

Guideline on Management of the Developing Dentition and Occlusion in Pediatric Dentistry

Originating Committee

Clinical Affairs Committee – Developing Dentition Subcommittee

Review Council

Council on Clinical Affairs

Adopted

1990

Revised

1991, 1998, 2001, 2005

Purpose

The American Academy of Pediatric Dentistry (AAPD) recognizes the importance of managing the developing dentition and occlusion and its effect on the well-being of infants, children, and adolescents. Management includes the recognition, diagnosis, and appropriate treatment of dentofacial abnormalities. This guideline is intended to set forth objectives for management of the developing dentition and occlusion in pediatric dentistry.

Methods

A MEDLINE literature search was conducted using the terms “ankylosis”, “anterior crossbite”, “Class II malocclusion”, “Class III malocclusion”, “dental crowding”, “ectopic eruption”, “impaction”, “obstruction sleep apnea syndrome (OSAS)”, “occlusal development”, “oligodontia”, “oral habits”, “posterior crossbite”, “space maintenance”, and “tooth size/arch length discrepancy”.

Background

Guidance of eruption and development of the primary, mixed, and permanent dentitions is an integral component of comprehensive oral health care for all pediatric dental patients. Such guidance should contribute to the development of a permanent dentition that is in a stable, functional, and esthetically acceptable occlusion. Early diagnosis and successful treatment of developing malocclusions can have both short-term and long-term benefits while achieving the goals of occlusal harmony and function and dentofacial esthetics.¹⁻⁴ Dentists have the responsibility to recognize, diagnose, and either appropriately manage or refer abnormalities in the developing dentition as dictated by the complexity of the problem and the individual clinician's training, knowledge, and experience.

Many factors can affect the management of the developing dental arches and minimize the overall success of any treatment. The variables associated with the treatment of the developing dentition that will affect the degree to which treatment is successful include, but are not limited to:

1. chronological/mental/emotional age of the patient and the patient's ability to understand and cooperate in the treatment;
2. intensity, frequency, and duration of an oral habit;
3. parental support for the treatment;
4. compliance with clinician's instructions;
5. craniofacial configuration;
6. craniofacial growth;
7. concomitant systemic disease or condition;
8. accuracy of diagnosis;
9. appropriateness of treatment.

A thorough clinical examination, appropriate pretreatment records, differential diagnosis, sequential treatment plan, and progress records are necessary to manage any condition affecting the developing dentition.

Clinical examination should include:

1. facial analysis to:
 - a. identify adverse transverse growth patterns including asymmetries (maxillary and mandibular);
 - b. identify adverse vertical growth patterns;
 - c. identify adverse sagittal (anteroposterior) growth patterns and dental anteroposterior (AP) occlusal disharmonies;
 - d. assess esthetics and identify orthopedic and orthodontic interventions that may improve esthetics and resultant self-image and emotional development.
2. intraoral examination to:
 - a. assess overall oral health status;
 - b. determine the functional status of the patient's occlusion.
3. functional analysis to:
 - a. determine functional factors associated with the malocclusion;
 - b. detect deleterious habits;
 - c. detect temporomandibular joint dysfunction, which may require additional diagnostic procedures.

Diagnostic records may be needed to assist in the evaluation of the patient's condition and for documentation purposes. Prudent judgment is exercised to decide the appropriate records required for diagnosis of the clinical condition.⁵

Diagnostic records may include:

1. extraoral and intraoral photographs to:
 - a. supplement clinical findings with oriented facial and intraoral photographs;
 - b. establish a database for documenting facial changes during treatment.
2. diagnostic dental casts to:
 - a. assess the occlusal relationship;
 - b. determine arch length requirements for intra-arch tooth size relationships;
 - c. determine arch length requirements for interarch tooth size relationships;
 - d. determine location and extent of arch asymmetry.
3. intraoral and panoramic radiographs to:
 - a. establish dental age;
 - b. assess eruption problems;
 - c. estimate the size and presence of unerupted teeth;
 - d. identify dental anomalies/pathology.
4. lateral and AP cephalograms to:
 - a. produce a comprehensive cephalometric analysis of the relative dental and skeletal components in the anteroposterior, vertical, and transverse dimensions;
 - b. establish a baseline growth record for longitudinal assessment of growth and displacement of the jaws.
5. other diagnostic views (eg, magnetic resonance imaging, computed tomographic scans) for hard and soft tissue imaging as indicated by history and clinical examination.

A differential diagnosis and diagnostic summary are completed to:

1. establish the relative contributions of the dental and skeletal structures to the patient's malocclusion;
2. prioritize problems in terms of relative severity;
3. detect favorable and unfavorable interactions that may result from treatment options for each problem area;
4. establish short-term and long-term objectives;
5. summarize the prognosis of treatment for achieving stability, function, esthetics.

A sequential treatment plan will:

1. establish timing priorities for each phase of therapy;
2. establish proper sequence of treatments to achieve short-term and long-term objectives;
3. assess treatment progress and update the biomechanical protocol accordingly on a regular basis.

Stages of development of occlusion

General considerations and principles of management: The stages of occlusal development include:

1. Primary dentition: Beginning in infancy with the eruption of the first tooth, usually about 6 months of age, and complete from approximately 3 to 6 years of age when all primary teeth are erupted.
2. Mixed dentition: From approximately age 6 to 13, primary and permanent teeth are present in the mouth.
3. Adolescent dentition: All primary teeth have exfoliated, second permanent molars may be erupted or erupting, and third molars have not erupted.

4. Adult dentition: All permanent teeth are present and eruptive growth is complete.⁶⁻⁹

These stages may further be divided and referenced as "early" and "late" (ie, early primary, late primary, early mixed, late mixed, etc).⁶⁻⁹

Evaluation and treatment of occlusal and skeletal disharmonies may be initiated at various stages of dental arch development, depending on the:

1. problems; growth, parental involvement, risks and benefits of treatment and of withholding treatment;
2. interest/ability of the practitioner.

Historically, orthodontic treatment was provided mainly for adolescents. During the last 2 decades, increased interest has been expressed in early treatment as well as in adult treatment. Treatment and timing options for the growing patient, especially in the mixed dentition and early permanent dentition, have increased and continue to be evaluated by the research community.^{8,10-12} Many clinicians seek to modify skeletal, muscular, and dentoalveolar abnormalities before the eruption of the full permanent dentition.⁶

A thorough knowledge of craniofacial growth and development of the dentition, as well as orthodontic treatment, must be used in diagnosing and reviewing possible treatment options before recommendations are made to parents.⁸ Early treatment is beneficial for many children but may not be indicated for every patient.

Treatment considerations: The developing dentition should be monitored throughout eruption. This monitoring at regular clinical examinations should include, but not be limited to diagnosis of missing, supernumerary, developmentally defective, and fused or geminated teeth; ectopic eruption; and space and tooth loss secondary to caries.

Radiographic examination, when appropriate¹³ and feasible, should accompany clinical examination. Diagnosis of anomalies of primary or permanent tooth development and eruption should be made to inform the patient's parent and to plan and recommend appropriate intervention. This evaluation is ongoing throughout the developing dentition, at all stages.⁶⁻⁹

1. Primary dentition stage: Anomalies of primary teeth and eruption may not be evident/diagnosable prior to eruption, due to the child's not presenting for dental examination or to a radiographic examination not being possible in a young child. Evaluation, however, should be accomplished when feasible. The objectives of evaluation include:

- a. identification of all anomalies of tooth number and size (as previously noted);
- b. anterior and posterior crossbites;
- c. presence of habits along with their dental and skeletal sequelae.

Radiographs are taken with appropriate clinical indicators or based upon risk assessment/history.

