

Management of the Developing Dentition and Occlusion in Pediatric Dentistry

Latest Revision

2019

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. These recommendations are intended to set forth objectives for management of the developing dentition and occlusion in pediatric dentistry.

Methods

Recommendations on management of the developing dentition and occlusion were developed by the Developing Dentition Subcommittee of the Clinical Affairs Committee and adopted in 1990.¹ This document by the Council of Clinical Affairs is a revision of the previous version, last revised in 2014.² This revision is based upon a new PubMed®/MEDLINE search using the terms: tooth ankylosis, Class II malocclusion, Class III malocclusion, interceptive orthodontic treatment, evidence-based, dental crowding, ectopic eruption, dental impaction, obstructive sleep apnea syndrome (OSAS), occlusal development, craniofacial development, craniofacial growth, airway, facial growth, oligodontia, oral habits, occlusal wear and dental erosion, anterior crossbite, posterior crossbite, space maintenance, third molar development, and tooth size/arch length discrepancy; fields: all; limits: within the last 10 years, humans, English, and birth through age 18. Papers for review were chosen from these searches and from references within selected articles. When data did not appear sufficient or were inconclusive, recommendations were based upon expert and/or consensus opinion by experienced researchers and clinicians.

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 and normal subsequent dentofacial development. 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.³⁻⁵ Dentists have the responsibility to recognize, diagnose, and 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.⁶

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.
10. timing 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[AP]) growth patterns and dental AP occlusal disharmonies; and
 - 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; and
 - b. determine the functional status of the patient's occlusion.

ABBREVIATIONS

AAPD: American Academy Pediatric Dentistry. **AP:** Anteroposterior. **CBCT:** Cone-beam computed tomography. **EE:** Ectopic eruption. **OSAS:** Obstructive sleep apnea syndrome. **PFE:** Primary failure of eruption. **TMD:** Temporomandibular joint dysfunction.

3. Functional analysis to:
 - a. determine functional factors associated with the malocclusion;
 - b. detect deleterious habits; and
 - c. detect temporomandibular joint dysfunction (TMD), 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.⁷

Diagnostic orthodontic evaluations fall into three major categories: (1) health of the teeth and oral structures, (2) alignment and occlusal relationships of the teeth, and (3) facial and jaw proportions.⁷

Diagnostic records may include:

1. Extraoral and intraoral photographs to:
 - a. supplement clinical findings with oriented facial and intraoral photographs; and
 - 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 intraarch tooth size relationships;
 - c. determine arch length requirements for interarch tooth size relationships; and
 - 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; and
 - 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 AP, vertical, and transverse dimensions;
 - b. establish a baseline growth record for longitudinal assessment of growth and displacement of the jaws; and
 - c. determine dental maturity relative to skeletal maturity and chronological age.
5. Other diagnostic views (e.g., magnetic resonance imaging, cone-beam computed tomographic images [CBCT]) 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 soft tissue and 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, and 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 six months of age, and complete from approximately three to six years of age when all primary teeth are erupted.
2. Mixed dentition: From approximately age six to 13, primary and permanent teeth are present in the mouth. This stage can be divided further into early mixed and late mixed dentition.
3. Adolescent dentition: All succedaneous teeth have erupted, second permanent molars may be erupted or erupting, and third molars have not erupted.
4. Adult dentition: All permanent teeth are present.^{7,8}

Historically, orthodontic treatment was provided mainly for adolescents. Interest continues to be expressed in the concept of interceptive (early) treatment as well as in adult treatment. Treatment and timing options for the growing patient have increased and continue to be evaluated by the research community.^{9,10} Many clinicians seek to modify skeletal, muscular, and dentoalveolar abnormalities before the eruption of the full permanent dentition.

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

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; space and tooth loss secondary to caries; and periodontal and pulpal health of the teeth.

Radiographic examination, when necessary¹¹ 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.^{7,8}

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 child due to age or behavior. Evaluation, however, should be accomplished when feasible. The objectives of evaluation include identification of:
 - a. 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;
 - d. openbite; and
 - e. airway problems.
 Radiographs are taken with appropriate clinical indicators or based upon risk assessment/history.
2. Early mixed dentition stage: The objectives of evaluation continue as noted for the primary 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. unerupted teeth;
 - b. missing, supernumerary, fused, and geminated teeth;
 - c. tooth size and shape (e.g., peg or small lateral incisors);
 - d. positions (e.g., ectopic first permanent molars);
 - e. developing skeletal discrepancies; and
 - f. periodontal health.
 Space analysis can be used to evaluate arch length at the time of incisor eruption.
3. Late mixed dentition stage: The objectives of the evaluations remain consistent with the prior stages, with an emphasis on evaluation for ectopic tooth positions, especially canines, premolars, 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 of the dentist's observations.
5. Early adult dentition stage: Third molars should be evaluated. If orthodontic diagnosis has not been accomplished, recommendations should be made as necessary.

Treatment objectives: At each stage, the objectives of intervention/treatment include managing adverse growth, correcting dental and skeletal disharmonies, improving esthetics of the smile and the accompanying positive effects on self-image, and improving the occlusion.

1. Primary dentition stage: Habits and crossbites should be diagnosed and, if predicted not likely to be self-correcting, they should be addressed as early as feasible to facilitate normal occlusal relationships. 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 dentition stage: Treatment consideration should address:
 - a. habits;
 - b. arch length shortage;
 - c. intervention for crowded incisors;
 - d. intervention for ectopic teeth;
 - e. holding of leeway space;
 - f. crossbites;
 - g. openbite;
 - h. surgical needs; and
 - i. adverse skeletal growth.
 Intervention for ectopic teeth may include extractions of primary teeth and space maintenance/regaining to aid erupting teeth and reduce the risk of need for permanent tooth extraction or surgical bracket placement for orthodontic traction. Treatment should take advantage of the child's growth and should be aimed at prevention of adverse dental relationships and skeletal growth.
3. Late mixed dentition stage: Intervention for treatment of skeletal disharmonies and crowding may be instituted at this stage.
4. Adolescent dentition stage: In full permanent dentition, orthodontic diagnosis and treatment can provide the most functional, stable, and esthetic occlusion.
5. Early adult dentition stage: Third molar position or space can be evaluated and, if indicated, the tooth/teeth 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 OSAS are discussed in these recommendations.

Oral habits may apply negative 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.^{12,13} Habits of sufficient frequency, duration, and intensity may be associated with dentoalveolar or skeletal deformations such as increased overjet, reduced overbite, openbite, posterior crossbite, or increased facial height. The duration of force is more important than its magnitude¹⁴; 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.^{15,16}

Nonnutritive sucking behaviors are considered normal in infants and young children. Long-term nonnutritive sucking habits (e.g., pacifier use, thumb/finger sucking) have been associated with anterior open bite and posterior crossbite.^{12,15-19} Some evidence indicates that changes resulting from sucking habits persist past the cessation of the habit; therefore, 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.^{12,15,16}

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 (e.g., emotional stress,²⁰ parasomnias,²¹ traumatic brain injury,²² neurologic disabilities²³) and morphologic factors (e.g., malocclusion²⁴, muscle recruitment²⁵). The occlusal wear that may result from bruxism is important to differentiate from other forms of occlusal loss of enamel (e.g., erosion caused by diet or gastroesophageal reflux).²⁶ Reported complications of bruxism include dental attrition, headaches, TMD, and soreness of the masticatory muscles.²⁰ Evidence indicates that juvenile bruxism is self-limiting and does not persist in adults.²⁷ The spectrum of bruxism management ranges from patient/parent education, occlusal splints, and psychological techniques to medications.^{21,22,28,29}

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.³⁰ 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.^{15,30} 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.¹⁵

Self-injurious or self-mutilating behavior (i.e., repetitive acts that result in physical injury to the individual) is extremely rare in the normal child. Such behavior, however, is a chronic condition more frequently seen in special needs populations, having been associated with developmental delay or disabilities, psychiatric disorders, traumatic brain injuries, and some syndromes.^{31,32} The spectrum of treatment options for developmentally disabled individuals includes pharmacologic management, behavior modification, and physical restraint.³³ Dental treatment modalities include, among others, lip-bumper and occlusal bite appliances, protective padding, and extractions. Some habits, such as lip-licking and lip-pulling, are relatively benign in relation to an effect on the dentition. Severe lip- and tongue-biting habits may be associated with profound neurodisability due to severe brain damage.³³ Management options include monitoring the lesion, odontoplasty, providing a bite-opening appliance, or extracting the teeth.³³

