Management of the Developing Dentition and Occlusion in Pediatric Dentistry

Latest Revision
2024

How to Cite: American Academy of Pediatric Dentistry. Management of the developing dentition and occlusion in pediatric dentistry. *The Reference Manual of Pediatric Dentistry*. American Academy of Pediatric Dentistry; 2026:525-543.

Abstract

This best practice asserts that the management of developing dentition and occlusion is an essential part of comprehensive oral health care and that early diagnosis and treatment of abnormalities can aid patients in achieving the goal of a stable, functional, and esthetic occlusion. The document outlines the components of the clinical examination and necessary diagnostic records and emphasizes the importance of the diagnostic summary for determining treatment priorities and timing. Considerations for management according to each stage of dentition (primary, mixed, adolescent, adult) are presented along with treatment objectives and recommendations for relevant dental concerns, including oral habits, congenitally missing or supernumerary teeth, ectopic eruption, and ankylosis or primary failure of eruption. Lastly, the document provides discussion of arch length discrepancy, space maintenance, space regaining, crossbites, and Class II and Class III malocclusions. Providers may use this document as a resource for gathering crucial diagnostic information and making informed decisions regarding the timing, sequence, and appropriateness of interventions.

This document was developed through a collaborative effort of the American Academy of Pediatric Dentistry Councils on Clinical Affairs and Scientific Affairs to offer updated information and recommendations regarding management of developing dentition and occlusion.

KEYWORDS: ANKYLOSIS; CROSSBITE; MALOCCLUSION; ORAL HABITS; SPACE MAINTENANCE; SUPERNUMERARY TEETH; TOOTH ERUPTION

Purpose

The American Academy of Pediatric Dentistry 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 on Clinical Affairs is a modification of the previous revision, last revised in 2021.[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*, *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. The initial search was modified to include only randomized clinical trials, systematic reviews, and meta-analyses, resulting in 6016 articles. Papers for review were chosen from these searches and from references within selected articles; textbooks also were used. 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 can contribute to the development of a permanent dentition that is in a stable, functional, and esthetically acceptable occlusion and to 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 scope of practice.[6]

ABBREVIATIONS
AP: Anteroposterior. **CBCT:** Cone-beam computed tomography. **EE:** Ectopic eruption. **OSA:** Obstructive sleep apnea. **PFE:** Primary failure of eruption.

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

- chronological/mental/emotional age of the patient and the patient's ability to understand and cooperate in the treatment
- intensity, frequency, and duration of an oral habit
- parental support for the treatment
- compliance with clinician's instructions
- craniofacial configuration
- craniofacial growth
- concomitant systemic disease or condition
- accuracy of diagnosis
- appropriateness of treatment
- timing of treatment

A comprehensive clinical examination with pretreatment diagnostic records (eg, radiographs, casts) is necessary to develop a differential diagnosis, treatment goals (both short- and long-term), and a sequential treatment plan to manage the developing dentition and occlusion. The practitioner can share findings of adverse growth and developing malocclusions with the patient/parent and, through the process of informed consent, establish treatment priorities. Including the patient and parent in treatment planning is critical for informed consent.[7(p208)] A thorough discussion of existing problems, treatment options, alternatives, and outcomes is necessary for the decision-making process not only at the beginning of treatment but ongoing during later stages.[7(p229)] Patient compliance is an important consideration for providers during treatment planning and informed consent as it can significantly affect the outcome.[7(p208)]

Clinical examination can include

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

Diagnostic records may be needed to assist in the evaluation of the patient's condition and for documentation purposes. Prudent judgment is exercised to decide the appropriate records required for diagnosis of the clinical condition.[8(pp140,166)]

Diagnostic orthodontic evaluations fall into 3 major categories: (1) health of the teeth and oral structures, (2) occlusal relationships and alignment of the teeth, and (3) proportions of the face and jaw.[8(p166)]

Diagnostic records may include

- extraoral and intraoral photographs to
  - supplement clinical findings with oriented facial and intraoral photographs; and
  - establish a database for documenting facial changes during treatment.
- diagnostic dental casts to
  - assess the occlusal relationship;
  - determine arch length requirements for tooth size discrepancies;
  - determine arch length requirements for interarch tooth size relationships; and
  - determine location and extent of arch asymmetry.
- intraoral and panoramic radiographs to
  - establish dental age;
  - assess eruption problems;
  - estimate the size and presence of unerupted teeth; and
  - identify dental anomalies/pathology.
- lateral and AP cephalograms to
  - analyze the relative dental and skeletal components in the AP, vertical, and transverse dimensions;
  - establish a baseline growth record for longitudinal assessment of growth and displacement of the jaws; and
  - determine dental maturity relative to skeletal maturity and chronological age.
- other diagnostic views (eg, magnetic resonance imaging, cone-beam computed tomography [**CBCT**]) for hard and soft tissue imaging as indicated by history and clinical examination.

A differential diagnosis and diagnostic summary are completed to

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

A sequential treatment plan will

- establish timing priorities for each phase of therapy;
- establish proper sequence of treatments to achieve short-term and long-term objectives; and

- assess treatment progress and update the biomechanical protocol accordingly on a regular basis.

Stages of development of occlusion

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

1. primary dentition: beginning in infancy with the eruption of the first tooth, usually about 6 months of age, and complete from approximately 2 to 6 years of age when all primary teeth are erupted.[9(p65)]
2. mixed dentition: from approximately age 6 to 11 years, primary and permanent teeth are present in the mouth. This stage can be divided further into early mixed and late mixed dentition.[9(p74)]
3. adolescent dentition: beginning at approximately age 11 to 12 years, all permanent teeth except the third molars may be erupted or erupting.[9(p77)]
4. adult dentition: when all permanent teeth are present.[10,11]

Interest continues to be expressed in the concept of interceptive (early) treatment as well as in adult treatment. 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, is essential in diagnosing and reviewing possible interceptive treatment options. Treatment is beneficial for many children but may not be indicated for every patient with a developing malocclusion.

Treatment considerations: Monitoring developing dentition at regular clinical examinations may 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 necessary[12] and feasible, augments clinical examination. Diagnosis of anomalies of primary or permanent tooth development and eruption is critical in establishing treatment goals and a sequential treatment plan. This evaluation is ongoing throughout the developing dentition, at all stages.[8(p167)]

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 a dental examination or to a radiographic examination not being possible in a child because of age or behavior. The objectives of evaluation during this stage, accomplished when feasible, include identification of[13] all anomalies of tooth number and size; crossbites; habits along with their dental and skeletal sequelae; open bite; and airway problems. Radiographs are taken with appropriate clinical indicators or based upon risk assessment/history.

Early mixed dentition stage: The objectives of evaluation continue as noted for the primary dentition stage as well as palpation for unerupted teeth. Panoramic, occlusal, and periapical radiographs, as indicated at the time of eruption of the lower incisors and first permanent molars, provide diagnostic information concerning unerupted teeth; missing, supernumerary, fused, and geminated teeth; tooth size and shape (eg, peg or small lateral incisors); root development/resorption; positions[14(pp425-434)] (eg, ectopic first permanent molars); developing skeletal discrepancies[14(pp423,424)]; and periodontal health[14(pp424,425)]. Space analysis can be used to evaluate arch length at the time of incisor eruption.[14(p440)]

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.[15(p524)]

Adolescent dentition stage: If not instituted earlier, orthodontic diagnosis and treatment planning for Class I crowded, Class II, and Class III malocclusions as well as posterior and anterior crossbites is indicated.[16(pp572-575)] Third molars should be monitored as to position and space.[16(p567)]

Early adult dentition stage: Evaluation of the third molars is indicated. If orthodontic diagnosis has not been accomplished, recommendations may be made as necessary.

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

Primary dentition stage: Addressing habits and crossbites not likely to self-correct facilitates normal occlusal relationships. Interventions/treatment can be recommended if diagnosis can be made and treatment is appropriate and possible.

Early mixed dentition stage: Treatment consideration include habits; arch length shortage; intervention for crowded incisors; intervention for ectopic teeth; intervention for premature loss of primary teeth; holding of leeway space; crossbites; open bite; surgical needs; and 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 needing 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.

Late mixed dentition stage: Holding of leeway space and intervention for treatment of skeletal disharmonies and crowding may be instituted at this stage.

Adolescent dentition stage: In full permanent dentition, orthodontic diagnosis and treatment can be considered to provide a functional, stable, and esthetic occlusion.

Early adult dentition stage: Third molar position or space can be evaluated and, if indicated, the tooth/teeth removed.

Recommendations

Oral habits

General considerations and principles of management: 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.[17,18(p488)] Habits of sufficient frequency, duration, and intensity may be associated with dentoalveolar or skeletal deformations such as increased overjet, reduced overbite, open bite, posterior crossbite, or increased facial height. The duration of force is more important than its magnitude; the resting pressure from the lips, cheeks, and tongue has the greatest impact on tooth position as these forces are maintained most of the time.[19(p127),20] Oral habits include, but are not limited to, nonnutritive sucking, bruxism, tongue thrust swallow and abnormal tongue position, self-injurious/self-mutilating behavior, and obstructive sleep apnea (**OSA**).