2. Early mixed dentition stage: Palpation for unerupted teeth should be part of every examination. Panoramic, occlusal, and periapical radiographs as indicated, at the time of eruption of the lower incisors and first permanent molars, provide diagnostic information concerning:

- a. anomalies of tooth numbers (eg, missing, supernumerary, fused, geminated);
 - b. tooth size and shape (eg, peg or small lateral incisors);
 - c. positions (eg, ectopic first permanent molars).
- Space analysis can be used to evaluate arch length/crowding at the time of incisor eruption.
3. Mid-to-late mixed dentition stage: Ectopic tooth positions should be diagnosed, especially canines, bicuspid, and second permanent molars.
 4. Adolescent dentition stage: If not instituted earlier, orthodontic diagnosis and treatment should be planned for Class I crowded, Class II, and Class III malocclusions as well as posterior and anterior crossbites. Third molars should be monitored as to position and space, and parents should be informed.
 5. Early adult dentition stage: Third molars should be evaluated. If orthodontic diagnosis has not been accomplished, recommendations should be made, as necessary.

Objectives: At each stage, the objectives of intervention/treatment include: (1) reducing adverse growth; (2) preventing increasing dental and skeletal disharmonies; (3) improving esthetics of the smile; (4) the accompanying positive effects on self-image; and (5) improving the occlusion.

1. Primary dentition stage: Habits and posterior crossbites should be diagnosed and addressed as early as feasible. Parents should be informed about findings of adverse growth and developing malocclusions. Interventions/treatment can be recommended if diagnosis can be made, treatment is appropriate and possible, and parents are supportive and desire to have treatment done.
2. Early mixed stage: Treatment should address: (1) habits; (2) arch length shortage; (3) prevention of crowded incisors; (4) intervention for ectopic molars and incisors; (5) holding of leeway space; (6) crossbites; and (7) adverse skeletal growth. Treatment should take advantage of high rates of growth and prevention of worsened adverse dental and skeletal growth.
3. Mid-to-late mixed dentition stage: Intervention for ectopic teeth may include extractions and space maintenance to aid eruption and reduce the risk of need for surgical bracket placement and orthodontic traction. Intervention for treatment of skeletal disharmonies and crowding may be instituted at this stage.
4. Adolescent dentition stage: In full permanent dentition, final orthodontic diagnosis and treatment can provide the most functional occlusion.
5. Early adult dentition stage: Third molar position or space can be evaluated and, if indicated, the tooth removed. Full orthodontic treatment should be recommended if needed.

Recommendations

Oral habits

General considerations and principles of management: The habits of nonnutritive sucking, bruxing, tongue thrust swallow and abnormal tongue position, self-injurious/self-mutilating behavior, and airway obstruction (OSAS) are discussed in this guideline.

Oral habits may apply forces to the teeth and dentoalveolar structures. The relationship between oral habits and unfavorable dental and facial development is associational rather than cause and effect.¹⁴⁻¹⁶ Habits of sufficient frequency, duration, and intensity may be associated with dentoalveolar or skeletal deformations such as increased overjet, reduced overbite, posterior crossbite, or long facial height. The duration of force is more important than its magnitude; the resting pressure from the lips, cheeks, and tongue has the greatest impact on tooth position, as these forces are maintained most of the time.^{17,18}

Nonnutritive sucking behaviors are considered normal in infants and young children. Prolonged nonnutritive sucking habits have been associated with decreased maxillary arch width, increased overjet, decreased overbite, anterior open bite, and posterior crossbite.^{15,17,18} As preliminary evidence indicates that some changes resulting from sucking habits persist past the cessation of the habit, it has been suggested that early dental visits provide parents with anticipatory guidance to help their children stop sucking habits by age 36 months or younger.^{15,17,18}

Bruxism, defined as the habitual nonfunctional and forceful contact between occlusal surfaces, can occur while awake or asleep. The etiology is multifactorial and has been reported to include central factors (eg, emotional stress,¹⁹ parasomnias,²⁰ traumatic brain injury,²¹ neurologic disabilities²²) and morphologic factors (eg, malocclusion,²³ muscle recruitment²⁴). Reported complications include dental attrition, headaches, temporomandibular dysfunction, and soreness of the masticatory muscles.²⁰ Preliminary evidence suggests that juvenile bruxism is a self-limiting condition that does not progress to adult bruxism.²⁵ The spectrum of bruxism management ranges from patient/parent education, occlusal splints, and psychological techniques to medications.^{21,26,27}

Tongue thrusting, an abnormal tongue position and deviation from the normal swallowing pattern, may be associated with anterior open bite, abnormal speech, and anterior protrusion of the maxillary incisors.¹⁶ There is no evidence that intermittent short-duration pressures, created when the tongue and lips contact the teeth during swallowing or chewing, have significant impact on tooth position.^{16,17} If the resting tongue posture is forward of the normal position, incisor displacement is likely, but if resting tongue posture is normal, a tongue thrust swallow has no clinical significance.¹⁷

Self-injurious or self-mutilating behavior (ie, repetitive acts that result in physical damage to the individual) is extremely rare in the normal child.²⁸ Such behavior, however, has been associated with mental retardation, psychiatric disorders, developmental disabilities, and some syndromes.²⁹ The spectrum of treatment options for developmentally disabled individuals includes pharmacologic management, behavior modification, and physical restraint.³⁰ Reported dental treatment modalities include, among others, lip-bumper and occlusal bite appliances, protective padding, and extractions.²⁸ Some habits, such as lip-licking and lip-pulling, are relatively benign in relation to an effect on the dentition.²⁸ More severe lip- and tongue-biting habits may be associated with profound neurodisability due to severe brain damage.³⁰ Management options include monitoring the lesion, odontoplasty, providing a bite-opening appliance, or extracting the teeth.³⁰

Research on the relationship between malocclusion and mouth breathing suggests that impaired nasal respiration may contribute to the development of increased facial height, anterior open bite, increased overjet, and narrow palate, but it is not the sole or even the major cause of these conditions.³¹

OSAS may be associated with narrow maxilla, crossbite, low tongue position, vertical growth, and open bite. History associated with OSAS may include snoring, observed apnea, restless sleep, daytime neurobehavioral abnormalities or sleepiness, and bedwetting. Physical findings may include growth abnormalities, signs of nasal obstruction, adenoidal facies, and/or enlarged tonsils.³¹⁻³³

The identification of an abnormal habit and the assessment of its potential immediate and long-term effects on the craniofacial complex and dentition should be made as early as possible. The dentist should evaluate habit frequency, duration, and intensity in all patients with habits. Intervention to terminate the habit should be initiated if indicated.¹⁶

Patients and their parents should be provided with information regarding consequences of a habit. Parents have a role in the correction of an oral habit as nagging or punishment may result in an increase in habit behaviors; change in the home environment may be necessary before a habit can be overcome.¹⁵

Treatment considerations: Management of an oral habit is indicated whenever the habit is associated with unfavorable dentofacial development or adverse effects on child health or when there is a reasonable indication that the oral habit will result in unfavorable sequelae in the developing permanent dentition. Any treatment must be appropriate for the child's development, comprehension, and ability to cooperate. Habit treatment modalities include patient/parent counseling, behavior modification techniques, myofunctional therapy, appliance therapy, or referral to other providers—including, but not limited to, orthodontists, psychologists, myofunctional therapists, or otolaryngologists. Use of an appliance to manage oral habits is indicated only when the child wants to stop the habit and would benefit from a reminder.¹⁶

Objectives: Treatment is directed toward decreasing or eliminating the habit and minimizing potential deleterious effects on the dentofacial complex.

Disturbances in number *Congenitally missing teeth*

General considerations and principles of management: Hypodontia, the congenital absence of 1 or more permanent teeth, has a prevalence of 3.5% to 6.5%.^{34,35} Excluding third molars, the most frequently missing permanent tooth is the mandibular second premolar followed by the maxillary lateral incisor.^{36,37} In the primary dentition, hypodontia occurs less (0.1% to 0.9% prevalence) and almost always affects the maxillary incisors and first primary molars.³⁸ The chance of familial occurrence of 1 or 2 congenitally missing teeth is to be differentiated from missing lateral incisors in cleft lip/palate³⁹ and multiple missing teeth (6 or more) due to ectodermal dysplasia or other syndromes⁴⁰ as the treatment usually differs. A congenitally missing tooth should be suspected in patients with cleft lip/palate, certain syndromes, and a familial pattern of missing teeth. In addition, patients with asymmetric eruption sequence or ankylosis of a primary mandibular second molar may have a congenitally missing tooth.