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.³⁴

OSAS may be associated with narrow maxilla, crossbite, low tongue position, vertical growth, increased overjet, and openbite.³⁵⁻³⁷ 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 enlarged tonsils.^{34,38,39}

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, and parents should be provided with information regarding consequences of a habit as well as tools to help in elimination of the habit.^{12,13}

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 (extraoral and intraoral), or referral to other providers including, but not limited to, orthodontists, psychologists, myofunctional therapists, or otolaryngologists. The child's desire to stop the habit is beneficial for managing oral habits.¹³

Treatment 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 one or more permanent teeth, has a prevalence of 3.5 to 6.5 percent.⁴⁰ Excluding third molars, the most frequently missing permanent tooth is the mandibular second premolar followed by the maxillary lateral incisor.⁴⁰ In the primary dentition, hypodontia occurs less frequently (0.1 to 0.9 percent prevalence) and almost always affects the maxillary incisors and first primary molars.⁴¹ The chance of familial occurrence of one or two congenitally missing teeth is to be differentiated from missing lateral incisors in cleft lip/palate⁴² and multiple missing teeth (six or more) due to ectodermal dysplasia or other syndromes⁴³ 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, over-retained primary teeth, or ankylosis of a primary mandibular second molar may have a congenitally missing tooth.^{42,44,45}

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 versus opening the space orthodontically and placing a prosthesis or implant depends on many factors. For maxillary laterals, the dentist may move the maxillary canine mesially and use the canine as a lateral incisor or create space for a future lateral prosthesis or implant.^{13,46}

Factors that influence the decision are: (1) patient age; (2) canine size and shape; (3) canine position; (4) child's occlusion and amount of crowding; (5) bite depth; (6) profile; (7) smile line; and (8) quality and quantity of bone in the edentulous area.^{46,47} Early extraction of the primary canine and/or lateral may be needed.⁴⁶ 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.^{43,46} 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.^{45,46} Patients generally prefer space closure over implants.⁴⁷

For a congenitally missing premolar, the primary molar may either be maintained or extracted with placement of a prosthesis, autotransplantation, or orthodontic space closure.⁴⁸⁻⁵⁴ Maintaining the primary second molar may cause occlusal problems due to its larger mesiodistal diameter, compared to the second premolar.⁴⁶ Reducing the width of the second primary molar is a consideration, but root resorption and subsequent exfoliation may occur.^{13,46} In crowded arches or with multiple missing premolars, extraction of the primary molar(s) can be considered, especially in mild Class III cases.^{13,46,50} For a single missing premolar, if maintaining the primary molar is not possible, placement of a prosthesis, autotransplantation, or implant should be considered.^{13,47,50} Preserving the primary tooth may be indicated in certain cases. However, maintaining a submerged/ankylosed tooth may increase the likelihood of an alveolar defect which can compromise later implant success.^{50,51} Consideration for extraction and space maintenance may be indicated.^{50,51} Consultation with an orthodontist and/or prosthodontist may be considered.

Treatment 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 five times more common in the

permanent.⁴⁴ Prevalence is reported in the primary dentition from 0.3-0.8 percent and the mixed dentition from 0.52 to two percent.⁵²⁻⁵⁵ Between 80 and 90 percent of all supernumeraries occur in the maxilla, with half in the anterior area and almost all in the palatal position.⁵² A supernumerary primary tooth is followed by a supernumerary permanent tooth in one-third of the cases.⁵⁶ Supernumerary teeth are classified according to their form and location.^{52,57}

During the early mixed dentition, 79 to 91 percent of anterior permanent supernumerary teeth are unerupted.^{45,53} While more erupt with age, only 25 percent of all mesiodens (a permanent supernumerary incisor located at the midline) erupt spontaneously.⁵² Mesiodens can prevent or cause ectopic eruption of a 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.⁵² 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 tooth can be suspected.^{41,42,53} Panoramic, occlusal, and periapical radiographs all can reveal a supernumerary tooth. To determine the supernumerary tooth's position, either a cone beam radiograph or two periapical or occlusal films reviewed by the parallax rule is recommended.^{52,54}

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.⁵⁶ Surgical extraction of unerupted anterior supernumerary teeth during the primary dentition can displace or damage the permanent incisor.⁵² Removal of an erupted mesiodens or other permanent supernumerary incisor results in eruption of the permanent adjacent normal incisor in 75 percent of the cases.⁵² Extraction of an unerupted supernumerary during the early mixed dentition (i.e., at age six to seven years when the permanent crown has formed completely and the root length is less than the crown height) allows for a normal eruptive force and eruption of the adjacent normal permanent incisor.^{52-54,58} 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.⁵² Inverted conical supernumerary teeth can be harder to remove if removal is delayed, as they can migrate deeper into the jaw.⁵³ After removal of the supernumerary tooth, clinical and radiographic follow-up is indicated in six months to determine if the normal incisor is erupting. If there is no eruption after six to 12 months and sufficient space exists, surgical exposure and orthodontic extrusion may be needed.^{52,59,60}

Treatment 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.^{61,62} 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.^{61,62}

EE of permanent molars can be diagnosed from bitewing or panoramic radiographs in the early mixed dentition.^{61,62} This condition occurs in up to three percent of the population.⁶¹ EE of first permanent molars has been associated with transverse and sagittal crowding and is more common in the maxillary arch and in children with cleft lip and palate.⁶²⁻⁶⁴ EE of second permanent molars occurs infrequently.⁶⁵ EE of permanent molars is classified into two types. There are those that self-correct and others that remain impacted. Previous data suggested that 66 percent of EE permanent molars self-correct by age seven;^{45,62} however, a recent cohort study demonstrated that 71 percent self-correct by age nine.⁶⁶ In some cases, definitive treatment is indicated to manage and/or avoid early loss of the primary second molar and space loss.^{61,62} Increased magnitude of impaction, increased resorption of the primary tooth, and bilateral occurrence were positively associated with irreversible ectopic eruption and may indicate the need for early intervention.⁶⁶

The maxillary canine appears in an impacted position in 1.5 - 2 percent of the population.⁶⁷ Maxillary canine impaction should be suspected when the canine bulge is not palpable, asymmetric canine eruption is evident, or peg shaped lateral incisors are present.⁶⁷⁻⁷¹ Panoramic radiographs may demonstrate that the canine has an abnormal inclination and/or overlaps the lateral incisor root. Additional potential radiographic signs of maxillary canine impaction include enlarged follicular sac, lack of root resorption of primary canines, and presence of premolar impaction.^{69,70,72}

Maxillary incisors can erupt ectopically or be impacted from supernumerary teeth in up to two percent of the population.⁵⁷ Incisors also can have altered eruption due to pulp necrosis (following trauma or caries) or pulpal treatment of the primary incisor.⁷³ EE of permanent incisors can be suspected after trauma to primary incisors, with pulpally-treated primary incisors, with asymmetric eruption, or if a supernumerary incisor is diagnosed.^{67,71}

Treatment considerations: Treatment for ectopic molars 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.⁶¹ For more severe impactions, distal tipping of the permanent molar is required.⁶¹ Tipping action can be accomplished with brass wires, removable appliances using springs, fixed appliances

such as sectional wires with open coil springs,⁷⁴ sling shot-type appliances,⁷⁵ or a Halterman appliance.⁷⁶

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.^{67,77,78} The use of rapid maxillary expansion alone^{79,80} or with cervical pull headgear⁸¹ in the early mixed dentition has been shown to increase the potential for eruption of palatally-displaced maxillary canines. When the impacted canine is diagnosed at a later age (11 to 16 years), if the canine is not horizontal, extraction of the primary canine lessens the severity of the permanent canine impaction and 75 percent will erupt.⁸² Extraction of the first primary molar also has been reported to allow eruption of first premolars and to assist in the eruption of the canines.⁸³ This need can be determined from a panoramic radiograph,^{84,85} although CBCT will provide greater localization of the impacted canine.⁸⁶ 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, and there is insufficient data to conclude the best type of surgical technique.^{87,88}

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.⁷³ Removal of supernumerary incisors in the early mixed dentition will lessen ectopic eruption of an adjacent permanent incisor.⁵² After incisor eruption, orthodontic treatment involving removable or banded therapy may be needed.