Nonnutritive sucking behaviors are considered normal in infants and young children. Long-term nonnutritive sucking habits (eg, pacifier use, thumb/finger sucking) have been associated with anterior open bite and posterior crossbite.[17,20-24] 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 nonnutritive sucking habits by age 36 months or younger.[17,20,21]

Bruxism, defined as repetitive jaw movements characterized by tooth clenching and grinding, occurs with variable intensity and frequency during periods of sleep and wakefulness.[25(p371)] Sleep bruxism, classified as a sleep-related masticatory muscle activity, has potential physiologic or protective relevance.[26,27] The etiology is multifactorial and has been reported to include emotional stress,[28] traumatic brain injury,[29] neurologic disabilities,[30] and sleep disturbances.[31] The occlusal wear that may result from bruxism is important to differentiate from other forms of occlusal loss of enamel (eg, erosion caused by diet, gastroesophageal reflux, eating disorders).[25(p374),32] Reported complications of bruxism include dental attrition, fractured teeth, tongue indentation, lip or cheek biting, headaches, and temporomandibular joint or facial pain.[25(p372),33] Evidence indicates that juvenile bruxism is self-limiting and does not persist in adults.[34] The spectrum of bruxism management ranges from patient/parent education, occlusal splints, behavioral strategies, and psychological techniques to medications.[35,36]

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.[37] 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.[19(p130),20]

Self-injurious or self-mutilating behavior is a chronic condition involving repetitive acts that result in physical injury to the individual.[38] Such behavior is seen more frequently in populations with special needs, having been associated with developmental delay or disabilities, psychiatric disorders, traumatic brain injuries, and some syndromes (eg, Lesch Nyhan, fragile X, Rett, Cornelia de Lange, Smith-Magenis).[38-40] The spectrum of treatment options for developmentally disabled individuals having oral self-injurious behaviors includes pharmacologic management, behavior modification, physical restraint, and chemodenervation using botulinum toxin.[41,42] 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.[41] Management options include monitoring the lesion, recontouring teeth, providing a bite-opening appliance, or extracting teeth.[41]

Research on the relationship between malocclusion and mouth breathing suggests impaired nasal respiration may contribute to the development of increased facial height, anterior open bite, increased overjet, and narrow palate but that it is not the sole or the major cause of these conditions.[43] Interdisciplinary care with medical colleagues or other dental specialists may be beneficial for these patients.[44]

OSA may be associated with narrow maxilla, crossbite, low tongue position, vertical growth, increased overjet, and open bite.[45-47] Historical findings associated with OSA may include snoring, observed apnea, restless sleep, daytime neurobehavioral abnormalities or sleepiness, and bedwetting.[48] Physical findings may include growth abnormalities, signs of nasal obstruction, adenoidal facies, enlarged tonsils, and hyponasal voice quality, in addition to possible retrognathia, micrognathia, macroglossia, and hypoplasia of the midface.[43,48-50] OSA is more prevalent in, but not limited to, children with craniofacial anomalies, neurologic disorders (eg, cerebral palsy, muscular dystrophy), sickle cell disease, or a history of prematurity.[48]

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. Anticipatory guidance on long-term sequelae should be provided and intervention to terminate the habit should be initiated if indicated.[17,18(p490)]

Treatment considerations: Management of an oral habit is indicated when 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.[18(p490)]

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 1 or more teeth, has a prevalence in the permanent dentition, depending on geographic location, of 4.4% to 13.4%.[51] Excluding third molars, the most frequently missing permanent tooth is the mandibular second premolar followed by the maxillary lateral incisor and then the maxillary second premolar.[51] The maxilla and mandible were found to have equal prevalence of hypodontia, with a higher prevalence in females compared to males.[51] In the primary dentition, hypodontia occurs less frequently (<1% prevalence) and almost always affects the maxillary incisors and first primary molars.[52,53] The association between a missing primary tooth and an increased prevalence of a missing succedaneous tooth is strong.[53] The chance of inherited occurrence of 1 or 2 congenitally missing teeth is to be differentiated from missing lateral incisors in cleft lip/palate[54] and multiple missing teeth (6 or more) due to ectodermal dysplasia or other syndromes[55] as the treatment usually differs. 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.[54,56,57]

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.[18(p515),58(p415),59]

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.[59,60] Early extraction of the primary canine and/or lateral may be needed.[59] 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.[55,59] 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.[57,59] Patients generally prefer space closure over implants.[60]

For a congenitally missing premolar, the primary molar may either be maintained or extracted in conjunction with treatment options such as placement of a prosthesis, orthodontic space closure, or autotransplantation.[61-67] Maintaining the primary second molar may cause occlusal problems due to its larger mesiodistal diameter, compared to the second premolar.[18(p516),59] Reducing the width of the second primary molar is a consideration, but root resorption and subsequent exfoliation may occur.[18(p516),59] In crowded arches or with multiple missing premolars, extraction of the primary molar(s) can be considered, especially mandibular molars in mild Class III cases.[18(p517),59,64] 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[18(p516),62,64,68(p383)] Consideration for extraction and space maintenance may be indicated.[18(p517),62,64] Interdisciplinary team care, including consultation with other dental specialists may be considered.

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

Supernumerary teeth

General considerations and principles of management: Supernumerary teeth, or hyperdontia, can occur in the primary or permanent dentition but are 5 times more common in the permanent.[56,69] Prevalence is reported in the primary dentition from 0.3%-0.8% and the mixed dentition from 0.52% to 2%.[61,66,67,70] Between 80% and 90% of all supernumeraries occur in the maxilla, with half in the anterior area and almost all in the palatal position.[18(p514),67] A supernumerary primary tooth is followed by a supernumerary permanent tooth in one-third of the cases.[71] Supernumerary teeth are classified according to their form and location.[67,72]

During the early mixed dentition, 79% to 91% of anterior permanent supernumerary teeth are unerupted.[57,66] While more erupt with age, only 25% of all mesiodens (a permanent supernumerary incisor located at the midline) erupt spontaneously.[67] 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.[67] If there is an asymmetric eruption pattern of the maxillary incisors, delayed or ectopic eruption of an incisor, or an over-retained primary incisor, a supernumerary tooth can be suspected.[52,54,66] Panoramic, occlusal, and periapical radiographs all can reveal a supernumerary tooth. To determine the supernumerary tooth's position, either a CBCT scan or 2 periapical or occlusal films reviewed by the parallax rule is recommended.[61,67]

Treatment considerations: Management and treatment of hyperdontia differ if the tooth is primary or permanent. Primary supernumerary teeth normally are accommodated into the arch and usually erupt and exfoliate without complications.[71] Surgical extraction of unerupted anterior supernumerary teeth during the primary dentition can displace or damage the permanent incisor.[67] Removal of an erupted mesiodens or other permanent supernumerary incisor results in eruption of the permanent adjacent normal incisor in 75% of the cases.[67] Extraction of an unerupted supernumerary during the early mixed dentition (ie, at age 6 to 7 years when the permanent crown has formed completely and the root lengthis less than the crown height) allows for a normal eruptive force and eruption of the adjacent normal permanent incisor.[61,67,73] Later removal of the mesiodens reduces the likelihood that the adjacent normal permanent incisor will eruption its own, especially if the apex is completed.[67] Inverted conical supernumerary teeth can be harder to remove if removal is delayed, as they can migrate deeper into the jaw.[66] After removal of the supernumerary tooth, clinical and radiographic follow-up is indicated in 6 months to determine if the normal incisor is erupting. If there is no eruption after 6 to 12 months and sufficient space exists, surgical exposure and orthodontic traction may be needed.[67,74,75]

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.[76,77] 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.[76,77]

EE of permanent molars can be diagnosed from bitewing or panoramic radiographs in the early mixed dentition.[76,77] This condition occurs in up to 3% of the population.[77] 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.[76,78,79] EE of second permanent molars occurs infrequently.[80] EE of permanent molars is classified into 2 types: those that self-correct and others that remain impacted. Recent data demonstrated that 71% self-correct by age 9.[81] In some cases, definitive treatment is indicated to manage and/or avoid early loss of the primary second molar and space loss.[76,77] 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.[81]

The maxillary canine appears in an impacted position in 1% to 3% of the population.[82-84] Females are 2 to 3 times more likely to experience canine impaction than males.[85] 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.[84,86-89] 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.[88-91]

Maxillary incisors can erupt ectopically or be impacted from supernumerary teeth in up to 2% of the population.[72] Incisors also can have altered eruption due to pulp necrosis (following trauma or caries) or pulpal treatment of the primary incisor.[92] 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.[84,87]

Treatment considerations: Treatment of 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.[77] For more severe impactions, distal tipping of the permanent molar is required.[77] Tipping action can be accomplished with brass wires, removable appliances using springs, fixed appliances such as sectional wires with open coil springs,[93] slingshot-type appliances,[94] or a Halterman appliance.[95]

Early diagnosis and treatment of impacted maxillary canines are important to reduce the risk of damage to adjacent permanent incisors and improve the chance for a successful intervention.[91,96] Extraction of the primary canine may stimulate eruption of the permanent canine and is indicated when the canine bulge cannot be palpated in the alveolar process and the canine radiographically overlaps the formed root of the lateral during the mixed dentition.[84,97,98] Extraction of the primary canine has been shown to correct the impacted canine in 67%-69% of cases compared to 39%-42% in a non-extraction control group.[99,100] The use of rapid maxillary expansion alone[101,102] or with cervical pull headgear[103] 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% will erupt.[104] 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.[105] This need can be determined from a panoramic radiograph,[106,107] although CBCT will provide greater localization[108] of the impacted canine. 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 non-impacted canines, and data is insufficient to conclude the best type of surgical technique.[109,110]

