MOSBY'S

ORTHODONTIC REVIEW

Jeryl D. English
Sercan Akyalcin
Timo Peltomäki
Kate Litschel

SECOND EDITION

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ORTHODONTIC REVIEW

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To my orthodontic family—faculty, colleagues, residents, and alumni—for their assistance and encouragement. To my family and especially to my wife, Kathy, whose love, encouragement, and support have helped make this book a reality.

—Jeryl D. English

To the three most influential women in my life; my mother, my sister, and my better half…

—Sercan Akyalcin

I want to thank my wife, Sari, and my children, Tuomo, Anna, and Saara, for reminding me that there are values more precious than the field of orthodontics.

—Timo Peltomäki

I want to show gratitude to my intelligent friend and Teacher, Reverend Wanarathana Kowlwewe for teaching me the true meaning of good work.

—Kate Litschel

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Preface

Orthodontics is an ever-developing and rapidly growing branch of dentistry. Therefore there is a high need for both the training students and practicing professionals to keep pace with the growth of this relatively young specialty. Moreover, orthodontics is a clinically-driven practice with the mentorship model using case studies being one of the most efficient ways to learn. *Mosby's Orthodontic Review* is designed to not only have answers to questions regarding what professionals need to know about orthodontics but also to provide a comprehensive understanding of clinical knowledge and excellent patient care. It should be the understanding of the reader that there is no specific "recipe" to use in a given case that makes orthodontics formulated. Malocclusions are composed of many aspects in all dimensions of the space, and all underlying tissues contribute to the complexity of the problem. It is the provider's ultimate responsibility to collect necessary information and to properly analyze the findings. This will eventually lead to correct diagnosis, well-established treatment goals, and systemized treatment mechanics. I, on behalf of the co-authors, would like to thank our readers for purchasing this textbook. We believe this new edition will provide an excellent review of orthodontic concepts that will help solidify your knowledge on clinical orthodontics and keep the reader up-to-date with new information and technologies.

Who is the intended audience for this book?

This book is intended for three different segments of the profession: students and orthodontic residents, general dentists, and orthodontists.

Senior dental students that are about to join the dental practice and community will find this textbook very useful as they prepare for the National Board Dental Exam. Orthodontic residents and recent graduates will also benefit from reviewing the text in preparation for the American Board of Orthodontics (ABO) written and clinical examinations. Second, we intend this book to be a good resource for general dentists in their clinical practices and in their discussion of cases with orthodontists. Basic cephalometric radiographs and treatment plans are included so that discussions are easily understood and communicated. Last but not least, experienced orthodontists will be provided updates in clinical issues and technological advancements in our profession.

What is unique about the format of this book?

We have chosen to use a question-and-answer format for each chapter. With this format, the reader can quickly focus on a specific area of interest to answer a question, such as the indication for removal of third molars, interpretation of three-dimensional images, or how long to wear a bonded lingual 3×3 retainer. Each chapter on treatment or treatment planning is subjective; we wanted expert clinicians to share their thoughts and treatment experiences when correcting various malocclusions. Numerous clinical case reports are presented, incorporating learning around real patient scenarios.

How is this book organized?

In organizing this book, we begin with basic foundational information first and then delve into more subjective areas of treatment planning and clinical treatment in the later chapters.

Chapter 1 is a review of craniofacial growth and development with current updates based on clinical research. Chapter 2 is a review of the development of the occlusion with a focus on arch development and eruption sequence. Chapter 3 focuses on the appropriate timing for early orthodontic intervention in specific malocclusions. Chapter 4 addresses orthodontic records and case review. Chapter 5 discusses three-dimensional imaging. Chapter 6 emphasizes the diagnosis of orthodontic problems in three tissues (dental, skeletal, and soft tissue) and in three planes of space (anteroposterior, transverse, and vertical). We have included a 3D-3T diagnostic grid to aid in creating a problem list. Diagnosis is objective, but all problems must be listed to avoid something being overlooked. Misdiagnosis is costly when one overlooks or ignores a patient's problem, such as periodontal disease. We have updated a section on specific objectives of treatment, as well as expanding on superimposition of cephalometric radiographs.

In Chapters 7 and 8, basic concepts in orthodontic appliances and biomechanics are discussed. The remaining 18 chapters focus on specific areas of orthodontic treatment; these areas are subjective and depend on both the training and experience of the clinician. Areas addressed in these chapters include the Invisalign system, minor tooth movement, implants, hygiene, craniofacial deformities, and more.

What is on the accompanying website?

Sample cases can be viewed on the ABO website under the Clinical Examination section by visiting www.americanboardortho.com/professionals/clinicalexam/default.aspx.

These cases represent the latest updates for cases required by the ABO.

Who are the contributors and why were they asked to participate?

Because we are targeting both general dentists and orthodontists for this book, we asked some of the very best clinicians and educators to write chapters. We also included younger faculty members so that their perspectives could be included. These authors understand the needs of prospective students and residents, as well as what information the practicing professional will find useful.

It has been challenging to select the chapter topics and to sequence them in a meaningful manner. Writing a book or a chapter in a book demands a great deal of time from the contributors. We appreciate their hard work, especially when faced with publisher deadlines. We are extremely pleased with the contributions to this book. We expected more than was reasonable and got more than we expected. The efforts of these authors are clear in their dedication to clinical excellence.

Jeryl D. English
Sercan Akyalcin
Timo Peltomäki
Kate Litschel

Note from the Editor

I would be remiss if I did not thank Adriana Cavender and Gloria Bailey for their help in typing and formatting the chapters. I would also like to thank the people at Elsevier, especially Brian S. Loehr and Sarah L. Vora for their advice and professionalism. This book would not have come to fruition without the contributions and support of my co-authors, Dr. Akyalcin and Dr. Peltomäki.

I am dedicated to contributing to the education of dental students, orthodontic residents, general dentists, and orthodontists, and I am confident that this book will serve as an excellent teaching resource on orthodontic diagnosis and treatment.

Jeryl D. English

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Craniofacial Growth and Development

CHAPTER 1

Peter H. Buschang

Clinicians require a basic understanding of growth and development in order to accurately perform diagnoses. According to the World Health Organization, growth and development are among the best measures available of individuals' health and well-being. Knowledgeable clinicians understand that general somatic growth provides important information about their patients' overall size, maturity status, and growth patterns. Because the timing of maturity events, such as the initiation of adolescent or attainment of peak growth velocity, is coordinated throughout the body, information derived from stature or weight—noninvasive and relatively easily obtained measures—can be applied to the craniofacial complex. In other words, the timing of peak height velocity (PHV) can be used to estimate the timing of peak mandibular growth velocity. Knowledge of general somatic growth is also useful when evaluating the size of patients' craniofacial dimensions. An individual's height and weight percentiles provide reliable measures of overall body size, against which craniofacial measures can be compared. For example, excessively small individuals (i.e., below the fifth percentile in body size) might also be expected to exhibit excessively small craniofacial features. Finally, the reference data available for somatic growth and maturation are based on large representative samples, making them more generally applicable and more precise at the extreme percentiles than available craniofacial reference data.

Postnatal craniofacial growth is a complex, but coordinated and ongoing process that clinicians must understand in order to properly plan treatments and evaluate treatment outcomes. The cranial structures are the most mature, followed by the cranial base, maxillary, and mandibular structures, which are the least mature and exhibit the greatest growth potential. Knowledge about a structure's relative growth is important because it serves, along with heritability, as an indicator of its response potential to treatment and other environmental influences. The fact that the mandible is the least mature structure helps to explain why it is the component of the craniofacial complex most often affected in individuals with Class II or Class III skeletal discrepancies. It is essential that clinicians understand that the maxilla and mandible, the two most important skeletal determinants of malocclusion, follow similar growth patterns. Both are displaced anteriorly and, especially, inferiorly; both tend to rotate forward or anteriorly; both rotate transversely; and both respond to displacement and rotation by characteristic patterns of growth and cortical drift. It is also useful to understand that patients should be expected to adapt skeletally to orthodontic, orthopedic, and surgical interventions, and that the adaptations mimic growth patterns exhibited by untreated patients. Perhaps most importantly, clinicians must understand the tremendous therapeutic potential that the eruption and drift of teeth provide. The maxillary molars and incisors, for example, undergo more eruption than inferior displacement of the maxilla, making them ideally suited for controlling vertical and anteroposterior (AP) growth.

Clinicians also often do not appreciate that adults show many of the same growth patterns exhibited by children and adolescents, simply in less exaggerated forms. It has been well established that craniofacial growth continues through the 20s and 30s, and perhaps beyond. Skeletal growth of adults appears to be predominantly vertical in nature, with forward mandible rotation in males and backward rotation in females. The teeth continue to erupt and compensate depending on the individual's growth patterns. Adults also exhibit important soft-tissue changes; the nose grows disproportionately and the lips flatten. Vertical relationships between the incisors and lips should also be expected to change with increasing age.

Finally, malocclusion must be considered as a multifactorial developmental process. Although genes have been linked with the development of Class III and perhaps Class II division 2 malocclusions, the most prevalent forms of malocclusions are largely environmentally determined. Equilibrium theory and the notion of dentoalveolar compensations provide the conceptual basis for understanding how closely linked tooth positions are with the surrounding soft tissues. Such an understanding makes it possible to predict the types of compensations that occur. For example, compensations explain why the development of malocclusion is associated with various habits, assuming the habits are of long enough duration. In fact, anything that alters mandibular posture might be expected to elicit skeletal and dentoalveolar compensations. This explains why individuals with chronic airway obstructions develop skeletal and dental malocclusions that are phenotypically similar to malocclusions associated with weak craniofacial musculature; both populations of patients posture their mandibles similarly and undergo similar dentoalveolar and skeletal compensations. Based on the foregoing, the following questions are intended to provide a basic—although only partial—understanding of growth and development and its application to clinical practice.

1. At what ages do most children enter adolescence, and when do they attain peak height velocity?

The adolescence growth spurt starts when decelerating childhood growth rates change to accelerating rates. During the first part of the growth spurt, statural growth velocities increase steadily until PHV is attained. Longitudinal assessments provide the best indicators of when adolescence is initiated and PHV is attained. Studies of North American and European children[1] show that girls are advanced by approximately 2 years compared with boys in the age of initiation of adolescence and age of PHV. Based on the 26 independent samples of girls and 23 samples of boys, the average ages of PHV are 11.9 and 14.0 years, respectively. Girls and boys initiate adolescence at 9.4 years and 11.2 years, respectively. Maximum adolescent growth velocity in body weight usually occurs 0.3 to 0.5 year after PHV (Fig. 1-1).

2. What is the mid-childhood growth spurt, and how does it apply to craniofacial growth?

The mid-childhood growth spurt refers to the increase in growth velocity that occurs in some, but not all, children several years before adolescence. Mid-childhood growth spurts in stature and weight have been reported to occur between 6.5 and 8.5 years of age; they tend to occur more frequently in boys than girls.[2,3] Based on yearly velocities, mid-childhood growth spurts have been demonstrated for a variety of craniofacial dimensions—also between 6.5 and 8.5 years of age—occurring simultaneously or slightly earlier for girls than boys.[4–7] Applying mathematical models to large longitudinal samples, Buschang and colleagues[8] reported mid-childhood growth spurts in mandibular growth for subjects with Class I and Class II molar relationships at approximately 7.7 years and 8.7 years of age for girls and boys, respectively.

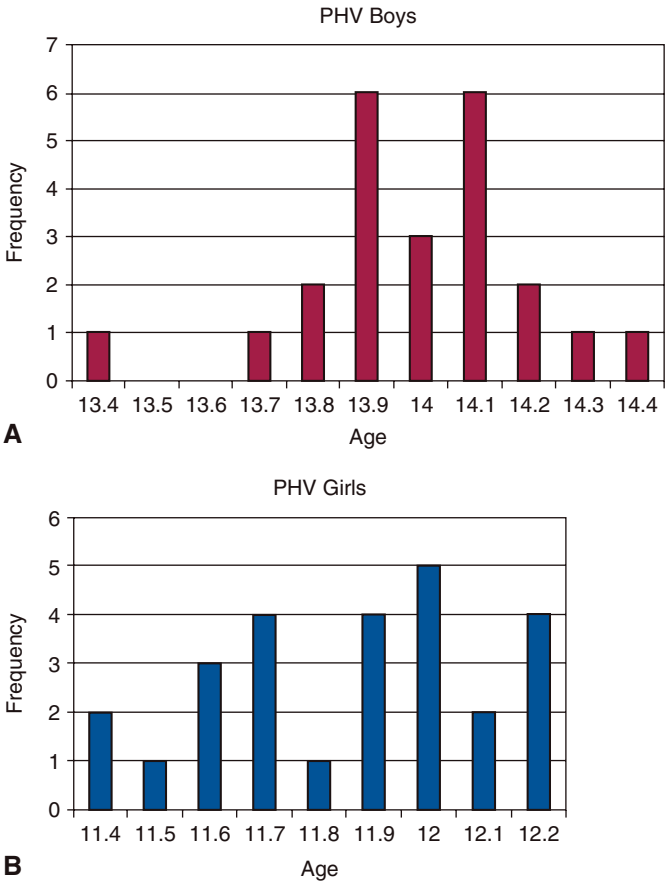

FIG 1-1 Frequency distribution of 26 sample ages of PHV for boys **(A)** and girls **(B).** (From Malina RM, Bouchard C, Beunen G: Human growth: selected aspects of current research on well-nourished children, *Ann Rev Anthropol* 17:187-219, 1988.)

3. Which skeletal indicators are most closely associated with peak height velocity?

According to Grave and Brown,[9] PHV in males and females occurs slightly after the appearance of the ulnar sesamoid and the hooking of the hamate, and slightly before capping of the third middle phalanx, the capping of the first proximal phalanx, and the capping of the radius. According to Fishman's[10] skeletal maturity indicators, capping of the distal phalanx of the third finger occurs less than 1 year before PHV, capping of the middle phalanx of the third finger occurs just after PHV, and capping of the middle phalanx of the fifth finger occurs less than ½ year after PHV. Based on the cervical vertebrae, PHV occurs between cervical vertebral maturation stage CS3 (concavities on the inferior borders of the second and third vertebrae, and both the third and fourth vertebrae are either trapezoid or rectangular horizontal in shape) and CS4 (concavities on the inferior borders of the second, third, and fourth vertebrae, and both the third and fourth vertebrae are rectangular horizontal in shape).[11]

4. What is the equilibrium theory of tooth position?

Although Brodie[12] was among the first to identify the relationship between muscles and tooth position, it was Weinstein and colleagues[13] who experimentally established that the teeth are maintained in a state of equilibrium between the soft-tissue forces. Based on a series of experiments, they concluded that:

1. The forces (produced naturally or by orthodontic appliances) exerted on the crowns of teeth are sufficient to cause tooth movements.
2. Each tooth may have more than one stable state of equilibrium.
3. Even small forces (3 to 7 gm), if applied over a long enough period, can cause tooth movements.