Treatment 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.⁸⁹ The periodontal ligament is replaced with osseous tissue, rendering the tooth immobile to eruptive change.⁸⁹ An ankylosed tooth stays at the same vertical level, yet in a growing child appears to submerge as the other teeth continue to erupt. Ankylosis can occur in the primary and permanent dentitions, with the most common incidence involving primary molars. The incidence is reported to be between seven and 14 percent in the primary dentition.⁹⁰ In the permanent dentition, ankylosis occurs most frequently following luxation injuries.⁹¹

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.^{90,91} Ankylosis can occur rapidly or gradually over time, in some cases as long as five years post trauma. It also may be transient if only a small bony bridge forms then is resorbed with subsequent osteoclastic activity.⁹²

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.⁹³ Radiographic examination also may reveal the loss of the periodontal ligament and bony bridging.⁸⁹

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^{4,93} or vertical occlusal discrepancies.⁹⁴ Replacement resorption of permanent teeth usually results in the loss of the involved tooth.⁹⁰

Mildly to moderately ankylosed primary molars without permanent successors may be retained and restored to function in arches without crowding.⁹⁴ Extraction of these molars can assist in resolving crowded arches in complex orthodontic cases.⁹⁵ Surgical luxation of ankylosed permanent teeth with forced orthodontic eruption has been described as an alternative to premature extraction.^{96,97}

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

Primary failure of eruption

General considerations and principles of management: Primary failure of eruption (PFE) is an eruption disorder characterized by partial or complete non-eruption of permanent teeth in the absence of any mechanical obstruction or syndrome.⁹⁸ Failure in eruptive mechanisms prevent permanent successors from following the eruption path after the exfoliation of deciduous teeth.⁹⁹ Posterior teeth are most commonly affected and one or all four quadrants may be involved.¹⁰⁰ Although typically associated with permanent teeth, examples in the primary dentition have been noted.¹⁰¹ Two main phenotypes of PFE have been identified: (1) All teeth distal to the most mesial non-erupted tooth are affected, or (2) unerupted teeth do not follow the pattern that all teeth distal to the most mesial involved tooth are also affected.¹⁰² Hallmark features of PFE include posterior open bite in the presence of normal vertical growth, infra-occlusion of affected teeth, and the inability to move affected teeth orthodontically.¹⁰³

The reported incidence of PFE is between 0.01 and 0.06 percent;^{104,105} however, some data suggests PFE may be misdiagnosed as infra-occlusion or ankylosis.^{106,107} PFE differs from

ankylosis in that eruption fails to occur due to an imbalance in resorptive and appositional factors related to tooth eruption.^{108,109} Teeth with PFE are not initially ankylosed but may become ankylosed when orthodontic forces are applied.¹¹⁰ A systematic review demonstrated 85 percent of patients with PFE have another family member with the condition.¹⁰⁰ PFE has variable expression and has been associated with mutations in the autosomal dominant parathyroid hormone receptor (PTH1R) gene.¹¹⁰⁻¹¹³ A sample of blood or saliva deoxyribonucleic acid (DNA) can be used to test for mutations in PTH1R.^{112,114}

Treatment considerations: Diagnosis of PFE should be based on a combination of clinical, radiographic, and genetic information.¹⁰⁸⁻¹¹⁰ A positive family history also supports a diagnosis of PFE.¹⁰² Other than a few anecdotal reports, PFE is strongly associated with the failure of orthodontically assisted eruption or tooth movement.^{108,109} To that point, early orthodontic intervention of the affected teeth should be avoided.^{103,108,109,114} To date there are no established mechanotherapeutic methods of modifying dentoalveolar growth for these patients.^{103,108,109,114} Space maintenance, up-righting adjacent teeth that have tipped into the sites, prevention of supra-eruption in opposing arch, or modification of lateral tongue thrust habits may be additional considerations.^{103,114} Once growth is complete, multidisciplinary treatment options such as single tooth or segmental osteotomies with immediate traction, or selective extractions followed by implants can be considered to create a functioning occlusion.¹⁰⁸ Early extraction of first molars allowing the second molars to drift forward has also been suggested.¹⁰³

Treatment objectives: Since best available evidence does not support early orthodontic intervention, treatment objectives of PFE should involve reassurance and education about the eruption disorder and preparation for future prosthetic rehabilitation.¹⁰³ In some cases, early extraction can improve normal development of the alveolus and permanent dentition.¹⁰³ Objectives include space and intra-arch maintenance in preparation for future implants, prosthetic rehabilitation, or corticotomy-assisted tooth movement.¹⁰³

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.¹¹⁵ 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 with resulting incisor crowding is a common occurrence with various negative sequelae and is particularly common in the early mixed dentition.¹¹⁶⁻¹¹⁹ 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.¹²⁰⁻¹²² 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.¹²¹⁻¹²³

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.^{116,118} Growth of the aging skeleton causes further crowding and incisor rotations.¹²⁴ Functional contacts are diminished where rotations of incisors, canines, and premolars exist.¹²⁵ Occlusal harmony and temporomandibular joint health are impacted negatively by less functional contacts.¹²⁵

Initial assessment may be done in early mixed dentition, when mandibular incisors begin to erupt.¹¹⁶ 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.^{116,125} It has been shown that the transseptal fibers do not develop until the cements-enamel junction of erupting teeth pass the bony border of the alveolar process.¹²⁵ Therefore, long-term stability of aligned incisors may be increased.¹²⁶

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

1. gaining space for permanent incisors to erupt and become straight naturally through primary canine extraction and space/arch length maintenance with holding arches. Extraction of primary or permanent teeth with the aim of alleviating crowding should not be undertaken without a comprehensive space analysis and a short- and long-term orthodontic treatment plan.
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 premolars and canines have erupted.
4. maintaining patient's original arch form.¹²⁵
5. interproximal stripping of the enamel of mandibular primary canines to allow alignment of crowded lower permanent lateral incisors.¹²⁷

Additional 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.

Treatment 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.^{116,128,129}

Space maintenance

General considerations and principles of management: The premature loss of primary teeth due to caries, infection, trauma, ectopic eruption, or crowding deviates from the normal exfoliation pattern and may lead to 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.¹³⁰ Whenever possible, restoration of carious primary teeth should be attempted to avoid malocclusions that could result from their extraction.¹³¹ The use of space maintainers to reduce the prevalence and severity of malocclusion following premature loss of primary teeth should be considered.^{13,132,133}

Adverse effects associated with space maintainers include: (1) dislodged, broken, and lost appliances; (2) plaque accumulation; (3) increase in microorganisms and increase in periodontal index scores; (4) caries; (5) damage or interference with successor eruption; (6) undesirable tooth movement; (7) inhibition of alveolar growth; (8) soft tissue impingement; and (9) pain.^{130,134-140} Premature loss of a primary tooth, especially in crowded dentitions, has the potential to cause loss of space available for the succeeding permanent tooth, but there is a lack of consensus or evidence regarding the effectiveness of space maintainers in preventing or reducing the severity of malocclusion.^{130,135,136,141-150}

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) occlusion and space assessment; (4) dental age; (5) presence and root development of permanent successor; (6) amount of alveolar bone covering permanent successor; (7) patient's health history and medical status; (8) patient's cooperative ability; (9) active oral habits; and (10) oral hygiene.^{13,130,131}

The literature pertaining to the use of space maintainers specific to the loss of a particular primary tooth type include expert opinion, case reports, and details of appliance design.^{13,132,133} Space maintainers can be designed as fixed unilateral (band and loop, crown and loop, distal shoe), fixed bilateral (lower lingual holding arch, Nance appliance, transpalatal arch), or removable (partial dentures, Hawley type appliance). Variations of these appliances have been described. Unilateral space maintainer kits as well as direct bonded techniques eliminate laboratory involvement and allow for single visit delivery; however, the literature describes mixed results on the longevity of these options compared to success rates of custom appliances.¹⁵²⁻¹⁵⁵

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.¹³⁹ The appliance should function until the succedaneous teeth have erupted into the arch. However, adjustment or new appliances may be necessary with continued development and changes in the dentition.

Treatment 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.^{13,132}

The AAPD recognizes the need for 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.^{13,130,133,156,157} Therefore, loss of space in the dental arch that interferes with the desired eruption of the permanent teeth may require evaluation.

