Orthodontic Considerations in Restorative Management of Hypodontia Patients With Endosseous Implants

Ali Borzabadi-Farahani, DDS, MScD*

The use of implant-supported restorations in patients with hypodontia remains challenging and requires a multistage treatment that begins in late mixed dentition and continues into late adolescence. The aim of this article is to review the role of orthodontics in endosseous implant rehabilitation of patients with hypodontia. The MEDLINE, Web of Science, Scopus, Cochrane databases, and necessary scientific textbooks were searched for relevant studies and reviews, and as far as possible, they were only included if they had been cited at least once in the literature. Dental implants are susceptible to overloading as the periodontal ligament is absent and the proprioceptive nerve endings are either lacking or very limited. Patients with hypodontia may present with skeletal features such as short and retrognathic maxilla, prognathic mandible, and shorter lower anterior facial height, and they sometimes need orthognathic correction as part of their overall treatment. Dental problems vary and include bimaxillary retroclination of incisors, spacing, centerline discrepancies, microdontia, hypoplastic enamels, ankylosis of the retained primary teeth, overeruptions, and volume deficiencies of alveolar ridges. The challenges mentioned, as well as bone volume deficiencies, compromise the successful placement of implants. Orthodontic strategies and techniques, such as uprighting mechanics, extrusion/intrusion, delayed space opening, and orthodontic implant site-switching, can be used to create, preserve, or augment the implant site. After orthodontic site development, the final planned position of the teeth should be maintained with a rigid bonded retainer; overlooking this stage may compromise the implant site and require orthodontic retreatment.

Key Words: hypodontia, craniofacial growth, implant-supported restorations, orthodontics, implantology

INTRODUCTION

Endosseous implants (EIs) were introduced more than 40 years ago,[1,2] and their use in the dental profession has grown exponentially ever since. EIs have become the standard of care, providing predictable and reliable treatment alternatives for rehabilitation of patients with edentulous or partially dentate dentitions and those who have congenitally absent teeth.[3–9] Despite the high success rate, implant failure still happens, and therefore, extensive research is targeted to identify the contributing causes.[10–15] Different shapes and sizes of EIs are available. The width of an implant is usually 3.75 mm, corresponding to a platform width of 4 mm. Implants can have a tapered or parallel shape, though the tapered shape is the most frequently used systems. The advantage of the tapered-shape implants is their ability for self-tapping, which is useful for softer bones or immediate loading. Implant dimensions range from 3 to 8 mm in diameter and 7 to 21 mm in length.

The current trends in dental implantology favor less patient comfort and shorter treatment times. Short and wide-diameter implants are associated with faster treatment and less morbidity, eliminating the need for vertical bone augmentation[16] or sinus lifting procedures.[17] Regrettably, the evidence-based practice is mainly available for the major implant brands.[18] Different dental implant systems have been presented in the market, such as

Craniofacial Orthodontics, Children's Hospital Los Angeles, and Center for Craniofacial Molecular Biology, University of Southern California, Los Angeles.
* *Corresponding author,* e-mail: farahani@faraortho.com
DOI: 10.1563/AAID-JOI-D-11-00022

temporary or provisional implants, flapless systems, immediate, immediate-delayed, and delayed implant systems. Flapless procedures are popular as they have been associated with less morbidity. According to recent 18-month to 3-year trials, implants successfully integrate using the flapless approach with immediate loading, similar to conventional protocols.[19,20] To date, there is not enough evidence to determine the possible advantages or disadvantages of specific systems or regimens.[21,22] Similarly, there is not sufficient evidence for or against performing augmentation procedures for immediate implants placed in fresh extraction sockets, or whether any of augmentation techniques is superior to others.[21–23]

Implant-supported restorations have become an increasingly accepted alternative for rehabilitation of dentition, but providing EIs for patients with hypodontia (PH) is not always clear-cut. Various factors should be evaluated before establishing the definitive treatment plan, and the objective of this article is to review the role of orthodontics in EI rehabilitation of PH.

Literature Search Method

As the material for this review was diverse and heterogeneous, it was not possible to perform a systematic review. Therefore, the MEDLINE, Web of Science, Scopus, and Cochrane databases, as well as necessary scientific textbooks, were searched for the relevant material, and as far as possible, studies and reviews were only included if they had been cited at least once in the literature.

Epidemiology and Terminology

Edentulous spaces, whether because of pathology or tooth agenesis, are common. The prevalence of hypodontia in permanent teeth, excluding third molars, ranges from 1.6% to 9.6%.[24] The term "hypodontia" is generally used to describe the absence of 1 to 6 teeth, excluding third molars.[24] However, most (80%) PH lack only 1 or 2 teeth,[25] mostly permanent second premolars and upper lateral incisors.[26] The severe form of hypodontia, oligodontia, refers to the absence of more than 6 teeth, excluding third molars, and anodontia refers to the complete developmental absence of primary and/or secondary dentitions.[24] Nearly, 1% (0.08%–1.1%) of the population has oligodontia.[27–31]