Treatment considerations: With congenitally missing permanent maxillary incisor(s) or mandibular second premolar(s), the decision to extract the primary tooth and close the space orthodontically vs opening the space orthodontically and placing a prosthesis or implant depends on many factors. For maxillary laterals, the dentist must move the maxillary canine mesially and use the canine as a lateral incisor or create space for a future lateral prosthesis or implant.^{16,40}

Factors that influence the decision are: (1) patient age; (2) canine shape; (3) canine position; (4) child's occlusion and amount of crowding; (5) bite depth; and (6) quality and quantity of bone in the edentulous area.^{41,42} Early extraction of the primary canine and/or lateral may be needed.^{41,42} Opening space for a prosthesis or implant requires less tooth movement, but the space needs to be maintained with an interim prosthesis, especially if an implant is planned.^{40,41} Moving the canine into the lateral position produces little facial change, but the resultant tooth size discrepancy often does not allow a canine guided occlusion.^{41,43}

For a congenitally missing premolar, the primary molar either may be maintained or extracted with subsequent placement of a prosthesis or by orthodontically closing the space. Maintaining the primary second molar may cause occlusal problems due to its larger mesiodistal diameter, compared to the second premolar. Reducing the width of the second primary molar is a consideration, but root resorption and subsequent exfoliation may occur.¹⁶ In crowded arches or with multiple missing premolars, extraction of the primary molar(s) can be considered, especially in mild Class III cases.¹⁶ For a single missing premolar, if maintaining the primary molar is not possible, placement

of a prosthesis or implant should be considered.^{16,40} Consultation with an orthodontist and/or prosthodontist may be required. In addition, preserving the primary tooth may be indicated in certain cases.

Objectives: Treatment is directed toward an esthetically pleasing occlusion that functions well for the patient.

*Supernumerary teeth
(primary, permanent, and mesiodens)*

General considerations and principles of management: Supernumerary teeth, or hyperdontia, can occur in the primary or permanent dentition but are 5 times more common in the permanent.³⁵ Prevalence is reported in the primary and mixed dentitions from 0.52% to 2%.^{35,37,45} Between 80% and 90% of all supernumeraries occur in the maxilla, with half in the anterior area and almost all in the palatal position.^{35,46} A supernumerary primary tooth is followed by a supernumerary permanent tooth in one third of the cases.⁴⁷

During the early mixed dentition, 79% to 91% of anterior permanent supernumerary teeth are unerupted.^{45,48} While more erupt with age, only 25% of all mesiodens (a permanent supernumerary incisor located at the midline) erupt spontaneously.⁴⁶ Mesiodens can prevent or cause ectopic eruption of the central incisor. Less frequently, a mesiodens can cause dilaceration or resorption of the permanent incisor's root. Dentigerous cyst formation involving the mesiodens, in addition to eruption into the nasal cavity, has been reported.^{46,49} If there is an asymmetric eruption pattern of the maxillary incisors, delayed eruption, an overretained primary incisor, or ectopic eruption of an incisor, a supernumerary can be suspected.^{35,44} Panoramic, occlusal, and periapical radiographs all can reveal a supernumerary, but the best way to locate the supernumerary is 2 periapical or occlusal films reviewed by the parallax rule.⁴⁶

Treatment considerations: Management and treatment of hyperdontia differs if the tooth is primary or permanent. Primary supernumerary teeth normally are accommodated into the arch and usually erupt and exfoliate without complications.⁴⁷ Extraction of an unerupted supernumerary tooth during the primary dentition usually is not done to allow it to erupt; surgical extraction of unerupted supernumerary teeth can displace or damage the permanent incisor.⁴⁶ Removal of a mesiodens or other permanent supernumerary incisor results in eruption of the permanent adjacent normal incisor in 75% of the cases.⁵⁰ Extraction of an unerupted supernumerary during the early mixed dentition allows for a normal eruptive force and eruption of the permanent adjacent normal incisor.^{51,52} Later removal of the mesiodens reduces the likelihood that the adjacent normal permanent incisor will erupt on its own, especially if the apex is completed.⁴⁶ Inverted conical supernumeraries can be harder to remove if removal is delayed, as they can migrate deeper into the jaw.⁴⁴ After removal of the supernumerary, clinical and radiographic follow-up is indicated in 6 months to determine if the normal incisor is erupting. If there is no eruption after 6 to 12 months and sufficient space exists, surgical exposure and orthodontic extrusion is needed.⁴⁶

Objectives: Removal of supernumerary teeth should facilitate eruption of permanent teeth and encourage normal alignment. In cases where normal alignment or spontaneous eruption does not occur, further orthodontic treatment is indicated.

*Localized disturbances in eruption
Ectopic eruption*

General considerations and principles of management: Ectopic eruption (EE) of permanent first molars occurs due to the molar's abnormal mesioangular eruption path, resulting in an impaction at the distal prominence of the primary second molar's crown. EE can be suspected if asymmetric eruption is observed or if the mesial marginal ridge is noted to be under the distal prominence of the second primary molar. EE of permanent molars can be diagnosed from bitewing or panoramic radiographs in the early mixed dentition. This condition occurs in up to 0.75% of the population,⁵³ but is more common in children with cleft lip and palate.⁵⁴ The maxillary canine appears in an impacted position in 1.5% to 2% of the population,^{55,56} while maxillary incisors can erupt ectopically or be impacted from supernumerary teeth in up to 2% of the population.⁴⁶ Incisors also can have altered eruption due to pulp necrosis (following trauma or caries) or pulpal treatment of the primary incisor.⁵⁷

EE of permanent molars is classified into 2 types. There are those that self correct or "jump" and others that remain impacted. In 66% of the cases, the molar jumps.⁵⁸ A permanent molar that presents with part of its occlusal surface clinically visible and part under the distal of the primary second molar normally does not jump and is the impacted type.⁵⁹ Nontreatment can result in early loss of the primary second molar and space loss.

Maxillary canine impaction should be suspected when the canine bulge is not palpable or when asymmetric canine eruption is evident. Panoramic radiographs would show the canine has an abnormal inclination and/or overlaps the lateral incisor root. EE of permanent incisors can be suspected after trauma to primary incisors, with pulpally treated primary incisors or asymmetric eruption, or if a supernumerary incisor is diagnosed.

Treatment considerations: Treatment depends on how severe the impaction appears clinically and radiographically. For mildly impacted first permanent molars, where little of the tooth is impacted under the primary second molar, elastic or metal orthodontic separators can be placed to wedge the permanent first molar distally.¹⁶ For more severe impactions, distal tipping of the permanent molar is required. Tipping action can be accomplished with brass wires, removable appliances using springs, fixed appliances such as sectional wires with open coil springs, sling shot-type appliances,⁵⁹ a Halterman appliance,⁶⁰ or surgical uprighting.⁶¹

Early diagnosis and treatment of impacted maxillary canines can lessen the severity of the impaction and may stimulate eruption of the canine. Extraction of the primary canine is indicated when the canine bulge cannot be palpated

in the alveolar process and there is radiographic overlapping of the canine with the formed root of the lateral during the mixed dentition.⁶² Even if the impacted canine is diagnosed at a later age (11 to 16), if the canine is not horizontal, extraction of the primary canine lessens the severity of the permanent canine impaction and 75% will erupt.⁶³ Extraction of the first primary molar also has been reported to allow eruption of first bicuspid and to assist in the eruption of the cuspids. This need can be determined from a panoramic radiograph.^{56,64} Bonded orthodontic treatment normally is required to create space or align the canine. Long-term periodontal health of impacted canines after orthodontic treatment is similar to nonimpacted canines.⁶⁵

Treatment of ectopically erupting incisors depends on the etiology. Extraction of necrotic or over-retained pulpally-treated primary incisors is indicated in the early mixed dentition.⁵⁷ Removal of supernumerary incisors in the early mixed dentition will lessen ectopic eruption of an adjacent permanent incisor.⁴⁶ After incisor eruption, orthodontic treatment involving removable or banded therapy may be needed. **Objectives:** Management of ectopically erupting molars, canines, and incisors should result in improved eruptive positioning of the tooth. In cases where normal alignment does not occur, subsequent comprehensive orthodontic treatment may be necessary to achieve appropriate arch form and intercuspation.