The degree to which space is affected varies according to the arch, site in the arch, and time elapsed since tooth loss.¹⁵⁸ The quantity and incidence of space loss are dependent upon which adjacent teeth are present in the dental arch and their status.^{13,130} The amount of crowding or spacing in the dental arch will determine the consequence of space loss.¹⁵⁷

Treatment considerations: Space can be maintained or regained with removable or fixed appliances.^{130,132} Some examples of fixed space regaining appliances are active holding arches, pendulum appliances, Halterman-type appliances, and Jones jig. Examples of removable space regaining appliances are Hawley appliance with springs, lip bumper, and headgear.¹³² If space regaining is planned, a comprehensive analysis should be completed prior to any treatment decisions. Some factors that should be considered in the analysis include: dentofacial development, age at time of tooth loss, tooth that has been lost, space available, and space needed.^{130,132}

Treatment 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: Crossbites are defined as any abnormal buccal-lingual relation between opposing incisors, molars, or premolars in centric relation.¹⁵⁹⁻¹⁶¹ If the mid lines undergo a compensatory or habitual shift when the teeth occlude in crossbite, this is termed a functional shift.¹⁵⁷ A crossbite can be of dental or skeletal origin or a combination of both.¹⁵⁷

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 and/or mandibular anterior teeth. This condition should be differentiated from a Class III skeletal malocclusion where the crossbite is the result of the basal bone position.¹⁵⁹ Posterior crossbites may be the result of bilateral or unilateral lingual position of the maxillary teeth relative to the mandibular posterior teeth due to tipping or alveolar discrepancy, or a combination. Most often, unilateral posterior crossbites are the manifestation of a bilateral crossbite with a functional mandibular shift.¹⁶¹ Dental crossbites may be the result of tipping or rotation of a tooth or teeth. In this case, the condition is localized and does not involve the basal bone. In contrast, skeletal crossbites involve disharmony of the craniofacial skeleton.^{161,162} Aberrations in bony growth may give rise to crossbites in two 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.^{160,163}

Such growth aberrations can be due to inherited growth patterns, trauma, or functional disturbances that alter normal growth.¹⁶¹⁻¹⁶³

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; (5) increase arch perimeter, (6) help avoid periodontal damage, and (7) prevent the potential for TMD.^{162,164} If enough space is available, a simple anterior crossbite can be aligned as soon as the condition is noted. Treatment options include acrylic incline planes, acrylic retainers with lingual springs, or fixed appliances with springs. If space is needed, an expansion appliance also is an option.¹⁶⁰ Posterior crossbite correction can accomplish the same objectives and can improve the eruptive position of the succedaneous teeth. Early correction of posterior crossbites with a mandibular functional shift has been shown to improve functional conditions significantly and largely eliminate morphological and positional asymmetries of the mandible.^{30,165,166} Contemporary evidence indicates a need for long-term studies to assess the possibility for spontaneous crossbite correction, as current proof is conflicting.¹⁶⁷ Functional shifts should be eliminated as soon as possible with early correction¹⁶³ to avoid TMD and/or asymmetric growth.^{161,167} Treatment can be completed with:

1. equilibration.

2. appliance therapy (fixed or removable).
3. extractions.
4. a combination of these treatment modalities to correct the alveolar constriction.¹⁶⁷

Skeletal expansion with fixed or removable palatal expanders can be utilized until mid line suture fusion occurs.^{157,159}

Treatment decisions depend on the:

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

Treatment objectives: Treatment of a crossbite should result in improved intramaxillary alignment and an acceptable interarch occlusion and function.¹⁶⁵

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 dental (malposition of the teeth in the arches), skeletal (mandibular retrusion and/or maxillary protrusion), or a combination of dental and skeletal factors.⁶

Results of randomized clinical trials indicate that Class II malocclusion can be corrected effectively with either a single or two-phase regimen.¹⁶⁸⁻¹⁷¹ Growth-modifying effects in some studies did not show an influence on the Class II skeletal pattern,¹⁷¹⁻¹⁷³ while other studies dispute these findings.^{174,175} 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.^{168,174} Some reports state interceptive treatment does not reduce the need for either premolar extractions or orthognathic surgery,^{169,171} while others disagree with these findings.¹⁷⁶ Two-phase treatment results in significantly longer treatment time^{163,169,177} although the time spent in full bonded appliance therapy in the permanent dentition can be significantly less.¹⁷⁸

Clinicians may decide to provide interceptive treatment based on other factors.^{169,174} Evidence suggests that, for some children, interceptive Class II treatment may improve self-esteem and decrease negative social experiences, although the improvement may not be different longterm.^{174,179} Early Class II correction may improve facial convexity and/or reduce incidence of maxillary anterior tooth trauma.¹⁸⁰⁻¹⁸⁵ An overjet

in excess of three millimeters is associated with an increased risk of incisor injury, with large overjets (>8 millimeters) resulting in trauma in more than 40 percent of children.^{186,187}

Treatment considerations: Factors to consider when planning orthodontic intervention for Class II malocclusion are: (1) facial growth pattern; (2) amount of AP 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.¹⁵⁷

Treatment objectives: Treatment of a developing Class II malocclusion should result in an improved overbite, overjet, and intercuspation 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 (mesio-occlusion) 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), anterior functional shift of the mandible, or a combination of these factors.¹⁸⁸

The etiology of Class III malocclusions can be hereditary, environmental, or both. Hereditary factors can include clefts of the alveolus and palate as well as other craniofacial anomalies that are part of a genetic syndrome.^{189,190} Some environmental factors are trauma, oral/digital habits, caries, and early childhood OSAS.¹⁹¹

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 compensating growth.¹⁹² Interceptive Class III treatment has been proposed for years and has been advocated as a necessary tool in contemporary orthodontics, with initiation in the primary-early mixed dentition recommended.¹⁹³⁻²⁰² 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; and (5) space analysis.

Treatment objectives: Interceptive Class III treatment may provide a more favorable environment for growth and may improve occlusion, function, and esthetics.²⁰³ Although interceptive 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 may be the best alternative to achieve a satisfactory result for some patients, especially if they exhibit facial characteristics as follows: mandible forward to cranial base, increase mandibular length, short ramal length, or obtuse gonial angle.^{59,204-206}

Treatment of a Class III malocclusion can be achieved using several modalities including protraction therapy with or without rapid palatal expansion, functional appliances, intermaxillary elastics with modified miniplates, or chin cup therapy.^{193-196,204,207-212} These interventions in a growing patient should result in improved overbite, overjet, and intercuspalation of posterior teeth and an esthetic appearance and profile compatible with the patient's skeletal morphology.

References

1. American Academy of Pediatric Dentistry. Guidelines for management of the developing dentition in pediatric dentistry. Chicago, Ill.: American Academy of Pediatric Dentistry; 1990.
2. American Academy of Pediatric Dentistry. Management of the developing dentition and occlusion in pediatric dentistry. *Pediatr Dent* 2014;36(special issue):250-63.
3. Woodside DG. The significance of late developmental crowding to early treatment planning for incisor crowding. *Am J Orthod Dentofacial Orthop* 2000;117(5):559-61.
4. Kurol J. Early treatment of tooth-eruption disturbances. *Am J Orthod Dentofacial Orthop* 2002;121(6):588-91.
5. 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(3):317-27.
6. American Academy of Pediatric Dentistry. Policy on the ethical responsibilities in the oral health care management of infants, children, adolescents, and individuals with special health care needs. *Pediatr Dent* 2018;40(special issue):142-3.
7. Proffit WR, Sarver DM, Fields HW Jr. Orthodontic diagnosis: The problem-oriented approach. In: Proffit WR, Fields HW Jr, Larson BE, Sarver DM, eds. *Contemporary Orthodontics*. 6th ed. Philadelphia, Pa.: Elsevier; 2019: 140-207.
8. Proffit WR. Later stages of development. In: Proffit WR, Fields HW Jr, Larson BE, Sarver DM, eds. *Contemporary Orthodontics*. 6th ed. Philadelphia, Pa.: Elsevier; 2019: 84-106.
9. International Symposium on Early Orthodontic Treatment. *Am J Orthod Dentofacial Orthop* 2002;121(6):552-95.
10. Ackerman M. Evidenced-based orthodontics for the 21st century. *J Am Dent Assoc* 2004;135(2):162-7.
11. American Dental Association, U.S. Department of Health and Human Services. Dental radiographic examinations: Recommendations for patient selection and limiting radiation exposure. Available at: "https://www.ada.org/-/media/ADA/Member%20Center/Files/Dental_Radiographic_Examinations_2012.pdf". Accessed July 25, 2019.
12. 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(12):1685-93.
13. Dean JA. Management of the developing occlusion. In: McDonald and Avery's *Dentistry for the Child and Adolescent*. 10th ed. Maryland Heights, Mo.: Mosby Elsevier; 2015:415-78.
14. Proffit WR. The etiology of orthodontic problems. In: Proffit WR, Fields HW Jr, Larson BE, Sarver DM, eds. *Contemporary Orthodontics*. 6th ed. Philadelphia, Pa.: Elsevier; 2019:107-36.
15. 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(2):161-6.
16. Warren JJ, Bishara SE. Duration of nutritive and non-nutritive sucking behaviors and their effects on the dental arches in the primary dentition. *Am J Orthod Dentofacial Orthop* 2002;121(4):347-56.
17. Adair SM, Milano M, Lorenzo I, Russell C. Effects of current and former pacifier use on the dentition of 24- to 59-month old. *Pediatric Dent* 1995;17(7):437-44.
18. Milink S, Vagner MV, Hocevar-Boltezar J, Ovsenick M. Posterior crossbite in the deciduous dentition period, its relation with sucking habits, irregular orofacial functions and otolaryngological findings. *Am J Orthod Dentofacial Orthop* 2010;138(1):32-40.
19. Dogramaci EJ, Rossi-Fedele G. Establishing the association between non-nutritive sucking behavior and malocclusions: A systematic review and meta-analysis. *J Am Dent Assoc* 2016;147(12):926-34.
20. Monaco A, Ciammella NM, Marci MC, Pirro R, Giannoni M. The anxiety in bruxer child: A case-control study. *Minerva Stomatol* 2002;51(6):247-50.
21. Weideman CL, Bush DL, Yan-Go FL, Clark GT, Gornbein JA. The incidence of parasomnias in child bruxers vs nonbruxers. *Pediatr Dent* 1996;18(7):456-60.
22. Ivanhoe CB, Lai JM, Francisco GE. Bruxism after brain injury: Successful treatment with botulinum toxin-A. *Arch Phys Med Rehabil* 1997;78(11):1272-3.
23. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. *Adv Neurol* 1988;49:329-41.
24. 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(3): 191-4.
25. 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. *ASDC J Dent Child* 1998;65(6):449-58.
26. Taji S, Seow WK. A literature review of dental erosion in children. *Aust Dent J* 2010;55(4):358-67.