Proffit,[14] who revisited the equilibrium theory 15 years later, noted that the primary factors involved were the resting pressures of the lips, cheeks, and tongue, as well as the eruptive forces produced by metabolic activity within the periodontal membrane. He further noted that extrinsic pressures, such as habits and orthodontic forces, can alter dentoalveolar equilibrium, provided that they are sustained for at least 6 hours each day. Proffit[14] also identified head posture and growth displacements/rotations as secondary factors determining equilibrium. As the mandible rotates, the incisors move and dental equilibrium is reestablished. Björk and Skieller,[15] for example,

have shown an association between changes in lower incisor angulation and true mandibular rotation (e.g., the greater the true forward mandibular rotation, the greater the lower incisor proclination).

5. What is the prevalence of Class II dental malocclusion among adolescents and young adults living in the United States?

The best direct epidemiologic evidence comes from the National Health Survey,[16,17] which evaluated approximately 7400 children between 6 and 11 years of age and over 22,000 youths 12 to 17 years of age. Unilateral and bilateral distoclusion occurred in approximately 16.1% and 22.7% of Caucasian children and 7.6% and 6.0% of African-American children, respectively. Comparable prevalence among Caucasian youths was 17.8% and 15.8%, and 12.0% and 6.0% among African-American youths. Based on overjet measurements provided by the National Health and Nutrition Examination Survey (NHANES) III, Proffit and associates[18] estimated that the prevalence of Class II malocclusion (overjet ≥ 5 mm) decreases from over 15.6%, for youths 12 and 17 years of age, to 13.4% for adults. They also showed that Class II malocclusion is more prevalent among African-Americans (16.5%) than Caucasians (14.2%) and Hispanics (9.1%).

6. What is the prevalence of incisor crowding among individuals living in the United States, and how does it change with age?

According to the initial NHANES III data,[19] incisor irregularities increase from an average of 1.6 mm for children 8 to 11 years, to 2.5 mm for youths 12 to 17 years, to 2.8 mm for adults 18 to 50 years of age. Although incidences are similar at the youngest age, African-American youths and adults show significantly less crowding than Caucasians and Hispanics. Based on the complete NHANES data set, including 9044 individuals between 15 and 50 years of age, approximately 39.5% of US adults have mandibular incisor irregularities ≥ 4 mm and 16.8% have irregularities ≥ 7.[20] Adult males tend to show greater crowding than females; Hispanics show greater crowding than Caucasians, who in turn display greater crowding than African-Americans. Based on the available data for untreated subjects followed longitudinally, rates of crowding increase precipitously between 15 and 50 years of age, especially during the late teens and early 20s (Fig. 1-2).[20]

7. What is the prevalence of Class III dental malocclusion among adolescents and young adults living in the United States?

Worldwide prevalence of Class III malocclusion has been estimated to be 6.8%, with higher prevalence in Southeast Asia (15.8%) and the Middle East (10.2%), than Europe (4.9%) and Africa (4.6%).[21] Based on the National Health Surveys[16,17] conducted on large samples of children and adolescents during the 1970s, which evaluated the subjects' molar relationships, approximately 4.9% of children 6 to 11 years of age and 6% of adolescents 12 to 17 years of age have bilateral Class III malocclusion. Based on overjet measurements provided by the NHANES III, approximately 4.9% of Caucasians, 8.1% of African-Americans, and 8.3% of Mexican-Americans have Class III malocclusion. Importantly, the majority (> 75%) of cases presents with mild (overjet = 0 mm) Class III malocclusions.

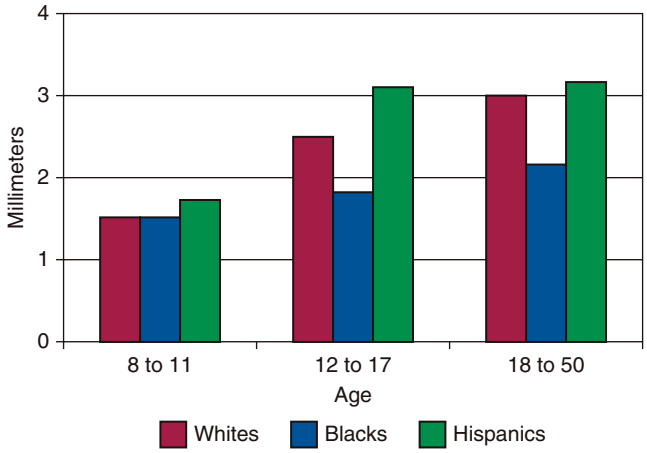

FIG 1-2 Average mandibular alignment scores; US persons, 1988-1991. (Adapted from Brunelle JA, Bhat M, Lipton JA: Prevalence and distribution of selected occlusal characteristics in the US population, 1988-1991, *J Dental Res* 75[special issue]:706-713, 1996.)

8. Skeletally, are Class III dental malocclusions primarily a problem of maxillary or mandibular growth?

Although the maxilla alone and the two jaws combined have both been shown to contribute to Class III skeletal discrepancies, the mandible has most often been cited as the primary determinant.[22–24] In their large cross-sectional study of 848 Class III's from 6 to 16 years of age, Reys and colleagues[25] showed that the sagittal position of the maxilla at all age intervals was normal, whereas the sagittal position of the mandible was abnormal and the mandibular dimensions were larger. Sugawara and Mitani[26] came to similar conclusions. Most recently, Wolfe and colleagues,[27] who followed Class III's longitudinally between 7 and 15 years, verified that the AP position of the maxilla and the changes in AP position over time were the same as in Class I dental malocclusions; the growth differences were in the mandible. Corpus length increased significantly more over time and the mandible became more divergent in Class III dental malocclusions than Class I dental malocclusions.

9. Do the third molars play a role in determining crowding?

Although third molars have been related with crowding,[28–31] most contemporary studies show little or no relationship. In 1979 a National Institutes of Health (NIH) conference came to the consensus that there is little or no justification for extracting third molars solely to minimize present or future crowding of the lower anterior teeth.[32] Ades and co-workers[33] found no differences between subjects whose third molars were impacted, erupted in function, congenitally absent, or

extracted at least 10 years before post-retention records were taken. Sampson and colleagues[34] also showed no differences in crowding between subjects whose third molars have erupted completely or partially, remained impacted, or were missing. A randomized controlled trial that followed 77 patients for 66 months showed a 1.0 mm difference in anterior crowding between patients whose third molars had and had not been removed; the authors concluded that removal of third molars to reduce or prevent late crowding cannot be justified.[35] Based on the NHANES data, individuals who had erupted third molars displayed significantly less crowding than those who did not have erupted third molars.[20]

10. Does horizontal or vertical mandibular growth affect crowding?

Based on the notion that the lower incisors are carried into the lower lip as the mandibe "grows forward," late mandibular growth has been suggested as a major contributor to post-retention crowding.[36] Although incisor compensation to backward mandibular rotation has been demonstrated,[15] crowding as a result of anterior growth displacements remains to be established. However, changes in lower incisor crowding have been shown to be related to vertical growth. Both treated and untreated patients who undergo greater inferior growth displacements of the mandible, and associated greater eruption of the lower incisors, show greater crowding than those who undergo less vertical growth and less eruption.[27,38] Supporting the idea that growth predisposes patients to crowding, Park and coworkers showed that adolescents undergo more post-retention crowding than similarly treated adults.[39] Since vertical mandibular growth continues well beyond the teen years, patients would be well advised to wear their retainers into their early and mid-20s.

11. How much should the maxillary and mandibular incisors and molars be expected to erupt during adolescence?

Based on natural structure superimpositions, the maxillary first molars and central incisors erupt approximately 5 to 6 mm and 4.5 to 5 mm, respectively, between 10 and 15 years of age.[40] In contrast, the mandibular molars and incisors erupt 3 to 5.5 mm and 2.5 to 4.5 mm, respectively. Males showed greater eruption than females for both the maxillary and mandibular teeth. Also using natural structure superimpositions, Watanabe and colleagues[41] demonstrated that the rates of eruption were greater in males than females, attaining peak velocities at approximately 12 and 14 years of age for females and males, respectively.

12. How does untreated arch perimeter change between the late primary dentition and the permanent dentition?

Based on a centenary curve extending between the mesial aspects of the first molars,[42] arch perimeter increases during the early mixed dentition and decreases during and after the transition to the permanent dentition. Maxillary perimeter increases 4 to 5 mm between 6 and 11 years of age and decreases 3 to 4 mm between 11 and 16 years. In contrast, mandibular arch perimeter increases approximately 2 to 3 mm initially and then decreases 4 to 7 mm, with greater decreases in females than males (Fig. 1-3).

13. How do untreated maxillary and mandibular intermolar widths change during childhood and adolescence?

Bishara and colleagues[43] reported that intermolar widths increase 7 to 8 mm between the deciduous dentition (5 years of age) and the early mixed (8 years of age) dentitions, and an additional 1 to 2 mm between the early mixed and early permanent (12½ years of age) dentitions, with little or no sex differences. Between 6 (first molar fully erupted) and 16 years of age, Moyers and colleagues[42] showed greater increases for males than females for both maxillary (4.1 versus 3.7 mm) and mandibular (2.6 versus 1.5 mm) intermolar widths. Based on a sample of 26 subjects followed longitudinally between 12 and 26 years of age, DeKock[44] reported no significant change for females and only slight increases (1.4 and 0.9 mm for maxilla and mandible, respectively) in intermolar width for males (Fig. 1-4).

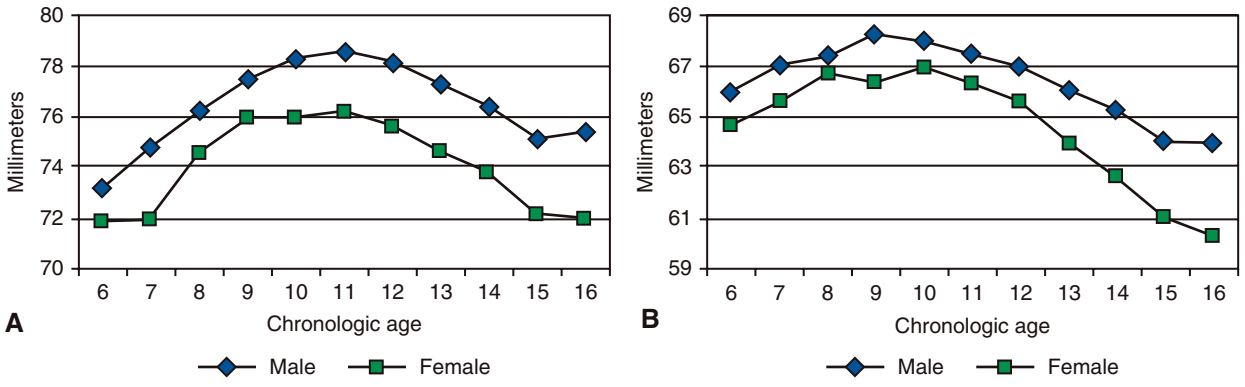

FIG 1-3 Maxillary **(A)** and mandibular **(B)** arch perimeters between 6 and 16 years of age. (Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA Jr: Standards of human occlusal development. Monograph #5, Craniofacial Growth Series, Center for Human Growth and Development, University of Michigan, Ann Arbor, Michigan, 1976.)

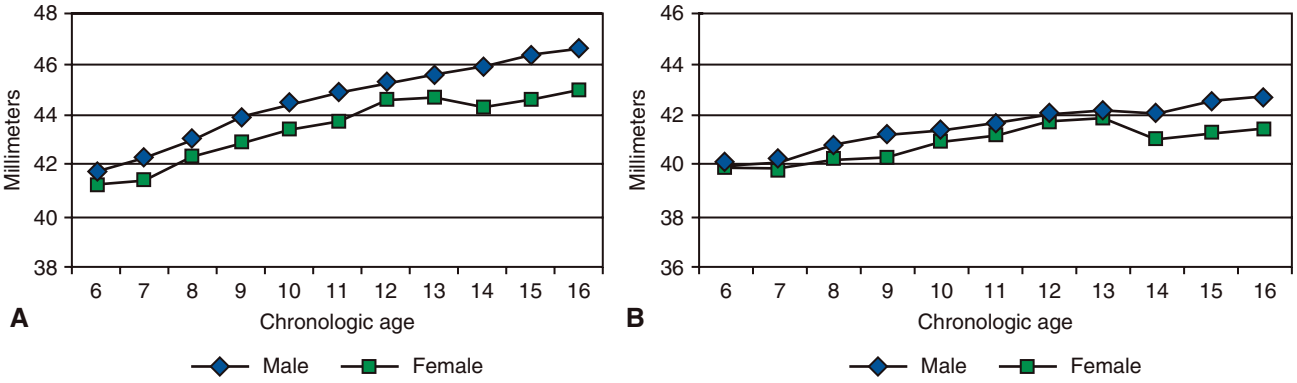

FIG 1-4 Maxillary **(A)** and mandibular **(B)** intermolar widths between 6 and 16 years of age. (Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA Jr. Standards of human occlusal development. Monograph #5, Craniofacial Growth Series, Center for Human Growth and Development, University of Michigan, Ann Arbor, Michigan, 1976.)

14. Without treatment, how do maxillary and mandibular arch depths change during childhood and adolescence?

Maxillary and mandibular arch depths, midline distances between the incisors and a line drawn tangent to the distal crown of the deciduous second molars or their permanent successors, show different growth patterns over time. Maxillary arch depth increases 1.4 and 0.9 mm in males and females, respectively, during the eruption of the permanent incisors.[45] Mandibular arch depth shows little change over the same period. With the loss of the deciduous molars, maxillary arch depth decreases 1.5 and 1.9 mm, whereas mandibular arch depth decreases 1.8 and 1.7 mm in males and females, respectively.[45] Based on subjects with normal occlusion, Bishara and co-workers[43] showed increases (1.1 to 2.8 mm) in maxillary and mandibular arch depths between the deciduous and early mixed dentitions; between the mixed and early permanent dentition, maxillary arch depths increased only slightly (0.5 to 0.7 mm) and mandibular depths decreased 2.6 to 3.3 mm (Fig. 1-5). DeKock[44] reported decreases (approximately 3.0 mm) in arch depth between 12 and 26 years of age, with rates diminishing over time.

