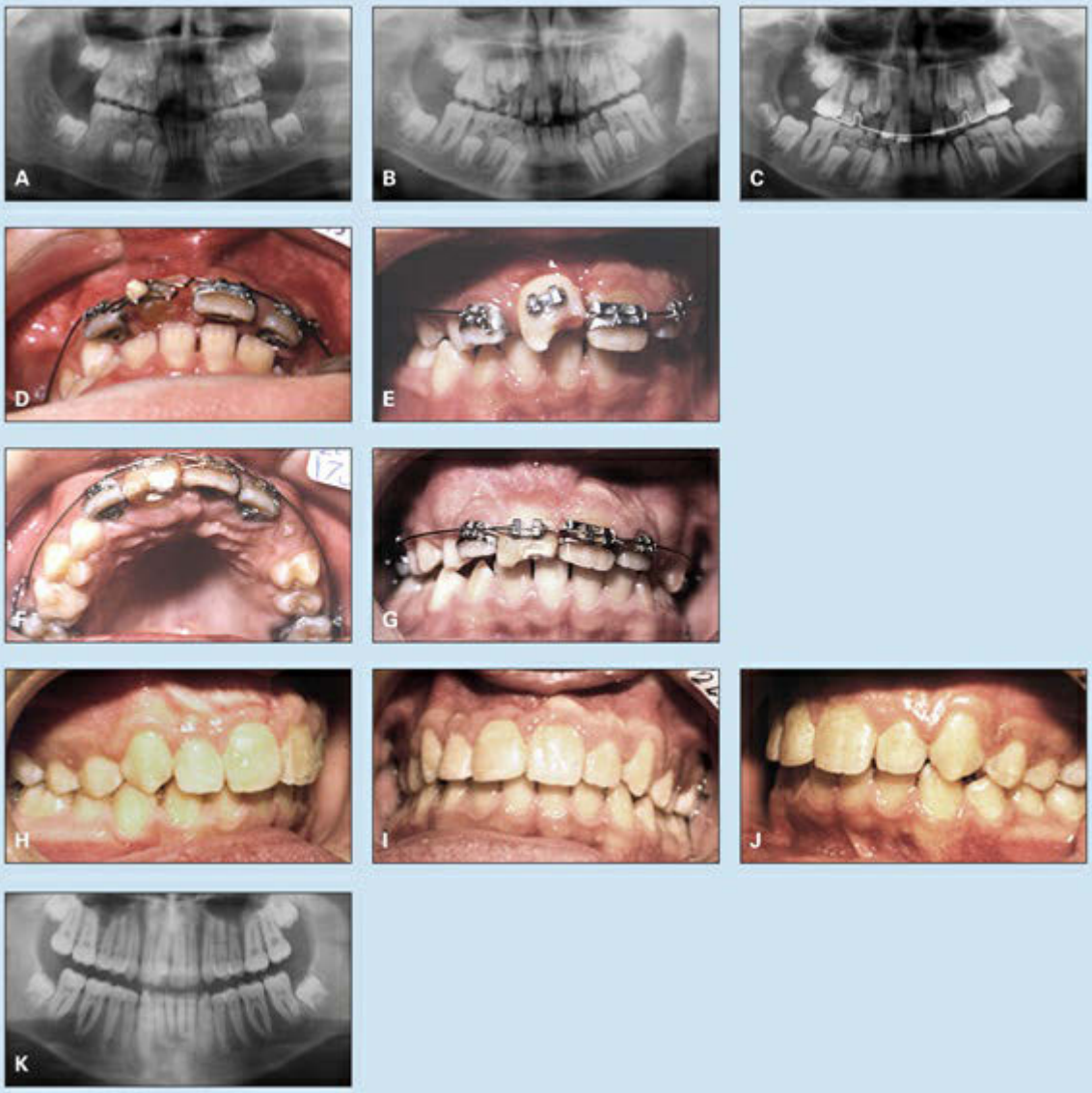


Fig 1 Treatment of a 9-year, 7-month-old boy with mesiodentes preventing eruption and damaging the maxillary right central incisor crown. *(A)* Pretreatment panoramic radiograph. *(B)* Panoramic radiograph after extraction of the supernumerary teeth and lateral incisor eruption. *(C)* Panoramic radiograph taken during the initial stage of orthodontic treatment and space opening. *(D to G)* Traction of the central incisor. Note the damage to the crown and pulp exposure caused by the supernumerary teeth; a hydroxyapatite restoration has been placed. *(H to J)* Posttreatment occlusion, after orthodontic treatment and final composite restoration. *(K)* Post-treatment panoramic radiograph.