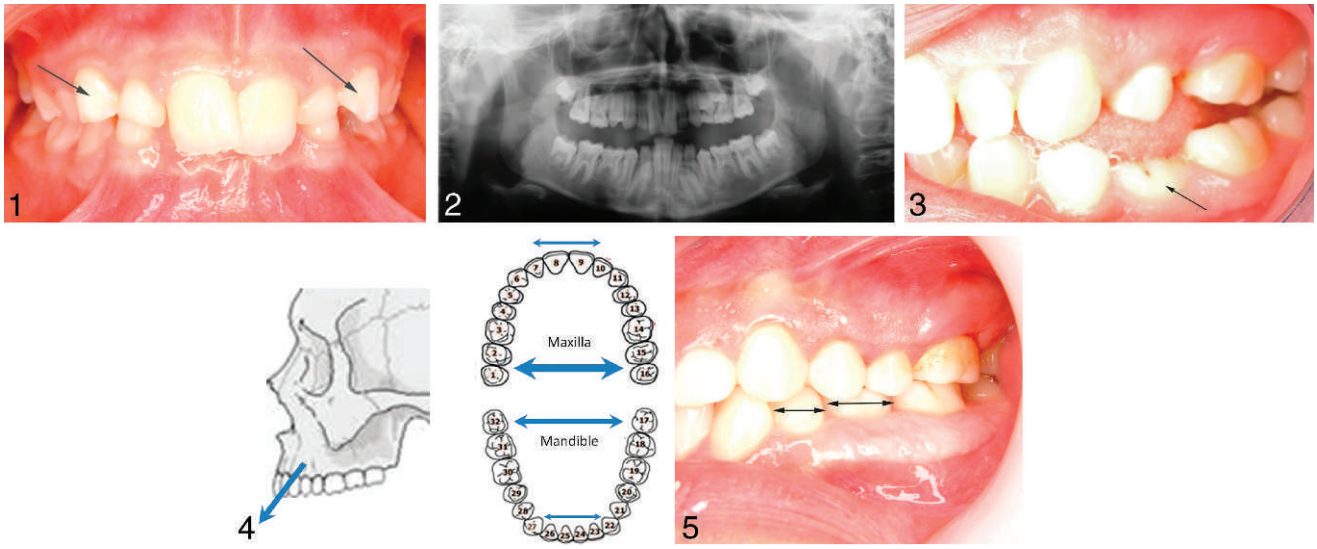
Associated Medical Problems

Few medical conditions can potentially complicate the use of dental implants,[32] such as uncontrolled diabetes mellitus, bleeding disorders, a weakened immune system, cognitive problems, and an acquired (ie, radiotherapy) or congenital bone disease.[32] Although it is not a medical condition, patients should be able to open their mouth wide enough at the implant site ($\geq$35 mm) to give the clinician enough working space for drilling and placing the implant within the bone.

The severe form of nonsyndromic hypodontia has been associated with the signs or symptoms of ectodermally derived diseases (ie, impaired structure or function of sweat glands, low salivary secretion, deficient hair or nails). Similarly, hypodontia, and particularly the severe form of it, has been related to more than 120 different syndromes, most often with X-linked ectodermal dysplasia and, to a lesser extent, with the autosomal recessive and dominant forms of hypohidrotic ectodermal dysplasia.[31.33] Syndromic hypodontia refers to tooth agenesis in patients with an underlying recognizable clinical syndrome, such as Down, Ehlers-Danlos (Type VII), Rieger (Type I), and Witkop syndrome.[34] Therefore, potential medical conditions should be excluded, and appropriate medical advice should be sought before drawing up a treatment plan (Figures 1 and 2).

Dental and Skeletal Characteristics of PH

Tooth agenesis has been associated with certain dental[35–44] (Tables 1 and 2; Figure 3) and skeletal features[45–52] (Table 2). These features become more noticeable in severe forms of hypodontia. Evaluation of patients with oligodontia[31] revealed the following features: maxillomandibular retrognathism, anterior rotation of the mandible, large inferior extensions of the maxillary sinus, receding midface, shorter anterior face height, increased nasolabial angle, deep labiomental fold, and decreased vertical and transversal dimensions of the alveolar process. Further, patients with multiple missing teeth have an increased freeway space that has been attributed to the lack of posterior support,[53] delayed devel-



Figures 1–5. Figure 1. Oligodontia, including agenesis of maxillary permanent canines and primary retained maxillary canines (arrows), is displayed. The patient was sent for medical examination as a mild form of ectodermal dysplasia was suspected. **Figure 2.** Panoramic radiograph of the patient seen in Figure 1. The following teeth were absent: 4, 6, 7, 10, 11, 20, 23, 29. **Figure 3.** Agenesis of the left maxillary and mandibular second premolars and the retained infraoccluded left mandibular primary molar (arrow) are shown. Nearly, 55% of retained primary mandibular second molars show signs of infraocclusion,[43] but only 2.5%–8.3% of retained primary molars are severely infraoccluded (positioned below the gingival margin of the adjacent teeth).[44] **Figure 4.** The maxilla grows in a downward and forward direction. The transverse growth of the maxilla occurs primarily because of growth at the midpalatal suture. This is greater posteriorly than anteriorly. After the age of 6–7 years, the transverse growth of the mandible in the incisor region is minimal.[54] Implants placed in the anterior mandible after the age of 11 years had a relatively stable position, not constraining the transverse growth.[139–142] However, the horizontal and vertical growth of the mandible continues through adolescence and early adulthood. With a Class III tendency and further horizontal growth of the mandible there may be a need for replacing implants or changing the abutments in the future. **Figure 5.** Agenesis of the mandibular left second premolar is shown. The size of the second retained primary mandibular molar is larger (9.5 mm) than that of the adjacent premolar (7.5–8 mm).[87]

opment,[54] and the relative absence of growth in that area.[54] In addition, severe hypodontia has been associated with deep overbite, disturbances of eruption, and microdontia.[31] These features can affect the interdisciplinary management of PH, making the restorative interventions in the edentulous area very challenging. Therefore, early orthodontic management of some of these problems (eg, deep overbite) has been recommended.[54]

Growth Considerations

The downward and forward growth of the facial skeleton is a well-known fact[55] (Figure 4). In the maxilla, bone is resorbed from the floor of the maxillary antrum and deposited in the palatal vault, translating the palate in a downward direction. Because there is no periodontal ligament, EIs behave like the ankylosed teeth.[55] This, whether caused by EIs or submerged teeth, exercises an inhibitory and detrimental effect on the eruption of adjacent teeth (ie, creates a posterior lateral open bite) (Figure 3) or, at least, generates some gingival level discrepancy. Animal and human studies showed that the early use of implants in a growing child led to submergence or positional changes of implants.[56–59] This complicates the prosthetic management of edentulous patients and creates esthetic or maintenance issues. The vertical growth of the face and the eruption of teeth, contrary to the sutural growth, continue past puberty.[60] As a rule, growth in width finishes first, and this is followed by growth in length and finally growth in height.[55] Except for the anterior mandibular region, where a very limited transverse and vertical growth occur after 6–7 years of age,[54] most regions of the dental arch experience some positional changes with growth. Placing implants in the maxillary anterior region of a growing patient may lead to the implants moving apart (because of the transverse growth of the maxilla), becoming infraoccluded, or occasionally being exposed to the nasal cavity.