15. How do untreated maxillary and mandibular intercanine widths change over time?

During the transition from the deciduous to permanent incisors, mandibular intercanine width increases approximately 3 mm.[45] Maxillary intercanine width also increases during that transition, and then again (approximately 1.5-2.0 mm) with the emergence of the permanent canines; mandibular intercanine widths decrease slightly after the emergence of the permanent canine.[45] Bishara and co-workers[43] reported similar—albeit somewhat smaller—increases in maxillary and mandibular intercanine widths between the deciduous and early mixed dentition; maxillary intercanine width increased 2-2.5 between the early mixed and early permanent dentitions; mandibular widths changed only slightly between the late mixed and early permanent dentitions. Intercanine widths of children followed by the University School Growth Study, Michigan,[42] increased approximately 3.0 mm between 6 and 9 years of age; maxillary widths increased an additional 2.5 mm with the emergence of the permanent canines (Fig. 1-6).

16. What differences exist in intermolar widths between subjects with normal and Class II malocclusion?

Lux and colleagues[46] reported that maxillary intermolar widths were significantly smaller in subjects with Class II division 1 malocclusion than subjects with Class II division 2, Class I malocclusion and normal occlusion. The narrow maxillary arch of division 1 subjects was apparent at 7 years of age and persisted through 15 years of age. Bishara and co-workers'[43] comparisons also showed that the differences between maxillary and mandibular intermolar widths were larger in subjects with normal occlusion than in their Class II division 1 counterparts.

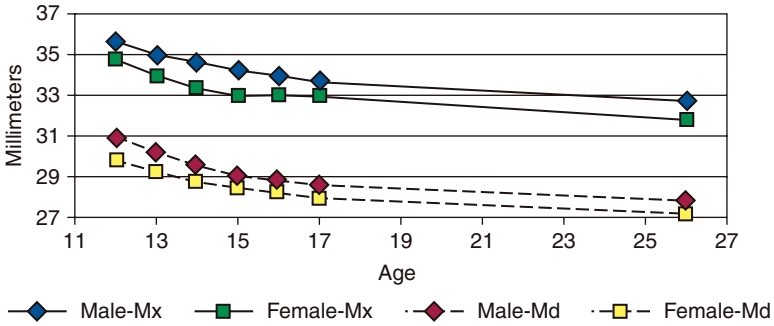

FIG 1-5 Maxillary *(Mx)* and mandibular *(Md)* molar arch depths between 11 and 27 years of age. (Adapted from DeKock WH: *Am J Orthod* 1972;62:56-66.)

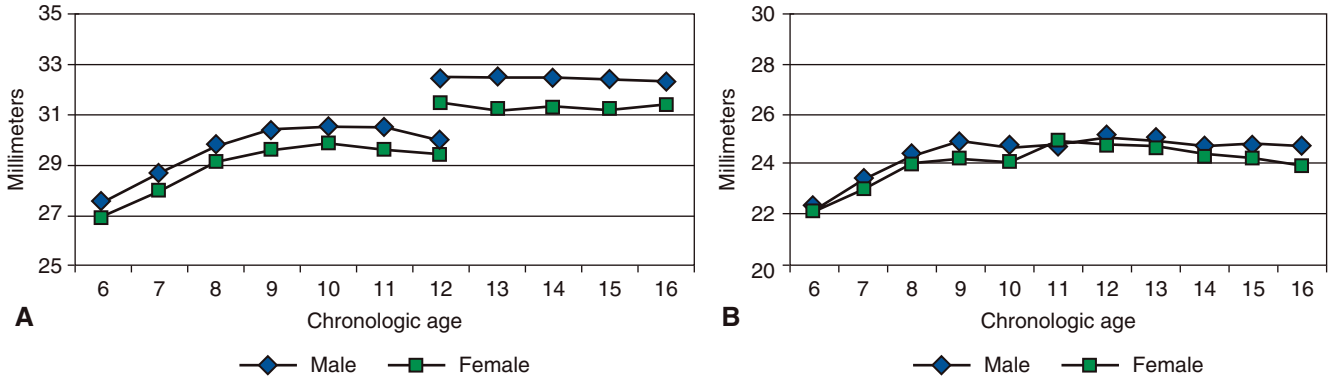

FIG 1-6 Maxillary **(A)** and mandibular **(B)** intercanine width between 6 and 16 years of age. (Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA Jr. Standards of human occlusal development. Monograph #5, Craniofacial Growth Series, Center for Human Growth and Development, University of Michigan, Ann Arbor, Michigan, 1976.)

Comparing arch shape of subjects with Class I and Class II malocclusions, Buschang and colleagues[47] showed that subjects with Class II division 2 malocclusion have the shortest and widest maxillary arches, whereas subjects with Class II division 1 had relatively longer and narrower maxillary arches.

17. Which craniofacial structures might be expected to be the least mature and show the greatest relative growth between 5 and 17 years of age?

Differences in the relative growth of the craniofacial structures have long been established. Hellman,[48] who was among the first to quantify relative growth, showed that cranial widths are consistently more mature than cranial depths, which are in turn more mature than cranial heights. Until the 1970s, growth of the splanchnocranium and neurocranium was categorized based on Scammon's[49] typology and was thought to follow either a general (i.e., somatic) or neural pattern. Baughan and co-workers[50] introduced three distinct growth patterns: a cranial pattern for the cranium and cranial base, a facial pattern for the maxilla and mandible, and a general pattern for the long bones of the body. Buschang and colleagues[51] demonstrated that the craniofacial complex is actually integrated between Scammon's neural and general growth curves. Accordingly, relative craniofacial growth and maturation cannot be neatly categorized; it follows a developmental gradient moving from the more mature measures, such as head height (B-Br; the most mature that they evaluated) through anterior cranial base (S-N), posterior cranial base (S-B), maxillary length (ANS-PNS), upper facial height (N-ANS), corpus length (Go-Gn), and ramus height (Ar-Go). After 9 to 10 years of age, ramus height is actually less mature than stature; it has approximately 10% of its growth remaining in boys 15½ years of age (Fig. 1-7).

18. What sex differences exist in facial heights during infancy, childhood, and adolescence?

Anterior and posterior facial heights are 3% to 5% larger in males than females between birth and 5 years of age.[52] Facial heights are 1% to 10% larger in males than females during childhood and adolescence. Sex differences during childhood are small but statistically significant.[53,54] Differences decrease slightly as females enter their adolescent phase of growth and then increase substantially after males enter adolescence. Male and female ratios of total anterior facial height to total posterior facial height remain similar throughout childhood and adolescence (Fig. 1-8).

19. What sex differences exist in mandibular size and position during infancy, childhood, and adolescence?

During the first 5 years of life, males have significantly larger mandibles than females, with sex differences increasing from 3% to 5% during the first year to 9% to 13% by age 5.[55] During childhood, males continue to exhibit significantly larger overall mandibular size (Co-Pg) than females, primarily due to increased corpus length (Co-Pg). Sex differences in ramus height (Co-Go) during childhood are smaller and increase through adolescence.[53,54] Sex differences in the Y-axis (N-S-Gn), the gonial angle (Co-Go-Me), and mandibular plane angles (S-N/Go-Me) are not statistically significant during childhood or adolescence (Fig. 1-9).

20. What craniofacial features characterize the morphology of hyperdivergent (skeletal open-bite) patients?

Compared with patients with Class I normal occlusion, hyperdivergent patients display decreased posterior-to-anterior face height ratios, smaller upper-to-lower facial height ratios, small ramus heights, larger anterior heights, as well as increased mandibular, gonial, and palatal planes.[56–60] Associated with increased lower face heights and steeper mandibular plane angles, patients with hyperdivergent tendencies demonstrate excessive dentoalveolar heights, especially in the maxilla.[29,58,59,61,62] Children 6 to 12 years of age with high mandibular plane angles undergo significantly less true and apparent forward rotation than children with low mandibular plane angles.[63]

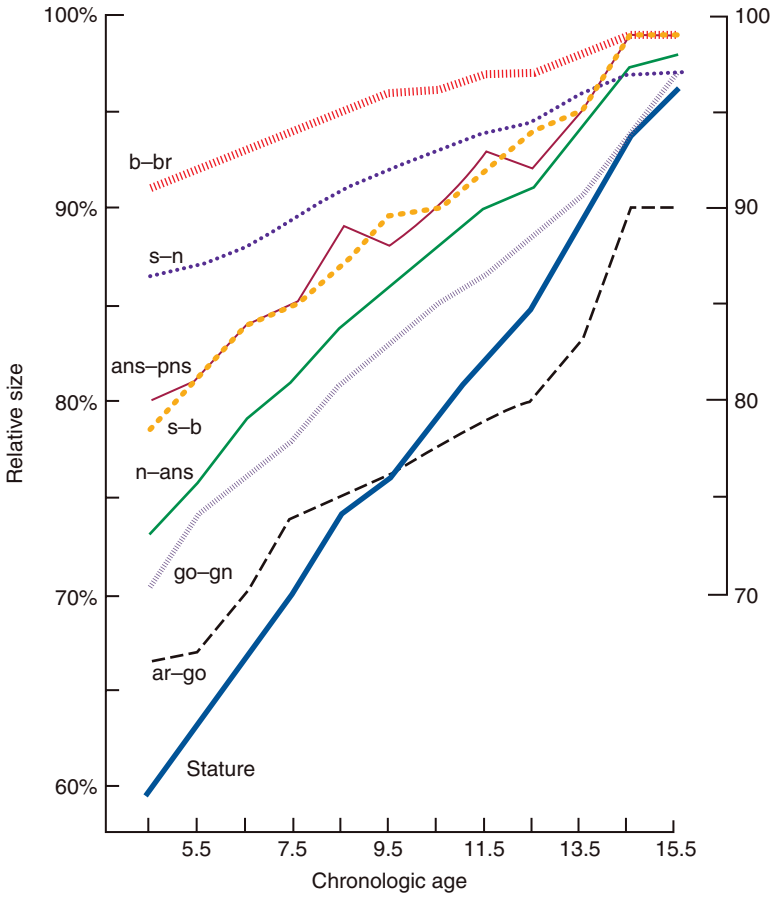

FIG 1-7 Relative (percentage of adult) size of seven craniofacial measures and stature for boys 4½ to 15½ years of age. (Adapted from Buschang PH, Baume RM, Nass GG: A craniofacial growth maturity gradient for males and females between 4 and 16 years of age, *Am J Phys Anthrop* 61:373-381, 1983.)

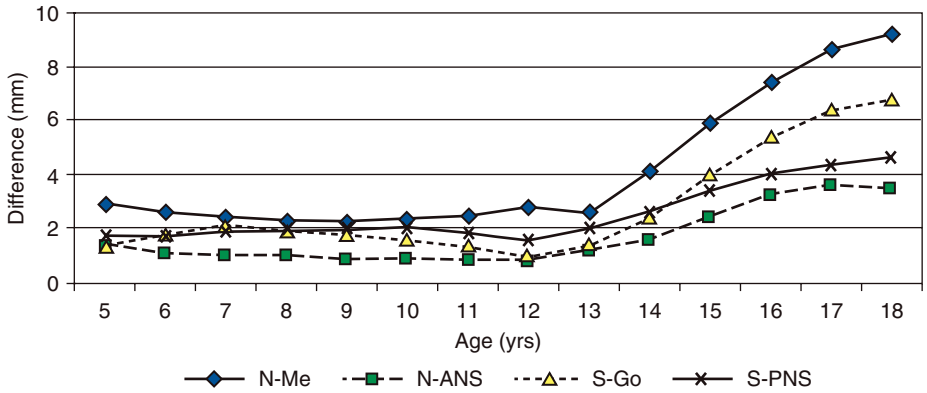

FIG 1-8 Sex differences (male minus female) in facial heights. (Modified from Bhatia SN, Leighton BC: *A manual of facial growth: a computer analysis of longitudinal cephalometric growth data,* New York, 1993, Oxford University Press.)

21. Which aspects of the maxilla and mandible undergo an adolescent growth spurt?

Treatments are often planned based on whether or not patients are approaching, or have attained, their maximum growth. This is one of the reasons why adolescence is commonly thought to be an optimal time to treat. As such, it is important to understand that growth spurts do *not* occur in the AP positions of either the maxilla[25,64,65] or the mandible.[65–68] In other words, the chin does not undergo an anteriorly directed growth spurt. However, the vertical aspects of both maxillary[65,68,70] and mandibular[25,65,68,69] growth exhibit adolescent spurts with peaks. Peak maxillary growth velocities are usually attained more

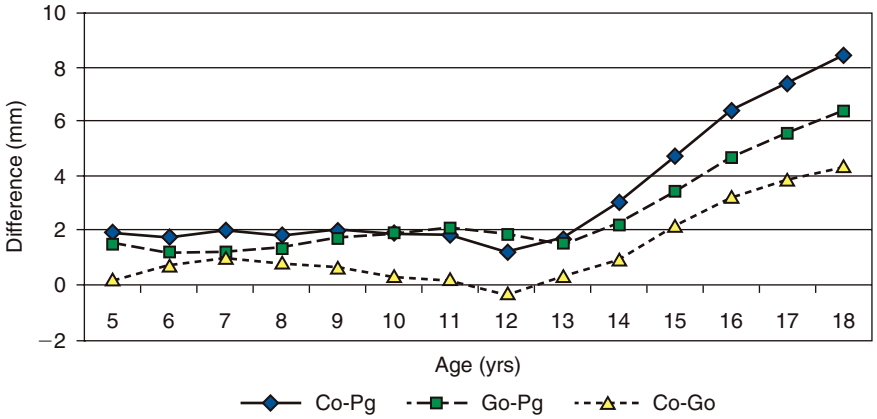

FIG 1-9 Sex differences (male minus female) in mandibular size. (Modified from Bhatia SN, Leighton BC: *A manual of facial growth: a computer analysis of longitudinal cephalometric growth data,* New York, 1993, Oxford University Press.)

than 6 month before peak mandibular velocities.[65] The maxilla tends to peak before PHV,[70] whereas the mandible peaks after PHV.[71,72]

22. How much change is expected in the anteroposterior maxillomandibular relationships of Caucasians during adolescence?

The University of Michigan's mixed-longitudinal study of untreated subjects[54] showed improvements of maxillomandibular skeletal relationships between 10 and 15 years of age; the ANB angle decreases 1 to 1.1 degrees and the A-N-Pg angle decreases 3 to 3.1 degrees. Adolescents followed by the Philadelphia Center for Research in Child Growth[73] demonstrated a decrease of 1.3 and 3.6 degrees for ANB and N-A-Pg angles, respectively, in males; these two measures decreased less than a degree in females between 10 and 15 years of age. The growth study conducted by King's College School of Medicine and Dentistry in London[56] showed a 0.5- to 0.8-degree decrease of ANB and 2 to 3 degrees of decrease of N-A-Pg between 10 and 15 years of age. Untreated French-Canadian males and females between 10 and 15 years show 0.6- and 0.2-degree decreases of the ANB angle, respectively.[74] Although the average changes are small, individual variation is large, with approximately 30% and 26% of 10-year-olds classified as prognathic and retrognathic, respectively, changing to orthognathic by 15 years of age. Similarly, approximately 30% of those who are orthognathic at 10 years of age become either prognathic or retrognathic at 15 years.[74]

23. Does the mandible undergo transverse rotation like the maxilla? If so, how are the two related?

Björk and Skieller[75] showed that posterior maxillary implant widths increased approximately 0.4 mm/year between 4 and 20 years of age. This compares well with the findings of Korn and Baumrind,[76] who reported increases of 0.43 mm/year in the posterior-most region of the maxilla for children 8½ to 15½ years of age. Korn and Baumrind[76] were also the first to document transverse widening of the mandible based on metallic bone markers; they showed that the mandible widened 0.28 mm/year or approximately 65% as much as the maxillary. Gandini and Buschang,[77] who evaluated 25 subjects 12 to 18 years of age with bone markers in both jaws, showed significant width increases between the posterior maxillary (0.27 mm/year) and mandibular (0.19 mm/year) bone markers. For every 1 mm that the maxillary width increased, mandibular width increased 0.70 mm. Iseri and Solow,[78] who followed children annually from 8 to 16 years of age, also reported bilateral width increases of the mandibular body in all subjects. Annual rates decreased from 0.34 mm/year at the younger ages to 0.11 mm/year at 15, demonstrating a clear age effect.