Chapter 11: Case 1

A 9-year, 3-month-old boy in the middle mixed dentition had a history of previous thumb sucking. He presented with a severe Class II division 1 malocclusion, an 11.3-mm overjet, and a 2.6-mm open bite. He exhibited severe maxillary and mild mandibular incisor proclination, a convex profile, and a retrognathic chin caused by mandibular clockwise rotation (Figs 1A to 1D).

Treatment:
Because of his vertical growth tendency and aligned mandibular incisors, the only appliance used in the mandibular dentition was a heavy lower holding arch to preserve leeway space and prevent vertical molar eruption, thereby controlling clockwise mandibular rotation. In the maxillary arch, a combined tongue crib–transpalatal arch appliance was inserted first to control anterior tongue movement and achieve some intrusion of the maxillary molars (for more detail on the tongue crib–transpalatal arch appliance, see chapter 6). Then high-pull headgear was applied to correct the Class II molar relationships and intrude the molars in order to achieve more counterclockwise rotation of the mandible. Treatment provided significant results in correcting all dentoskeletal aberrations and improving the profile (Figs 1E to 1J). Treatment resulted in a Class I dentition; a significant change in the A-B discrepancy from 9.1 to 4.3 degrees; correction of the overjet, from 11.3 to 2.1 mm; and a change in incisor overlap from a –2.6 to 1.4 mm. In addition, significant changes in the retruded chin were achieved by mandibular growth and counterclockwise rotation of the mandible; these changes were confirmed by cephalometric improvement in the facial angle, lower facial height, and profile convexity.

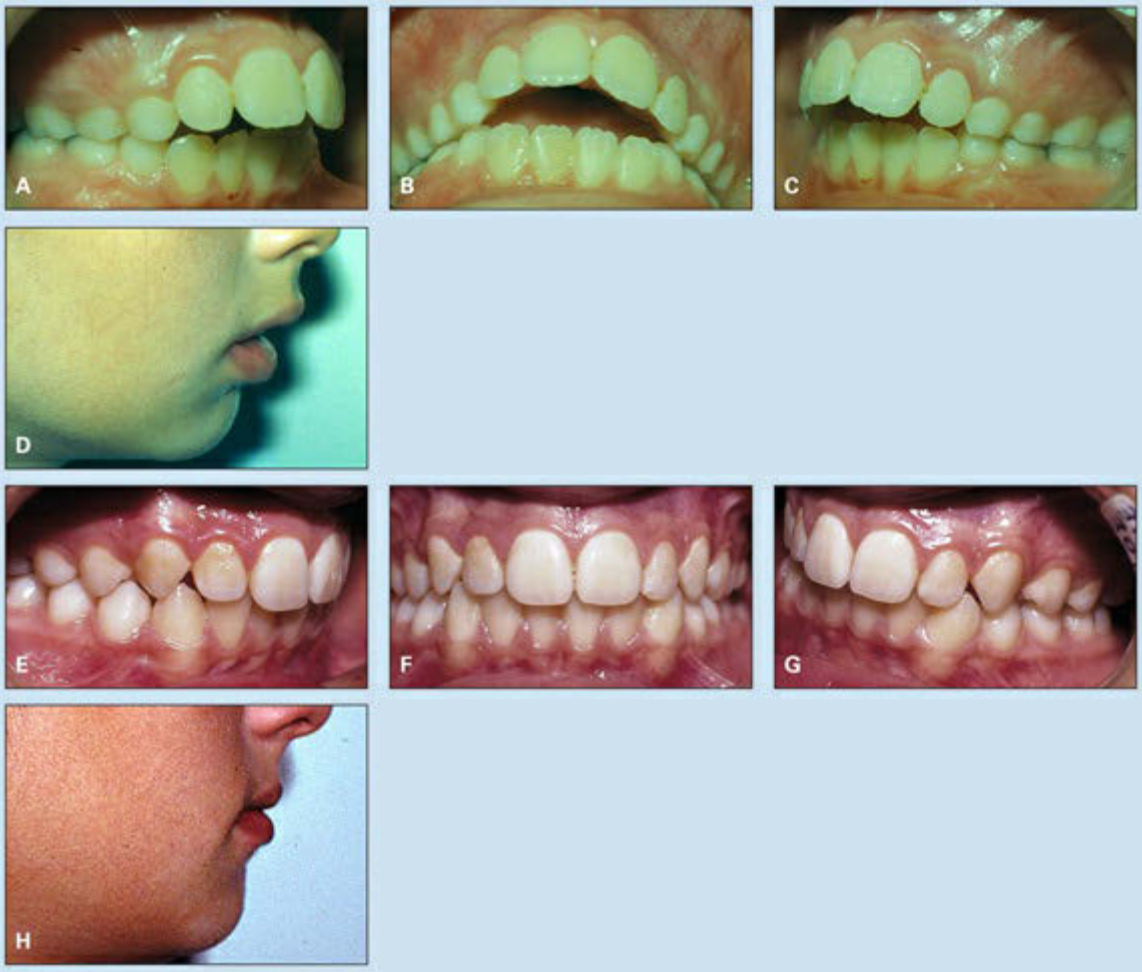
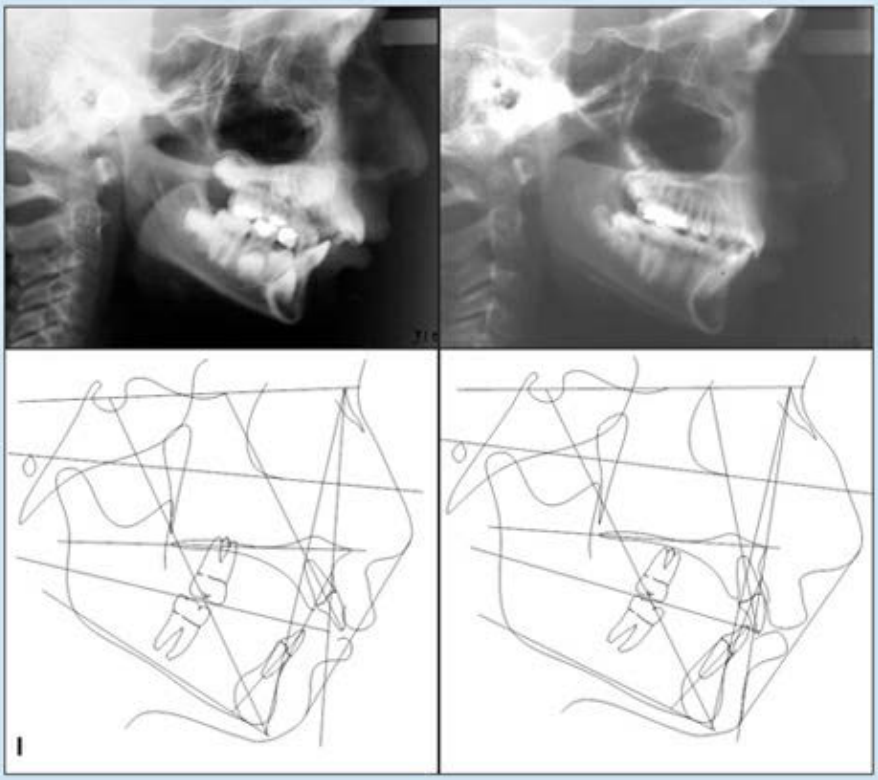