TABLE 1

Examples of dental features associated with hypodontia

Feature
Small teeth (microdontia)[35–38]
Infraocclusion of primary molars[38,39]
Spacing in dental arches, malpositioned teeth, centerline discrepancies, overeruption of teeth
Uneven gingival margins
Enamel hypoplasia[38]
Taurodontism[40,41]
Delayed dental development[42]

Further, because of the maxillomandibular transverse growth, early implant placement in the posterior maxilla and, to a lesser extent, in the posterior mandible can lead to transverse displacement of the implants.[61,62] In addition, the anterior or posterior rotational growth changes of the facial skeleton may alter the inclination of dental implants.[63–65]

To reduce the psychological distress in growing patients and increase their self-esteem and social acceptance, early placement of EIs may take place in extreme forms of hypodontia, such as hypohidrotic ectodermal dysplasia, severe oligodontia, or anodontia.[31] This may take place in children as young as 3 years old to provide support for overdentures or bridge restorations,[66,67] usually by placing 2 or more EIs between the mandibular canines. However, the overall growth of the face may dictate placing provisional implants or revising the abutments and restorations periodically.[68] The positional growth changes of implants and abutments must be addressed properly to prevent overloading and possible implant failure. In general, the recommendation is to delay placing EIs until most growth is complete. This tends to be earlier in females (17 years) than males (18–21 years),[69,70] and can be assessed by serial lateral cephalometric evaluations. To conclude, there are also reports of submergence or positional changes of implants due to late adult growth,[71,72] and consequently, long-term evaluation of occlusion and follow-up of patients with implant-supported restorations are recommended.

TABLE 2

Skeletal and dental features of patients with hypodontia

Skeletal and Dental Features
Smaller cranial base length[45,46] and angle[45,47]
More retrognathic[47–50] and shorter maxilla[45,46,49,51]
More prognathic mandible[45,46,52]
Smaller mandibular plane[46,47,52] and sagittal jaw relationship angles[47,48]
Straighter facial convexity[47,49,50]
Shorter lower anterior facial height[46,47]
Bimaxillary retroclination
Greater retroclination of maxillary[45,47,48,50] and mandibular incisors[45,47,48]
Larger interincisal angle[45,47,50]

MULTISTAGE ORTHODONTIC TREATMENT AND RETAINED PRIMARY TEETH

The primary objective of orthodontic treatment in PH is to minimize and consolidate edentulous spaces.[73] If implant-supported restorations are planned, the objective is to use the minimum required number of EIs without compromising the function, facial profile, and dental esthetics.[74] The interdisciplinary management of PH involves different stages, and orthodontics can correct some alveolar bone deficiencies and achieve root parallelism adjacent to the implant site. Patients with severe forms of hypodontia and fully edentulous arches occasionally benefit from an implant-supported overdenture during the primary dentition.[66] This is to provide function, prevent distortion of the occlusal plane, eliminate overeruption of opposing dentition,[72] and improve the child's self-esteem and social acceptance.[31]

For most PH, orthodontic management starts in the mixed dentition, usually after radiographic screening around the age of 8 years,[75] and assessment of records. Radiographic screening with a panoramic radiograph at age 8 years identified 65% and 85% of patients with $\geq$6 and $\geq$9 absent permanent teeth, respectively.[75] This first stage of treatment may coincide with some restorative management of microdontia, which is often associated with hypodontia.[35–38] Interceptive measures, such as simple orthodontic space redistribution, ease the guided eruption of teeth. The persistence of the primary teeth is the result of congenital absence of successors[76–81] or the impaction of successor teeth.[81–83] Mandibular primary second molars and maxillary primary canines are the most cited.[81] At this stage, detecting any infraocclusion of retained primary teeth is imperative to prevent any future vertical bone deficiency.[84,85] The infraocclu-

sion can be caused by ankylosis, the most likely reason, or tipping of adjacent permanent teeth, leading to impaction of the primary tooth.[86] A bitewing radiograph shows the bone level around the suspected ankylosed tooth, and a flat bone level is usually a good sign, but vertical bone defects most likely indicate an ankylosed tooth.[87] This is imperative to keep the length of this early orthodontic treatment to a minimum, avoiding losing the patient's compliance.