24. Does the glenoid fossa change its position during postnatal growth?

Inferior and posterior displacement of the glenoid fossa should be expected to occur along with growth at the spheno-occipital synchondrosis, elongation of the posterior cranial base, and associated displacement of the temporal bone.[79] Using articulare as a surrogate measure of the glenoid fossa, Björk[80] reported that the distance between the fossa and nasion increases 7.5 mm between 12 and 20 years of age. Based on superimpositions performed on naturally stable cranial base reference structures of 118 children and 155 adolescents, Buschang and Santos-Pinto[81] demonstrated that the glenoid fossa was displaced 0.45 to 0.53 mm/year posteriorly and 0.25 to 0.45 mm/year inferiorly, with greater displacements during adolescence than childhood.

25. How much and in what direction should condylion and gonion be expected to grow and remodel during childhood and adolescence?

The condyle grows superiorly and slightly posteriorly, whereas gonion drifts superiorly and posteriorly in approximately equal amounts. Björk and Skieller's[15] implant studies showed that, depending on the type of true rotation that occurs, the condyle

is capable of growing in both anterior (forward rotators) and posterior directions (backward rotators). Also using metallic implants for superimposing, Baumrind and colleagues[8/2] demonstrated that the condyle grows predominantly in a superior (2.5 mm/year) and slightly posterior (0.3 mm/year) direction between 8½ and 15½ years of age; gonion drifts superiorly (0.9 mm/year) and posteriorly (1.0 mm/year) at similar rates. Using naturally stable mandibular reference structures for superimpositions, Buschang and Santos-Pinto[81] reported 2.3 to 2.7 mm/year superior and 0.2 to 0.3 mm/year posterior growth of the condyle for large samples of children 6 to 15 years of age. Peak adolescent condylar growth velocities approximated 3.1 mm/year (at 14.3 years) and 2.3 mm/year (at 12.2 years) for males and females, respectively.[83]

26. How does the bony chin remodel during childhood and adolescence?

Relative to metallic bone markers inserted into the mandible, each of the 21 cases evaluated by Björk and Skieller[15] demonstrate stability (i.e., lack of modeling) of the cortical region located slightly above pogonion. The remainder of the mandible's external surface models, with both the type and amount of modeling depending on the individual's rotational pattern. On average, there is vertical bone growth associated with the eruption of the teeth; the anterior cortical region demarcated vertically by infradentale and inferiorly by the incisor apex undergoes resorption (but this is highly variable), and the cortical bone below the pogonion and below the symphysis is depository.[82] The same modeling patterns are evident when the mandible is superimposed on naturally stable reference structures.[84] The lingual surface of the symphysis undergoes substantially greater amounts of bony deposition than the anterior or inferior surfaces.

27. At what age might the craniofacial sutures be expected to start closing?

The age at which sutures begin to close is variable and depends largely on how closure is measured. Todd and Lyon[85] were among the first to evaluate sutural closure. Based on a series of 514 male skulls, they described the closure based on gross examination of the ectocranial and endocranial surfaces. They showed that closure begins at approximately the same time on both surfaces, but that ectocranial closure progresses more slowly. Gross examination of 538 male and 127 female skulls demonstrated that the cranial sutures can start closing as early as the late teens or as late as over 60 years of age.[86] By the early 30s or 40s, most people can be expected to show signs of sagittal, coronal, and lambdoid suture closure. Behrents and Harris[87] identified remnants of the premaxillary-maxillary suture in 50 skulls and showed that the facial aspect of the suture was already closed in children 3 to 5 years of age. Using stained sections from 24 subjects, Persson and Thilander[88] reported that closure of the midpalatal and transverse sutures can begin as early as 15 years of age but can be delayed in some individuals into the late 20s or early 30s. Based on histological and microradiographic evaluations of growth activity, Melsen[89] showed that the midpalatal sutures showed evidence of growth through 16 years of age in girls and 18 years of age in boys. Kokich's[90] histological, radiographic, and gross examinations of 61 individuals showed no evidence of bony union of the frontozygomatic suture before 70 years of age (Table 1-1). While sutures become more complex during childhood and adolescence, they show little change in adults.[91] Even though they start closing in adults, only relatively small portions (3-7%) of the sutures exhibit true fusion.[91,92]

28. How much do lip length and thickness change during childhood and adolescence?

Subtelny[93] showed that upper and lower lip lengths increase similarly (approximately 4.5 mm) and progressively between 6 and 15 years of age. After full eruption of the central incisors, the vertical relationship of the maxillary incisor and upper lip is maintained through 18 years of age. Vig and Cohen,[94] who measured upper and lower lip heights relative to the palatal and mandibular planes, respectively, reported increases of approximately 5 mm for the upper and 9 mm for the lower lip between 5 and 15 years of age. Subtelny[93] also showed that increases in lip thickness were considerably greater in the vermilion regions than in the regions overlying skeletal structures. During the first 18 years of life, upper lip

TABLE 1-1 Estimated Ages for the Initiation of Sutural Closure

REFERENCES	SUTURE	AGE OF MALES	AGE OF FEMALES
Todd and Lyon[66]	Sagittal and sphenofrontal	22	N/A
Todd and Lyon[66]	Coronal	24	N/A
Todd and Lyon[66]	Lambdoidal and occiptomastoid	26	N/A
Todd and Lyon[66]	Sphenoparietal	29	N/A
Todd and Lyon[66]	Sphenotemporal, maso-occipital	30-31	N/A
Todd and Lyon[66]	Squamosal, parietomastoid	37	N/A
Sahni et al.[67]	Sagittal	31-35	41-45
Sahni et al.[67]	Coronal	31-35	31-35
Sahni et al.[67]	Lambdoid	41-45	31-35
Behrents and Harris[68]	Premaxillary-maxillary	3-5	3-5
Persson and Thilander[69]	Midpalatal and transpalatal	20-25	20-25
Melsen[70]	Midpalatal and transpalatal	15-16	17-18
Kokich[71]	Frontozygomatic	80s	80s

thickness at Point A increased approximately 7.8 and 6.5 mm in males and females, respectively. Nanda and colleagues[95] showed that upper lip length (Sn-Sto$_{upper}$) increased 2.7 mm (males) and 1.1 mm (females) between 7 and 18 years of age; lower lip length (ILS-Sto$_{lower}$) increased 4.3 mm in males and 1.5 mm in females.

29. Does the soft-tissue facial profile change during childhood and adolescence?

The changes that occur depend on whether or not the nose is included when measuring the soft-tissue profile. Subtelny[93] reported that total facial convexity (N′-Pr-Pog′) decreased 5 to 6 degrees between 6 and 15 years of age; soft-tissue profile (N′-Sn-Pog′) showed little or no change over the same time period. Bishara and colleagues[96] showed that the angle of total facial convexity (Gl′-Pr-Pog′) decreased approximately 7 degrees between 6 and 15 years of age. In contrast, the angle of facial convexity, which does not include the nose, maintained or increased slightly.

30. How does the nose change shape during childhood and adolescence?

It was originally reported that the "hump" on the nasal dorsum develops during the adolescent growth spurt[93] and that nasal shape changes were due to the elevation of the nasal bone.[97] Similar types of shape changes actually take place during childhood (6 to 10 years) and adolescence (10 to 14 years).[98] The upper portion of the dorsum rotates upward and forward (counterclockwise) approximately 10 degrees between 6 and 14 years of age. The lower dorsum shows both downward and backward (clockwise) and upward and forward (counterclockwise) rotation, depending on the relative vertical/horizontal growth changes of the midface.[98] Changes in the nasal dorsum are more closely associated with angular changes of the lower dorsum than of the upper dorsum.

31. According to present evidence, when does growth of the craniofacial skeleton cease?

Behrents[99] reported both size and shape changes in adults. Based on 70 distances and 69 angular measures, he showed growth changes after 17 years of age for 91% of the distances and 70% of the angular measures evaluated. Eighty percent of the distances and 41% of the angles showed growth changes after 30 years of age; 61% and 28% of the distances and angles, respectively, showed growth changes after 35 years of age. Lewis and Roche,[100] who evaluated 20 adults followed between 17 and 50 years of age, showed that cranial base lengths (S-N, Ba-N, Ba-S) and mandibular lengths (Ar-Go, Go-Gn, Ar-Gn) attained their maximum lengths between 29 and 39 years of age, after which they shortened slightly.

32. How does the mandible rotate during adulthood?

Behrents[99] reported that the mandible rotates in a counterclockwise manner in adult males and clockwise in adult females, with associated compensatory alterations of the dentition. He also showed that the Y-axis (N-S-Gn) decreases slightly in males and does not change in females. Relative to the pterygomaxillary (PM) vertical, the mandible comes forward in adult males (approximately 2 mm) but not in females. The mandibular plane angle (S-N/Go-Gn) decreases in males and increases in females. Behrents also showed greater posterior vertical development of the mandible in adult males than adult females. Bishara and colleagues[101] showed that adult males 25 to 46 years of age undergo greater increases of SNB and S-N-Pg than females, whereas females undergo significant increases of N-S-Gn. Forsberg and colleagues[102] reported an increase (0.3 mm) of the mandibular plane angle in males and females between 25 and 45 years of age.

33. What generally happens to the nose during adulthood?

The nose develops substantially during adulthood, with the tip growing forward and downward an average of 3 mm after 17 years of age.[99] Individual adults can exhibit much greater amounts of nasal growth. Males display significantly more nasal growth than females. Formby and colleagues[103] showed that nose height increases 0.6 mm, nose length increases 1.7 mm, and nose depth increases 2.3 mm between 18 and 42 years of age. Between 21 and 26 years of age, Sarnas and Solow[104] demonstrated 0.8- to 1.0-mm increases in nose length.

34. What generally happens to the upper lip length during adulthood?

Upper lip length increases 0.5 to 0.6 mm between 21 and 26 years of age.[104] Over the same period, upper incisor display (Sto-OP$_{max}$) decreases slightly (0.3 mm) in males and does not change in females. Formby and colleagues[103] showed that upper lip length increases 0.8 to 1.7 mm and upper incisor display (lip to incisal edge) decreases 1.0 mm between 18 and 42 years of age. Behrents[99] demonstrated that upper lip length (ANS-Sto) increases significantly in both males (2.8 mm) and females (2.2 mm), whereas the maxillary incisor to palatal plane distance increases only 0.06 to 0.08 mm after 17 years of age, thereby supporting an even greater decrease in upper incisor display.

35. How does the soft-tissue profile change during adulthood?

Sarnas and Solow[104] showed that the soft-tissue profile angle (including the nose) increased (0.3 degree) in males and decreased (0.4 degree) in females between 21 and 26 years of age. Behrents[99] provides the best longitudinal data demonstrating a straightening and flattening of the soft-tissue lip profile during adulthood. The lips become substantially less pronounced with increasing age.[99,101,102] The perpendicular distances of the upper and lower lips relative to the soft tissue plane decreased approximately 1 mm in adults; angular changes indicate approximately 4- to 6-degree flattening of the lips.[99]

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CHAPTER

2

Development of the Occlusion

Timo Peltomäki

Development of the occlusion, in other words, eruption of the teeth and formation of the interrelationship between the teeth of the upper and lower jaws, is a genetically and environmentally regulated process. Coordination between tooth eruption and facial growth is essential to achieve a functionally and esthetically acceptable occlusion. Most orthodontic problems arise through variations in the normal tooth eruption/occlusal developmental process. Therefore, every developing malocclusion and dentofacial deformity must be evaluated against normal development.

In this chapter, normal eruption timing and sequence of primary and permanent teeth are discussed. Since occlusion is regarded as a dynamic rather than a static structure, changes in the dental arch dimensions are then discussed. Finally, various common deviations in the occlusal development are addressed.

1. What are the stages of tooth development?

Tooth development is a genetically regulated process characterized by interactions between the oral epithelium and the underlying mesenchymal tissue.[1] During the first stage of tooth development, called the *initiation stage*, a plate-like thickening of the oral epithelium (dental placodes) can be seen in histological examination. This is followed by the bud stage with epithelial ingrowth and formation of bud-shaped tooth germs. Next, the mesenchymal tissue condenses around the epithelial buds and progressively forms the dental papilla. Gradually the dental epithelial tissue grows to surround the dental papilla.

From this stage the epithelium can be called the *enamel organ*. It gains a concave structure; therefore, this stage is called the *cap stage*. A third structure, the dental follicle, originates from the dental mesenchyme and surrounds the developing enamel organ. During this stage the shape of the crown becomes evident, but the final shaping of a tooth occurs during the next stage, called the *bell stage*. During the bell stage, cytodifferentiation begins and tooth-specific cell populations are formed. Some of these cells differentiate into specific dental tissue-forming cells. During the secretory stage, the differentiated cells start to deposit the specific dental matrix and minerals. Once the dental hard tissue in the crown has been formed and completely calcified, tooth development continues with the root formation and tooth eruption.

Root formation takes place concomitantly with the development of the supporting structures of the teeth (periodontal ligament, cement, alveolar bone). The epithelial buds of the permanent teeth (except permanent molars) develop from the dental lamina of the primary teeth.