Fig 1 Management of severe overjet and overbite in a 9-year, 3-month-old boy with a history of thumb sucking. He has a Class II division 1 malocclusion, severe maxillary incisor proclination, and a retrognathic chin. *(A to C)* Pretreatment occlusion. *(D)* Pretreatment soft tissue profile. *(E to G)* Posttreatment occlusion. *(H)* Posttreatment soft tissue profile.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.6	85.9
SNB (°)	78.0	74.6	77.2
SNA (°)	82.0	83.7	81.5
Maxillary depth (FH-NA) (°)	90.0	91.3	89.6
ANB (°)	2.3	9.1	4.3
FMA (MP-FH) (°)	25.9	27.2	19.8
Y-axis (SGn-SN) (°)	59.4	66.9	65.4
Interincisal angle (U1-L1) (°)	135.0	112.0	128.0
U1-FH (°)	116.2	121.8	108.0
IMPA (L1-MP) (°)	95.0	99.1	104.2
Overbite (mm)	2.5	–2.6	1.4
Overjet (mm)	2.5	11.3	2.1

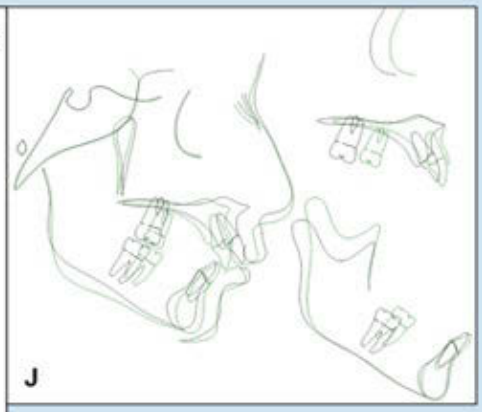


Fig 1 *(cont) (I)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs and tracings. *(J)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Chapter 11: Case 2

An 11-year, 3-month-old girl in the late mixed dentition presented with a Class II division 1 malocclusion, an 11.6-mm overjet, impinging deep bite, and maxillary and mandibular incisor crowding. The maxillary incisors were crowded and proclined, and the mandibular incisors were retroclined and overerupted, creating a two-step occlusion (Figs 2A to 2D).