27. Kieser JA, Groeneveld HT. Relationship between juvenile bruxing and craniomandibular dysfunction. *J Oral Rehabil* 1998;25(9):662-5.
28. 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(9):354-60.
29. Nissani M. A bibliographical survey of bruxism with special emphasis on nontraditional treatment modalities. *J Oral Sci* 2001;43(2):73-83.
30. Bell RA, Kiebach TJ. Posterior crossbites in children: Developmental based diagnosis and implications to normative growth patterns. *Semin Orthod* 2014;20(2):77-113.
31. Shapira J, Birenboim R, Shoshani M, et al. Overcoming the oral aspects of self-mutilation: Description of a method. *Spec Care Dent* 2016;36(5):282-7.
32. Saemundsson SR, Roberts MW. Oral self-injurious behavior in the developmentally disabled: Review and a case. *ASDC J Dent Child* 1997;64(3):205-9.
33. Millwood J, Fiske J. Lip biting in patients with profound neurodisability. *Dent Update* 2001;28(2):105-8.
34. Fields HW Jr, Warren DW, Black B, Phillips CL. Relationship between vertical dentofacial morphology and respiration in adolescents. *Am J Orthod Dentofacial Orthop* 1991;99(2):147-54.
35. Katyál V, Pamula Y, Daynes CN, et al. Craniofacial and upper airway morphology in pediatric sleep-disordered breathing and changes in quality of life with rapid maxillary expansion. *Am J Orthod Dentofacial Orthop* 2013;144(6):860-71.
36. Pirilä-Parkkinen K, Pirttiniemi P, Nieminen P, Tolonen U, Pelttari U, Löppönen H. Dental arch morphology in children with sleep disordered breathing. *Eur J Orthod* 2009;31(2):160-7.
37. Pirilä-Parkkinen K, Löppönen H, Nieminen P, Tolonen U, Pirttiniemi P. Cephalometric evaluation of children with nocturnal sleep disordered breathing. *Eur J Orthod* 2010;32(6):662-71.
38. Marcus CL, Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2012;130(3):e714-55.
39. Ward T, Mason TB II. Sleep disorders in children. *Nurs Clin North Am* 2002;37(4):693-706.
40. Polder BJ, Van't Hof MA, Van der Linden FP, Kuijpers-Jagtman AM. A meta-analysis of the prevalence of dental agenesis of permanent teeth. *Community Dent Oral Epidemiol* 2004;32(3):217-26.
41. Whittington BR, Durward CS. Survey of anomalies in primary teeth and their correlation with the permanent dentition. *NZ Dent J* 1996;92(407):4-8.
42. Shapira Y, Lubit E, Kuftinec MM. Hypodontia in children with various types of clefts. *Angle Orthod* 2000;70(1):16-21.
43. Worsaae N, Jensen BN, Holm B, Holsko J. Treatment of severe hypodontia-oligodontia—An interdisciplinary concept. *Int J Oral Maxillofac Surg* 2007;36(6):473-80.
44. Garib DG, Peck S, Gomes SC. Increased occurrence of dental anomalies associated with second-premolar agenesis. *Angle Orthod* 2009;79(3):436-41.
45. 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(6):697-710.
46. Spear FM, Mathews DM, Kokich VG. Interdisciplinary management of single-tooth implants. *Semin Orthod* 1997;3(1):45-72.
47. Schneider U, Moser L, Fornasetti M, Piattella M, Siciliani G. Esthetic evaluation of implants vs canine substitution in patients congenitally missing maxillary incisors: Are there any new insights? *Am J Orthod Dentofacial Orthop* 2016;150(3):416-42.
48. Park SY, Tai K, Yuasa K, Hayashi D. The autotransplantation and orthodontic treatment of multiple congenitally missing and impacted teeth. *J Clin Pediatr Dent* 2012;36(4):329-34.
49. Ko JM, Palk CH, Choi S, Baek AH. A patient with protrusion and multiple missing teeth treated with autotransplantation and space closure. *Angle Orthod* 2014;84(3):561-7.
50. Kokich VG, Kokich VO. Congenitally missing mandibular second premolars: Clinical options. *Am J Orthod Dentofacial Orthop* 2006;130(4):437-44.
51. Kennedy DB. Review: Treatment strategies for ankylosed primary molars. *Eur Arch Paediatr Dent* 2009;10(4):201-10.
52. Russell KA, Folwarczna MA. Mesiodens: Diagnosis and management of a common supernumerary tooth. *J Can Dent Assoc* 2003;69(6):362-6.
53. Primosch RE. Anterior supernumerary teeth: Assessment and surgical intervention in children. *Pediatr Dent* 1981;3(2):204-15.
54. He D, Mei L, Wang Y, Li J, Li H. Association between maxillary anterior supernumerary teeth and impacted incisors in the mixed dentition. *J Am Dent Assoc* 2017;148(8):595-603.
55. Anthonappa RP, King NM. Prevalence of supernumerary teeth based on panoramic radiographs revisited. *Pediatr Dent* 2013;35(3):257-61.
56. Taylor GS. Characteristics of supernumerary teeth in the primary and permanent dentition. *Dent Pract Dent Rec* 1972;22(5):203-8.
57. Garvey MT, Barry HJ, Blake M. Supernumerary teeth – An overview of classification, diagnosis and management. *J Can Dent Assoc* 1999;65(11):612-6.
58. Omer RS, Anthonappa RP, King NM. Determination of the optimum time for surgical removal of unerupted anterior supernumerary teeth. *Pediatr Dent* 2010;32(1):14-20.

References continued on the next page.