The definitive restorative occlusal objectives are reviewed and established during the late mixed dentition. The final position of the teeth and the future implant sites are established during orthodontic treatment in the permanent dentition. This is followed by placing a rigid bonded retainer or a resin bonded bridge to maintain the teeth and their roots in the correct position. If implant-supported restorations are the treatment of choice, all retained primary teeth without successors should be preserved to maintain the buccolingual width of the alveolar ridge. A primary molar is usually much wider than the successive premolar.[87] This discrepancy in size requires some mesiodistal tooth reduction and restorative buildup of the retained primary molar (Figure 5).[70,88–90] This lessens the occlusal load on the primary tooth; increases the long-term survival, thereby preparing the implant site for a properly sized implant-supported restoration; and preserves the buccolingual width of the alveolar ridge.[70]

Limited evidence is available for timing of primary tooth extraction and placement of implant, and recommendations are based mainly on clinical experience.[74] The retained primary molars are not very good at maintaining the vertical height of the alveolar ridge, particularly if infraoccluded, which leads to creation of vertical bone deficiencies; thus, vertical bone augmentation of the implant site may be needed in the future (Figure 3). The ankylosed primary molars either need interproximal reduction and restoration of the occlusal surface to maintain the occlusion or should be extracted where severe infraocclusion prevails. There is limited evidence on the long-term survival of primary teeth,[74,91] and the longest life span is reported for the mandibular deciduous canines followed by maxillary canines.[92] Retention of primary mandibular second molars beyond the age of 20 usually conveys a good long-term survival.[86] Some aspects of treatment involving placement of EIs or orthognathic surgery should be delayed until most of the maxillomandibular growth is complete, and therefore, preserving primary retained teeth without successors, at least until the late teens, is advisable.[86]

Biological Considerations

There are distinct differences between EIs and human teeth; for example, EIs have no, or a very limited, proprioceptive nerve ending to protect them against excessive occlusal forces and overloading. The direct relationship between overloading and implant failure has been debated,[93] but the consensus is that implant overloading during function increases the risk of implant failure.[94] An increase in occlusal loading often leads to hypermobility of teeth and periodontal ligament widening, a protective mechanism that is present in all permanent teeth. Nonetheless, EIs have a very limited capacity to displace axially (3–5 μm).[95] Because of the nonexistent periodontal ligament and the absence of proprioceptive protective mechanisms, EIs are more vulnerable to occlusal overloading and failure.[95] Therefore, it is imperative to avoid placing any form of occlusal overload or laterally inclined forces on the implant.[95] The occlusal loads should be directed down the long axis of implants.[96] The EIs are much narrower (3.75 mm) than the roots of teeth they replace, and forces that are not in line with the long axis of the implant result in greater lateral forces being delivered to implant compared with that of replacing teeth.

Light infraocclusions[93] cusp-to-fossa occlusal relationships,[96] as well as light or no occlusal contacts in eccentric excursions should be planned for implant-supported restorations, so far as practicable, to reduce the occlusal overloading and laterally inclined forces. An 8-μm clearance on firm clench and light passive occlusal contacts between implant prosthesis and opposing teeth have been recommended.[93] Placing implants with an oversized occlusal table can lead to cantilevers, inclined occlusal forces, overloading, premature failure of abutments, and eventually implant failures.[94,96] An example is a patient with a missing mandibular premolar, where the mesiodistal size of the retained primary tooth is larger than the successive premolar (Figure 3).[70] This long edentulous space should be shortened to the size of a premolar tooth to avoid having an oversized occlusal table.

Orthodontic Implant Site Development and Bone Volume Deficiencies

The adequate mesiodistal, buccolingual, and vertical spaces for an EI should be planned ahead of placement. Providing space for the implant and prosthetic crown are equally important, and as a rule, for an implant 4 mm in diameter, approximately 2 mm of bone support is needed on either side of the implant.[97] This translates to a minimum of 8 mm of bone thickness mesiodistally or buccolingually. However, for esthetic results and proper appearance of the interproximal papilla, the recommendations go beyond the 2 mm bone thickness in the mesiodistal direction, that is, an interimplant and implant-tooth distances of 3 and 3–4 mm, respectively.[98,99] A minimum of 8 mm vertical space is required to fabricate and design a prosthetic crown (from the crest of alveolar bone to the occlusal surface of the opposing teeth). Violating these simple rules can lead to inadequate blood supply to the thin bone plate that surrounds implant and may result in bone resorption and bone dehiscences. This is particularly important in the maxillary anterior region, where immediate or delayed loading can be the treatment of choice after extraction of periodontally compromised or traumatized teeth. The so-called "bundle bone" is the portion of alveolar bone surrounding teeth, and into which the collagen fibers of the periodontal ligament are embedded.[100] The bundle bone is functionally dependent on blood supply from the periodontal ligament and periosteum. The maxillary anterior buccal crestal bone thickness can be very thin and deficient in vertical and buccolingual dimensions, much less than 2 mm.[101,102] In fact, similar to findings in cadaver[103] and clinical[104] studies, a recent cone-beam study[101] revealed that facial bone thickness $\geq$2 mm at levels 1, 2, 3, 4, and 5 mm from the bone crest was present in 0%, 1.5%, 2.0%, 3.0%, and 2.5% of patients, respectively. After removal of teeth, blood supply to this predominantly thin facial bone overlying maxillary anterior teeth can be disrupted, leading to detrimental bone loss or soft-tissue recession.[101,102,105,106] Therefore, atraumatic procedures, use of alternative implant sites with adequate bone volume, and delayed placement of implants are recommended until enough bone volume is generated at the implant site by performing bone grafting, socket preservation techniques, or orthodontic bone generation (Figures 6 and 7).[107–110]

When the teeth are extracted or in adult PH, opposing or adjacent teeth may overerupt or drift into the edentulous space and compromise the space needed for implant-supported restoration (Figures 8 and 9). Orthodontic therapy is often required to create enough space for the implant and the implant-supported restoration. The space creation involves space opening in mesiodistal dimension, or in vertical dimension with overerupted opposing teeth. Correcting teeth that are tilted or that have drifted into the edentulous space usually involves some form of uprighting mechanics (Figure 8). Mini-implants and EIs[111] may be used as temporary or absolute source of anchorage to facilitate tooth movement (Figure 8). The mini-implants, or the so-called "temporary anchorage devices," are usually 1.2 mm in diameter and 6–8 mm long, small enough to be inserted between the roots of teeth and used for molar intrusion. The overeruption of opposing teeth can be successfully corrected using mini-implant–assisted intrusive mechanics (Figure 9).[111,112] The accelerated intrusion of overerupted molars can be achieved by combining the use of mini-implants and selective alveolar corticotomies[113] or osteotomies.[114,115] When orthodontic implant site development is completed, a rigid fixed retainer is often required to stabilize and retain the teeth, restraining any root reapproximation or unwanted tooth movement.