Treatment:

Because of the patient's severe maxillary incisor crowding, the HLH approach was modified to include an inclined bite plane and, instead of a labial bow, 2 × 4 bonding, plus cervical headgear and a mandibular lip bumper. The first goal of treatment was to align the maxillary incisors by bonding. The second goal was to distalize and extrude the maxillary molars by the use of cervical headgear in conjunction with an anterior inclined bite plane to disocclude the posterior segments to reduce the overbite. Third, the curve of Spee was reduced by use of the lip bumper to upright the proclined mandibular incisors. After some reduction of overbite was achieved by the previous three steps, 2 × 4 bonding of the mandibular incisors was started. Later, a utility arch was placed to intrude the mandibular incisors for complete leveling of the mandibular arch.

The treatment approach was a one-phase treatment that consisted of the aforementioned steps, which were continued until normal interdigitation was achieved and the crowding, overjet, and overbite were corrected. Active treatment was finished with 2 × 6 bonding followed by placement of a maxillary Hawley retainer and mandibular fixed retainer extending from canine to canine. Figures 2E to 2J show the results of treatment.

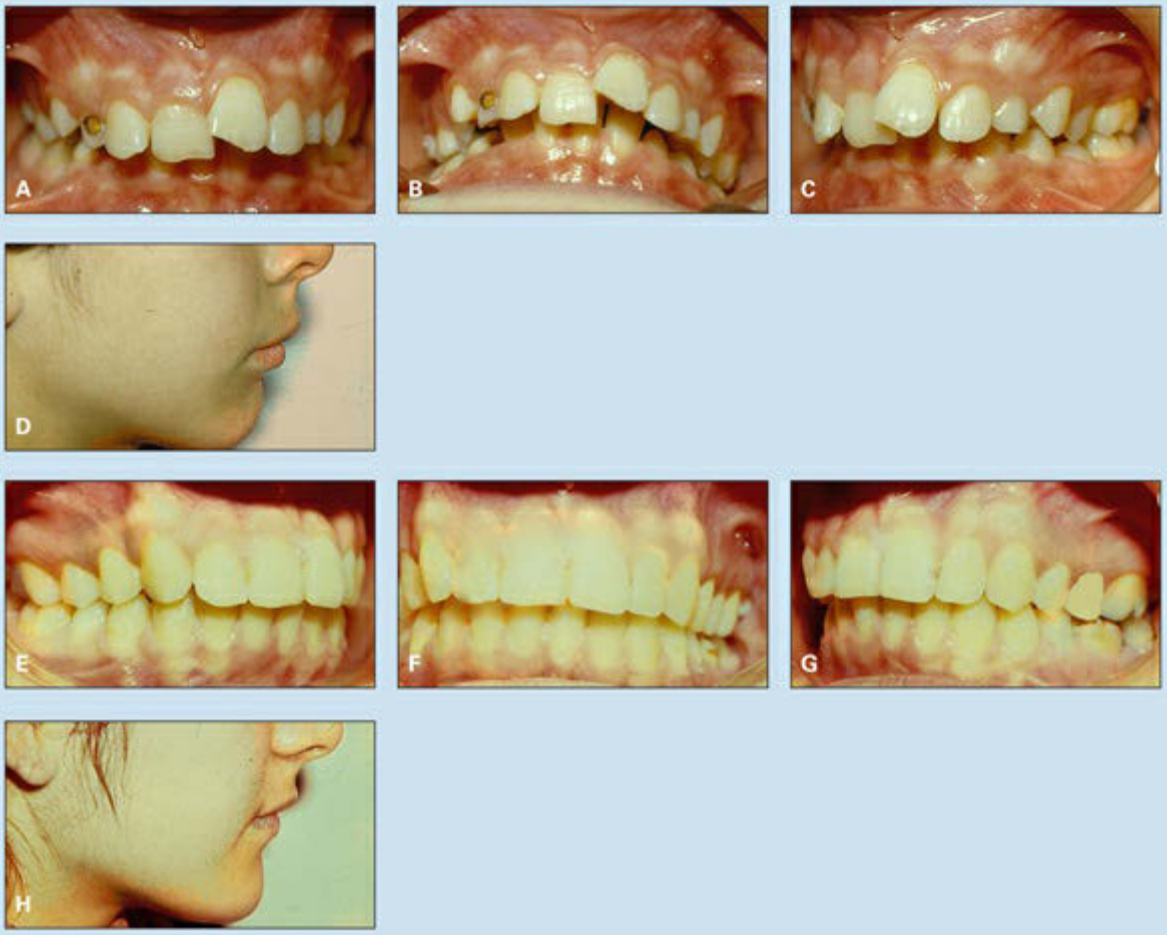
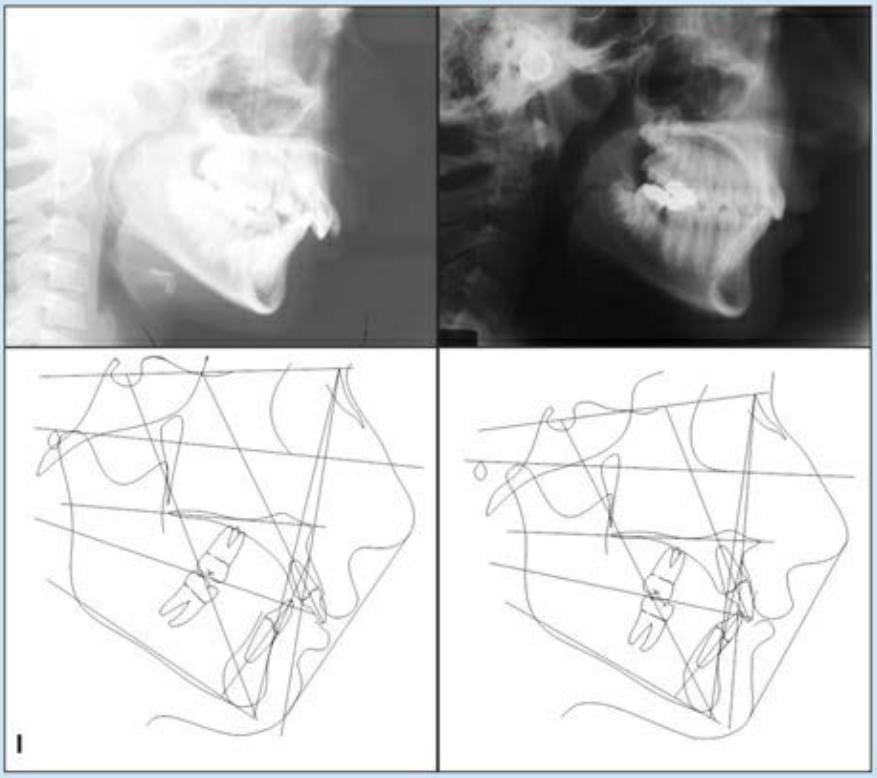


Fig 2 Management of severe overjet, impinging deep bite, and incisor crowding in an 11-year, 3-month-old girl with a Class II division 1 malocclusion. *(A to C)* Pretreatment occlusion. *(D)* Pretreatment soft tissue profile. *(E to G)* Posttreatment occlusion. *(H)* Posttreatment soft tissue

profile.



Landmark	Norm	Pretreatment	Posttreatment
Facial angle (FH-NPo) (°)	87.0	82.7	85.8
SNB (°)	78.0	74.6	74.6
SNA (°)	82.0	81.1	78.1
Maxillary depth (FH-NA) (°)	90.0	88.5	88.2
ANB (°)	2.3	6.4	3.4
FMA (MP-FH) (°)	25.9	28.0	27.8
Y-axis (SGn-SN) (°)	59.4	70.8	71.3
Interincisal angle (U1-L1) (°)	135.0	117.6	121.8
U1-FH (°)	116.2	121.8	113.2
IMPA (L1-MP) (°)	95.0	92.6	97.2
Overbite (mm)	2.5	3.4	3.1
Overjet (mm)	2.5	11.6	3.5

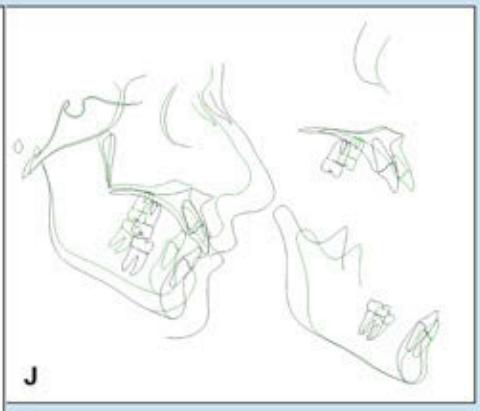


Fig 2 *(cont) (I)* Pretreatment *(left)* and posttreatment *(right)* cephalometric radiographs and tracings. *(J)* Changes in cephalometric measurements and superimposition of pretreatment *(black)* and posttreatment *(green)* tracings.