59. Foley J. Surgical removal of supernumerary teeth and the fate of incisor eruption. *Eur J Paediatr Dent* 2004;5(1):35-40.
60. Ayers E, Kennedy D, Wiebe C. Clinical recommendations for management of mesiodens and unerupted permanent maxillary incisors. *Eur Arch Pediatr Dent* 2014;15(6):421-8.
61. Yaseen SM, Naik S, Uloopr KS. Ectopic eruption – A review and case report. *Contemp Clin Dent* 2011;2(1):3-7.
62. Barberia-Leache E, Suarez-Clus MC, Seavedra-Ontiveros D. Ectopic eruption of the maxillary first permanent molar: Characteristics and occurrence in growing children. *Angle Orthodont* 2005;75(4):610-5.
63. Salbach A, Schremmer B, Grabowski R, Stahl de Castrillon F. Correlation between the frequency of eruption disorders for first permanent and the occurrence of malocclusions in early mixed dentition. *J Orofac Orthop* 2012;73(4):298-306.
64. Carr GE, Mink JR. Ectopic eruption of the first permanent maxillary molar in cleft lip and palate children. *ASDC J Dent Child* 1965;32(3):179-88.
65. Hwang S, Choi YJ, Lee JY, Chung C, Kim KH. Ectopic eruption of maxillary second molar: Predictive factors. *Angle Orthodont* 2017;87(4):583-9.
66. Dabbaugh B, Sigal MJ, Thompson BD, Titley K, Andrews P. Ectopic eruption of the permanent maxillary first molar: Predictive factors for irreversible outcome. *Pediatr Dent* 2017;39(3):215-8.
67. Richardson G, Russell KA. A review of impacted permanent maxillary cuspids – Diagnosis and prevention. *J Can Dent Assoc* 2000;66(9):497-501.
68. Uribe P, Ransjo M, Westerlund AG. Clinical predictors of maxillary canine impaction: A novel approach using multivariate analysis. *Eur J Orthodont* 2017;39(2):153-60.
69. Sherwood K. Evidence-based surgical-orthodontic management of impacted teeth. *Atlas Oral Maxillofac Surg Clin North Am* 2013;21(2):199-210.
70. Garib DG, Leonardi M, Giuntini V, Alencar BM, Lauris JRP, Bacetti T. Agenesis of maxillary lateral incisors and associated dental anomalies. *Am J Orthodont and Dentofacial Orthop* 2010;137(6):732.e1-6.
71. Sachan A, Chatunedi TP. Orthodontic management of buccally erupted ectopic canine with two case reports. *Contemp Clin Dent* 2012;3(1):123-8.
72. Bacetti T, Leonardi M, Giuntini V. Distally displaced premolars: A dental anomaly associated with palatally-displaced canines. *Am J Orthodont Dentofacial Orthoped* 2010;138(3):318e22.
73. Coll JA, Sadrian R. Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition. *Pediatr Dent* 1996;18(1):57-63.
74. Seehra J, Winchester L, Dibase A, Cobourne MT. Orthodontic management of ectopic maxillary first permanent molars: A case report. *Aust Orthodont J* 2011;27(1):57-62.
75. Gehm S, Crespi PV. Management of ectopic eruption of permanent molars. *Compend Cont Educ Dent* 1997;18(6):561-9.
76. Halterman CW. A simple technique for the treatment of ectopically erupting first permanent molars. *J Am Dent Assoc* 1982;105(6):1031-3.
77. Bedoya MM, Park JH. A review of the diagnosis and management of impacted maxillary canines. *J Am Dent Assoc* 2009;140(12):1485-93.
78. Litsas G, Acar A. A review of early displaced maxillary canines: Etiology, diagnosis and interceptive treatment. *Open Dent J* 2011;5(3):39-47.
79. Baccetti T, Mucedero M, Leonardi M, Cozza P. Interceptive treatment of palatal impaction of maxillary canines with rapid maxillary expansion: A randomized clinical trial. *Am J Orthodont Dentofacial Orthop* 2009;136(5):657-61.
80. O'Neill J. Maxillary expansion as an interceptive treatment for impacted canines. *Evid Based Dent* 2010;11(3):86-7.
81. Ami P, Cozza P, Baccetti T. Effect of RME and headgear treatment on the eruption of palatally-displaced canines: A randomized clinical study. *Angle Orthodont* 2011;81(3):370-4.
82. Olive RJ. Orthodontic treatment of palatally impacted maxillary canines. *Aust Orthodont J* 2002;18(2):64-70.
83. Bonetti A, Incerti Parenti S, Zanarini M, Marini I. Double vs primary single teeth extraction approach as a prevention of permanent maxillary canine ectopic eruption. *Pediatr Dent* 2010;32(5):407-12.
84. D'Amico RM, Bjerklin K, Kurol J, Falahat B. Long-term results of orthodontic treatment of impacted maxillary canines. *Angle Orthodont* 2003;73(3):231-8.
85. Bonetti G, Sanarini M, Parenti SI, Marini I, Gatto MR. Preventive treatment of ectopically erupting maxillary permanent canines by extraction of deciduous canines and first molars: A randomized clinical trial. *Am J Orthodont Dentofacial Orthop* 2011;139(3):316-23.
86. Serrant PS, McIntyre GT, Thomson DJ. Localization of ectopic maxillary canines – Is CBCT more accurate than conventional horizontal or vertical parallax? *J Orthodont* 2014;41(1):13-8.
87. Parkin NA, Milner RS, Deery C, et al. Periodontal health of palatally displaced canines treated with open or closed surgical technique: A multicenter, randomized controlled trial. *Am J Orthodont Dentofacial Orthop* 2013;144(2):176-84.
88. Incerti-Parenti S, Checchi V, Ippolito R, et al. Periodontal status after surgical-orthodontic treatment of labially impacted canines with different surgical techniques: A systematic review. *Am J Orthodont Dentofacial Orthop* 2016;149(4):463-72.
89. Ducommun F, Bornstein MM, Bosshardt D, Katsaros C, Dula K. Diagnosis of tooth ankylosis using panoramic views, cone beam computed tomography and histological data: A retrospective observational case series study. *Eur J Orthodont* 2018;40(3):231-8.

90. McKibben DR, Brearley LJ. Radiographic determination of the prevalence of selected dental anomalies in children. *ASDC J Dent Child* 1971;28(6):390-8.
91. Malmgren B, Malmgren O, Andreaswn JO. Long-term follow up of 103 ankylosed permanent incisors surgically treated with decoronation—A retrospective cohort study. *Dent Traumatol* 2015;31(3):184-9.
92. Kokich VO. Congenitally missing teeth: Orthodontic management in the adolescent patient. *Am J Orthod Dentofacial Orthop* 2002;121(6):594-5.
93. Mishra SK, Jindal MK, Singh RP, Stark TR. Submerged and impacted primary molars. *Int J Clin Pediatr Dent* 2010;3(3):211-3.
94. Proffit WR. Moderate nonskeletal problems in pre-adolescent children: Preventive and interceptive treatment in family practice. In Proffit W, Fields HW Jr, and Sarver DM, eds. *Contemporary Orthodontics*, 5th ed. St. Louis, Mo.: Mosby; 2012:426-7.
95. Sabri R. Management of congenitally missing second premolars with orthodontics and single-tooth implants. *Am J Orthod Dentofacial Orthop* 2004;125(5):634-42.
96. Shi KK, Kim JY, Choi TH, Lee KJ. Timely relocation of subapically impacted maxillary canines and replacement of an ankylosed mandibular molar are the keys to eruption disturbances in a prepubertal patient. *Am J Orthod Dentofacial Orthop* 2014;145(2):228-37.
97. Geiger AM, Brunsky MJ. Orthodontic management of ankylosed permanent posterior teeth: A clinical report of three cases. *Am J Orthod Dentofacial Orthop* 1994;106(5):543-8.
98. Proffit WR, Vig KW. Primary failure of eruption: A possible cause of posterior open bite. *Am J Orthod* 1981; 80(2):73-90.
99. Mubeen S, Seehrab J. Failure of eruption of first permanent molar teeth: A diagnostic challenge. *J Orthod* 2018; 45(2):129-34.
100. Hanisch M, Hanisch L, Kleinheinz J, Jung S. Primary failure of eruption (PFE): A systematic review. *Head Face Med* 2018;14(1):5.
101. Ahmad S, Brister D, Cobourne MT. The clinical features and aetiological basis of primary eruption failure. *Eur J Orthod* 2006;28(6):535-40.
102. Hartsfield JK, Jacob GJ, Morford LA. Heredity, genetics and orthodontics: How much has this research really helped? *Semin Orthod* 2017;23(4):336-47.
103. Frazier-Bowers SA, Long S, Tucker M. Primary failure of eruption and other eruption disorders—Considerations for management by the orthodontist and oral surgeon. *Semin Orthod* 2016;22(1):34-44.
104. Grover PS, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. *Oral Surg Oral Med Oral Pathol* 1985;9(4):420-5.
105. Baccetti T. Tooth anomalies associated with failure of eruption of first and second permanent molars. *Am J Orthod Dentofacial Orthop* 2000;118(6):608-10.
106. Anthonappa RP, King NM. Primary failure of eruption or severe infra-occlusion: A misdiagnosis? *Eur Arch Paediatr Dent* 2013;14:267-70.
107. Pilz P, Meyer-Marcotty P, Eigenthaler M, Roth H, Weber BH, Stellzig-Eisenhauer A. Differential diagnosis of primary failure of eruption (PFE) with and without evidence of pathogenic mutations in the PTHR1 gene. *J Orofac Orthop* 2014;75(3):226-39.
108. Frazier-Bowers SA, Puranik CP, Mahaney MC. The etiology of eruption disorders—Further evidence of a “genetic paradigm”. *Semin Orthod* 2010;16(3):180-5.
109. Frazier-Bowers SA, Simmons D, Wright JT, Proffit WR, Ackerman J. Primary failure of eruption and PTHR1: The importance of a genetic diagnosis for orthodontic treatment planning. *Am J Orthod Dentofacial Orthop* 2010;137(2):160.e1-160.e7.
110. Rhoades SG, Hendricks HM, Frazier-Bowers SA. Establishing the diagnostic criteria for eruption disorders based on genetic and clinical data. *Am J Orthod Dentofacial Orthop* 2013;144(2):194-202.
111. Decker E, Stellzig-Eisenhauer A, Fiebig BS, et al. PTHR1 loss of function mutations in familial nonsyndromic primary failure of tooth eruption. *Am J Hum Gen* 2008; 83(6):781-6.
112. Jelani M, Kang C, Mohamoud HIS, et al. A novel homozygous PTHR1 variant identified through whole exome sequencing further expands the clinical spectrum of primary failure of tooth eruption in a consanguineous Saudi family. *Arch Oral Biol* 2016;67:28-33.
113. Submaranian H, Doring F, Kollert S, et al. PTHR1 mutants found in patients with primary failure of tooth eruption disrupt G-protein signaling. *PLoS One* 2016;11(11):1-16.
114. Grippaudoa C, Cafierob C, D’Apolitic I, Riccio B, Frazier-Bowers SA. Primary failure of eruption: Clinical and genetic findings in the mixed dentition. *Angle Orthod* 2018;88(3):275-82.
115. Bolton WA. The clinical application of a tooth-size analysis. *Am J Orthod* 1962;48(7):504-29.
116. Dugoni SA, Lee JS, Varela J, Dugoni AA. Early mixed dentition treatment: Post-retention evaluation of stability and relapse. *Angle Orthod* 1995;65(5):311-20.
117. Foster H, Wiley W. Arch length deficiency in the mixed dentition. *Am J Orthod* 1958;44:61-8.
118. Little RM, Riedel RA, Stein A. Mandibular arch length increase during the mixed dentition: Post-retention evaluation of stability and relapse. *Am J Orthod Dentofacial Orthop* 1990;97(5):393-404.
119. Little RM. Stability and relapse of mandibular anterior alignment: University of Washington studies. *Semin Orthod* 1999;5(3):191-204.
120. 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):1-18.