Ankylosis

General considerations and principles of management: Ankylosis is a condition in which the cementum of a tooth's root fuses directly to the surrounding bone. The periodontal ligament is replaced with osseous tissue, rendering the tooth immobile to eruptive change. Ankylosis can occur in the primary and permanent dentitions, with the most common incidence involving primary molars. The incidence is reported to be between 7% and 14% in the primary dentition.⁴⁸ In the permanent dentition, ankylosis occurs most frequently following luxation injuries.⁶⁶

Ankylosis is common in anterior teeth following trauma and is referred to as replacement resorption. Periodontal ligament cells are destroyed and the cells of the alveolar bone perform most of the healing. Over time, normal bony activity results in the replacement of root structure with osseous tissue.⁶⁷ Ankylosis can occur rapidly or gradually over time, in some cases as long as 5 years posttrauma. It also may be transient if only a small bony bridge forms that can be resorbed with subsequent osteoclastic activity.⁶⁸

Ankylosis can be verified by clinical and radiographic means. Submergence of the tooth is the primary recognizable sign, but the diagnosis also can be made through percussion and palpation. Radiographic examination also may reveal the loss of the periodontal ligament and bony bridging.

Treatment considerations: With ankylosis of a primary molar, exfoliation usually occurs normally. Extraction is recommended if prolonged retention of the primary molar is noted. If a severe marginal ridge discrepancy develops, extraction should be considered to prevent the adjacent teeth

from tipping and producing space loss.³ Replacement resorption of permanent teeth usually results in the loss of the involved tooth.⁶⁶

Mildly to moderately ankylosed primary molars without permanent successors may be retained and restored to function in arches without crowding. Extraction of these molars can assist in resolving crowded arches in complex orthodontic cases.^{64,69} Surgical luxation of ankylosed permanent teeth with forced eruption has been described as an alternative to premature extraction.⁷⁰

Objectives: Treatment of ankylosis should result in the continuing normal development of the permanent dentition. Or, in the case of replacement resorption of a permanent tooth, appropriate prosthetic replacement should be planned.

Tooth size/arch length discrepancy and crowding

General considerations and principles of management: Arch length discrepancies include inadequate arch length and crowding of the dental arches, excess arch length and spacing, and tooth size discrepancy, often referred to as a Bolton discrepancy.⁷¹ These arch length discrepancies may be found in conjunction with complicating and other etiological factors including missing teeth, supernumerary teeth, and fused or geminated teeth. Inadequate arch length and resulting incisor crowding is a common occurrence with various negative sequelae, and is particularly common in the early mixed dentition.⁷²⁻⁷⁵ Studies of arch length in today's children compared to their parents and grandparents of 50 years ago indicate less arch length, more frequent incisor crowding, and stable tooth sizes.⁷⁶⁻⁸⁰ This implies that the problem of incisor crowding and ultimate arch length discrepancies may be increasing in numbers of patients and in amount of arch length shortage.^{76,77}

Arch length and especially crowding must be considered in the context of the esthetic, dental, skeletal, and soft tissue relationships. Mandibular incisors have a high relapse rate in rotations and crowding.^{72,74} Growth of the aging skeleton causes further crowding and incisor rotations.⁸¹ Functional contacts are diminished where rotations of incisors, canines, and bicuspid exist.⁸² Occlusal harmony and temporomandibular joint health are impacted negatively by less functional contacts.⁸²

Initial assessment may be done in early mixed dentition, when mandibular incisors begin to erupt.⁷² Evaluation of available space and consideration of making space for permanent incisors to erupt may be done initially utilizing appropriate radiographs to ascertain the presence of permanent successors. Comprehensive diagnostic analysis is suggested, with evaluation of maxillary and mandibular skeletal relationships, direction and pattern of growth, facial profile, facial width, muscle balance, and dental and occlusal findings including tooth positions, arch length analysis, and leeway space.

Derotation of teeth just after emergence in the mouth implies correction before the transseptal fiber arrangement has been established.^{72,82} It has been shown that the transseptal fibers do not develop until the cemento-enamel junction of erupting teeth pass the bony border of the alveolar process.⁸² Long-term stability of aligned incisors may be increased.⁸³

Treatment considerations: Treatment considerations may include, but are not limited to:

1. making space for permanent incisors to erupt and become straight naturally through primary canine extraction and space/arch length maintenance;
2. orthodontic alignment of permanent teeth as soon as erupted and feasible, expansion and correction of arch length as early as feasible;
3. utilizing holding arches in the mixed dentition until all permanent bicuspid and canines have erupted;
4. extractions of permanent teeth;
5. maintaining patient's original arch form.⁸²

Other treatment modalities may include, but are not limited to: (1) interproximal reduction; (2) restorative bonding; (3) veneers; (4) crowns; (5) implants; and (6) orthognathic surgery.

Objectives: Well-timed intervention can:

1. prevent crowded incisors;
2. increase long-term stability of incisor positions;
3. decrease ectopic eruption and impaction of permanent canines;
4. reduce orthodontic treatment time and sequelae;
5. improve gingival health and overall dental health.^{72,84,85}

Space maintenance

General considerations and principles of management: The premature loss of primary teeth due to caries, trauma, ectopic eruption, or other causes may lead to undesirable tooth movements of primary and/or permanent teeth including loss of arch length. Arch length deficiency can produce or increase the severity of malocclusions with crowding, rotations, ectopic eruption, crossbite, excessive overjet, excessive overbite, and unfavorable molar relationships.⁸⁶ The dental profession has recommended the use of space maintainers to reduce the prevalence and severity of malocclusion following premature loss of primary teeth.^{16,87,88} Space maintenance may be a consideration in the primary dentition after early loss of a maxillary incisor when the child has an active digit habit. An intense habit may reduce the space for the erupting permanent incisor.

Adverse effects associated with space maintainers include: (1) dislodged, broken, and lost appliances; (2) plaque accumulation; (3) caries; (4) interference with successor eruption; (5) undesirable tooth movement; (6) inhibition of alveolar growth; (7) soft tissue impingement; and (8) pain.^{86,89-91} Premature loss of a primary tooth of any type has the potential to cause loss of space available for the succeeding permanent tooth, but there is a lack of consensus regarding the effectiveness of space maintainers in preventing or reducing the severity of malocclusion.^{86,92-94}

Treatment considerations: It is prudent to consider space maintenance when primary teeth are lost prematurely. Factors to consider include: (1) specific tooth lost; (2) time elapsed since tooth loss; (3) pre-existing occlusion; (4) favorable space analysis; (5) presence and root development of permanent successor; (6) amount of alveolar bone covering permanent successor; (7) patient's health status; (8)

cooperative ability; (9) active oral habits; and (10) oral hygiene.^{16,86} If a space analysis is required prior to the placement of a space maintainer, appropriate radiographs and study models should be considered.⁹⁵

The literature pertaining to the use of space maintainers specific to the loss of a particular primary tooth type includes: expert opinion, case reports, and details of appliance design.^{16,87,88} Treatment modalities may include, but are not limited to:

1. fixed appliances (eg, band and loop, crown and loop, passive lingual arch, distal shoe, Nance appliance, transpalatal arch);
2. removable appliances (eg, partial dentures, Hawley appliance).^{16,87,88}

The placement and retention of space maintaining appliances requires ongoing compliant patient behavior. Follow-up of patients with space maintainers is necessary to assess integrity of cement and to evaluate and clean the abutment teeth.⁹⁰ The appliance should function until the succedaneous teeth have erupted into the arch.