Chapter 11: Case 3

A 14-year-old girl presented with a neglected pseudo–Class III malocclusion, space deficiency for the maxillary second premolar, and a mandibular shift that was causing temporomandibular dysfunction (Figs 3A to 3D). She exhibited some damage to the incisor structure and minor spacing in the mandibular anterior segment. The patient's chief complaint was temporomandibular joint pain.

Treatment:
The treatment approach involved use of only one removable appliance in the maxilla and no treatment in the mandible. The appliance was a modified Hawley appliance with a special labial bow with two horizontal loops on the buccal side of the canines to provide good retention in the anterior segment without touching incisors; occlusal coverage; two Z-springs for labial movement of the maxillary incisors out of crossbite; and a jackscrew for distalization of the maxillary left molars (Fig 3E). Figures 3F to 3I show the posttreatment occlusion. The crossbite was corrected, the maxillary second premolar was aligned, and minor mandibular incisor spacing was spontaneously closed.

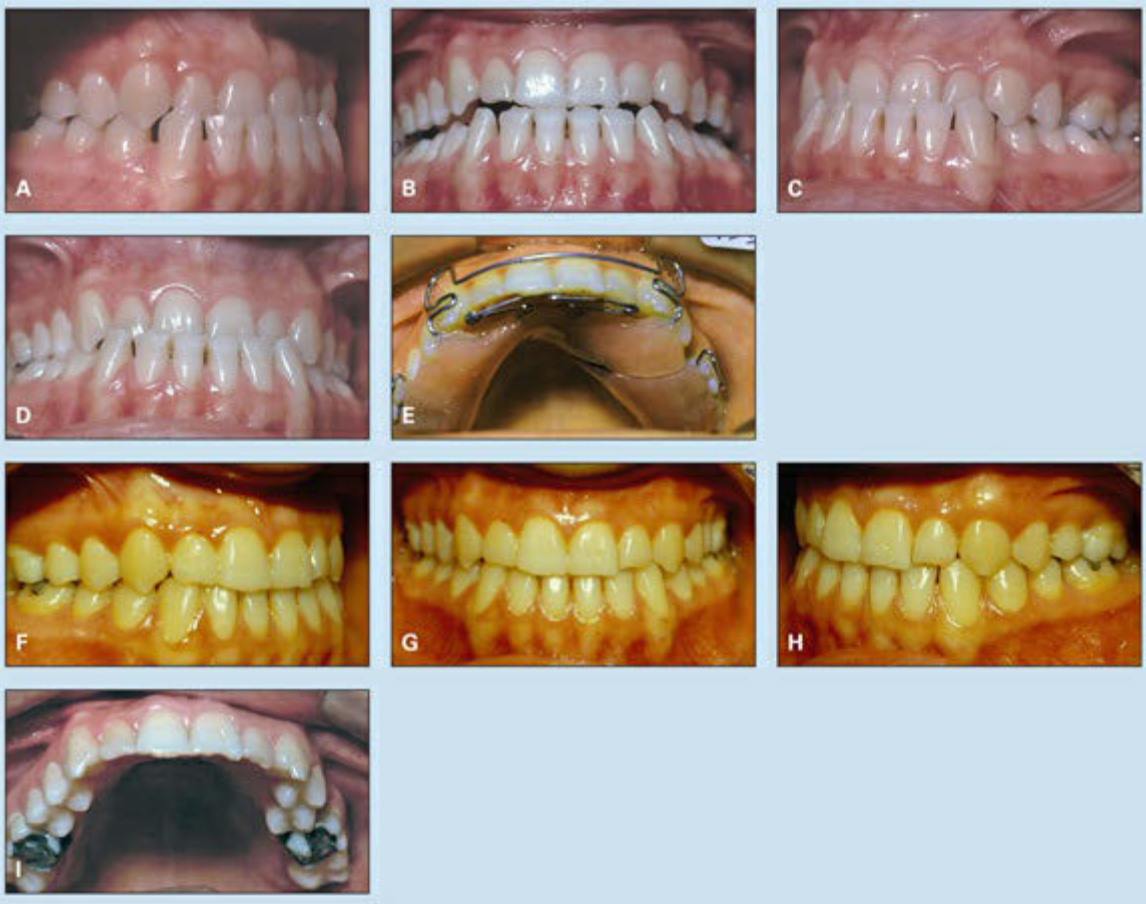


Fig 3 Management of a neglected pseudo–Class III malocclusion with mandibular shift and space deficiency. The neglected problems have caused temporomandibular dysfunction and pain. *(A to C)* Pretreatment occlusion in centric occlusion. *(D)* Pretreatment occlusion in centric relation. *(E)* Modified Hawley appliance with occlusal coverage, modified labial bow, and jackscrew to regain space for the maxillary left second premolar. *(F to I)* Posttreatment occlusion.

Chapter 11: Case 4

A 5-year-old girl in the primary dentition presented with a complete unilateral and anterior crossbite as well as a mandibular functional shift (Figs 4A to 4F).

Treatment:
This patient was treated with a removable Hawley appliance. The maxillary primary second molars were bonded with a buccal tube and C-clasps over the tube for better retention of the appliance. Two Z-springs were constructed to procline the maxillary incisors out of crossbite, while posterior occlusal coverage was used to disocclude the anterior segment.

Figures 4G to 4I show different stages of anterior crossbite correction. The posterior segments exhibited open occlusion before settling. Figure 4J shows the appliance after removal of the occlusal acrylic resin. By 10 months after the end of retention, all mandibular permanent incisors and the maxillary central incisors had erupted (Figs 4K to 4O).

The type of appliance used in this patient must be worn 24 hours a day except during toothbrushing. As mentioned earlier, the first tactic in such cases is to jump the incisors out of crossbite. In some patients, after the maxillary incisors are corrected, open occlusion may result in the posterior segments if the appliance is worn continuously.

In this situation, the occlusal acrylic resin should be left in place and gradually reduced in thickness at each visit. Once the posterior dentition has erupted and settled, the occlusal coverage can be removed or the appliance can be stopped, if required.