Treatment of ectopically erupting incisors depends on etiology. Extraction of necrotic or over-retained, pulpally-treated primary incisors is indicated in the early mixed dentition.[92] Removal of supernumerary incisors in the early mixed dentition will lessen ectopic eruption of an adjacent permanent incisor.[67] After incisor eruption, orthodontic treatment 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.[111] The periodontal ligament is replaced with osseous tissue, rendering the tooth immobile to eruptive change.[111] 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 reported incidence in the primary dentition is between 7% and 14%.[112] In the permanent dentition, ankylosis occurs most frequently following luxation injuries.[113]

Ankylosis is common in anterior teeth following trauma (eg, avulsion) or injury to periodontal ligament cells and is the process of pathological fusion of the external root surface to the surrounding alveolar bone.[114] The degree of replacement resorption and infraocclusion contribute to the severity of ankylosis. Over time, normal bony activity may result in the replacement of root structure with osseous tissue.[112,113] Ankylosis can occur rapidly or gradually, in some cases as long as 5 years post trauma, and also may be transient if only a small bony bridge forms then is resorbed with subsequent osteoclastic activity.[114,115]

Ankylosis can be verified by clinical and radiographic means. Submergence of the tooth, or infraocclusion, is the primary recognizable sign, but the diagnosis also can be made through palpation and percussion.[116] Lack of physiologic mobility and the presence of a dull tone (in comparison to adjacent teeth) upon percussion with a metal instrument such as a dental mirror handle are indicative of ankylosis. Intraoral radiographic examination, while limited in its 2-dimensional view, may show the loss of the periodontal ligament, external resorption, and alveolar replacement.[111]

Treatment considerations: Management of an ankylosed primary molar with a successor consists of maintaining it until an interference with eruption or tipping/drifting of adjacent teeth occurs. If associated problems occur, the practitioner should extract the ankylosed primary molar and place a lingual arch or other fixed appliance if needed. Management of ankylosed primary molars without successors should take into consideration the patient's age, specific tooth condition, comprehensive orthodontic treatment plan including future prosthodontic considerations, and parental preferences. If severe infraocclusion is anticipated, ankylosed primary molars without a permanent successor should either undergo extraction before a large vertical occlusal discrepancy develops or decoronation to maintain alveolar width and prevent further loss of vertical height.[58(p408),68(p384)] Decoronation is the removal of the clinical crown and root structure below the soft tissue level and necessitates removal of the remaining vital pulp tissue. It reduces the chance of ridge resorption and the need for bone grafting[14(p454),58(p408)] following a surgical extraction. Decoronation helps preserve bone until an implant can be placed.[117] Extraction of ankylosed primary molars without a succedaneous tooth can assist in resolving crowded arches in complex orthodontic cases.[58(p408,413),118] Consultation with other dental specialists may assist clinicians in their treatment decision making.

Surgical luxation of ankylosed permanent teeth with forced orthodontic eruption has been described as an alternative to premature extraction.[119] Management of ankylosed permanent anterior teeth can include build-up of minor infraocclusion, intentional repositioning (surgical or orthodontic) with splinting, autotransplantation, decoronation,[113,120,121] or extraction with prosthetic rehabilitation. In permanent incisor decoronation, the tooth undergoes endodontic treatment and then removal of the clinical crown and the cervical portion of the root to a level 2 mm below marginal bone height, followed by reflecting, repositioning, and suturing a mucoperiosteal flap over the root.[122] Additional research on management of ankylosed permanent anterior teeth is needed.[114]

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.[123] Failure in eruptive mechanisms prevent permanent successors from following the eruption path after the exfoliation of deciduous teeth.[124] Posterior teeth are most commonly affected, and 1 or all 4 quadrants may be involved.[125] Although typically associated with permanent teeth, examples in the primary dentition have been noted.[126] Two main phenotypes of PFE have been identified: (1) all teeth distal to the most mesial non-erupted tooth are affected, and (2) unerupted teeth do not follow the pattern that all teeth distal to the most mesial involved

tooth are also affected.[127] Hallmark features of PFE include posterior open bite in the presence of normal vertical growth, infraocclusion of affected teeth, and the inability to move affected teeth orthodontically.[128]

The reported incidence of PFE is between 0.01 and 0.06%,[129,130] although some data suggests PFE may be misdiagnosed as infraocclusion or ankylosis.[70,131] PFE differs from ankylosis in that eruption fails to occur due to an imbalance in resorptive and appositional factors related to tooth eruption.[132,133] Teeth with PFE are not initially ankylosed but may become ankylosed when orthodontic forces are applied.[134] A systematic review demonstrated 85% of patients with PFE have a family member with the condition.[134]

Treatment considerations: Diagnosis of PFE should be based on a combination of clinical, radiographic, and genetic information.[132.134] A positive family history also supports a diagnosis of PFE.[127] Other than a few anecdotal reports, PFE is strongly associated with the failure of orthodontically-assisted eruption or tooth movement.[127,128] To that point, early orthodontic intervention of the affected teeth should be avoided.[128,132,133,135] To date there are no established mechanotherapeutic methods of modifying dentoalveolar growth for these patients.[128,132,133,135] Space maintenance, uprighting adjacent teeth that have tipped into the sites, prevention of supraeruption in opposing arch, or modification of lateral tongue thrust habits may be additional considerations.[128,135] Once growth is complete, multidisciplinary treatment options (eg, single tooth or segmental osteotomies with immediate traction, selective extractions followed by implants) can be considered to create a functioning occlusion.[132] Early extraction of first molars allowing the second molars to drift forward also has been suggested.[128]

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.[128] In some cases, early extraction can improve normal development of the alveolus and permanent dentition.[128] Objectives include space and intraarch maintenance in preparation for future implants, prosthetic rehabilitation, or corticotomy-assisted tooth movement.[128]

Tooth size/arch length discrepancy and crowding

General considerations and principles of management: Tooth size/arch length discrepancy (TSALD) is defined as the difference between the space required to align teeth and the space available in each arch.[136] Arch length discrepancies include inadequate arch length (crowding), excess arch length (spacing), and tooth size discrepancy (referred to as a Bolton discrepancy.)[137] These arch length discrepancies may be found in conjunction with other etiological factors including missing, supernumerary, fused, and geminated teeth. Inadequate arch length with resulting incisor crowding is a common occurrence, particularly in the early mixed dentition.[138,139] Studies of arch length in contemporary children suggest less arch length, more frequent incisor crowding, and slightly larger tooth sizes compared to children measured in past decades.[140-142] This implies that the amount of incisor crowding may be increasing in present-day patients creating more arch length shortage.[142,143]

Arch length and 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.[144,145] Growth of the aging skeleton causes further crowding and incisor rotations.[146] Functional contacts are diminished where rotations of incisors, canines, and premolars exist.[147] Therefore, aligning crowded and rotated incisors in the early mixed dentition is of interest.

Initial assessment of arch length may be performed in early mixed dentition when mandibular incisors begin to erupt.[144] Evaluation of available space and consideration of making space for permanent incisors includes a comprehensive clinical examination with radiographs to ascertain the presence of successors and a thorough diagnostic analysis.

A systematic review assessing the physiologic behavior of lower incisor position from the early mixed to the permanent dentition in children beginning between ages 7 and 9 over a 4-to-6-year period demonstrated mandibular incisor crowding tended to decrease over time and the amount of reduction was related to the initial amount of incisor crowding.[148] Data suggest the greater the amount of crowding in the mixed dentition, the greater its self-correction in the permanent dentition.[148] The systematic review cautioned against early orthodontic treatment due to self-correction; however, there was great variability in the data, and several patients showed increased crowding over time.[148] A passive lingual arch was suggested if the clinician was concerned about the worsening in crowding over time.[148]

Some have suggested removal of mandibular primary canines to provide permanent incisors more room to align in a favorable position.[99,149] While the decrease in incisor irregularity indices is statistically significant, this comes at the expense of a statistically significant loss in arch length.[150,151] The loss in arch length is reported to be up to 2.7 mm. Studies suggest one should consider whether the decrease in lower irregularity index loss is more important than the loss of arch length before removing lower primary canines.[150]

Because premature loss of a mandibular primary canine has been reported to lead to an immediate midline shift, removal of the contralateral primary canine to correct the shift is often recommended.[18(p476)] However, a review of patients in the Iowa and Toronto growth studies found the lower dental midline did not change significantly after premature unilateral loss of a primary canine.[152,153]

If primary mandibular canines are removed to resolve lower incisor alignment problems, placement of a passive lingual arch has been suggested.[154] In approximately 9 of 10 patients, all permanent teeth can be accommodated if arch length is expanded 2 mm after a passive lingual arch is employed to save leeway space.[154] Sixty percent of patients will have adequate

space for tooth alignment without any arch expansion following the same protocol.[154] Early placement of a passive lingual arch can increase the long term stability of lower incisor position.[144]

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

- 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.
- orthodontic alignment of permanent teeth as soon as erupted and feasible, expansion and correction of arch length as early as feasible.
- utilizing holding arches in the mixed dentition to manage leeway space until all premolars and permanent canines have erupted.
- maintaining patient's original arch form.[147]
- interproximal stripping of the enamel of mandibular primary canines to allow alignment of crowded lower permanent lateral incisors.[155]

Additional treatment modalities may include but are not limited to interproximal reduction; restorative bonding; veneers; crowns; implants; and orthognathic surgery.