2. What are the stages of tooth eruption?

Eruption of teeth can be divided into different stages.[2] The first stage is called preemergent eruption when the developing tooth moves inside the alveolar bone but cannot yet be seen clinically. This movement begins once the root formation has started. Resorption of bone, and in the case of a permanent tooth, resorption of the roots of the primary teeth, is necessary to allow preemergent eruption. In addition, an eruption force (origin still unknown) must exist to move the tooth. Emergence, the moment when a cusp or an incisal edge of a tooth first penetrates the gingiva, usually occurs when 75% of the final root length is established. Next, postemergent eruption follows and a tooth erupts until it reaches the occlusal level (Fig. 2-1). Eruption speed is faster during this stage and therefore the stage term *postemergent spurt* is sometimes used. Eruption does not stop once the tooth has come to occlusion but continues to equal the rate of the vertical growth of the face. On average, a molar tooth erupts about 10 mm after having reached the occlusal contact. It is also important to know that eruption of a tooth causes the alveolar bone to grow. In other words, each tooth makes its own alveolar bone. This has a clinical bearing: if a tooth fails to erupt, no alveolar bone develops; if a tooth is lost, alveolar bone is also gradually lost.

Short-term eruption of teeth seems to follow day-night (circadian) rhythm.[3] Eruption occurs mainly during early hours of sleep, although some intrusion can happen during the day, particularly after meals. Furthermore, it has been found that tooth eruption and secretion of growth and thyroid hormones have a similar circadian pattern.[3]

3. What are the eruption timing and sequence of primary teeth?

There is a large individual variation in the eruption schedule of both primary and permanent teeth. Delay or acceleration of 6 months from the average eruption timetable is still within the normal range. Despite variation in the eruption schedule, the eruption sequence of teeth is usually preserved.

Generally the first primary teeth to erupt are the lower central incisors (on average at 7 months), followed soon by the upper central incisors (on average at 10 months). Thereafter, the upper and lower lateral incisors emerge (on average at 12 months), then the upper and lower first molars (on average

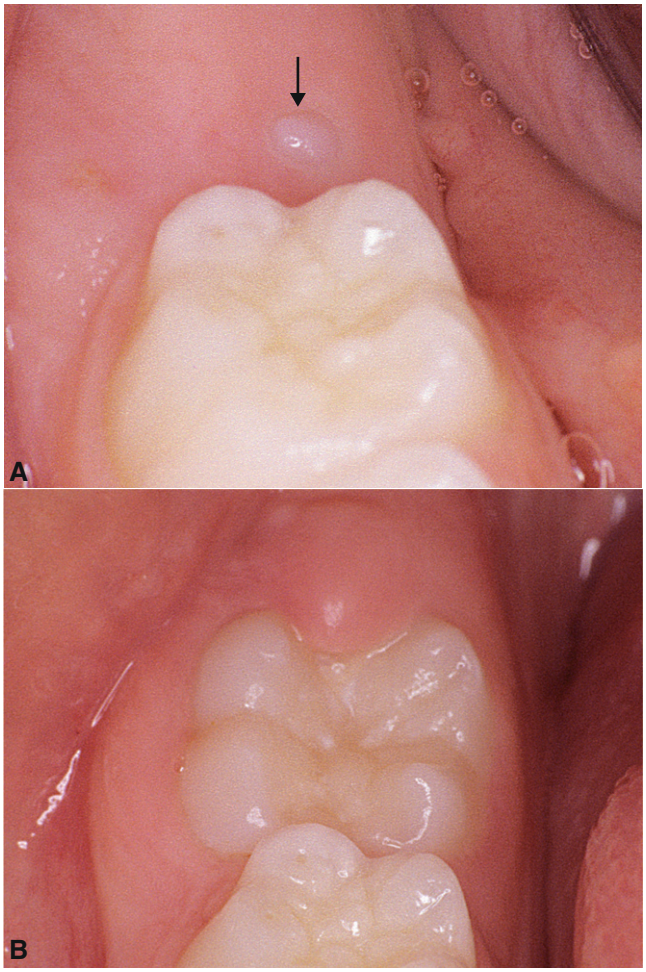

FIG 2-1 **A**, The mesiolingual cusp of the lower right first permanent molar *(arrow)* has emerged. **B**, Two months later the occlusal surface can be seen. Next, postemergent eruption follows and a tooth erupts until it reaches the occlusal level.

TABLE 2-1 Average Eruption Timing and Sequence of Primary Teeth

TOOTH	TIME (IN MONTHS)
Lower central incisors	7
Upper central incisors	10
Upper and lower lateral incisors	12
Upper and lower first molars	16
Upper and lower canines	20
Upper and lower second molars	28

at 16 months). Primary canines erupt on average at 20 months and finally the second molars on average at 28 months. Primary dentition is thus fully formed by the age of 2½ years with calcification of the roots of the primary teeth completed 1 year later (Table 2-1).

4. What are typical features of primary dentition?

Spacing in the primary dentition is a typical feature and a requirement to secure space for the larger permanent incisors (Fig. 2-2, *A*). About 70% of children have spaces in the

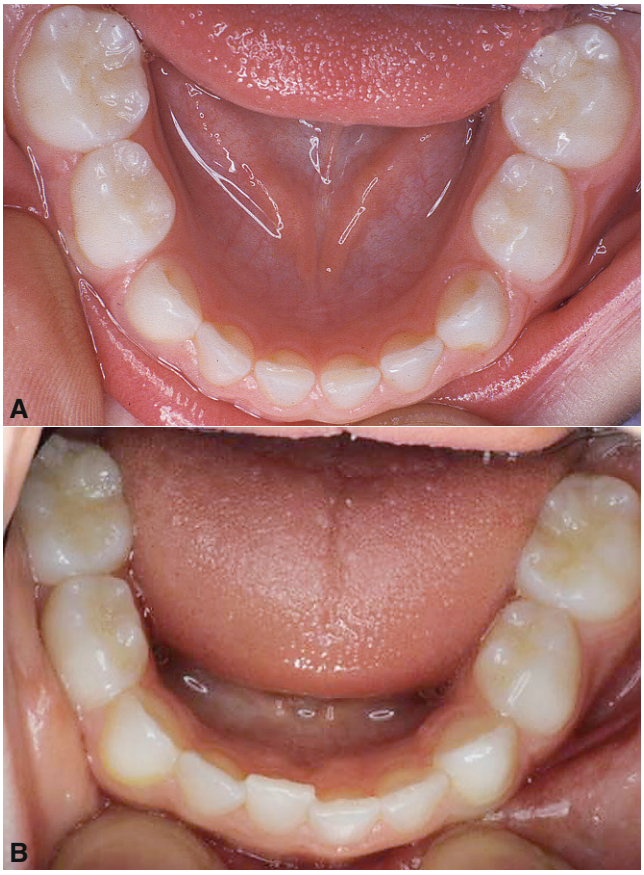

FIG 2-2 **A**, Spacing in the primary dentition is a typical feature and is a requirement to secure space for the larger permanent incisors. **B**, If there is crowding in the primary dentition, crowding is inevitable in the permanent dentition.

front area of primary teeth. The largest spaces, called *primate spaces*, are located between the upper primary laterals and canines and between the lower primary canines and first molars. It is estimated that if the total amount of space per dental arch is 0 to 3 mm, there is 50% probability of crowding in the permanent dentition. If there are no spaces or even crowding in the primary dentition, crowding is inevitable in the permanent dentition (see Fig. 2-2, *B*).[4] During the full primary dentition stage (3 to 6 years), not much happens in the dimensions of the dental arches; however, overjet and overbite may decrease.[5]

5. What is the terminal plane, and what are the different terminal plane relationships in the primary dentition?

Terminal plane denotes the anteroposterior relationship (discrepancy) between the distal surfaces of the upper and lower second primary molars. It can be a flush terminal plane, or there may be a mesial or a distal step (Fig. 2-3). Occurrence of different terminal planes differs greatly according to the method used to define terminal plane and the population studied. In the Caucasian (European descent) population, about 60% of children exhibit mesial step (in about 40% the mesial step is less than 2 mm and in 20% more than 2 mm), about 30% exhibit

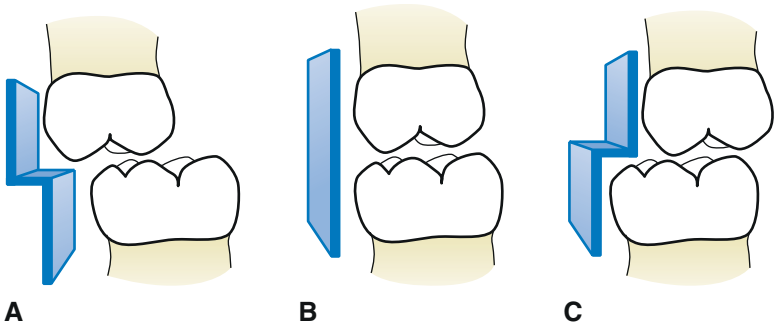

FIG 2-3 Terminal plane denotes the anteroposterior relationship between the distal surfaces of the upper and lower second primary molars. In the Caucasian population about 60% of children exhibit mesial step **(A)**, about 30% flush terminal plane **(B)**, and about 10% distal step **(C)**. (From Bath-Balogh M, Fehrenbach MF: *Illustrated dental embryology, histology, and anatomy*, ed 2, St Louis, 2006, Saunders.)

flush terminal plane, and about 10% distal step.[6] In children of African-American descent, the prevalence of distal step is lower (5%) and mesial step higher (89%).[7]

6. What does the terminal plane relationship of the primary second molars predict on the permanent molar relationships?

The terminal plane relationship determines the anteroposterior position of the permanent first molars at the time of their eruption. Differential forward drift of the lower and upper first permanent molars (generally more forward drift of the lower molar) and differential maxillary and mandibular forward growth (generally more forward growth of the mandible) play a role in this transition. In about 80% of the individuals with mesial step less than 2 mm, Angle's Class I molar relationship results. If the mesial step is more than 2 mm, a Class III molar relationship results in 20% of the subjects. The flush terminal plane results in either a Class I (56% of subjects) or Class II (44% of subjects) molar relationship, depending on the amount of mandibular anterior growth and forward drift of the lower first primary molars in relation to the upper ones. Distal step of the primary second molars almost invariably results in a Class II molar relationship in the permanent dentition.[6]

7. How is Angle's classification of occlusion defined?

Angle's original classification of occlusion is based on the anteroposterior relationship between the upper and lower first permanent molars. In Class I occlusion, the mesiobuccal cusp of the upper first molar occludes with the buccal groove of the lower first molar. Class I occlusion can further be divided into normal occlusion and malocclusion. Both subtypes have the same molar relationship, but the latter is also characterized by crowding, rotations, and other positional irregularities.

Class II occlusion is when the mesiobuccal cusp of the upper first molar occludes anterior to the buccal groove of the lower first molar. Two subtypes of Class II occlusion exist. Both have a Class II molar relationship, but the difference lies in the position of the upper incisors. In Class II division 1 malocclusion, the upper incisors are labially tilted, creating significant overjet. On the contrary, in Class II division 2 malocclusion, the upper central incisors are lingually inclined and the lateral incisors are labially inclined. When measured from the first incisors, overjet is within normal limits in individuals with Class II division 2 malocclusion.

Class III malocclusion is opposite to Class II; the mesiobuccal cusp of the upper first molar occludes more posterior than the buccal groove of the lower first molar.

8. What are the eruption timing and sequence of permanent teeth?

The eruption sequence can be checked with the help of eruption charts and is a useful tool for the orthodontist to assess the dental age of a patient (Table 2-2). As a general rule, a tooth should erupt once two-thirds of its root is formed.

Permanent teeth erupt in two different stages. The first transitional period occurs between the ages of 6 and 8 and is followed by an approximately 2-year intermediate period. The second transitional period starts on average at the age of 10 years and lasts around 2 years. In general, teeth erupt earlier in girls than in boys. As in the primary dentition, there is a great individual variation in the eruption timing of permanent teeth. Delay or acceleration of 12 months from the average eruption timetable is still within the normal range.

The first transitional period, between 6 and 8 years, can be divided further into three yearly stages. At 6 years the upper and lower first molars (also called *6-year molars*) and the permanent lower central incisors erupt (Fig. 2-4). At 7 years the upper central and the lower lateral incisors emerge and erupt. The first transitional period is completed by the eruption of the upper lateral incisors at the age of 8 years. By this time all the permanent upper and lower incisors and first molars have erupted, for a total of 12 permanent teeth. The term *mixed dentition* is used to describe a dentition containing both primary and permanent teeth.

The second transitional period can also be divided into three yearly stages. The first period is characterized by the eruption of the lower canines and lower and upper first premolars within the same time frame at about 10½ years of age. This is followed

TABLE 2-2 Average Eruption Timing and Sequence of Permanent Teeth

TRANSITION PERIOD	AGE	TEETH	FEMALE (TIME IN YEARS)	MALE (TIME IN YEARS)
First				
	6 years	Lower first molars	5.9	6.2
		Upper first molars	6.2	6.4
		Lower central incisors	6.3	6.5
	7 years	Upper central incisors	7.2	7.5
		Lower central incisors	7.3	7.7
	8 years	Upper lateral incisors	8.2	8.3
Second				
	10 years	Lower canines	9.9	10.8
		Upper first premolars	10.0	10.4
		Lower first premolars	10.2	10.8
	11 years	Upper second premolars	10.9	11.2
		Lower second premolars	10.9	11.5
		Upper canines	11.0	11.7
	12 years	Lower second molars	11.7	12.1
		Upper second molars	12.3	12.7
		Upper and lower third molars	17-25	17-25

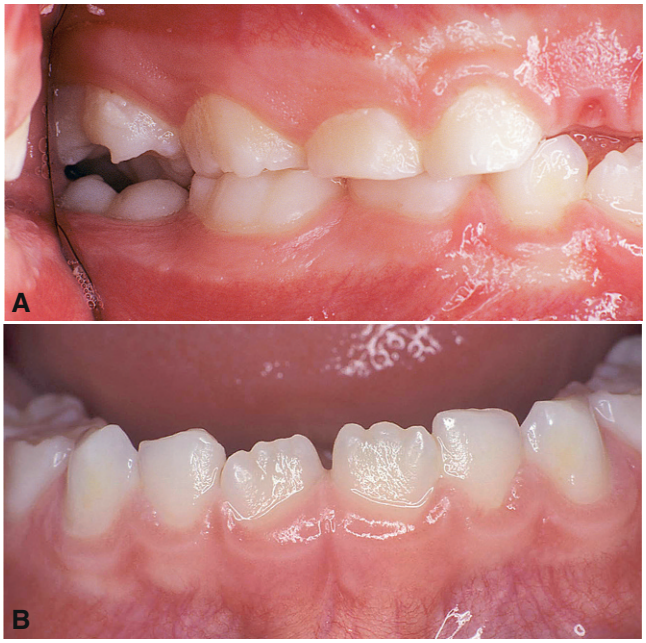

FIG 2-4 The first transitional period starts at approximately the age of 6 years with the eruption of the upper and lower first molars **(A)** and the lower central incisors **(B)**.

soon by the eruption of the upper and lower second premolars and usually somewhat later by the upper canines (at the age of 11 years). The second molars (12-year molars) complete the second transitional period at the age of 12 years.