Orthodontic Strategies to Augment or Preserve the Implant Site

A minimum bone volume is required for successful placement of EIs. There are varieties of surgical techniques to augment the alveolar bone but discussing these techniques is beyond the scopes of this article. Briefly, these include, but are not limited to, bone grafting procedures, ridge expansion techniques, osteodistraction, and sinus lifting procedures. Some orthodontic strategies to augment or preserve the implant site are briefly discussed in the following.

Orthodontic extrusion

The orthodontic extrusion of nonrestorable or periodontally compromised teeth increases the hard- and soft-tissue volume in the future implant site[116–119] and may eliminate the vertical bone volume deficiencies. Alveolar ridge augmentation techniques are more predictable in restoring the width of an alveolar

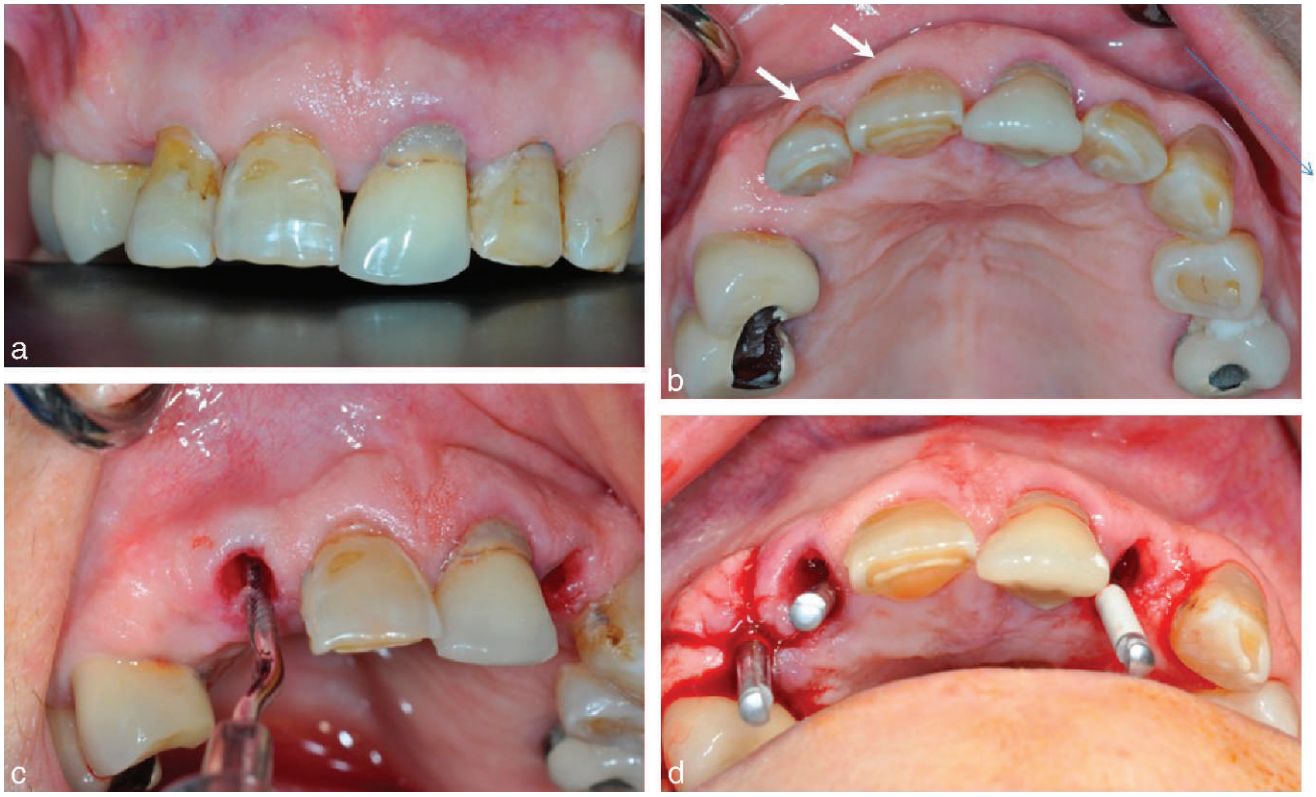


Figure 6. A thin maxillary buccal cortical bone (a,b) (arrows) is a common occurrence[101] and associated with a detrimental bone loss after placing an implant. A recent study[107] revealed that the crestal labial soft tissue thickness and implant's labial bone thickness were highly associated in the anterior maxillary region. If immediate loading is the treatment of choice, atraumatic extractions and implant site preparations (c) (eg, piezoelectric systems), as well as placement of implants more palatally (d), help avoid complications.

ridge than its height[120]; nonetheless, orthodontic extrusion is one of the most reliable means of gaining vertical bone augmentation. This is particularly true in the maxillary anterior region, where vertical bone augmentation is difficult. Good plaque control, the existence of at least one-third to one-fourth of the apical attachment, and a sufficient stabilization period are necessary for a successful forced eruption.[117,118,121] The orthodontic extrusion is done at a rate of 1 mm per week, and a stabilization period of 1 month for each millimeter extruded has been recommended.[122] When a periodontally compromised tooth is extruded, torquing[118] and tipping of the tooth toward an angular bone defect increase the alveolar bone volume in the future implant site.[119] With this strategy, some improvement of the interproximal papillary height can be expected.[119]