References continued on the next page.

121. 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(3):211-5.
122. 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(6):614-9.
123. Turpin DL. Where has all the arch length gone? (editorial) *Am J Orthod Dentofacial Orthop* 2001;119(3):201.
124. 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.
125. Zachrisson BU. Important aspects of long-term stability. *J Clin Orthod* 1997;31(9):562-83.
126. Kusters ST, Kuijpers-Jagman AM, Maltha JC. An experimental study in dogs of transseptal fiber arrangement between teeth which have emerged in rotated and non-rotated positions. *J Dent Res* 1991;70(3):192-7.
127. Nakhjavani Y, Nakhjavani F, Jaferi A. Mesial stripping of mandibular deciduous canines for correction of permanent lateral incisors. *Int J Clin Pediatr Dent* 2017;10(3):229-33.
128. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. *Eur J Orthod* 1988;10(4):283-95.
129. Ericson S, Kurol J. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbances. *Eur J Orthod* 1986;8(3):133-40.
130. Brothwell DJ. Guidelines on the use of space maintainers following premature loss of primary teeth. *J Can Dent Assoc* 1997;63(10):753-66.
131. Northway WM. The not-so-harmless maxillary primary first molar extraction. *J Am Dent Assoc* 2000;131(12):1711-20.
132. Ngan P, Alkire RG, Fields HW Jr. Management of space problems in the primary and mixed dentitions. *J Am Dent Assoc* 1999;130(9):1330-9.
133. Terlaje RD, Donly KJ. Treatment planning for space maintenance in the primary and mixed dentition. *ASDC J Dent Child* 2001;68(2):109-14.
134. Kirshenblatt S, Kulkarni GV. Complications of surgical extraction of ankylosed primary teeth and distal shoe space maintainers. *J Dent Child* 2011;78(1):57-61.
135. Sonis A, Ackerman M. E-space preservation. *Angle Orthod* 2011;81(6):1045-9.
136. Rubin RL, Baccetti T, McNamara JA. Mandibular second molar eruption difficulties related to the maintenance of arch perimeter in the mixed dentition. *Am J Orthod Dentofacial Orthop* 2012;141(2):146-52.
137. Dincer M, Haydar S, Unsal B, Turk T. Space maintainer effects on intercanine arch width and length. *J Clin Pediatr Dent* 1996;21(1):47-50.
138. Qudeimat MA, Fayle SA. The longevity of space maintainers: A retrospective study. *Pediatr Dent* 1998;20(4):267-72.
139. 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(2):117-20.
140. Arika V, Kizilci E, Ozalp N, Ozcelik B. Effects of fixed and removable space maintainers on plaque accumulation, periodontal health, candidal and *Enterococcus Faecalis* carriage. *Med Princ Pract* 2015;24(4):311-7.
141. Rajab LD. Clinical performance and survival of space maintainers: Evaluation over a period of 5 years. *ASDC J Dent Child* 2002;69(2):156-60.
142. 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(1):37-49.
143. Kisling E, Hoffding J. Premature loss of primary teeth. Part IV, a clinical control of Sannerud's space maintainer, type I. *ASDC J Dent Child* 1979;46(2):109-13.
144. Brennan MM, Gianelly A. The use of the lingual arch in the mixed dentition to resolve incisor crowding. *Am J Orthod Dentofacial Orthop* 2000;117(1):81-5.
145. Gianelly AA. Treatment of crowding in the mixed dentition. *Am J Orthod Dentofacial Orthop* 2002;121(6):569-71.
146. Lin YT, Lin WH, Lin YT. Twelve-month space changes after premature loss of a primary maxillary molar. *Int J Paediatr Dent* 2011;21(3):161-6.
147. Tunison W, Flores-Mir C, ElBadrawy H, Nassar U, El-Bialy T. Dental arch space changes following premature loss of a primary first molars: A systematic review. *Pediatr Dent* 2008;30(4):297-302.
148. Laing E, Ashley P, Naini FB, et al. Space maintenance. *Int J Pediatr Dent* 2009;19(3):155-62.
149. Lin YT, Lin WH, Lin YT. Immediate and six-month space changes after premature loss of a primary maxillary first molar. *J Am Dent Assoc* 2007;138(3):362-8.
150. Canadian Agency for Drugs and Technologies in Health. Dental space maintainers for the management of premature loss of deciduous molars: A review of clinical effectiveness and guidelines. Ottawa (ON): 2016. Available at: "<https://www.ncbi.nlm.nih.gov/books/NBK401552/>". Accessed July 25, 2019.
151. Law CS. Management of premature primary tooth loss in the child patient. *J Calif Dent Assoc* 2013;41(8):612-8.
152. Kara NB, Cehreli S, Sagirkaya E, Karasoy D. Load distribution in fixed space maintainers: A strain gauge analysis. *Pediatr Dent* 2013;35(1):19-22.
153. Kulkarni G, Lau D, Hafezi S. Development and testing fiber-reinforced composite space maintainers. *J Dent Child* 2009;76(3):204-8.