Treatment objectives: Well-timed intervention should be directed towards improved positioning of succedaneous teeth by arch length preservation and, when indicated, space gaining. In cases where normal alignment or spontaneous eruption does not occur, further orthodontic treatment may be indicated.

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.[156] Whenever possible, restoration of carious primary teeth should be attempted to return the tooth to its original morphology to avoid malocclusions that could result from space loss due to an under contoured restoration or extraction.[157] The use of space maintainers to reduce the prevalence and severity of malocclusion following premature loss of primary teeth should be considered.[18(p474),139,158]

Adverse effects associated with space maintainers include dislodged, broken, and lost appliances; plaque accumulation; caries; damage or interference with successor eruption; undesirable tooth movement; inhibition of alveolar growth; soft tissue impingement; and pain.[156,159-165] Premature loss of a primary tooth has the potential to cause loss of space available for the succeeding permanent tooth, but consensus or evidence regarding the effectiveness of space maintainers in preventing or reducing the severity of malocclusion is lacking.[156]

Treatment considerations: It is prudent to consider space maintenance when primary teeth are lost prematurely. Factors to consider include specific tooth lost; time elapsed since tooth loss; occlusion and space assessment; dental age; presence and root development of permanent successor; amount of alveolar bone covering permanent successor; patient's health history and medical status; patient's cooperative ability; active oral habits; and oral hygiene.[18(pp473,474),156,157]

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.[139,158] 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).[166] 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.[167-170] A systematic review of space maintenance for premature loss of a primary molar showed mean survival times for all (except crown and loop) space maintainers did not exceed 2 years; decementation or loss of cement was the most common reason for failure.[171]

The placement and retention of space maintaining appliances requires ongoing compliant patient behavior. Periodic assessment of appliance integrity and retention as well as monitoring the succedaneous tooth's eruption help maximize effectiveness. Clinicians should prepare for repair, recementation, or replacement of a space maintainer. If the appliance is cemented, the band and cement should be inspected to prevent problems with leakage due to inadequate seals.[160] The appliance should function until the permanent tooth has erupted into the correct position.[15(p522)] Appliance adjustment or a new appliance may be necessary with continued growth and changes in the developing 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.[18(p473),139]

Space regaining

General considerations and principles of management: Some of the more common causes of space loss within an arch are primary teeth with interproximal caries; ectopically erupting teeth; alteration in the sequence of eruption; ankylosis of a primary molar; dental impaction; transposition of teeth; loss of primary molars without proper space management; congenitally missing teeth; abnormal resorption of primary molar roots; premature and delayed eruption of permanent teeth;

and abnormal dental morphology.[18(pp473,474),68(p362),156,158] 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.[172] The quantity and incidence of space loss are dependent upon which adjacent teeth are present in the dental arch and their status.[18(p470),156] The amount of crowding or spacing in the dental arch will determine the consequence of space loss.[68(p362)]

Treatment considerations: Space can be maintained or regained with removable or fixed appliances.[139,156] Some examples of fixed space regaining appliances are active lingual arches, pendulum appliances, Halterman-type appliances, and lip bumpers. Examples of removable space regaining appliances are Hawley appliance with springs or screws and extraoral appliances such as headgear.[139] If space regaining is planned, a diagnostic analysis including records should be completed prior to any treatment decisions. The clinician should consider dentofacial development, age at time of tooth loss, the tooth that has been lost, the space available, and space needed in addition to other existing orthodontic problems.[139,156]

Treatment objectives: The goal of space regaining intervention is the recovery of lost arch width and perimeter to improved the eruptive position of succedaneous teeth. Regained space should be maintained until adjacent permanent teeth have erupted completely[173] 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.[174-176] If the midlines undergo a compensatory or habitual shift when the teeth occlude in crossbite, this is termed a functional shift.[18(pp500-501),173] A crossbite can be of dental or skeletal origin, or a combination of both.[8(p201),18(p498)]

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.[174] 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.[18(p500),175] Dental crossbites may be the result of tipping or rotation of a tooth or teeth.[8(p201)] In this case, the condition is localized and does not involve the basal bone. In contrast, skeletal crossbites involve disharmony of the craniofacial skeleton.[18(p500),175,177] Aberrations in bony growth may give rise to crossbites in 2 ways: (1) adverse transverse growth of the maxilla and mandible, and (2) disharmonious or adverse growth in the sagittal (AP) length of the maxilla and mandible.[176,178]

Such growth aberrations can be due to inherited growth patterns, trauma, or functional disturbances that alter normal growth.[175,177,178]

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 temporomandibular disorder (TMD).[18(p494),177,179] 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.[176] 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.[37,180,181] Functional shifts should be eliminated as soon as possible with early correction[178] to avoid temporomandibular disorder and/or asymmetric growth.[175,182] Treatment can be completed with

- equilibration.
- appliance therapy (fixed or removable).
- extractions.
- a combination of these treatment modalities to correct the alveolar constriction.[182]

Skeletal expansion with fixed or removable palatal expanders can be utilized until midline suture fusion occurs.[68(p364),174] Treatment decisions depend on the

- amount and type of movement (tipping versus bodily movement, rotation, or dental versus orthopedic movement).
- space available.
- AP, transverse, and vertical skeletal relationships.
- growth status.
- 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.[181]

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.[183(p455)]

Results of randomized clinical trials indicate that Class II malocclusion can be corrected effectively with either a single or 2-phase regimen.[184-187] Growth-modifying effects in some studies did not show an influence on the Class II skeletal pattern,[185,188,189] while other studies dispute these findings.[190,191] 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.[177,184] Some reports state interceptive treatment does not reduce the need for either premolar extractions or orthognathic surgery,[185,187] while others disagree with these findings.[192] Two-phase treatment results in significantly longer treatment time,[177,187,193] although the time spent in full bonded appliance therapy in the permanent dentition can be significantly less.[194]

Clinicians may decide to provide interceptive treatment based on other factors.[186,190] Evidence suggests that, for some children, interceptive Class II treatment may improve self-esteem and decreases negative social experiences, although the improvement may not be different longterm.[190,195] Early Class II correction may improve facial convexity and/or reduce incidence of maxillary anterior tooth trauma.[196,197] An overjet in excess of 3 mm is associated with an increased risk of incisor injury, with large overjets (>8 mm) resulting in trauma in more than 40% of children.[198,199] Interceptive treatment in patients with an overjet of 5 mm or greater has the potential to decrease the risk of traumatic injury to the permanent incisors.[200]

Treatment considerations: Factors to consider when planning orthodontic intervention for Class II malocclusion are facial growth pattern; amount of AP discrepancy; patient age; projected patient compliance; space analysis; and anchorage requirements. Treatment modalities include extraoral appliances/headgear; functional appliances; fixed appliances; tooth extraction and interarch elastics; and orthodontics with orthognathic surgery.[18(pp520-524),183(pp457-62),201]

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.[8(p149),168, 201]

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.[195,196] Some environmental factors are trauma, oral/digital habits, caries, and early childhood OSA.[204(p440)]

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.[205] 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.[206-214] Factors to consider when planning orthodontic intervention for Class III malocclusion are: (1) facial growth pattern; (2) amount of AP discrepancy; (3) patient age; (4) projected patient compliance; (5) space analysis.; and (6) anchorage requirements.

Treatment objectives: Interceptive Class III treatment may provide a more favorable environment for growth and may improve occlusion, function, and esthetics.[128] 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, a surgical procedure combined with orthodontics may be the best alternative to achieve a satisfactory result for some patients, especially if they exhibit facial characteristics as follow: mandible forward to cranial base, increase mandibular length, short ramal length, or obtuse gonial angle.[75,215-217]

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.[204(pp430-452),206-209,218-222] These interventions in a growing patient should result in improved overbite, overjet, and intercuspation 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. American Academy of Pediatric Dentistry; 1990.

References continued on the next page.