Delayed orthodontic space opening

Reductions in the buccolingual and vertical dimensions of the alveolar ridge occur after extractions[123–127] or in patients with congenitally missing teeth.[128] In one study,[128] after extraction of primary mandibular second molars, the ridge narrowed by 25% during the first 4 years, and after 7 years, it narrowed by an overall of 30%. This ridge defect that is mainly in the buccal side[128] requires a bone graft or necessitates more lingual or palatal implant placement. The orthodontic space opening has been associated with decreases in bone width in the newly opened sites,[129,130] though, changes 2 years after finishing the space opening were very minimal.[130] This bone defect may be seen in such areas as the missing maxillary lateral incisor or mandibular premolars. The limited available evidence suggests that, to avoid the surgical ridge augmentation at the site of missing maxillary lateral incisors, the distalization of the canine should be postponed until after the age of 13 years[131] or near the end of skeletal growth[132] (Figure 10); however, this has been disputed.[130]

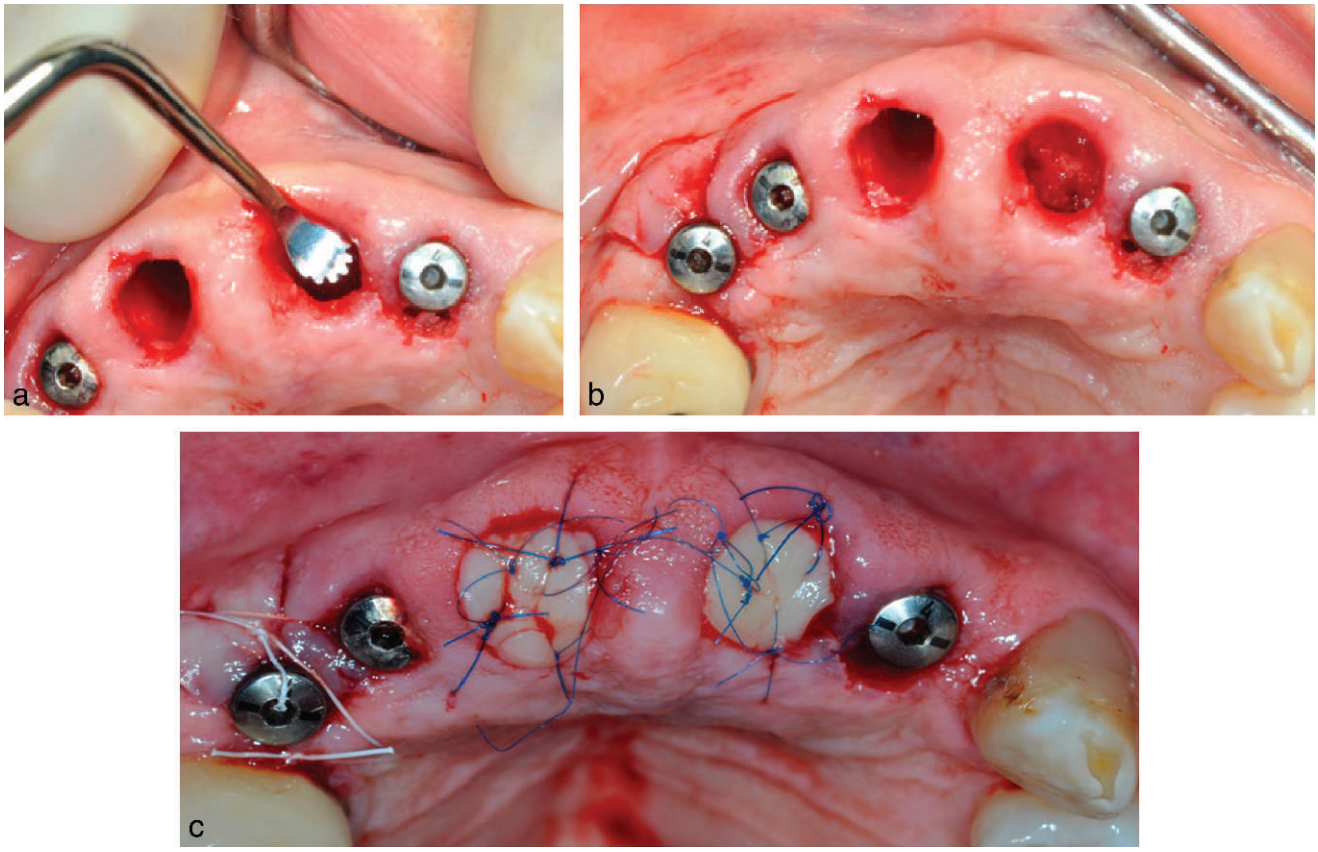


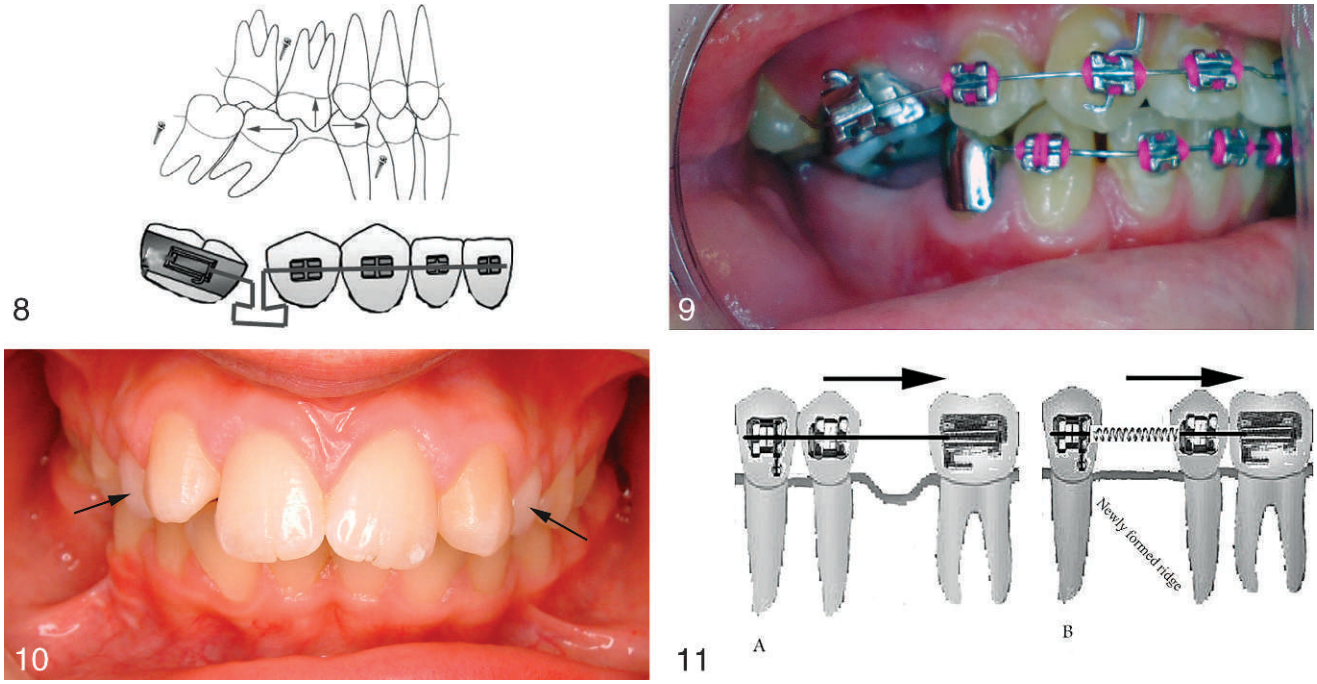
Figure 7. The patient in Figure 6 showed signs of periodontal bone loss around maxillary central incisors (a,b) and it was decided to perform socket preservation in those areas (c) and implants placed in tooth number 6, 7, and 10 areas instead.

Orthodontic implant site-switching technique

The orthodontic site-switching technique involves moving the adjacent teeth into the bone-deficient edentulous area, closing the edentulous space, and creating an implant site adjacent to the original edentulous area.[70,133] This newly generated ridge often has adequate bone volume, eliminating further bone grafting (Figure 11). Therefore, by using the adjacent tooth as a stimulus for alveolar site development, the need for bone graft may be eliminated.[70] This technique is valuable for generating enough bone volume in the maxillary and mandibular lateral incisor or premolar regions, common sites for congenital tooth agenesis.[24] For instance, the mesialization of the second premolar into the narrowed and deficient site of a missing first premolar (or vice versa) leaves behind an alveolar ridge with an adequate bone volume, eliminating bone grafting before implant placement.[60,70,132,134]

Orthodontic Retention Stage

Replacing congenitally missing teeth often involves some orthodontic space opening or space redistribution during adolescence. Due to continued facial growth and tooth eruption, several years may elapse between the completion of treatment and the provision of implant therapy. Unless a rigid bonded retainer or a resin bonded bridge is provided, positional changes of the teeth adjacent to edentulous spaces are common.[135] The other potential problem is overeruption of unopposed teeth into the edentulous space, which compromises the future prosthetic treatment.[136] Positional changes should be prevented, such as root reapproximation of teeth adjacent to the edentulous area,[137,138] tilting of adjacent crowns into edentulous space,[137,138] and overeruption of unopposed molars.[136] A study of the post-retention root position of maxillary central incisors and canines revealed that 11% of patients experienced a relapse that was significant enough to prevent implant placement in