Objectives: The goal of space maintenance is to prevent loss of arch length, width, and perimeter by maintaining the relative position of the existing dentition.^{16,87}

The AAPD supports controlled randomized clinical trials to determine efficacy of space maintainers as well as analysis of costs and side effects of treatment.

Space regaining

General considerations and principles of management: Some of the more common causes of space loss within an arch are: (1) primary teeth with interproximal caries; (2) ectopically erupting teeth; (3) alteration in the sequence of eruption; (4) ankylosis of a primary molar; (5) dental impaction; (6) transposition of teeth; (7) loss of primary molars without proper space management; (8) congenitally missing teeth; (9) abnormal resorption of primary molar roots; (10) premature and delayed eruption of permanent teeth; and (11) abnormal dental morphology.^{16,86,96} Loss of space in the dental arch that interferes with the desired eruption of the permanent teeth may require evaluation.

Space loss may occur unilaterally or bilaterally and may result from teeth tipping, rotating, extruding, being ankylosed, or translating or from extrusion of teeth and the deepening of the curve of Spee.⁹⁷

The degree to which space is affected varies according to the: arch affected, site in the arch, and time elapsed since tooth loss. The quantity and incidence of space loss also are dependent upon which adjacent teeth are present in the dental arch and their status.^{16,86} The amount of crowding or spacing in the dental arch will determine the degree to which space loss has a significant consequence.^{97,98}

Treatment considerations: Treatment modalities may include, but are not limited to, fixed appliances or removable appliances (eg, Hawley appliance, lip bumper, headgear). Space loss and dentofacial skeletal development may dictate that space regaining not be indicated. This should be

determined as the result of a comprehensive analysis. The timing of clinical intervention subsequent to premature loss of a primary molar is critical.⁹⁸

Objectives: The goal of space regaining intervention is the recovery of lost arch width and perimeter and/or improved eruptive position of succedaneous teeth. Space regained should be maintained until adjacent permanent teeth have erupted completely and/or until a subsequent comprehensive orthodontic treatment plan is initiated.

Crossbites (dental, functional, and skeletal)

General considerations and principles of management:

Anterior and posterior crossbites are malocclusions which involve one or more teeth in which the maxillary teeth occlude lingually with the mandibular antagonistic teeth.^{99,100} If the midlines undergo a compensatory or habitual shift when the teeth occlude in crossbite, this is termed a functional shift.¹⁰¹ A crossbite can be of dental or skeletal origin or a combination of both.¹⁰²

A simple anterior crossbite is of dental origin if the molar occlusion is Class I and the malocclusion is the result of an abnormal axial inclination of maxillary anterior teeth. This condition should be differentiated from a Class III skeletal malocclusion where the crossbite is the result of the basal bone position.⁹⁹ Dental crossbites result from the tipping or rotation of a tooth or teeth. The condition is localized and does not involve the basal bone. Skeletal crossbites involve disharmony of the craniofacial skeleton. Aberrations in bony growth may give rise to crossbites in 2 ways:

1. adverse transverse growth of the maxilla and mandible;
2. disharmonious or adverse growth in the sagittal (AP) length of the maxilla and mandible.^{99,101}

Such growth aberrations can be due to inherited growth patterns, trauma, or functional disturbances that alter normal growth.

Treatment considerations: Crossbites should be considered in the context of the patient's total treatment needs. Anterior crossbite correction can: (1) reduce dental attrition; (2) improve dental esthetics; (3) redirect skeletal growth; (4) improve the tooth-to-alveolus relationship; and (5) increase arch perimeter. A simple anterior crossbite can be aligned as soon as the condition is noted, if there is sufficient space; otherwise, space needs to be created first. Such appliances as acrylic incline planes, acrylic retainers with lingual springs, or fixed appliances all have been effective. If space is needed, an expansion appliance also is required.⁹⁹ Posterior crossbite correction can accomplish the same objectives and can improve the eruptive position of the succedaneous teeth. Early correction of unilateral posterior crossbites has been shown significantly to improve functional conditions and largely eliminate morphological and positional asymmetries of the mandible.^{103,104} Functional shifts should be eliminated as soon as possible with early correction¹⁰¹ to avoid asymmetric growth. Treatment can be completed with:

1. equilibration;
2. appliance therapy (fixed or removable);
3. extractions; or

4. a combination of these treatment modalities to correct the palatal constriction.

Fixed or removable palatal expanders can be utilized until midline suture fusion occurs.^{100,101} Treatment decisions depend on the:

1. amount and type of movement (tipping vs bodily movement, rotation, or dental vs orthopedic movement);
2. space available;
3. AP, transverse, and vertical skeletal relationships;
4. growth status;
5. patient cooperation.

Patients with crossbites and concomitant Class III skeletal patterns and/or skeletal asymmetry should receive comprehensive treatment as covered in the Class III malocclusion section.

Objectives: Treatment of a crossbite should result in improved intramaxillary alignment and an acceptable interarch occlusion and function.¹⁰³

Class II malocclusion

General considerations and principles of management:

Class II malocclusion (distocclusion) may be unilateral or bilateral and involves a distal relationship of the mandible to the maxilla or the mandibular teeth to maxillary teeth. This relationship may result from: (1) dental (malposition of the teeth in the arches); (2) skeletal (mandibular retrusion and/or maxillary protrusion); or (3) a combination of dental and skeletal factors.¹⁰⁵

Results of randomized clinical trials indicate that Class II malocclusion can be corrected effectively with either a single or 2-phase regimen.¹⁰⁶⁻¹⁰⁹ Growth modifying effects in some studies did not show an influence on the Class II skeletal pattern,^{108,110,111} while other studies dispute these findings.^{112,113} There is substantial variation in treatment response to growth modification treatments (headgear or functional appliance) and no reliable predictors for favorable growth response have been found.^{106,112} Some reports state early treatment does not reduce the need for either premolar extractions or orthognathic surgery^{107,108} while others disagree with these findings.¹¹⁴ Two-phase treatment results in significantly longer treatment time.^{101,107,115}

Clinicians may decide to provide early treatment based on other factors.^{107,111} Preliminary evidence suggests that, for some children, early Class II treatment improves self-esteem and decreases negative social experiences.¹¹⁶ Incisor injury that is more severe than simple enamel fractures has been associated positively with increased overjet and prognathic position of the maxilla.¹¹⁷ Some studies indicate early treatment for Class II malocclusions can be initiated, depending upon patient cooperation and management.¹¹⁸

Treatment considerations: Factors to consider when planning orthodontic intervention for Class II malocclusion are: (1) facial growth pattern; (2) amount of anterior-posterior discrepancy; (3) patient age; (4) projected patient compliance; (5) space analysis; (6) anchorage requirements; and (7) patient and parent desires. Treatment modalities include:

(1) extraoral appliances (headgear); (2) functional appliances; (3) fixed appliances; (4) tooth extraction and interarch elastics; and (5) orthodontics with orthognathic surgery.¹⁰²

Objectives: Treatment of a developing Class II malocclusion should result in an improved overbite, overjet, and intercuspatation of posterior teeth and an esthetic appearance and profile compatible with the patient's skeletal morphology.