2. American Academy of Pediatric Dentistry. Management of the developing dentition and occlusion in pediatric dentistry. *The Reference Manual of Pediatric Dentistry.* American Academy of Pediatric Dentistry; 2021:408-425.
3. Kurol J. Early treatment of tooth-eruption disturbances. *Am J Orthod Dentofacial Orthop.* 2002;121(6):588-591. DOI: 10.1067/mod.2002.124173
4. Sankey WL, Buschang PH, English J, Owen AH III. Early treatment of vertical skeletal dysplasia: The hyperdivergent phenotype. *Am J Orthod Dentofacial Orthop.* 2000; 118(3):317-327. DOI: 10.1067/mod.2000.106068
5. Woodside DG. The significance of late developmental crowding to early treatment planning for incisor crowding. *Am J Orthod Dentofacial Orthop.* 2000;117(5):559-561. DOI: 10.1016/s0889-5406(00)70199-5
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. *The Reference Manual of Pediatric Dentistry.* American Academy of Pediatric Dentistry; 2024:23-24.
7. Proffit WR, Fields HW, Jr, Larson BE, Sarver DM. Orthodontic treatment planning: From problem list to specific plan. In: Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM, eds. *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:208-229.
8. 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. Elsevier; 2019:140-201.
9. Proffit WR. Early stages of development. In: Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM, eds. *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:65-77.
10. Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM. Introduction to diagnosis and treatment planning. In: *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:137.
11. Proffit WR. Later stages of development. In: Proffit WR, Fields HW, Jr, Larson BE, Sarver DM, eds. *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:84.
12. American Dental Association; US Department of Health and Human Services. *The Selection of Patients for Dental Radiographic Examinations: Recommendations for Patient Selection and Limiting Radiation Exposure*. US Food and Drug Administration; 2012. Accessed February 24, 2024. https://www.fda.gov/radiation-emitting-products/medical-x-ray-imaging/selection-patients-dental-radiographic-examinations
13. Quiñonez RB, Christensen JR, Fields H. Examination, diagnosis, and treatment planning. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy Through Adolescence.* 6th ed. Elsevier; 2019:273-276.
14. Schwartz SB, Christensen JR. Examination, diagnosis, and treatment planning. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry: Infancy through Adolescence.* 6th ed. Elsevier; 2019: 423-454.
15. Christensen JR, Fields HW, Jr, Sheats RD. Treatment planning and management of orthodontic problems. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy Through Adolescence.* 6th ed. Elsevier; 2019:522-524.
16. Brecher E, Stark TR, Christensen JR, Sheats RD, Fields H. Examination, diagnosis, and treatment planning for general and orthodontic problems. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy Through Adolescence.* 6th ed. Elsevier; 2019:567-575.
17. 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;13 2(12):1685-1693. DOI: 10.14219/jada.archive.2001.0121
18. Dean JA, Walsh JS. Managing the developing occlusion. In: Dean JA, ed. *McDonald and Avery's Dentistry for the Child and Adolescent.* 11th ed. Elsevier; 2022:470-524.
19. Proffit WR. The etiology of orthodontic problems. In: Proffit WR, Fields HW, Jr, Larson BE, Sarver DM, eds. *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:127-130.
20. 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):161-166. DOI: 10.1016/S0889-5406(94)70034-6
21. Warren JJ, Bishara SE. Duration of nutritive and nonnutritive sucking behaviors and their effects on the dental arches in the primary dentition. *Am J Orthod Dentofacial Orthop.* 2002;121(4):347-356. DOI: 10.1067/mod.2002.121445
22. 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):437-444.
23. 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):926-934. DOI: 10.1016/j.adaj.2016.08.018
24. 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. DOI: 10.1016/j.ajodo.2008.09.029
25. American Academy of Sleep Medicine. Sleep related bruxism. In: *International Classification of Sleep Disorders.* 3rd ed., text revision. American Academy of Sleep Medicine; 2023:371-374.
26. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil.* 2018;45(11):837-844. DOI: 10.1111/joor.12663

27. Manfredini D, Ahlberg J, Lobbezoo F. Bruxism definition: Past, present, and future – What should a prosthodontist know? *J Prosthet Dent.* 2022;128(5):905-912. DOI: 10.1016/j.prosdent.2021.01.026
28. Monaco A, Ciammella NM, Marci MC, Pirro R, Giannoni M. The anxiety in bruxer child: A case-control study. *Minverva Stomatol.* 2002;51(6):247-250.
29. Ivanhoe CB, Lai JM, Francisco GE. Bruxism after brain injury: Successful treatment with botulinum toxin-A. *Arch Phys Med Rehabil.* 1997;78(11):1272-1273. DOI: 10.1016/s0003-9993(97)90343-9
30. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. *Adv Neurol.* 1988;49:329-341.
31. Castroflorio T, Bargellini A, Rossini G, Cugliari G, Rainoldi A, Deregibus A. Risk factors related to sleep bruxism in children: A systematic literature review. *Arch Oral Biol.* 2015;60(11):1618-1624. DOI: 10.1016/j.archoralbio.2015.08.014
32. Taji S, Seow WK. A literature review of dental erosion in children. *Aust Dent J.* 2010;55(4):358-367. DOI: 10.1111/j.1834-7819.2010.01255.x
33. Ierardo G, Mazur M, Luzzi V, Calcagnile F, Ottolenghi L, Polimeni A. Treatments of sleep bruxism in children: A systematic review and meta-analysis. *Cranio.* 2021;39(1):58-64. DOI: 10.1080/08869634.2019.1581470
34. Kieser JA, Groeneveld HT. Relationship between juvenile bruxing and craniomandibular dysfunction. *J Oral Rehabil.* 1998;25(9):662-665. DOI: 10.1046/j.1365-2842.1998.00304.x
35. Casazza E, Giraudeau A, Payet A, Orthlieb JD, Camoin A. Management of idiopathic sleep bruxism in children and adolescents: A systematic review of the literature. *Arch Pediatr.* 2022;29(1):12-20. DOI: 10.1016/j.arcped.2021.11.014
36. Bulanda S, Ilczuk-Rypuła D, Nitecka-Buchta A, Nowak Z, Baron S, Postek-Stefańska L. Sleep bruxism in children: Etiology, diagnosis, and treatment–A literature review. *Int J Environ Res Public Health.* 2021;18(18):9544. DOI: 10.3390/ijerph18189544
37. Bell RA, Kiebach TJ. Posterior crossbites in children: Developmental based diagnosis and implications to normative growth patterns. *Semin Orthod.* 2014;20(2):77-113. DOI: 10.1053/j.sodo.2014.04.002
38. Rosenberg DR, Chiriboga JA. Anxiety disorders. In: Kliegman R, Stanton B, St Geme JW, Schor NF, Behrman RE, eds. *Nelson Textbook of Pediatrics.* 21st ed. Elsevier; 2020:209.
39. Saemundsson SR, Roberts MW. Oral self-injurious behavior in the developmentally disabled: Review and a case. *ASDC J Dent Child.* 1997;64(3):205-209.
40. 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):282-287. DOI: 10.1111/scd.12181
41. Millwood J, Fiske J. Lip biting in patients with profound neurodisability. *Dent Update.* 2001;28(2):105-108. DOI: 10.12968/denu.2001.28.2.105
42. Garcia-Romero MDM, Torres RJ, Garcia-Puig J, Pascual-Pascual SI. Safety and efficacy of botulinim toxin in the treatment of self-biting behavior in Lesch-Nyhan disease. *Pediatr Neurol.* 2022;127:6-10. DOI: 10.1016/j.pediatrneurol.2021.10.018
43. 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):147-154. DOI: 10.1016/0889-5406(91)70117-F
44. Grippaudo C, Paolantonio EG, Antonini G, Saulle R, La Torre G, Deli R. Association between oral habits, mouth breathing and malocclusion. *Acta Otorhinolaryngol Ital.* 2016;36(5):386-394. DOI: 10.14639/0392-100X-770
45. Katyal 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):860-871. DOI: 10.1016/j.ajodo.2013.08.015
46. 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):662-671. DOI: 10.1093/ejo/cjp162
47. 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):160-167. DOI: 10.1093/ejo/cjn061
48. Bitners AC, Arens R. Evaluation and management of children with obstructive sleep apnea syndrome. *Lung.* 2020;198(2):257-270. DOI: 10.1007/s00408-020-00342-5
49. Marcus CL, Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics.* 2012;130(3):e714-755. DOI: 10.1542/peds.2012-1671
50. Ward T, Mason TB II. Sleep disorders in children. *Nurs Clin North Am.* 2002;37(4):693-706. DOI: 10.1016/s0029-6465(02)00032-4
51. Khalaf K, Miskelly J, Voge E, Macfarlane TV. Prevalence of hypodontia and associated factors: A systematic review and meta-analysis. *J Orthod.* 2014;41(4):299-316. DOI: 10.1179/1465313314Y.0000000116
52. 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.
53. Neville BW, Damm DD, Allen CM, Chi AC. Abnormalities of teeth. In: *Oral and Maxillofacial Pathology.* 4th ed. Elsevier; 2016:72.
54. Shapira Y, Lubit E, Kuftinec MM. Hypodontia in children with various types of clefts. *Angle Orthod.* 2000;70(1):16-21. DOI: 10.1043/0003-3219(2000)070<0016:HICWVT>2.0.CO;2

References continued on the next page.