Figures 8–11. Figure 8. Overeruption of opposing teeth or drift of adjacent teeth into the edentulous space is a common problem associated with patients with hypodontia. For a 4 mm diameter implant, a minimum mesiodistal space of 8 mm is required for implant placement. The fixed appliances may be used for uprighting and space opening. Mini-implants can be used with fixed appliances to intrude the overerupted teeth, open up space mesiodistally, and upright the tilted teeth adjacent to edentulous space. **Figure 9.** Overeruption of right maxillary first and second permanent molars into the opposing mandibular edentulous area can be seen. Mini-implants, in combination with selective alveolar corticotomies[113] or osteotomies,[114,115] may be used to intrude the overerupted molars. **Figure 10.** A patient with retained maxillary primary canines (arrows) and congenitally absent maxillary lateral incisors. If replacements of maxillary lateral incisors with implants are planned, the distalization of the maxillary canines can be done after the age of 13 years or near the end of skeletal growth.[131] This approach may preserve the buccolingual bone volume in the maxillary lateral incisor regions. **Figure 11.** The orthodontic implant site-switching technique uses tooth movement to generate new bone. (A) A first premolar is pushed distally into the second premolar position, where bone volume deficiency exists. (B) New bone is generated in the first premolar position and can be used for implant placement, obviating the need for bone grafting.

that area.[138] These positional changes compromise or prevent the future implant placement, requiring orthodontic retreatment to facilitate implant placement. Apparently, removable retainers are not very efficient in maintaining dimensions of edentulous space[138]; thus, placing a rigid bonded wire or a resin bonded bridge has been recommended. This prevents root approximation during the retention stage or overeruption of unopposed molars after tooth loss.[70,138]

In summary, this review has described several orthodontic considerations in planning implant-supported restoration for PH. The orthodontic and restorative management of PH, using EIs, requires early diagnosis and multistage orthodontic and restorative treatment. A minimum required bone volume is needed for successful placement of implants and should be considered when formulating the treatment plan. With congenitally missing teeth, adjacent or opposing teeth may tip, drift, or overerupt, leaving edentulous spaces that are not favorable to replacement of missing teeth. Collectively, this affects the space and bone volume required for implant placement or the implant-supported restoration. The staged orthodontic treatment and some orthodontic strategies, such as the orthodontic extrusion, delayed orthodontic space opening, and the orthodontic implant site-switching technique, can preserve or augment the future implant site.