Eruption of the third molars occurs much later with large individual variation (range, 17 to 25 years).

9. When does the mineralization of the permanent teeth occur?

Radiologically visible mineralization of the permanent first molars starts approximately at the time of birth and is followed 6 months later by the upper and lower central and lower lateral incisors. The long canines require a long time to become fully mineralized and therefore start the mineralization early (at 12 months) despite late eruption. Upper lateral incisors have an opposite mineralization/eruption pattern: a fairly late start of mineralization at 18 months and much earlier eruption than canines. The mineralization of premolars and second molars begins between ages 2½ and 3½ years. Signs of mineralization of the third molars can be seen at approximately 10 years, with particularly large variation. As a general rule, completion of crown formation (mineralization) takes 4 years, and the root formation takes another 5 years ±1 year, depending on the size of the tooth.

10. How do the initial location and size of the permanent incisors compare with the primary teeth?

In the maxilla and mandible, the permanent incisors develop on the palatal/lingual side of the roots of the primary incisors with considerable crowding. Upper lateral incisors are located even more palatally than the central ones. Total mesiodistal dimension of the upper permanent incisors is about 8 mm larger than that of the primary incisors. In other words, in the upper front area there is lack of space, approximately the size of an upper lateral incisor. In the lower arch, the difference is less (5 to 6 mm), approximately the mesiodistal dimension of a lower incisor.

11. How is the space deficit between the primary and permanent incisors solved?

For the upper permanent incisors, several factors are available to regain this 8 mm or so space deficit. First, the upper incisors generally erupt to a wider dental arch circumference than the primary incisors, which is the most effective way to gain space for these teeth. Second, when the central permanent incisors erupt, they push the primary lateral incisors distally. The same "pushing effect" repeats when the permanent laterals erupt and push the primary canines distally. With this "pushing effect" the

existing spaces of primary dentition are also closed and used for the larger permanent incisors to accommodate. Another mechanism of space-gaining in the permanent dentition is the transverse growth of the maxilla at its midpalatal suture. Thus, despite the initial lack of space in the maxillary anterior area, space conditions are generally resolved for the permanent incisors. Naturally, if the above factors are not available or working, crowding and/or crossbite, particularly of the upper laterals, can be seen.

In the mandibular anterior area, comparable pushing takes place as in the maxillary anterior area to make space for the erupting permanent incisors. However, lower anterior teeth do not generally erupt to a wider dental arch circumference than the primary ones, and no transverse growth can take place in the anterior area of the mandible. If considerable spacing in the primary dentition (> 5 to 6 mm) does not exist, crowding is commonly seen once the permanent lower incisors have erupted. This is called *physiological crowding*.

12. Is anterior spacing common once permanent incisors have erupted?

Despite the initial crowding of the permanent incisors in the maxillary bone, spacing is a common finding in the upper anterior area once the incisors have erupted. A large space (> 2 mm) between the upper central incisors, called *midline diastema*, may exist due to a strong labial frenum. Upper lateral incisors may be inclined distally due to the pressure of the erupting canines on their roots. This normal spacing condition in the upper front area is called *ugly duckling*. Once the permanent canines erupt, upper spaces usually close and uprighting of the lateral incisors can be seen. On the other hand, spacing in the mandibular anterior area is very seldom seen. Rather, some crowding is typical for this developmental stage.

13. What are nonsuccedaneous teeth, and how is space secured for them?

Nonsuccedaneous teeth are teeth that do not succeed deciduous teeth (i.e., all permanent molars). In the upper dental arch, space is created for the molars by bone apposition at the free posterior border of the maxilla. Also, the transverse palatal suture may make a contribution. For the lower molars, bone apposition occurs on the posterior side of the mandibular ramus, and bone resorption occurs on the anterior portion of the ramus. During normal occlusal development, upper and lower first molars usually drift forward because of excess space due to the leeway space. This anterior drift of the first molars opens up space for the second molars to erupt.

14. What is leeway space, and what is its importance?

The space occupied by the primary canines and molars is greater than that required for the corresponding permanent teeth. This size difference of the primary and permanent teeth is known as the *leeway space*. On average, 1 to 1.5 mm of excess space exists in each upper quadrant and 2 to 2.5 mm in the lower quadrants with large individual variation. A significant contribution of the leeway space comes from the difference in the second primary molars and their counterparts. The primary molars are on average 2 mm larger than the second premolars. During normal occlusal development, about 2 mm of the leeway space is used by the anterior drift of the molars. Lower molars usually drift more mesially than the upper ones, which often strengthens the Class I molar relationship. Physiological crowding in the lower front area may also be reduced from the leeway space, allowing the permanent canines to drift distally.

15. Is the eruption sequence of teeth important?

The eruption sequence presented in Question 8 is the most optimal one for a proper occlusion to develop. However, variations from this normal sequence are frequently seen during the second transitional period, and these variations may have clinical significance.

Sometimes the lower second molars erupt before the second premolars. This may cause anterior drift of the first permanent molars too early and, as a consequence, space loss for the second permanent premolars. Therefore, it is preferable that the second premolars erupt before the second permanent molars.

Since the leeway space provides the space needed by the upper canines, they should erupt after the permanent premolars. If not, lack of space may cause the upper canines to erupt too labially.

16. What changes occur in the dental arch length during occlusal development?

Dental arch length has a special meaning in orthodontics. Arch length denotes the distance from the most labial surfaces of the central incisors to the line connecting the mesial (or distal) points of the first permanent molars in the midsagittal plane.

Measurements and changes in the dental arch dimensions are largely based on the studies of Moorrees.[5] Changes in the arch length occur in two different phases during occlusal development. During the first transitional period, upper dental arch length increases slightly (on average 0.5 mm) because of the more labial eruption of the upper permanent central incisors. Essentially, this eruption pattern creates a larger dental arch circumference compared with the positions of the primary incisors. An additional increase of approximately 1 mm can be seen when the permanent lateral incisors erupt. During the second transitional period, arch length commonly decreases because the leeway space allows permanent premolars and first molars to drift forward. Therefore, the average upper dental arch length is slightly longer or the same at 3 years than at 15 years.

In the lower dental arch, no clinically significant changes occur in the arch length during the first transitional period because lower permanent incisors erupt into the same arch circumference as the primary incisors. A considerable shortening of the lower dental arch length takes place during the second transitional period. As discussed earlier, larger leeway space in the lower compared with the upper dental arch allows more anterior migration of the premolars and molars, which leads to the shortening of the arch length. The average lower dental arch length is thus slightly longer at 3 years than at 15 years.

According to Moorrees,[5] 2- to 3-mm shortening of the lower dental arch length can be seen from the full primary dentition to the permanent dentition.

17. What changes occur in the dental arch width during occlusal development?

During the eruption of the maxillary permanent incisors, intercanine dimension (measured between primary canines) increases on average by 3 mm. Before or at the time of eruption of the permanent canines, another increase of approximately 2 mm takes place in canine-to-canine distance. The increase in the upper intercanine distance may be caused by the distalizing pressure of the erupting permanent incisors on the permanent canines and growth in width of the maxilla at the midpalatal suture. A steady increase (total 4 to 5 mm) in the distance between the upper first permanent molars can be seen after their emergence.

In the lower dental arch, a comparable increase of the intercanine distance as in the upper arch occurs during the eruption of the permanent incisors (3 mm on average). However, unlike in the upper arch, no additional increase in the canine-canine distance takes place in the lower arch during the later stages of dental development. This early establishment of the lower intercanine distance has an important clinical bearing in that attempts to increase lower intercanine distance by orthodontic means usually leads to relapse.[8] After the emergence of the molars, the distance between the lower first molars increases steadily corresponding to the upper arch.

There are two ways to measure dental arch width. The more common method is to measure the distance between the corresponding contralateral teeth at the cusp tips (e.g., intercanine or intermolar width). Another measurement can be made at the palatal/lingual gingival level of the teeth; this measurement describes the width of the bony arch.[5] The increase in the intercanine distance is greater when measured from the cusp tips of the teeth than at the gingival level, particularly in the upper dental arch. This may be because the labio-lingual crown diameter of the permanent canines is greater than that of the primary canines.

18. What changes occur in the dentition once permanent teeth (excluding wisdom teeth) have erupted?

Appearance of, or actual increase of, already existing crowding, called *late* or *secondary crowding*, in the lower anterior area is a typical finding in late dental development in the late teens and early 20s. This crowding occurs before or simultaneously with the emergence of wisdom teeth and may take place both in orthodontically untreated or treated subjects. Several factors are thought to play a role in this crowding in the lower anterior area.[9] Maxillary and mandibular differential growth is considered to have an effect on the late crowding. Growth of the maxilla ceases earlier than growth of the mandible. Because of overbite, lower anterior teeth cannot move forward to the extent of the lower jaw growth but tilt lingually to a smaller circumference, which results in crowding. In addition, the maturation of soft tissues that occurs during the teenage period may increase the pressure from lips, causing crowding. More forward drift takes place in the lower dentition than in the upper, which also increases crowding.

19. Do wisdom teeth play a role in the lower anterior crowding?

Eruption of wisdom teeth often occurs simultaneously with the appearance or increase in lower anterior crowding. It is a common belief that this is because of pressure created by the erupting wisdom teeth. However, a randomized controlled study suggests that wisdom teeth play a minor role, if any, in the late lower incisor crowding.[10] Individuals with congenitally missing third molars may also have this crowding. Thus, there is no evidence to support a recommendation to extract third molars in order to prevent late incisors from crowding.[11]

20. What are the most common reasons for interference with normal tooth eruption?

As stated earlier, great individual variation occurs in the timing of eruption of permanent teeth. Premature tooth eruption is possible, but delayed tooth eruption is more common. This may occur only on one side or on both sides of the dental arch.

Reasons for the delayed tooth eruption may be divided into rare systemic factors and more frequent local factors.[12] Systemic factors usually involve a disease process with the whole dentition commonly affected. Bone metabolism for necessary resorption of the alveolar bone and/or roots of the primary tooth may be disturbed, and eruption may therefore be delayed or even hindered. If a permanent tooth fails to fully or partially move from its crypt position in the alveolar process into the oral cavity without evident cause (presumably due to malfunction of the eruption mechanism), this condition is called *primary failure of tooth eruption (PFE)*.[13] PFE is rare and usually affects posterior teeth. Due to incomplete eruption of posterior teeth, severe lateral open bite is seen. Recent studies suggest that parathyroid hormone receptor 1 gene is causative for PFE.[14]

Local factors that delay tooth eruption may be mechanical in nature, and once the obstruction is eliminated, further tooth eruption may take place. Local factors include supernumerary teeth, heavy fibrous gingival tissue because of premature loss of a primary tooth, crowding, and sclerotic alveolar bone. Ankylosis of a tooth also causes delay or prevention of a tooth eruption. As a general rule, if a permanent tooth has erupted but its counterpart does not within 6 months, an eruption problem is evident and further investigation is recommended.

21. What is tooth ankylosis, and what is its clinical significance?

Ankylosis of a tooth is defined as the union/fusion between a tooth and alveolar bone. This means that the periodontal ligament is obliterated in one or more locations, and there is contact between the cementum of a tooth and alveolar bone. Ankylosis is more common in the primary, particularly primary molars, than in the permanent dentition (Fig. 2-5). Prevalence of primary molar ankylosis is 5% to 10%. Ankylosis is thought to be related to the noncontinuous resorption process of the

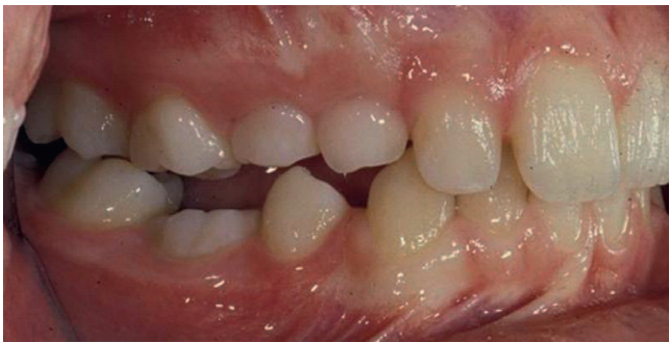

FIG 2-5 Because of ankylosis of the lower right primary second molar, a vertical deficiency in the occlusal level developed since the ankylosed teeth could not erupt and the adjacent teeth continued erupting.

roots of the primary teeth. In other words, during the resorption phase of the root, there are periods of rest and reparation. During the reparative phase, fusion of the cementum and alveolar bone may develop. Causative factors for ankylosis are currently unknown.

An ankylosed tooth cannot erupt; consequently, the tooth appears to submerge with continued alveolar growth. In reality, an ankylosed tooth does not submerge, but when it fails to erupt, a vertical deficiency in the occlusal level develops as the adjacent teeth continue erupting. The term *infraocclusion* is used to describe this condition and the amount of infraocclusion of an ankylosed tooth depends on when the ankylosis occurred. It is known that a molar erupts on average 1 mm yearly. This means that if the vertical defect is large, one may speak about *early ankylosis*. On the other hand, *late ankylosis* denotes infraocclusion as minor (1 to 2 mm), and ankylosis had evidently occurred near the time of exfoliation of a primary molar.

22. What is ectopic eruption?

Ectopic eruption of a tooth means that the tooth erupts away from the normal position. This condition can have a multifactorial underlying etiology. Sometimes a tooth erupts ectopically because of an abnormal initial position of the tooth bud. Upper first molars and canines are most commonly observed to erupt ectopically, followed by lower canines, upper premolars, lower premolars, and upper lateral incisors. In the permanent dentition, the upper first molars erupt most commonly ectopically (prevalence approximately 4%) (Fig. 2-6). The molar may then erupt too far anteriorly and make contact with the distal root of the second primary molar. As a consequence, the first permanent molar may fail to erupt on both sides or only on one side. It may also happen that an ectopically erupting first permanent molar causes severe resorption (called *undermining resorption*) of the roots of the second primary molar, leading to early exfoliation of that primary molar. This causes a more anterior eruption of the first permanent molar, resulting in space loss and future crowding of that quadrant. Because of insufficient space, the upper and lower lateral incisors may also erupt ectopically and too distally. The clinical significance of this may be an early loss of the primary canines from undermining resorption.