Class III malocclusion

General considerations and principles of management: Class III malocclusion (mesocclusion) may be unilateral or bilateral and involves a mesial relationship of the mandible to the maxilla or mandibular teeth to maxillary teeth. This relationship may result from dental factors (malposition of the teeth in the arches), skeletal factors (asymmetry, mandibular prognathism, and/or maxillary retrognathism), or a combination of these factors.¹¹⁹

The etiology of Class III malocclusions can be hereditary, environmental, or both. In a study of 320 orthodontic patients in 155 sibships, the hereditary effect on molar relationship was determined to be 56%.¹²⁰ Hereditary factors include clefts of the alveolus and palate and other craniofacial anomalies that are part of a genetic syndrome. Some environmental factors are trauma, oral/digital habits, caries, and early childhood OSAS.¹²¹

Treatment considerations: Treatment of Class III malocclusions is indicated to provide psychosocial benefits for the child patient by reducing or eliminating facial disfigurement and to reduce the severity of malocclusion by promoting harmonious growth. Early Class III treatment has been proposed for several years and has been advocated as a necessary tool in contemporary orthodontics.¹²²⁻¹²⁵ Factors to consider when planning orthodontic intervention for Class III malocclusion are: (1) facial growth pattern; (2) amount of AP discrepancy; (3) patient age; (4) projected patient compliance; (5) space analysis; (6) anchorage (headgear); (7) functional appliances; (8) fixed appliances; (9) tooth extraction; (10) interarch elastics; and (11) orthodontics with orthognathic surgery.¹²⁶

Objectives: Early Class III treatment may provide a more favorable environment for growth and may improve occlusion, function, and esthetics. Although early treatment can minimize the malocclusion and potentially eliminate future orthognathic surgery, this is not always possible. Typically, Class III patients tend to grow longer and more unpredictably and, therefore, surgery combined with orthodontics is the best alternative to achieve a satisfactory result for some patients.¹⁰²

Treatment of a developing Class III malocclusion should result in improved overbite, overjet, and intercuspatation of posterior teeth and an esthetic appearance and profile compatible with the patient's skeletal morphology.

References

1. Kanellis MJ. Orthodontic Treatment in the Primary Dentition. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa: WB Saunders Co; 2001:248-256.
2. Woodside DG. The significance of late developmental crowding to early treatment planning for incisor crowding. *Am J Orthod Dentofacial Orthop* 2000; 117:559-561.
3. Kurol J. Early treatment of tooth-eruption disturbances. *Am J Orthod Dentofacial Orthop* 2002;121:588-591.
4. Sankey WL, Buschang PH, English J, Owen AH III. Early treatment of vertical skeletal dysplasia: The hyperdivergent phenotype. *Am J Orthod Dentofacial Orthop* 2000;118:317-327.
5. Proffit WR, Ackerman JL. The development of a problem list. In: Proffit WR, Fields HW Jr, eds. *Contemporary Orthodontics*. 3rd ed. St. Louis, Mo: Mosby; 2000:133-156.
6. Graber TM, Vanarsdall RL. Orthodontics: Current Principles and Techniques. 3rd ed. St. Louis, Mo: Mosby; 2000:399-411.
7. Ferguson DJ. Growth of the face and dental arches. In: McDonald RE, Avery DR, Dean JA, eds. *Dentistry for the Child and Adolescent*. 8th ed. St. Louis, Mo: Mosby; 2004:590-596.
8. McNamara JA, Brudon WL. Dentitional development. In: *Orthodontics and Dentofacial Orthopedics*. Ann Arbor, Mich: Needham Press, Inc; 2001:31-38.
9. Proffit WR, Fields HW. The later stages of development. In: *Contemporary Orthodontics*. 3rd ed. St. Louis, Mo: Mosby; 2000:75-93.
10. Hunt O, Hepper P, Johnston C, Stevenson M, Burden D. Professional perceptions of the benefits of orthodontic treatment. *Eur J Orthod* 2001;23:315-323.
11. International Symposium on Early Orthodontic Treatment. *Am J Orthod Dentofacial Orthop* 2002;121: 552-595.
12. Ackerman M. Evidenced-based orthodontics for the 21st century. *J Am Dent Assoc* 2004;135:162-167.
13. American Dental Association, US Dept of Health and Human Services. The selection of patients for dental radiographic examinations—2004. Available at: <http://www.ada.org/prof/resources/topics/radiography.asp>. Accessed February 15, 2005.
14. Proffit WR, Fields HW Jr. The etiology and development of orthodontic problems. In: *Contemporary Orthodontics*. 3rd ed. St. Louis, Mo: Mosby; 2000:109-114.
15. Warren JJ, Bishara SE, Steinbock KL, Yonezu T, Nowak AJ. Effects of oral habits' duration on dental characteristics in the primary dentition. *J Am Dent Assoc* 2001;132:1685-1693.
16. Dean JA. Management of the developing occlusion. In: Dean JA, McDonald RE, Avery DA, eds. *Dentistry for the Child and Adolescent*. 8th ed. St. Louis, Mo: Mosby; 2004:631-668.

17. Ogaard B, Larsson E, Lindsten R. The effect of sucking habits, cohort, sex, intercanine arch widths, and breast or bottle feeding on posterior crossbite in Norwegian and Swedish 3-year-old children. *Am J Orthod Dentofacial Orthop* 1994;106:161-166.
18. Warren JJ, Bishara SE. Duration of nutritive and nonnutritive sucking behaviors and their effects on the dental arches in the primary dentition. *Am J Orthod Dentofacial Orthop* 2002;121:347-356.
19. Monaco A, Ciammella NM, Marci MC, Pirro P, Giannoni M. The anxiety in bruxer child: A case-control study. *Minerva Stomatol* 2002;51:247-250.
20. Weideman CL, Bush DL, Yan-Go FL, Clark GT, Gornbein JA. The incidence of parasomnias in child bruxers vs nonbruxers. *Pediatr Dent* 1996;18:456-460.
21. Ivanhoe CB, Lai JM, Francisco GE. Bruxism after brain injury: Successful treatment with botulinum toxin-A. *Arch Phys Med Rehabil* 1997;78:1272-1273.
22. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. *Adv Neurol* 1988;49:329-341.
23. Sari S, Sonmez H. The relationship between occlusal factors and bruxism in permanent and mixed dentition in Turkish children. *J Clin Pediatr Dent* 2001;25:191-194.
24. Negoro T, Briggs J, Plesh O, Nielsen I, McNeill C, Miller AJ. Bruxing patterns in children compared to intercuspal clenching and chewing as assessed with dental models, electromyography, and incisor jaw tracing: Preliminary study. *J Dent Child* 1998;65:449-458.
25. Kiesser JA, Groeneveld HT. Relationship between juvenile bruxing and craniomandibular dysfunction. *J Oral Rehabil* 1998;25:662-665.
26. Restrepo CC, Alvarez E, Jaramillo C, Velez C, Valencia I. Effects of psychological techniques on bruxism in children with primary teeth. *J Oral Rehabil* 2001;28:354-360.
27. Nissani M. A bibliographical survey of bruxism with special emphasis on nontraditional treatment modalities. *J Oral Sci* 2001;43:73-83.
28. Christensen J, Fields HW Jr, Adair S. Oral habits. In: Pinkham JR, Casamassimo PS, McTigue DJ, Fields HW Jr, Nowak AJ, eds. *Pediatric Dentistry: Infancy Through Adolescence*. 4th ed. St. Louis, Mo: Elsevier Saunders; 2005:431-439.
29. Saemundsson SR, Robers MW. Oral self-injurious behavior in the developmentally disabled: Review and a case. *J Dent Child* 1997;64:205-209.
30. Milwood J, Fiske J. Lip biting in patients with profound neurodisability. *Dent Update* 2001;28:105-108.
31. Fields HW Jr, Warren DW, Black BK, Phillips C. Relationship between vertical dentofacial morphology and respiration in adolescents. *Am J Orthod Dentofacial Orthop* 1991;99:147-154.
32. American Academy of Pediatrics. Clinical practice guideline: Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2002;109:704-712.
33. Ward T, Mason TB II. Sleep disorders in children. *Nurs Clin North Am* 2002;37:693-706.
34. Brook AH. Dental anomalies of number, form, and size: Their prevalence in British school children. *J Int Assoc Dent Child* 1974;5:37-53.
35. Byrd ED. Incidence of supernumerary and congenitally missing teeth. *J Dent Child* 1943;10:84-86.
36. Rose JS. A survey of congenitally missing teeth, excluding third molars, in 6,000 orthodontic patients. *Dent Pract Dent Rec* 1966;17:107-114.
37. Clayton JM. Congenital dental anomalies occurring in 3,552 children. *J Dent Child* 1956;23:206-286.
38. Brabant H. Comparison of the characteristics and anomalies of the deciduous and permanent dentitions. *J Dent Res* 1967;46:897-902.
39. Graber LW. Congenital absence of teeth: A review with emphasis on inheritance patterns. *J Am Dent Assoc* 1978;96:266-275.
40. Hobkirk JA, Brook AH. The management of patients with severe hypodontia. *J Oral Rehabil* 1980;7:289-298.
41. Riolo ML, Avery DR. Preadolescent orthodontic treatment and orthodontic treatment during active stages of growth and development. In: *Essentials for Orthodontic Practice*. Ann Arbor & Grand Haven, Mich: ESOP Press; 2003:48-49.
42. Woodworth DA, Sinclair PM, Alexander RG. Bilateral congenital absence of maxillary lateral incisors: A craniofacial and dental cast analysis. *Am J Orthod* 1985;87:280-293.
43. Robertson S, Mohlin B. The congenitally missing upper lateral incisor. A retrospective study of orthodontic space closure vs restorative treatment. *Eur J Orthod* 2000;22:697-710.
44. Primosch RE. Anterior supernumerary teeth: Assessment and surgical intervention in children. *Pediatr Dent* 1981;3:204-215.
45. Luten JR. The prevalence of supernumerary teeth in primary and mixed dentitions. *J Dent Child* 1967;34:346-353.
46. Russell KA, Folwarczna MA. Mesiodens: Diagnosis and management of a common supernumerary tooth. *J Can Dent Assoc* 2003;69:362-366.
47. Taylor GS. Characteristics of supernumerary teeth in primary and permanent dentition. *Trans Br Soc Study Orthod* 1970-71;57:123-128.
48. McKibben DR, Brearley LJ. Radiographic determination of the prevalence of selected dental anomalies in children. *J Dent Child* 1971;28:390-398.
49. Humerfelt D, Hurlen B, Humerfelt S. Hyperdontia in children below four years of age: A radiographic study. *J Dent Child* 1985;52:121-124.
50. Howard RD. The unerupted incisor. A study of the postoperative eruptive history of incisors delayed in their eruption by supernumerary teeth. *Dent Pract Dent Rec* 1967;17:332-341.