55. Worsaae N, Jensen BN, Holm B, Holsko J. Treatment of severe hypodontia-oligodontia—An interdisciplinary concept. *Int J Oral Maxillofac Surg.* 2007;36(6):473-480. DOI: 10.1016/j.ijom.2007.01.021
56. Garib DG, Peck S, Gomes SC. Increased occurrence of dental anomalies associated with second-premolar agenesis. *Angle Orthod.* 2009;79(3):436-441. DOI: 10.2319/021308-87.1
57. 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. DOI: 10.1093/ejo/22.6.697
58. Fields HW, Jr, Proffit WR. Complex nonskeletal problems in preadolescent children: Preventive and interceptive treatment. In: Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM, eds. *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:408-415.
59. Spear FM, Mathews DM, Kokich VG. Interdisciplinary management of single-tooth implants. *Semin Orthod.* 1997;3(1):45-72. DOI: 10.1016/s1073-8746(97)80039-4
60. 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):416-442. DOI: 10.1016/j.ajodo.2016.02.025
61. He D, Mei L, Wang Y, Li J, Li H. Association between maxillary ankylosed anterior supernumerary teeth and impacted incisors in the mixed dentition. *J Am Dent Assoc.* 2017;148(8):595-603. DOI: 10.1016/j.adaj.2017.05.017
62. Kennedy DB. Review: Treatment strategies for ankylosed primary molars. *Eur Arch Paediatr Dent.* 2009;10(4):201-210. DOI: 10.1007/BF03262683
63. 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):561-567. DOI: 10.2319/070813-498.1
64. Kokich VG, Kokich VO. Congenitally missing mandibular second premolars: Clinical options. *Am J Orthod Dentofacial Orthop.* 2006;130(4):437-444. DOI: 10.1016/j.ajodo.2006.05.025
65. 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):329-334. DOI: 10.17796/jcpd.36.4.n3013h2j15v35030
66. Primosch RE. Anterior supernumerary teeth: Assessment and surgical intervention in children. *Pediatr Dent.* 1981;3(2):204-215.
67. Russell KA, Folwarczna MA. Mesiodens: Diagnosis and management of a common supernumerary tooth. *J Can Dent Assoc.* 2003;69(6):362-366.
68. Fields HW, Jr, Proffit WR. Moderate nonskeletal problems in preadolescent children: Preventive and interceptive treatment in family practice. In: Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM, eds. *Contemporary Orthodontics.* 6th ed. Elsevier; 2019:362-384.
69. Hartsfield JK, Jr, Morford LA. Acquired and developmental disturbances of the teeth and associated oral structures. In: Dean JA, ed. *McDonald and Avery's Dentistry for the Child and Adolescent.* 11th ed. Elsevier; 2022:69.
70. Anthonappa RP, King NM. Prevalence of supernumerary teeth based on panoramic radiographs revisited. *Pediatr Dent.* 2013;35(3):257-261.
71. Taylor GS. Characteristics of supernumerary teeth in the primary and permanent dentition. *Dent Pract Dent Rec.* 1972;22(5):203-208.
72. Garvey MT, Barry HJ, Blake M. Supernumerary teeth – An overview of classification, diagnosis and management. *J Can Dent Assoc.* 1999;65(11):612-616.
73. 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.
74. Ayers E, Kennedy D, Wiebe C. Clinical recommendations for management of mesiodens and unerupted permanent maxillary incisors. *Eur Arch Pediatr Dent.* 2014;15(6):421-428. DOI: 10.1007/s40368-014-0132-1
75. Foley J. Surgical removal of supernumerary teeth and the fate of incisor eruption. *Eur J Paediatr Dent.* 2004;5(1):35-40.
76. 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):610-615. DOI: 10.1043/0003-3219(2005)75[610:EEOTMF]2.0.CO;2
77. Yaseen SM, Naik S, Uloopr KS. Ectopic eruption – A review and case report. *Contemp Clin Dent.* 2011;2(1):3-7. DOI: 10.4103/0976-237X.79289
78. 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):179-188.
79. 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.
80. Hwang S, Choi YJ, Lee JY, Chung C, Kim KH. Ectopic eruption of maxillary second molar: Predictive factors. *Angle Orthod.* 2017;87(4):583-589. DOI: 10.2319/020917-95
81. 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):215-218.
82. Almasoud NN. Extraction of primary canines for interceptive orthodontic treatment of palatally displaced permanent canines: A systematic review. *Angle Orthod.* 2017;87(6):878-885. DOI: 10.2319/021417-105.1
83. Naoumova J, Kurol J, Kjellberg H. A systematic review of the interceptive treatment of palatally displaced maxillary canines. *Eur J Orthod.* 2011;33(2):143-149. DOI: 10.1093/ejo/cjq045

84. Richardson G, Russell KA. A review of impacted permanent maxillary cuspids–Diagnosis and prevention. *J Can Dent Assoc.* 2000;66(9):497-501.
85. Becker A, Chaushu S. Etiology of maxillary canine impaction: A review. *Am J Orthod Dentofac Orthop.* 2015; 148(4):557-567. DOI: 10.1016/j.ajodo.2015.06.013
86. Uribe P, Ransjo M, Westerlund AG. Clinical predictors of maxillary canine impaction: A novel approach using multivariate analysis. *Eur J Orthod.* 2017;39(2):153-160. DOI: 10.1093/ejo/cjw042
87. Sachan A, Chatunedi TP. Orthodontic management of buccally erupted ectopic canine with two case reports. *Contemp Clin Dent.* 2012;3(1):123-128. DOI: 10.4103/0976-237X.94563
88. Sherwood K. Evidence-based surgical-orthodontic management of impacted teeth. *Atlas Oral Maxillofac Surg Clin North Am.* 2013;21(2):199-210. DOI: 10.1016/j.cxom.2013.05.006
89. Garib DG, Leonardi M, Giuntini V, Alencar BM, Lauris JRP, Bacetti T. Agenesis of maxillary lateral incisors and associated dental anomalies. *Am J Orthod and Dentofacial Orthop.* 2010;137(6):732.e1-6. DOI: 10.1016/j.ajodo.2009.12.024
90. Bacetti T, Leonardi M, Giuntini V. Distally displaced premolars: A dental anomaly associated with palatally-displaced canines. *Am J Orthod Dentofacial Orthoped.* 2010;138(3):318e22. DOI: 10.1016/j.ajodo.2008.09.036
91. Raes M, Cadenas De Llano-Pérula M, Alqerban A, Laenen A, Verdonck A, Willems G. Prediction of maxillary canine impaction based on panoramic radiographs. *Clin Exp Dent Res.* 2020;6(1):44-50. DOI: 10.1002/cre2.246
92. Coll JA, Sadrian R. Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition. *Pediatr Dent.* 1996;18(1):57-63.
93. 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.
94. Gehm S, Crespi PV. Management of ectopic eruption of permanent molars. *Compend Cont Educ Dent.* 1997;18(6):561-569.
95. Halterman CW. A simple technique for the treatment of ectopically erupting first permanent molars. *J Am Dent Assoc.* 1982;105(6):1031-1033. DOI: 10.14219/jada.archive.1982.0381
96. American Academy of Pediatric Dentistry. Management considerations for pediatric oral surgery and oral pathology. *The Reference Manual of Pediatric Dentistry.* American Academy of Pediatric Dentistry; 2024:523-532.
97. Bedoya MM, Park JH. A review of the diagnosis and management of impacted maxillary canines. *J Am Dent Assoc.* 2009;140(12):1485-1493. DOI: 10.14219/jada.archive.2009.0099
98. Litsas G, Acar A. A review of early displaced maxillary canines: Etiology, diagnosis and interceptive treatment. *Open Dent J.* 2011;5(3):39-47. DOI: 10.2174/1874210601105010039
99. Bazargani F, Magnuson A, Lennartsson B. Effect of interceptive extraction of deciduous canine on palatally displaced maxillary canine: A prospective randomized controlled study. *Angle Orthod.* 2014;84(1):3-10. DOI: 10.2319/031013-205.1
100. Naoumova J, Kurol J, Kjellberg H. Extraction of the deciduous canine as an interceptive treatment in children with palatal displaced canines – Part I: Shall we extract the deciduous canine or not? *Eur J Orthod.* 2015;37(2):209-218. DOI: 10.1093/ejo/cju040
101. 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 Orthod Dentofacial Orthop.* 2009;136(5):657-661. DOI: 10.1016/j.ajodo.2008.03.019
102. O'Neill J. Maxillary expansion as an interceptive treatment for impacted canines. *Evid Based Dent.* 2010;11(3):86-87. DOI: 10.1038/sj.ebd.6400742
103. Ami P, Cozza P, Baccetti T. Effect of RME and headgear treatment on the eruption of palatally-displaced canines: A randomized clinical study. *Angle Orthod.* 2011;81(3):370-374. DOI: 10.2319/062210-339.1
104. Olive RJ. Orthodontic treatment of palatally impacted maxillary canines. *Aust Orthod J.* 2002;18(2):64-70.
105. Alessandri Bonetti G, 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):407-412.
106. Alessandri Bonetti G, Zanarini 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 Orthod Dentofacial Orthop.* 2011;139(3):316-323. DOI: 10.1016/j.ajodo.2009.03.051
107. D'Amico RM, Bjerklin K, Kurol J, Falahat B. Long-term results of orthodontic treatment of impacted maxillary canines. *Angle Orthod.* 2003;73(3):231-238. DOI: 10.1043/0003-3219(2003)073<0231:LROOTO>2.0.CO;2
108. Serrant PS, McIntyre GT, Thomson DJ. Localization of ectopic maxillary canines—Is CBCT more accurate than conventional horizontal or vertical parallax? *J Orthod.* 2014;41(1):13-18. DOI: 10.1179/1465313313Y.0000000076
109. Incerti-Pareti 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 Orthod Dentofacial Orthop.* 2016;149(4):463-472. DOI: 10.1016/j.ajodo.2015.10.019
110. 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 Orthod Dentofacial Orthop.* 2013;144(2):176-184. DOI: 10.1016/j.ajodo.2013.03.016
111. 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 Orthod.* 2018;40(3):231-238. DOI: 10.1093/ejo/cjx063

References continued on the next page.