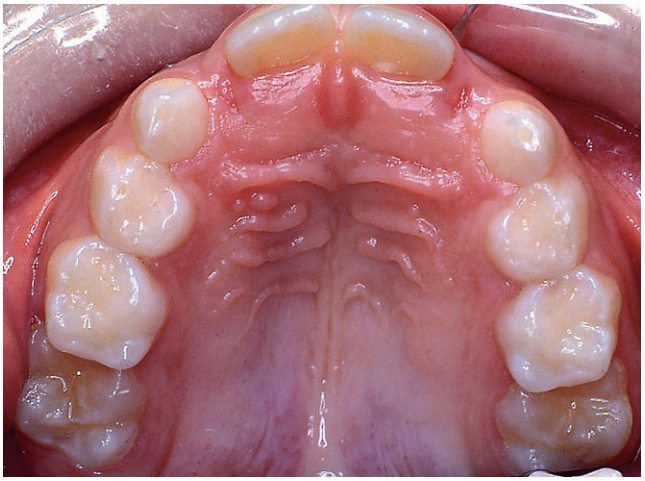

FIG 2-6 Both upper first molars have erupted ectopically, too far anteriorly. This may lead to early exfoliation of the upper second primary molars by undermining resorption and space loss in these quadrants.

23. What are eruption problems of the upper permanent canines?

Canines, particularly maxillary canines, have the longest way of all teeth to erupt from their initial position to the occlusion. Initially the upper canines are located high in the maxilla, in the canine fossa, close to the base of the nose. In preemergent eruption, they move downward along the distal aspect of the roots of the lateral incisors. When the child is 9 to 10 years old, these teeth should be palpable in the fornix between the permanent lateral incisor and the primary first molar. If not, ectopic eruption or impaction may be expected. Maxillary canines are the last teeth to erupt and are therefore strongly influenced by spacing conditions. The canines' long path of eruption, coupled with their late emergence timing, causes their high prevalence of impaction (about 2%).

Most of the impacted upper canines are palatally located. Interestingly, nearly 50% of patients with palatally located upper canines present with anomalous (peg shaped) or congenitally missing upper lateral incisors. Because of this clinical link, it has been proposed that a common genetic etiology may be responsible for canine impaction and hypodontia.[15] Another explanation for this observation could be that a guiding structure for the proper eruption of the canine is missing, and, therefore, the canine is palatally displaced.

In a computed tomography (CT) study, researchers found that even in cases of normal eruption of upper canines, the continuity of the periodontal ligament of the lateral incisor may be temporarily lost with no resorption sign in the root.[15] When the path of eruption abnormally diverges so that the canines make contact with the roots of the lateral incisors, resorption of the incisor may be expected unrelated to the size of the dental follicle of the canine.[16]

24. What is a typical eruption problem of the second permanent molars?

If space is not adequate for the upper second permanent molars, they often tilt buccally and distally before their emergence

and eventually erupt too buccally. On the contrary, the lower second permanent molars tend to tilt lingually because of insufficient space. When the second molars erupt like this, they may not occlude properly and a scissor-bite or buccal crossbite may develop. In the scissor-bite, the upper second molar is positioned too far to the buccal and the lower second molar is too far to the lingual.

25. Which factors have an effect on tooth position?

When a tooth is erupting, it is affected by two forces that dictate its vertical position: a force causing eruption brings a tooth to the oral cavity, but a force from the occlusion has an opposing effect. In addition, external forces from the cheeks and lips and internal forces from the tongue play a role in the buccolingual position of a tooth. According to Proffit,[17] forces from the cheeks, lips, and tongue are not in balance; however, periodontally healthy teeth do not move. The balancing factor is probably the periodontal ligament, an active element capable of stabilizing tooth position. On the other hand, if support from alveolar bone and periodontal ligament is reduced, teeth are prone to move.

Light but long-lasting forces (force from the soft tissues at rest, periodontal ligament, and gingival fibers) are more important than heavy but short-lasting forces (biting, swallowing) to cause a tooth to move or to maintain its position.

26. What is the relationship between occlusal development and facial growth?

Eruption of permanent teeth does not stop once a tooth has reached occlusion. Eruption of teeth causes an elongation of dentoalveolar processes that continues at a rate that parallels the rate of vertical growth of the face, and vertical growth of the mandibular ramus in particular. In an optimally growing individual, growth of the anterior and posterior face height is approximately equal. This means that the amount of eruption of the anterior and posterior teeth that have already reached the occlusal contact is in balance. During the period between 8 and 18 years of age, anterior and posterior face heights increase about 20 mm.[18,19] At the same time, each tooth erupts about 10 mm (1.0 mm/yr) to keep contact with its opposing tooth. In some individuals, however, growth of the anterior and posterior face is not in balance, and either anterior or posterior growth rotation of the mandible occurs. This is followed by overeruption of posterior or anterior teeth in posterior rotation pattern versus anterior rotation pattern, respectively.

27. When is occlusal development completed, and can possible continued occlusal development cause adverse effects when teeth are replaced by dental implants?

It has been found that anterior facial height may continue to increase still between ages 25 and 45 years (and probably beyond) in healthy individuals. At the same time overjet and overbite remain the same, indicating continuous eruption of incisors to adapt face height increase.[20] A dental implant, which does not have a periodontal ligament to allow movement, can be compared to an ankylosed tooth. In individuals with post-adolescence changes in the occlusion, a dental implant remains stable while the adjacent teeth erupt, causing a vertical step in the incisal and gingival lines (Fig. 2-7).[21] No reliable methods are available to predict in whom continued occlusal and facial development takes place in clinically significant amounts and causes adverse effects with dental implants. Interestingly, it has been found that dental implants in the upper front area may exhibit major vertical steps in the same amount in persons with early (15½ to 21 years) or late (40 to 55 years) implant placement.[22] Therefore, from the occlusal development point of view, placement of dental implants should be postponed as long as possible. It is advisable to inform the patient of the possibility of adverse infraocclusion due to continued unpredictable occlusal development.

28. Can individuals be found with variations in the number of teeth?

Variation in the number of teeth is a frequent finding in any patient population. Instead of the normal 20 primary teeth and 32 permanent teeth, individuals with excessive or reduced numbers of teeth can be seen. In the permanent dentition, one or two teeth are often congenitally missing. This condition is called *hypodontia* or *agenesis of teeth*. If more than six permanent teeth are missing, the condition is called *oligodontia*. *Anodontia*, which is characterized by complete failure of tooth development, is extremely rare. If supernumerary teeth are present, it is called *hyperdontia*.

29. How common is hypodontia, and which teeth are most often affected?

Based on epidemiological studies worldwide, the prevalence of congenitally missing permanent teeth has been found to vary according to the population studied as well as to gender. Studies from Europe and Australia show the prevalence of hypodontia ranging between 5.5% and 6.3%, whereas in North America (both Caucasians and African-Americans), the prevalence is 3.9%.[23] These numbers exclude the third molars, but when they are included the prevalence is considerably higher, since one or more wisdom teeth are missing in about 20% to 25% of the subjects. On the other hand, prevalence of congenitally missing primary teeth is only 0.1% to 0.4%. The prevalence of hypodontia is significantly higher (1.37 times) in girls than in boys.[23]

Hypodontia commonly runs in families, an indication that genetic factors are involved. Missing teeth can be inherited as part of a syndrome or isolated in an autosomal-dominant or autosomal-recessive way. Several gene defects have been found to be associated with hypodontia. The main genes known today to be involved in hypodontia are MSX1, PAX9, and AXIN2.[1] Individuals who are missing several teeth often have disturbances in other organs of ectodermal origin (e.g., a condition called *ectodermal dysplasia*).

The most commonly missing permanent teeth are the lower second premolars (more than 40% of the missing teeth), followed by the upper laterals and upper second molars. The number of other congenitally missing teeth is considerably

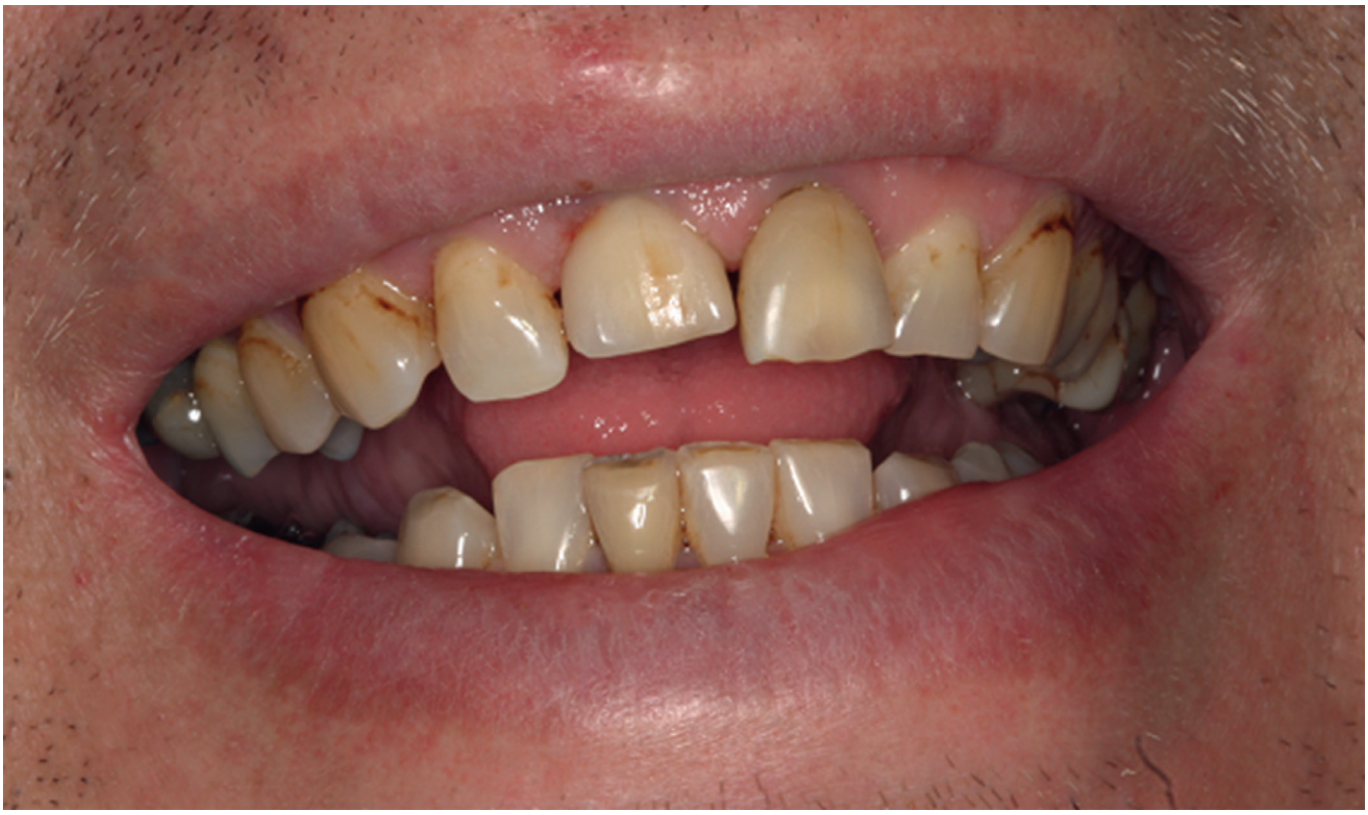

FIG 2-7 Upper right incisor was replaced with an implant at the age of 33 years. Because of continued facial growth and eruption of teeth, the implant (comparable to an ankylosed tooth) became gradually infraoccluded.

lower. As a general rule, the last tooth within its dental group is the one most likely to be congenitally missing. In other words, third molars are more likely to be missing than the first and second molars, second premolars more often than the first ones, and lateral incisors more often than the central incisors.

30. Can hypodontia be associated with other dental anomalies?

Different tooth and eruption anomalies are found together more frequently in some individuals than can be explained by chance alone. Hypodontia, small teeth (peg-shaped upper lateral incisors), delay in tooth formation and eruption, infraocclusion of primary molars, palatal displacement of upper canines, transposition of teeth, and distally displaced unerupted premolars have been found to be associated.[15,24–26] These interrelated anomalies are examples of dental anomaly patterns (DAP).[27] Understanding of DAP calls for a closer look of a patient who only has one missing tooth, for example.

31. How common is hyperdontia?

Prevalence of hyperdontia is lower than that of hypodontia. In the primary dentition, the prevalence of hyperdontia is about 0.5% and in the permanent dentition about 1%. Supernumerary teeth are most often (85%) located in the upper jaw, particularly in the premaxilla area. A supernumerary tooth may be typical or atypical in shape. An atypical supernumerary tooth is often found in the midline of the premaxilla and is called a *mesiodens* (Fig. 2-8). Overall, mesiodens is the most prevalent supernumerary tooth, followed by extra molars and lower second premolars.[28]

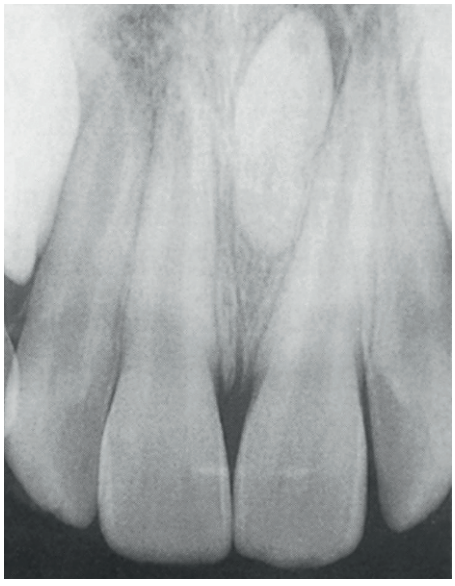

FIG 2-8 Supernumerary teeth are most often located in the upper jaw. A supernumerary tooth is seen in the midline of the premaxilla and is called a *mesiodens*.

32. Does variation in tooth size have an effect on occlusion?

Variation in tooth size is a relatively common finding and may have an effect on occlusion. It is estimated that the prevalence of "tooth size discrepancy" (also called *Bolton discrepancy*[29]) is about 5%.[30] Upper permanent lateral incisors show the largest variation in size. If they are significantly smaller or larger than average, ideal occlusion is difficult to establish. As a general rule, if the mesiodistal dimension of an upper lateral incisor is smaller than that of a lower incisor, normal overjet and overbite are difficult to obtain.

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CHAPTER

3

Appropriate Timing for Correction of Malocclusions

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Proffit[1] states that "in determining the optimal timing for orthodontic treatment, two considerations are important: effectiveness (how well does it work?) and efficiency (what is the cost-benefit ratio?)." Both must be kept in mind when deciding when to treat various orthodontic problems. A child who has a malocclusion that interferes with facial growth, dentitional development, and/or has a negative impact on psychosocial status should have treatment initiated in the primary or mixed dentition. Otherwise, treatment of the malocclusion can be delayed until the child is in the permanent dentition. An understanding of craniofacial growth and development and dentitional development is essential to differentiate the timing of orthodontic treatment for different problems. If treatment is started too early, it is not efficient (high cost-benefit ratio) because of extended treatment time. If treatment is started too late, it may not be effective because the opportunity for modifying skeletal growth may be missed; moreover, it can be more extensive and difficult, requiring a higher incidence of extraction and/or orthognathic surgery. This chapter addresses the appropriate timing for the commonly seen orthodontic problems from primary dentition to permanent dentition.