51. Tay F, Pang A, Yuen S. Unerupted maxillary anterior supernumerary teeth: A report of 204 cases. *J Dent Child* 1984;51:289-294.
52. Witsenburg B, Boering G. Eruption of impacted permanent upper incisors after removal of supernumerary teeth. *Int J Oral Surg* 1981;10:423-431.
53. Chintakanon K, Boonpinon P. Ectopic eruption of the first permanent molars: Prevalence and etiology factors. *Angle Orthod* 1998;68:153-160.
54. Carr GE, Mink JR. Ectopic eruption of the first permanent maxillary molar in cleft lip and palate children. *J Dent Child* 1965;32:179-188.
55. Grover P, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. *Oral Surg Oral Med Oral Pathol* 1985;59:420-425.
56. Ericson S, Kurol J. Radiographic examination of ectopically erupting maxillary canines. *Am J Orthod Dentofacial Orthop* 1987;91:483-492.
57. Coll JA, Sadrian R. Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition. *Pediatr Dent* 1996;18:57-63.
58. Young DH. Ectopic eruption of the first permanent molar. *J Dent Child* 1957;24:153-162.
59. Gehm S, Crespi PV. Management of ectopic eruption of permanent molars. *Compend Cont Educ Dent* 1997;18:561-569.
60. Halterman CW. A simple technique for the treatment of ectopically erupting first permanent molars. *J Am Dent Assoc* 1982;105:1031-1033.
61. Terry BC, Hegtvædt AK. Self-stabilizing approach to surgical uplifting of the mandibular second molar. *Oral Surg Oral Med Oral Pathol* 1993;75:674-676.
62. Fernandez E, Bravo LA, Canteras M. Eruption of the permanent upper canine: A radiographic study. *Am J Orthod Dentofacial Orthop* 1998;113:414-420.
63. Olive RJ. Orthodontic treatment of palatally impacted maxillary canines. *Aust Orthod J* 2002;18:64-70.
64. D'Amico RM, Bjerklin K, Kurol J, Falahat B. Long-term results of orthodontic treatment of impacted maxillary canines. *Angle Orthod* 2003;73:231-238.
65. McTigue DJ. Managing traumatic injuries in the young permanent dentition. In: Pinkham JR, Casamassimo PS, McTigue DJ, Fields HW Jr, Nowak AJ, eds. *Pediatric Dentistry Infancy Through Adolescence*. 4th ed. St Louis, Mo: Elsevier Saunders; 2005:593-607.
66. Andreasen JO, Andreasen FM. Intrusion, general prognosis. In: *Essentials of Traumatic Injuries to the Teeth*. Copenhagen, Denmark: Munksgaard; 1994:111.
67. Andreasen JO, Andreasen FM. Avulsion injuries: Pattern of injury and diagnosis. In: *Essentials of Traumatic Injuries to the Teeth*. Copenhagen, Denmark: Munksgaard; 1994:115-120.
68. Kokich VO. Congenitally missing teeth: Orthodontic management in the adolescent patient. *Am J Orthod Dentofacial Orthop* 2002;121:594-595.
69. Sabri R. Management of congenitally missing second premolars with orthodontics and single-tooth implants. *Am J Orthod Dentofacial Orthop* 2004;125:634-642.
70. Geiger AM, Brunsby MJ. Orthodontic management of ankylosed permanent posterior teeth: A clinical report of three cases. *Am J Orthod Dentofacial Orthop* 1994;106:543-548.
71. Bolton WA. The clinical application of a tooth-size analysis. *Am J Orthod* 1962;48:504-529.
72. Dugoni SA, Lee JS, Varela J, Dugoni AA. Early mixed dentition treatment: Postretention evaluation of stability and relapse. *Angle Orthod* 1995;65:311-320.
73. Foster H, Wiley W. Arch length deficiency in the mixed dentition. *Am J Orthod* 1958;68:61-68.
74. Little RM. Mandibular arch length increase during the mixed dentition: Postretention evaluation of stability and relapse. *Am J Orthod Dentofacial Orthop* 1990;97:393-404.
75. Little RM. Stability and relapse of mandibular anterior alignment: University of Washington studies. *Semin Orthod* 1999;5:191-204.
76. Warren JJ, Bishara SE, Yonezu T. Tooth size-arch length relationships in the deciduous dentition: A comparison between contemporary and historical samples. *Am J Orthod Dentofacial Orthop* 2003;123:614-619.
77. Warren JJ, Bishara SE. Comparison of dental arch measurements in the primary dentition between contemporary and historic samples. *Am J Orthod Dentofacial Orthop* 2001;119:211-215.
78. Moorrees CF. Register of longitudinal growth studies of facial and dental development. *J Dent Res* 1967;46:1206-1207.
79. Moorrees CF, Burstone CJ, Christiansen RL, Hixon EH, Weinstein S. Research related to malocclusion. A "state-of-the-art" workshop conducted by the Oral-Facial Growth and Development Program, The National Institute of Dental Research. *Am J Orthod* 1971;59:1-18.
80. Turpin DL. Where has all the arch length gone? (editorial) *Am J Orthod Dentofacial Orthop* 2001;119:201.
81. Behrents, RG. Growth in the aging craniofacial skeleton. Monograph 17. Craniofacial Growth Series. Ann Arbor, Mich: University of Michigan, Center for Human Growth and Development; 1985.
82. Zachrisson BU. Important aspects of long-term stability. *J Clin Orthod* 1997;31:562-583.
83. Kusters ST, Kuijpers-Jagman AM, Maltha JC. An experimental study in dogs of transseptal fiber arrangement between teeth which have emerged in rotated and nonrotated positions. *J Dent Res* 1991;70:192-197.
84. Ericson S, Kurol J. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbances. *Eur J Orthod* 1986;8:133-140.
85. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. *Eur J Orthod* 1988;10:283-295.