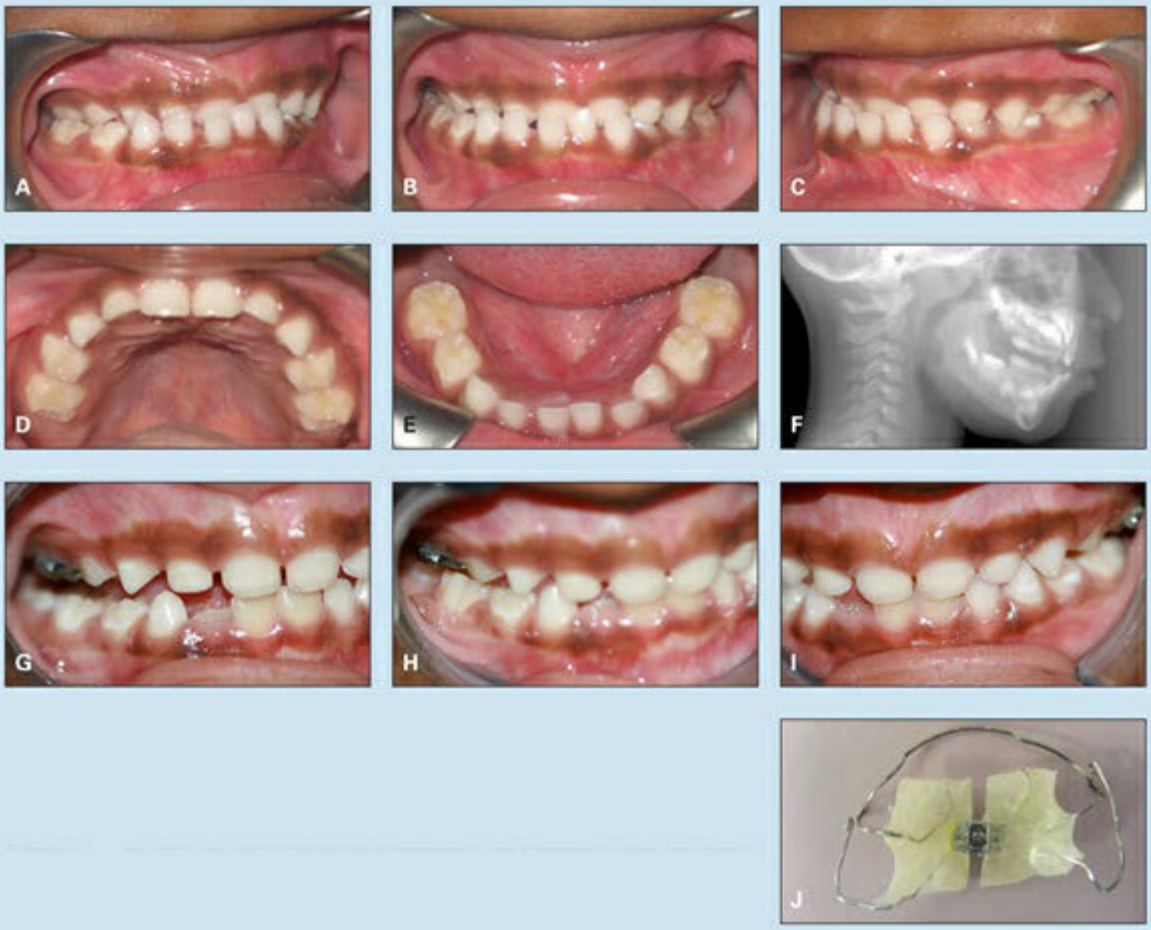


Fig 4 Management of complete unilateral and anterior crossbite and mandibular functional shift in a 5-year-old girl in the primary dentition. *(A to E)* Pretreatment occlusion. *(F)* Pretreatment cephalometric radiograph. *(G)* Occlusion after correction of anterior crossbite but before correction of posterior occlusion. *(H and I)* Occlusion after more occlusal contact has been established in the posterior segments. *(J)* Appliance used in treatment, after removal of occlusal coverage.

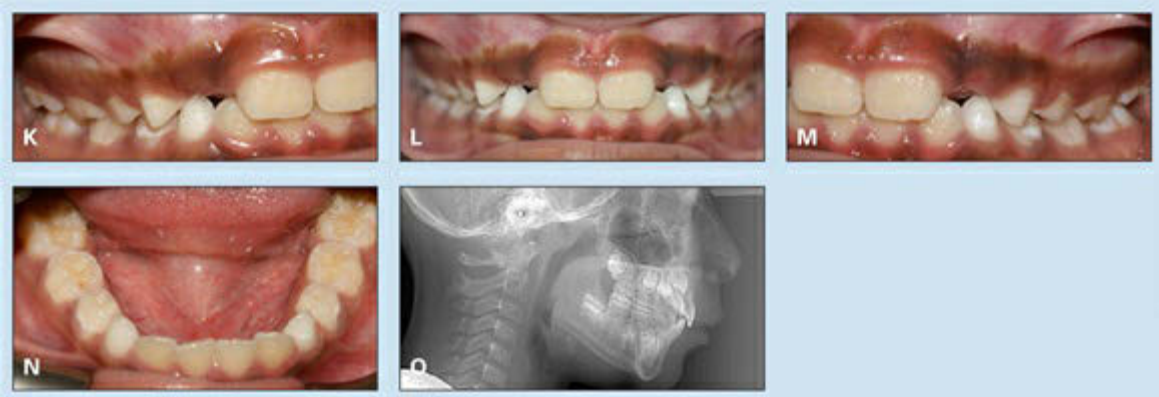


Fig 4 *(cont) (K to N)* Posttreatment occlusion. *(O)* Posttreatment cephalometric radiograph.

About the Author

Aliakbar Bahreman, DDS, MS, currently serves as a clinical professor in the Pediatric and Orthodontic Programs at the Eastman Institute for Oral Health at the University of Rochester in New York. After graduating from dental school at Tehran University of Iran in 1961. he completed a pediatric dentistry fellowship (1964) and a master's degree in orthodontic and dentofacial orthopedics (1967) at the Eastman Institute. He then started his career at the dental school at Shahid Beheshti University (former National University of Iran) as founder and chairman of the Departments of Orthodontics and Pediatric Dentistry. Later, as the dean of the dental school, he presented complete postgraduate curriculum to the Iranian Ministry of Higher Education and, for the first time in Iran, started postgraduate training. He returned to the Eastman Institute for Oral Health as a visiting professor in 1999 and started as a full-time clinical professor and clinical supervisor of the orthodontic, pediatric dentistry, and advanced education of dentistry programs in 2003. Dr Bahreman is the founder of the Iranian Orthodontic Association and was the first president of the Iranian International Orthodontic Meeting, and he is a fellow of the International College of Dentists, the American Association of Orthodontists, and the International Federation of Orthodontics. He received many awards and medals both as a student and as a faculty member in Iran, and in June 2010, he received the Iranpour Award for excellence in clinical education from the University of Rochester.

Learn more about Quintessence Publishing Co., Inc

www.quintpub.com