154. Setia V. Banded vs bonded space maintainers: Finding a better way out. *Int J Clin Pediatr Dent* 2014;7(2):97-104.
155. Kargul B, Caglar E, Kabalay U. Glass fiber-reinforced composite resin as fixed space maintainers in children: 12-month clinical follow up. *J Dent Child* 2005;72(3):109-12.
156. Christensen JR, Fields HW Jr. Space maintenance in the primary dentition. In: Casamassimo PS, McTigue DJ, Fields HW Jr, Nowak AJ, eds. *Pediatric Dentistry Infancy Through Adolescence*. 5th ed. St. Louis, Mo.: Elsevier Saunders; 2013:379-84.
157. Proffit WR, Fields HW Jr, Sarver DM. Orthodontic treatment planning: From problem list to specific plan. In: *Contemporary Orthodontics*. 5th ed. St. Louis, Mo.: Mosby; 2012:220-75.
158. Finucane D. Rationale for restoration of carious primary teeth: A review. *Eur Arch of Pediatr Dent* 2012;13(6):281-92.
159. Bishara SE, Staley RN. Maxillary expansion: Clinical implications. *Am J Orthod Dentofacial Orthop* 1987;91(1):3-14.
160. Richards B. An approach to the diagnosis of different malocclusions. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa.: Saunders Co.; 2001:157-8.
161. Da Silva Andrade, A, Gameiro G, DeRossi, M, Gaviao, M. Posterior crossbite and functional changes. *Angle Orthod* 2009;79(2):380-6.
162. Borrie F, Stearn D. Early correction of anterior crossbites: A systematic review. *J Orthod* 2011;38(3):175-84.
163. Kluemper GT, Beeman CS, Hicks EP. Early orthodontic treatment: What are the imperatives? *J Am Dent Assoc* 2000;131(5):613-20.
164. Noar J. Managing the developing occlusion: Anterior crossbites. In: *Interceptive Orthodontics: A Practical Guide to Occlusal Management*. Chichester, UK. Wiley Blackwell; 2014:29-73.
165. Sonnesen L, Bakke M, Solow B. Bite force in preorthodontic children with unilateral crossbite. *Eur J Orthod* 2001;23(6):741-9.
166. 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(5):513-20.
167. Agostino P, Ugolini A, Signori A, Silvestrini-Biavati A. Orthodontic treatment for posterior crossbites. *Cochrane Database Syst Rev* 2014;1-52. Available at: "https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD000979.pub2/epdf/full". Accessed October 12, 2019.
168. 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(1):51-61.
169. 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(1):62-72.
170. 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(6):657-67.
171. 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(1):40-50.
172. Chen JY, Will LA, Niederman R. Analysis of efficacy of functional appliances on mandibular growth. *Am J Orthod Dentofacial Orthop* 2002;122(5):470-6.
173. 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(3):234-43.
174. McNamara JA, Brookstein FL, Shaughnessy TG. Skeletal and dental changes following regulatory therapy on Class II patients. *Am J Orthod Dentofacial Orthop* 1985;88(2):91-110.
175. 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(6):597-609.
176. Carapezza L. Early treatment vs late treatment Class II closed bite malocclusion. *Gen Dent* 2003;51(5):430-4.
177. Von Bremen J, Panherz H. Efficiency of early and late Class II division 1 treatment. *Am J Orthod Dentofacial Orthop* 2002;121(1):31-7.
178. Oh H, Baumrind S, Korn EL. A retrospective study of Class II mixed dentition treatment. *Angle Orthod* 2017;87(1):56-67.
179. 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(5):488-95.
180. Kirjavainen M, Hurmerinta K, Kiravainen T. Facial profile changes in early Class II correction with cervical headgear. *Angle Orthod* 2007;77(6):960-7.
181. Kalha AS. Early orthodontic treatment reduced incisal trauma in children with class II malocclusions. *Evid Based Dent* 2014;15(1):18-20.
182. Thiruvankatachari B, Harrison JE, Worthington HV, O'Brien KD. Early orthodontic treatment for Class II malocclusion reduces the chance of incisal trauma: Results of a Cochrane systematic review. *Am J Orthod Orthop* 2015;148(1):47-59.
183. Batista K, Thiruvankatachari B, Harrison JE, O'Brien KD. Orthodontic treatment for prominent front teeth (Class II malocclusion) in children and adolescents. *Cochrane Database Syst Rev* 2018;13:3.

References continued on the next page.

184. Kania MJ, Keeling SD, McGorray SP, Wheeler TT, King GJ. Risk factors associated with incisor injury in elementary school children. *Angle Orthod* 1996;66(6):423-31.
185. 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(5):502-9.
186. Nguyen QV, Bezemer PD, Habets L, Prah-Andersen B. A systematic review of the relationship between overjet size and traumatic dental injuries. *Eur J Orthod* 1999;21(5):503-15.
187. Cameron AC. Trauma management. In: *Handbook of Pediatric Dentistry*. Angus Cameron, Richard Widmer, eds. 4th ed. Maryland Heights, Mo.: Mosby Elsevier; 2013:149-207.
188. Staley RN. Orthodontic diagnosis and treatment planning: Angle's classification system. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa.: Saunders Co.; 2001:102-3.
189. Xue F, Wong RWK, Rabie ABM. Genes, genetics, and Class III malocclusion. *Orthod Craniofacial Res* 2010;13(2):69-74.
190. Cassidy KM, Harris EF, Tolley EA, Keim RG. Genetic influences on dental arch in orthodontic patients. *Angle Orthod* 1998;68(5):445-54.
191. Staley RN. Etiology and prevalence of malocclusion. In: Bishara SE, ed. *Textbook of Orthodontics*. Philadelphia, Pa.: Saunders Co.; 2001:84.
192. Celikoglu M, Oktay H. Effects of maxillary protraction for early correction class III malocclusion. *Eur J Orthod* 2014;36(1):86-92.
193. Baccetti T, Tollaro I. A retrospective comparison of functional appliance treatment of Class III malocclusions in the deciduous and mixed dentitions. *Eur J Orthod* 1998;20(3):309-17.
194. Saadia M, Torres E. Vertical changes in Class III patients after maxillary protraction expansion in the primary and mixed dentitions. *Pediatr Dent* 2001;23(2):123-30.
195. Franchi L, Baccetti T, McNamara JA. Postpubertal assessment of treatment timing for maxillary expansion and protraction therapy followed by fixed appliances. *Am J Orthod Dentofacial Orthop* 2004;126(5):555-68.
196. Lione R, Buongiorno M, Lagana G, Cozza P, Franchi L. Early treatment of Class III malocclusion with RME and facial mask: Evaluation of dentoalveolar effects on digital dental casts. *Eur J Pediatr Dent* 2015;16(3):217-20.
197. Campbell PM. The dilemma of Class III treatment. Early or late? *Angle Orthod* 1983;53(3):175-91.
198. Kim JH, Viana MA, Graber TM, Omerza FF, BeGole EA. The effectiveness of protraction face mask therapy: A meta-analysis. *Am J Orthod Dentofacial Orthop* 1999;115(6):675-85.
199. Jager A, Braumann B, Kim C, Wahner S. Skeletal and dental effects of maxillary protraction in patients with Angle class III malocclusions. A meta-analysis. *J Orofac Orthop* 2001;62(4):275-84.
200. Page DC. Early orthodontics: 5 new steps to better care. *Dent Today* 2004;23(2):1-7.
201. Stahl F, Grabowski R. Orthodontic findings in the deciduous and early mixed dentition: Inferences for a preventive strategy. *J Orofac Orthop* 2003;64(6):401-16.
202. Ricketts RM. A statement regarding early treatment. *Am J Orthod Dentofacial Orthop* 2000;117(5):556-8.
203. Toffol LD, Pavoni C, Baccetti T, Franchi L, Cozza P. Orthopedic treatment outcomes in Class III malocclusion. *Angle Orthod* 2008;78(3):561-73.
204. Franchi L, Baccetti T, McNamara JA. Predictable variables for the outcome of early functional treatment of Class III malocclusion. *Am J Orthod Dentofacial Orthop* 1997;112(1):60-6.
205. Ghiz MA, Ngan P, Gunei E. Cephalometric variables to predict future success of early orthopedic Class III treatment. *Am J Orthod Dentofacial Orthop* 2005;127(3):301-6.
206. Tahmina K, Tanaka E, Tanne K. Craniofacial morphology in orthodontically treated patients of Class III malocclusion with stable and unstable treatment outcomes. *Am J Orthod Dentofacial Orthop* 2000;117(6):681-90.
207. Coscia G, Addabbo F, Peluso V, D'Ambrosio E. Use of intermaxillary forces in early treatment of maxillary deficient class III patients: Results of a case series. *J Craniomaxillofac Surg* 2012;40(8):350-4.
208. Deguchi T, Kuroda T, Minoshima Y, Graber T. Craniofacial features of patients with Class III abnormalities: Growth-related changes and effects of short term and long-term chin cup therapy. *Am J Orthod Dentofacial Orthop* 2002;121(1):84-92.
209. Ferro A, Nucci LP, Ferro F, Gallo C. Long term stability of skeletal Class III patients treated with splints, Class III elastics and chin cup. *Am J Orthod Dentofacial Orthop* 2003;123(4):423-34.
210. Palma JC, Tejedor-Sanz N, Oteo D, Alarcon JA. Long-term stability of rapid maxillary expansion combined with chin cup protraction followed by fixed appliances. *Angle Orthod* 2015;85(2):270-7.
211. Wendl B, Kamenica A, Droshci H. Retrospective 25 year follow up of treatment outcomes in angle Class II patients: Early vs late treatment. *J Orofac Orthop* 2017;78(3):201-10.
212. Proffit WR, Fields HW Jr. Treatment of skeletal transverse and class III problems. In: Proffit WR, Fields HW Jr, Larson BE, Sarver DM, eds. *Contemporary Orthodontics*. 6th ed. Philadelphia, Pa.: Elsevier; 2019:440-53.