86. Brothwell DJ. Guidelines on the use of space maintainers following premature loss of primary teeth. *J Can Dent Assoc* 1997;63:753-766.
87. Ngan P, Alkire RG, Fields H Jr. Management of space problems in the primary and mixed dentitions. *J Am Dent Assoc* 1999;130:1330-1339.
88. Terlaje RD, Donly KJ. Treatment planning for space maintenance in the primary and mixed dentition. *J Dent Child* 2001;68:109-114.
89. Dincer M, Haydar S, Unsal B, Turk T. Space maintainer effects on intercanine arch width and length. *J Clin Pediatr Dent* 1996;21:47-50.
90. Qudeimat MA, Fayle SA. The longevity of space maintainers: A retrospective study. *Pediatr Dent*. 1998;20:267-272.
91. Cuoghi OA, Bertoz FA, de Mendonca MR, Santos EC. Loss of space and dental arch length after the loss of the lower first primary molar: A longitudinal study. *J Clin Pediatr Dent* 1998;22:117-120.
92. Rajab LD. Clinical performance and survival of space maintainers: Evaluation over a period of 5 years. *J Dent Child* 2002;69:156-160.
93. Owen DG. The incidence and nature of space closure following the premature extraction of deciduous teeth: A literature survey study. *Am J Orthod Dentofacial Orthop* 1971;59:37-49.
94. Kisling E, Hoffding J. Premature loss of primary teeth. Part IV. *J Dent Child* 1979;46:109-113.
95. Qudeimat MA, Fayle SA. The use of space maintainers at a UK pediatric dentistry department. *J Dent Child* 1999;66:383-386.
96. Christensen JR, Fields HW Jr. Space maintenance in the primary dentition. In: Pinkham JR, Casamassimo PS, McTigue DJ, Fields HW Jr, Nowak AJ, eds. *Pediatric Dentistry Infancy Through Adolescence*. 4th ed. St Louis, Mo: Elsevier Saunders; 2005:423-430.
97. Proffit WR, Fields HW Jr. Orthodontic treatment planning: From problem list to final plan. In: *Contemporary Orthodontics*. 3rd ed. St. Louis, Mo: Mosby; 2000: 180-181.
98. Kanellis MJ. Orthodontic treatment in the primary dentition. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa: Saunders Co; 2001:248-250.
99. Richards B. An approach to the diagnosis of different malocclusions. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa: Saunders Co; 2001:157-158.
100. Bishara SE, Staley RN. Maxillary expansion: Clinical implications. *Am J Orthod Dentofacial Orthop* 1987;91:3-14.
101. Kluemper GT, Beeman CS, Hicks, EP. Early orthodontic treatment: What are the imperatives? *J Am Dent Assoc* 2000;131:613-620.
102. Proffit WR, Fields HW Jr. Orthodontic treatment planning: Limitations and special problems. In: *Contemporary Orthodontics*. 3rd ed. St Louis, Mo: Mosby; 2000:198-217.
103. Sonnesen L, Bakke M, Solow B. Bite force in pre-orthodontic children with unilateral crossbite. *Eur J Orthod* 2001;23:741-749.
104. Pinto AS, Bushang PH, Throckmorton GS, Chen P. Morphological and positional asymmetries of young children with functional unilateral posterior crossbites. *Am J Orthod Dentofacial Orthop* 2001;120:513-520.
105. Proffit WR, Ackerman JL. Orthodontic diagnosis: The development of a problem list. In: Proffit WR, Fields HW Jr, eds. *Contemporary Orthodontics*. 3rd ed. St. Louis, Mo: Mosby; 2000:186.
106. Ghafari J, Shofur FS, Jacobsson-Hunt U, Markowitz DL, Laster LL. Headgear vs functional regulator in the early treatment of Class II, division 1 malocclusion: A randomized clinical trial. *Am J Orthod Dentofacial Orthop* 1998;113:51-61.
107. Tulloch JF, Proffit WR, Phillips C. Benefit of early Class II treatment: Progress report of a two-phase randomized clinical trial. *Am J Orthod Dentofacial Orthop* 1998;113:62-72.
108. Keeling SD, Wheeler TT, King GJ, et al. Anteroposterior skeletal and dental changes after early Class II treatment with bionators and headgear. *Am J Orthod Dentofacial Orthop* 1998;113:40-50.
109. Tulloch JF, Phillips C, Proffit WR. Outcomes in a 2-phase randomized clinical trial of early Class II treatment. *Am J Orthod Dentofacial Orthop* 2004;125:657-667.
110. Chen JY, Will LA, Niederman R. Analysis of efficacy of functional appliances on mandibular growth. *Am J Orthod Dentofacial Orthop* 2002;122:470-476.
111. O'Brien K, Wright J, Conboy F, et al. Effectiveness of early orthodontic treatment with the twin-block appliance: A multicenter, randomized, controlled trial. Part 1: Dental and skeletal effects. *Am J Orthod Dentofacial Orthop* 2003;124:234-243.
112. McNamara JA, Brookstein FL, Shaughnessy TG. Skeletal and dental changes following regulatory therapy on Class II patients. *Am J Orthod Dentofacial Orthop* 1985;88:91-110.
113. Toth LR, McNamara JA Jr. Treatment effects produced by the twin-block appliance and the FR-2 appliance of Frankel compared with untreated Class II sample. *Am J Orthod Dentofacial Orthop* 1999;116:597-609.
114. Carapezza L. Early treatment vs late treatment Class II closed bite malocclusion. *Gen Dent* 2003;51:430-434.
115. Von Bremen J, Pancherz H. Efficiency of early and late Class II division 1 treatment. *Am J Orthod Dentofacial Orthop* 2002;121:31-37.
116. O'Brien K, Wright J, Conboy F, et al. Effectiveness of early orthodontic treatment with the twin-block appliance: A multicenter, randomized, controlled trial. Part 2: Psychosocial effects. *Am J Orthod Dentofacial Orthop* 2003;124:488-495.

117. Kania MJ, Keeling SD, McGorray SP, Wheeler TT, King GJ. Risk factors associated with incisor injury in elementary school children. *Angle Orthod* 1996; 66:423-431.
118. Baccetti T, Franchi L, McNamara JA Jr, Tollaro I. Early dentofacial features of Class II malocclusion: A longitudinal study from the deciduous through the mixed dentition. *Am J Orthod Dentofacial Orthop* 1997;111:502-509.
119. Staley RN. Orthodontic diagnosis and treatment planning: Angle's classification system. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa: Saunders Co; 2001:102-103.
120. Cassidy KM, Harris EF, Tolley EA, Keim RG. Genetic influences on dental arch in orthodontic patients. *Angle Orthod* 1998;68:445-454.
121. Staley RN. Etiology and prevalence of malocclusion. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa: Saunders Co; 2001:84.
122. Campbell P. Early Class III treatment. *Angle Orthod* 1983;53:175-191.
123. Page DC. Early orthodontics: 5 new steps to better care. *Dent Today* 2004;23:1-7.
124. Stahl F, Grabowski R. Orthodontic findings in the deciduous and early mixed dentition: Inferences for a preventive strategy. *J Orofac Orthop* 2003;64:401-416.
125. Ricketts RM. A statement regarding early treatment. *Am J Orthod Dentofacial Orthop* 2000;117:556-558.
126. Thomas PM, Proffit WR. Combined surgical and orthodontic treatment. In: Proffit WR, Fields HW Jr, eds. *Contemporary Orthodontics*. 3rd ed. St. Louis, Mo: Mosby; 2000:519-541.

Copyright of Pediatric Dentistry is the property of American Society of Dentistry for Children and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.