1. What is early treatment, and at what age is early treatment indicated?

Early treatment (Phase I) can be defined as "orthodontic treatment started in either primary or mixed dentition that is performed to enhance the dental and skeletal development before the eruption of the permanent dentition. Its purpose is to either correct or intercept a malocclusion and to reduce the need or the time for treatment in the permanent dentition."[2] It is typically a short duration (a few months to 1 year) of treatment, and then the child is monitored until the late mixed dentition or early permanent dentition for possible comprehensive orthodontics known as *Phase II treatment*.

Two-phase treatment is not needed for the majority of children who present in the primary or mixed dentition stage of development. It has been reported that about one-third of children are treated with two phases of orthodontic care, whereas the other two-thirds are treated with one-phase treatment (Phase II only) in the late mixed dentition or permanent dentition.[3]

2. What is the appropriate timing for the treatment of an anterior crossbite with a functional shift (pseudo Class III)?

Children who have an anterior crossbite with a functional shift should be treated early due to the negative impact on facial growth and development. The incisors are usually in edge-to-edge bite in centric relation (CR); however, in centric occlusion (CO) the child has to shift the mandible forward into incisal crossbite so that the posterior teeth can occlude. A child could be Class I in CR but a Class III in CO (pseudo Class III). A proper diagnosis and careful documentation of the CR-CO discrepancy is essential, with records that can include clinical measurements, photographs, models, and a lateral headfilm.[4] The treatment can be started as early as 5 to 6 years old in the primary dentition to correct the anterior crossbite and eliminate the functional shift. This correction helps to establish normal function and allows normal growth and development of the maxilla and mandible. Fig. 3-1 shows Patient 1, a 5 yr:5 mo child in primary dentition, with anterior crossbite and functional shift. The patient was treated with a removable appliance with finger springs to push upper incisors labially. The crossbite was corrected in 3 months, and 2 years later significant forward growth of the maxilla was noted (Fig. 3-2). At age 13 before Phase II treatment, a Class I molar and canine relationship was maintained (Fig. 3-3).

3. What is the appropriate timing for treatment of a skeletal Class III malocclusion, and what kind of treatment is involved?

For a skeletal Class III malocclusion, treatment with orthopedic appliances should be started in the early mixed dentition (age 6 to 8) to obtain optimal results.[5] The orthopedic skeletal changes from treatment diminish when the child enters adolescence. However, studies have shown that some skeletal modification can still be accomplished using orthopedic appliances in the early permanent dentition.[6]

A common treatment protocol for a skeletal Class III malocclusion in children would utilize a protraction facemask with a rapid palatal expander (RPE) to advance the maxilla forward. The mandible typically moves downward and backward accompanied by a slight increase in lower facial

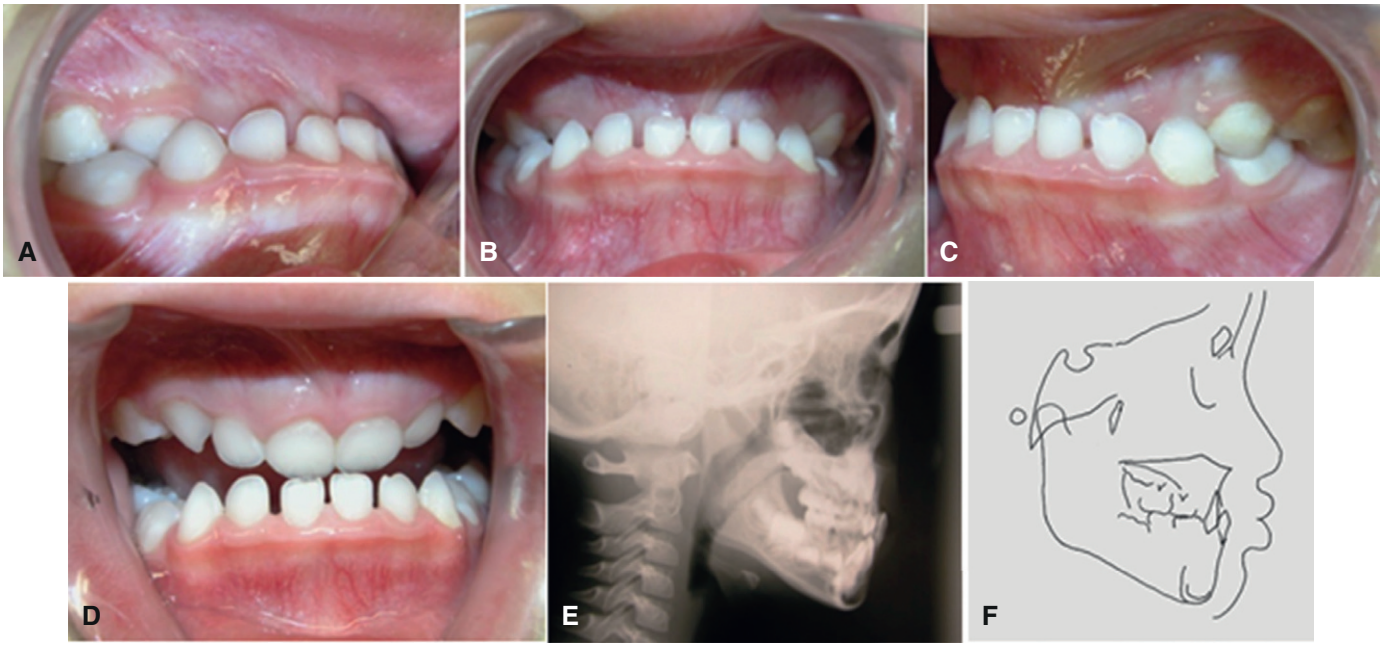

FIG 3-1 A 5 yr:5 mo child in primary dentition presented with anterior crossbite in centric occlusion (CO), retroclined upper incisors, extruded upper and lower incisors, and a deep bite **(A-C)**. An anterior edge-to-edge bite and posterior open bite were noted in centric relation (CR) **(D)**. The CO-CR discrepancy (functional shift) was about 2 mm. A lateral cephalogram was taken in CO **(E)** and cephalometric tracing showed SNA 80°, SNB 81.5°, ANB -1.5°, SN-MP 30° **(F)**.

height.[7–11] Efforts to restrain mandibular growth (i.e., chin-cup) may not be effective long-term because the adolescent mandibular growth spurt is very significant and the skeletal Class III can return.[12] Fig. 3-4 shows Patient 2, a 7-year-old child, with skeletal Class III (Wits: □11 mm). The patient was treated with RPE and facemask, and the results showed maxillary forward movement and significant improvement of skeletal Class III (Wits: □4 mm) (Fig. 3-5). It should be noted that occasionally Class III orthopedic treatment is required more than once for the skeletal Class III cases because of the significant mandibular forward growth tendency throughout adolescence.

4. What is the timing of treatment for a Class II malocclusion, and what kind of treatment is involved?

Recent randomized clinical trials have suggested that skeletal effects of early treatment using headgear or functional appliances at age 9 (Phase I) generally are positively impacted; however, this improvement cannot be sustained over time. They found that by the end of Phase II orthodontic treatment, the differences between those who had received Phase I treatment and those who had not were indistinguishable.[13–19] Thus, they suggested that moderate to severe Class II malocclusions do not benefit more from two-phase treatment than from a conventional one-phase treatment started in the late mixed dentition. However, it should be noted that the stages of tooth eruption do not correlate very well with the stages of skeletal growth. The timing of treatment often must be adjusted because skeletal and dental developments are not synchronized.

Children requiring Class II skeletal correction require treatment with growth modification, which is most successful if started at the beginning of the adolescent growth spurt and ended about the time rapid growth subsides. There is considerable individual variation, but puberty and the adolescent growth spurt occur on average nearly 2 years earlier in females than in males.[19] This has an important impact on the timing of orthodontic treatment, which should be initiated earlier in females than in males to take advantage of the adolescent growth spurt. For girls the growth spurt starts at about age 10½ to 11, and for boys it starts at about age 12½ to 13.[20] Thus, for girls the timing for skeletal Class II correction should be approximately 2 years earlier than for boys. For boys the growth spurt starts usually in the late mixed dentition or early permanent dentition stage; however, for girls it may start 2 years before the permanent dentition stage. If treatment for skeletal modification for a girl starts at age 10 when her growth spurt initiates, a first phase would be needed for about 1 year and then continue with a second phase of treatment.

It should be noted that treatment of Class II malocclusion should typically be delayed until the initiation of the growth spurt, but a Phase I (7 to 9 years old) treatment is indicated if the child has a psychosocial issue due to the malocclusion. Parents should know the later Phase II treatment is very possible and that this two-stage treatment will be more costly and time consuming.

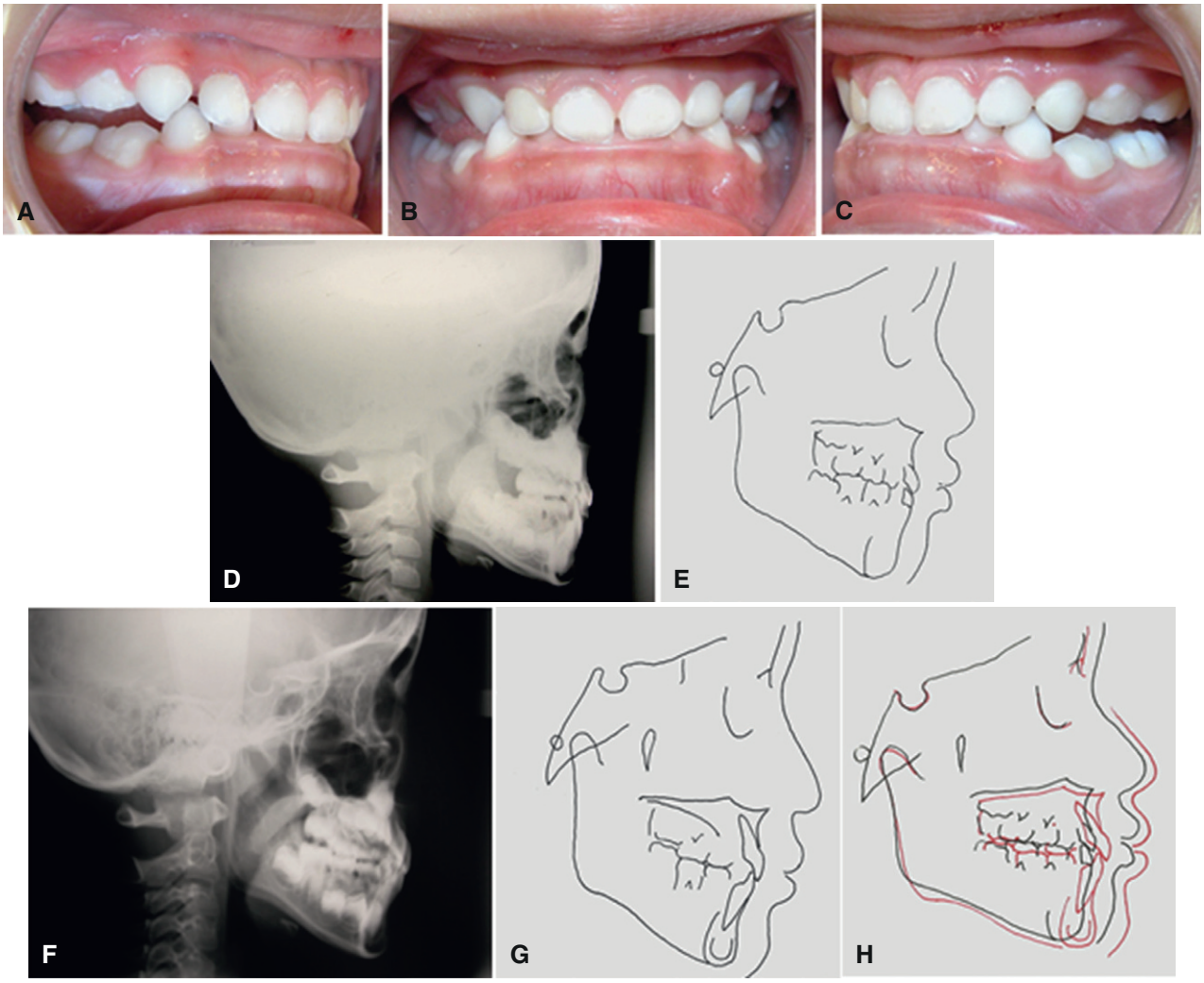

FIG 3-2 Patient 1: Anterior crossbite was corrected in 3 months at age 5 yr:8 mo **(A-C)**. Note the posterior open bite appeared **(B-C)**. A month later at age 5 yr:9 mo, the posterior occlusion was reestablished from eruption of posterior teeth as shown on the lateral cephalogram **(D)**. The tracing showed an SNA 80°, SNB 78.5°, ANB 1.5°, and SN-MP 36° **(E)**. To evaluate the growth, a cephalogram was taken at age 7 yr:7 mo **(F)**, and the cephalometric tracing showed significant forward maxillary growth. The SNA was 82°, SNB 79° (ANB: 3°), and SN-MP 33° **(G)**. Superimposition of ceph tracings is from age 5 yr:11 mo to 7 yr:6 mo **(H)**.

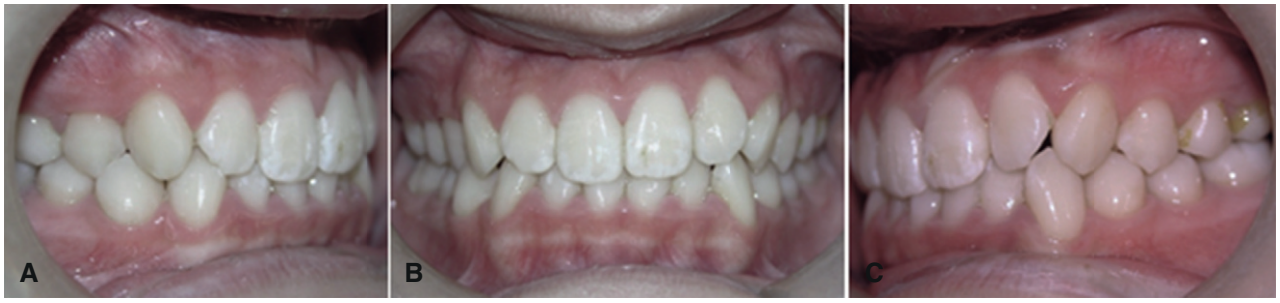

FIG 3-3 Patient 1 at age 13 before Phase II treatment **(A-C)**.