112. McKibben DR, Brearley LJ. Radiographic determination of the prevalence of selected dental anomalies in children. *ASDC J Dent Child.* 1971;28(6):390-398.
113. Malmgren B, Malmgren O, Andreasen JO. Long-term follow up of 103 ankylosed permanent incisors surgically treated with decoronation–A retrospective cohort study. *Dent Traumatol.* 2015;31(3):184-189. DOI: 10.1111/edt.12166
114. de Souza RF, Travess H, Newton T, Marchesan MA. Interventions for treating traumatized ankylosed permanent front teeth. *Cochrane Database Syst Rev.* 2015;(12):CD007820. DOI: 10.1002/14651858.CD007820.pub3
115. Kokich VO. Congenitally missing teeth: Orthodontic management in the adolescent patient. *Am J Orthod Dentofacial Orthop.* 2002;121(6):594-595. DOI: 10.1067/mod.2002.124174
116. Mishra SK, Jindal MK, Singh RP, Stark TR. Submerged and impacted primary molars. *Int J Clin Pediatr Dent.* 2010;3(3):211-213. DOI: 10.5005/jp-journals-10005-1080i
117. Hua L, Thomas M, Bhatia S, Bowkett A, Merrett S. To extract or not to extract? Management of infraoccluded second primary molars without successors. *Br Dent J.* 2019;227(2):93-98. DOI: 10.1038/s41415-019-0207-9
118. Sabri R. Management of congenitally missing second premolars with orthodontics and single-tooth implants. *Am J Orthod Dentofacial Orthop.* 2004;125(5):634-642. DOI: 10.1016/j.ajodo.2003.05.010
119. 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):543-548. DOI: 10.1016/S0889-5406(94)70078-8
120. Malmgren B. Ridge preservation/decoronation. *Pediatr Dent.* 2013;35(2):164-169.
121. Sapir S, Shapira J. Decoronation for the management of ankylosed young permanent tooth. *Dent Traumatol.* 2008;24(1):131-135. DOI: 10.1111/j.1600-9657.2006.00506.x
122. Malmgren B, Malmgren O, Andersson L. Dentoalveolar ankylosis, decoronation, and alveolar bone preservation. In: Andreasen JO, Andreasen FM, Andersson L, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*. 5th ed. Wiley-Blackwell; 2019:852.
123. Proffit WR, Vig KW. Primary failure of eruption: A possible cause of posterior open bite. *Am J Orthod.* 1981;80(2):73-90. DOI: 10.1016/0002-9416(81)90217-7
124. Mubeen S, Seehrab J. Failure of eruption of first permanent molar teeth: A diagnostic challenge. *J Orthod.* 2018;45(2):129-134. DOI: 10.1080/14653125.2018.1462902
125. Hanisch M, Hanisch L, Kleinheinz J, Jung S. Primary failure of eruption (PFE): A systematic review. *Head Face Med.* 2018;14(1):5. DOI: 10.1186/s13005-018-0163-7
126. Ahmad S, Brister D, Cobourne MT. The clinical features and aetiological basis of primary eruption failure. *Eur J Orthod.* 2006;28(6):535-540. DOI: 10.1093/ejo/cjl033
127. Hartsfield JK, Jacob GJ, Morford LA. Heredity, genetics and orthodontics: How much has this research really helped? *Semin Orthod.* 2017;23(4):336-347. DOI: 10.1053/j.sodo.2017.07.003
128. 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. https://doi.org/10.1053/j.sodo.2015.10.006
129. Baccetti T. Tooth anomalies associated with failure of eruption of first and second permanent molars. *Am J Orthod Dentofacial Orthop.* 2000;118(6):608-610. DOI: 10.1067/mod.2000.97938
130. Grover PS, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. *Oral Surg Oral Med Oral Pathol.* 1985;9(4):420-425. DOI: 10.1016/0030-4220(85)90070-2
131. 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):226-239. DOI: 10.1007/s00056-014-0215-y
132. Frazier-Bowers SA, Puranik CP, Mahaney MC. The etiology of eruption disorders—Further evidence of a "genetic paradigm". *Sem Orthod.* 2010;16(3):180-185. DOI: 10.1053/j.sodo.2010.05.003
133. Frazier-Bowers SA, Simmons D, Wright JT, Proffit WR, Ackerman J. Primary failure of eruption and PTH1R: The importance of a genetic diagnosis for orthodontic treatment planning. *Am J Orthod Dentofacial Orthop.* 2010;137(2):160.e1-160.e7; discussion 160-161. DOI: 10.1016/j.ajodo.2009.10.019.
134. 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. DOI: 10.1016/j.ajodo.2013.03.015
135. Grippaudoa C, Cafierob C, D'Apolitoc I, Riccic B, Frazier-Bowers SA. Primary failure of eruption: Clinical and genetic findings in the mixed dentition. *Angle Orthod.* 2018;88(3):275-282. DOI: 10.2319/062717-430.1
136. Correia GD, Habib FA, Vogel CJ. Tooth-size discrepancy: A comparison between manual and digital methods. *Dent Press J Orthod* 2014;19:107-113. DOI: 10.1590/2176-9451.19.4.107-113.oar
137. Bolton WA. The clinical application of a tooth-size analysis. *Am J Orthod.* 1962;48(7):504-529. https://doi.org/10.1016/0002-9416(62)90129-X
138. Carneiro DP, Venezian GC, Valdrighi HC, de Castro Meneghim M, Vedovello SA. Esthetic impact of maxillary midline diastema and mandibular crowding in children in the mixed dentition. *Am J Orthod Dentofacial Orthop.* 2022;161(3):390-395. DOI: 10.1016/j.ajodo.2020.09.031

139. Ngan P, Alkire RG, Fields HW, Jr. Management of space problems in the primary and mixed dentitions. *J Am Dent Assoc.* 1999;130(9):1330-1339. DOI: 10.14219/jada.archive.1999.0403
140. Allen TR, Trojan TM, Harris EF. Evidence favoring a secular reduction in mandibular leeway space. *Angle Orthod.* 2017;87(4):576-582. DOI: 10.2319/091416-688.1
141. Makiguchi T, Imai H, Arakawa A, Tashiro A, Yonezu T, Shintani S. Development of jaw and deciduous teeth in Japanese children–Comparing size of crown and alveolar area between today and 40 years ago. *Bull Tokyo Dent Coll.* 2018;59(3):171-181. DOI: 10.2209/tdcpublication.2017-0033
142. 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):614-619. DOI: 10.1016/s0889-5406(03)00053-2
143. Turpin DL. Where has all the arch length gone? (editorial) *Am J Orthod Dentofacial Orthop.* 2001;119(3):201. DOI: 10.1067/mod.2001.114410
144. Dugoni SA, Lee JS, Varela J, Dugoni AA. Early mixed dentition treatment: Post-retention evaluation of stability and relapse. *Angle Orthod.* 1995;65(5):311-320. DOI: 10.1043/0003-3219(1995)065<0311:EMDTPE>2.0.CO;2
145. 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. DOI: 10.1016/S0889-5406(08)70111-O
146. Behrents RG. Growth in the aging craniofacial skeleton. Monograph 17. Craniofacial Growth Series. University of Michigan, Center for Human Growth and Development; 1985:123.
147. Zachrisson BU. Important aspects of long-term stability. *J Clin Orthod.* 1997;31(9):562-583.
148 Dos Santos CCO, da Rosa Moreira Bastos RT, Bellini-Pereira SA, Garib D, Normando D. Spontaneous changes in mandibular incisor crowding from mixed to permanent dentition: A systematic review. *Prog Orthod.* 2023;24(1):15. DOI: 10.1186/s40510-023-00466-3
149. Espinosa DG, Cruz CM, Normando D. The effect of extraction of lower primary canines on the morphology of dental arch: A systematic review and meta-analysis. *Int J Paediatr Dent.* 2021;31(5):583-597. DOI: 10.1111/ipd.12726
150. Kau CH, Durning P, Richmond S, Miotti FA, Harzer W. Extractions as a form of interception in the developing dentition: A randomized controlled trial. *J Orthod.* 2004;31(2):107-114. DOI: 10.1179/146531204225020391
151. Sjögren A, Arnrup K, Lennartsson B, Huggare J. Mandibular incisor alignment and dental arch changes 1 year after extraction of deciduous canines. *Eur J Orthod.* 2012;34(5):587-594. DOI: 10.1093/ejo/cjr028
152. Bishara SE, Jakobsen JR, Treder J, Nowak A. Arch width changes from 6 weeks to 45 years of age. *Am J Orthod Dentofacial Orthop.* 1997;111(4):401-409. DOI: 10.1016/s0889-5406(97)80022-4
152. Christensen RT, Fields HW, Christensen JR, Beck FM, Casamassimo PS, McTigue DJ. The effects of primary canine loss on permanent lower dental midline stability. *Pediatr Dent.* 2018;40(4):279-84.
153. Thompson GW, Popovich F. A longitudinal evaluation of the Burlington growth centre data. *J Dent Res.* 1977;56(Spec No):C71-78. DOI: 10.1177/00220345770560032 1011
154. 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):81-85. DOI: 10.1016/s0889-5406(00)70252-6
155. 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):229-233. DOI: 10.5005/jp-journals-10005-144l
156. Brothwell DJ. Guidelines on the use of space maintainers following premature loss of primary teeth. *J Can Dent Assoc.* 1997;63(10):753-766.
157. Northway WM. The not-so-harmless maxillary primary first molar extraction. *J Am Dent Assoc.* 2000;131(12):1711-1720. DOI: 10.14219/jada.archive.2000.0117
158. Terlaje RD, Donly KJ. Treatment planning for space maintenance in the primary and mixed dentition. *ASDC J Dent Child.* 2001;68(2):109-114.
159. 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):311-317. DOI: 10.1159/000430787
160. 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):117-120.
161. 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.
162. Kirshenblatt S, Kulkarni GV. Complications of surgical extraction of ankylosed primary teeth and distal shoe space maintainers. *J Dent Child (Chic).* 2011;78(1):57-61.
163. Qudeimat MA, Fayle SA. The longevity of space maintainers: A retrospective study. *Pediatr Dent.* 1998;20(4):267-72.
164. 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):146-152. DOI: 10.1016/j.ajodo.2011.06.040
165. Sonis A, Ackerman M. E-space preservation. *Angle Orthod.* 2011;81(6):1045-1049. DOI: 10.2319/030711-165.1

References continued on the next page.

166. Law CS. Management of premature primary tooth loss in the child patient. *J Calif Dent Assoc.* 2013;41(8):612-618.
167. 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.
168. Kargul B, Cagler E, Kabalay U. Glass fiber-reinforced composite resin as fixed space maintainers in children: 12-month clinical follow up. *J Dent Child (Chic).* 2005; 72(3):109-112.
169. Kulkarni G, Lau D, Hafezi S. Development and testing fiber-reinforced composite space maintainers. *J Dent Child (Chic).* 2009;76(3):204-208.
170. Setia V. Banded vs bonded space maintainers: Finding a better way out. *Int J Clin Pediatr Dent.* 2014;7(2):97-104. DOI: 10.5005/jp-journals-10005-1245
171. Ahmad AJ, Parekh S, Ashley PF. Methods of space maintenance for premature loss of a primary molar: A review. *Eur Arch Pediatr Dent.* 2018;19(5):311-320. DOI: 10.1007/s40368-018-0357-5
172. Finucane D. Rationale for restoration of carious primary teeth: A review. *Eur Arch of Pediatr Dent.* 2012;13(6): 281-292. DOI: 10.1007/BF03320828
173. Ugolini A, Agostino P, Silvestrini-Biavati A, Harrison JE, Batista KB. Orthodontic treatment for posterior crossbites. *Cochrane Database Syst Rev.* 2021;12(12):CD000979. DOI: 10.1002/14651858.CD000979.pub3
174. Bishara SE, Staley RN. Maxillary expansion: Clinical implications. *Am J Orthod Dentofacial Orthop.* 1987;91(1): 3-14. DOI: 10.1016/0889-5406(87)90202-2
175. Da Silva Andrade A, Gameiro G, DeRossi, M, Gaviao M. Posterior crossbite and functional changes. *Angle Orthod* 2009;79(2):380-386. DOI: 10.2319/030708-137.1
176. Richards B. An approach to the diagnosis of different malocclusions. In: Bishara SE, ed. *Textbook of Orthodontics*. Saunders Co.; 2001:157-158.
177. Borrie F, Stearn D. Early correction of anterior crossbites: A systematic review. *J Orthod.* 2011;38(3):175-184. DOI: 10.1179/14653121141443
178. Kluemper GT, Beeman CS, Hicks EP. Early orthodontic treatment: What are the imperatives? *J Am Dent Assoc.* 2000;131(5):613-620. DOI: 10.14219/jada.archive.2000.0235
179. Noar J. Managing the developing occlusion: Anterior crossbites. In: *Interceptive Orthodontics: A Practical Guide to Occlusal Management*. Wiley-Blackwell; 2014:35.
180. 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):513-520. DOI: 10.1067/mod.2001.118627a
181. Sonnesen L, Bakke M, Solow B. Bite force in pre-orthodontic children with unilateral crossbite. *Eur J Orthod.* 2001;23 (6):741-749. DOI: 10.1093/ejo/23.6.741
182. Agostino P, Ugolini A, Signori A, Silvestrini-Biavati A. Orthodontic treatment for posterior crossbites. *Cochrane Database Syst Rev.* 2014;(8):CD000979. DOI: 10.1002/14651858.CD000979.pub2
183. Fields HW, Jr, Proffit WR. Growth modification in Class II, open bite/deep bite, and multidimensional problems. In: Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM, eds. *Contemporary Orthodontics*. 6th ed. Elsevier; 2019: 455-462.
184. 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. DOI: 10.1016/s0889-5406(98)70276-8
185. 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. DOI: 10.1016/S0889-5406(98)70275-6
186. 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):657-667. DOI: 10.1016/j.ajodo.2004.02.008
187. 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. DOI: 10.1016/S0889-5406(98)70277-X
188. Chen JY, Will LA, Niederman R. Analysis of efficacy of functional appliances on mandibular growth. *Am J Orthod Dentofacial Orthop.* 2002;122(5):470-476. DOI: 10.1067/mod.2002.126730
189. 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):234-243; quiz 339. DOI: 10.1016/S0889540603003524
190. 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. DOI: 10.1016/0002-9416(85)90233-7
191. 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. DOI: 10.1016/s0889-5406(99)70193-9
192. Carapezza L. Early treatment vs late treatment Class II closed bite malocclusion. *Gen Dent.* 2003;51(5):430-434.
193. Von Bremen J, Pancherz H. Efficiency of early and late Class II division 1 treatment. *Am J Orthod Dentofacial Orthop.* 2002;121(1):31-37. DOI: 10.1067/mod.2002120016
194. Oh H, Baumrind S, Korn EL. A retrospective study of Class II mixed dentition treatment. *Angle Orthod.* 2017; 87(1):56-67. DOI: 10.2319/012616-72.1
195. 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):488-495. DOI: 10.1016/S0889540603006425

196. 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):502-509. DOI: 10.1016/s0889-5406(97)70287-7
197. Kirjavanien M, Hurmerinta K, Kiravainen T. Facial profile changes in early Class II correction with cervical headgear. *Angle Orthod.* 2007;77(6):960-967. DOI: 10.2319/092106-384
198. Cameron AC. Trauma management. In: *Handbook of Pediatric Dentistry*. Cameron A, Widmer R, eds. 4th ed. Mosby Elsevier; 2013:150.
199. Nguyen QV, Bezemer PD, Habets L, Prahl-Andersen B. A systematic review of the relationship between overjet size and traumatic dental injuries. *Eur J Orthod.* 1999;21(5):503-515. DOI: 10.1093/ejo/21.5.503
200. Arraj GP, Rossi-Fedele G, Doğramacı EJ. The association of overjet size and traumatic dental injuries–A systematic review and meta-analysis. *Dent Traumatol.* 2019;35(4-5):217-232. DOI: 10.1111/edt.12481
201. Proffit WR, Larson BE. Comprehensive treatment in adolescence: Space closure and Class II/Class III correction. In: Proffit WR, Fields HW, Jr, Larson BE, Sarvar DM, eds. *Contemporary Orthodontics*. 6th ed. Elsevier; 2019:541-551.
202. Cassidy KM, Harris EF, Tolley EA, Keim RG. Genetic influences on dental arch in orthodontic patients. *Angle Orthod.* 1998;68(5):445-454. DOI: 10.1043/0003-3219(1998)068<0445:GIODAF>2.3.CO;2
203. Xue F, Wong RWK, Rabie ABM. Genes, genetics, and Class III malocclusion. *Orthod Craniofacial Res*. 2010;13(2):69-74. DOI: 10.1111/j.1601-6343.2010.01485.x
204. 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. Elsevier; 2019:430-452.
205. Celikoglu M, Oktay H. Effects of maxillary protraction for early correction class III malocclusion. *Eur J Orthod*. 2014;36(1):86-92. DOI: 10.1093/ejo/cjt006
206. 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):309-317. DOI: 10.1093/ejo/20.3.309
207. Franchi L, Bacetti 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):555-568. DOI: 10.1016/j.ajodo.2003.10.036
208. Lione R, Buongiomo 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):217-220.
209. 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):123-130.
210. 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):275-284. DOI: 10.1007/pl00001935
211. 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):675-685. DOI: 10.1016/s0889-5406(99)70294-5
212. Page DC. Early orthodontics: 5 new steps to better care. *Dent Today*. 2004;23(2):112-116.
213. Stahl F, Grabowski R. Orthodontic findings in the deciduous and early mixed dentition: Inferences for a preventive strategy. *J Orofac Orthop*. 2003;64(6):401-416. DOI: 10.1007/s00056-003-0313-8
214. Campbell PM. The dilemma of Class III treatment. Early or late? *Angle Orthod*. 1983;53(3):175-191. DOI: 10.1043/0003-3219(1983)053<0175:TDOCIT>2.0.CO;2
215. Franchi L, Bacetti T, McNamara JA. Predictable variables for the outcome of early functional treatment of Class III malocclusion. *Am J Orthod Dentofacial Orthop*. 1997;112(1):80-86. DOI: 10.1016/s0889-5406(97)70277-4
216. 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):301-306. DOI: 10.1016/j.ajodo.2004.02.014
217. 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):681-690.
218. 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 Cranio maxillofac Surg.* 2012;40(8):e350-354. DOI: 10.1016/j.jcms.2012.01.019
219. 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. DOI: 10.1067/mod.2002.120359
220. 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):423-434. DOI: 10.1067/mod.2003.70
221. 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):270-277. DOI: 10.2319/012514-68.1
222. Wendl B, Kamenica A, Droshci H. Retrospective 25-year follow-up of treatment outcomes in angle Class III patients: Early vs late treatment. *J Orofac Orthop*. 2017;78(3):201-210. DOI: 10.1007/s00056-016-0076-7