Abbreviations

EI: endosseous implant
PH: patients with hypodontia

Acknowledgment

The author was supported by a research grant from the European Orthodontic Society and a Post-Doctoral Research Fellowship from the Children's Hospital Los Angeles.

References

1. Branemark P-I, Breine U, Adell R, et al. Intra-osseous anchorage of dental prostheses. I. Experimental studies. *Scand J Plast Reconstr Surg*. 1969;3:81–100.

2. Branemark PI, Hansson BO, Adell R, et al. Osseointegrated implants in the treatment of the edentulous jaw. Experience from a 10-year period. *Scand J Plast Reconstr Surg Suppl*. 1977;16:1–132.

3. Adell R, Erikkson B, Lekholm U, Branemark PI, Jemt T. Long term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws. *Int J Oral Maxillofac Implants*. 1990;5:347–359.

4. Van Steenberghe D, Lekholm U, Bolender C, et al. The applicability of osseointegrated oral implants in the rehabilitation of partially edentulism: a prospective multicenter study on 558 fixtures. *Int J Oral Maxillofac Implants*. 1990;5:272–281.

5. Jemt T, Lekholm U. Oral implant treatment in posterior partially edentulous jaws: a 5-year follow-up report. *Int J Oral Maxillofac Implants*. 1993;8:635–640.

6. Lekholm U, Gunne J, Henry P, et al. Survival of the Branemark implant in partially edentulous jaws: a 10-year prospective multicenter study. *Int J Oral Maxillofac Implants*. 1999;14:639–645.

7. Hardt CR, Gröndahl K, Lekholm U, Wennström JL. Outcome of implant therapy in relation to experienced loss of periodontal bone support: a retrospective 5-year study. *Clin Oral Implants Res*. 2002;13:488–494.

8. Lekholm U, Gröndahl K, Jemt T. Outcome of oral implant treatment in partially edentulous jaws followed 20 years in clinical function. *Clin Implant Dent Relat Res*. 2006;8:178–186.

9. Scholander S. A retrospective evaluation of 259 single-tooth replacements by the use of Branemark implants. *Int J Prosthodont*. 1999;12:483–491.

10. Esposito M, Hirsch J-M, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (II). Etiopathogenesis. *Eur J Oral Sci*. 1998;106:721–764.

11. Taylor TD, Agar JR, Vogiatzi T. Implant prosthodontics: current perspective and future directions. *Int J Oral Maxillofac Implants*. 2000;15:66–75.

12. Gianserra R, Cavalcanti R, Oreglia F, Manfredonia MF, Esposito M. Outcome of dental implants in patients with and without a history of periodontitis: a 5-year pragmatic multicentre retrospective cohort study of 1727 patients. *Eur J Oral Implantol*. 2010;3:307–314.

13. Capelli M, Esposito M, Zuffetti F, et al. A 5-year report from a multicentre randomised clinical trial: immediate non-occlusal versus early loading of dental implants in partially edentulous patients. *Eur J Oral Implantol*. 2010;3:209–219.

14. Cho-Lee GY, Naval-Gias L, Castrejon-Castrejon S, et al. A 12-year retrospective analytic study of the implant survival rate in 177 consecutive maxillary sinus augmentation procedures. *Int J Oral Maxillofac Implants*. 2010;25:1019–1027.

15. Checchi L, Felice P, Antonini ES, et al. Crestal sinus lift for implant rehabilitation: a randomised clinical trial comparing the Cosci and the Summers techniques. A preliminary report on complications and patient reference. *Eur J Oral Implantol*. 2010;3:221–232.

16. Felice P, Cannizzaro G, Checchi V, et al. Vertical bone augmentation versus 7-mm-long implants in posterior atrophic mandibles. Results of a randomised controlled clinical trial of up to 4 months after loading. *Eur J Oral Implantol*. 2009;2:7–20.

17. Cannizzaro G, Felice P, Leone M, Viola P, Esposito M. Early loading of implants in the atrophic posterior maxilla: lateral sinus lift with autogenous bone and Bio-Oss versus crestal mini sinus lift and 8-mm hydroxyapatite-coated implants. A randomised controlled clinical trial. *Eur J Oral Implantol*. 2009;2:25–38.

18. Bhatavadekar N. Helping the clinician make evidence-based implant selections. A systematic review and qualitative analysis of dental implant studies over a 20 year period. *Int Dent J*. 2010;60:359–369.

19. Van de Velde T, Sennerby L, De Bruyn H. The clinical and radiographic outcome of implants placed in the posterior maxilla with a guided flapless approach and immediately restored with a provisional rehabilitation: a randomized clinical trial. *Clin Oral Implants Res*. 2010;21:1223–1233.

20. Cannizzaro G, Leone M, Consolo U, Ferri V, Esposito M. Immediate functional loading of implants placed with flapless surgery versus conventional implants in partially edentulous patients: a 3-year randomized controlled clinical trial. *Int J Oral Maxillofac Implants*. 2008;23:867–875.

21. Esposito M, Grusovin MG, Polyzos IP, Felice P, Worthington HV. Interventions for replacing missing teeth: dental implants in fresh extraction sockets (immediate, immediate-delayed and delayed implants). *Cochrane Database Syst Rev*. 2010;9:CD005968.

22. Esposito M, Grusovin MG, Polyzos IP, Felice P, Worthington HV. Timing of implant placement after tooth extraction: immediate, immediate-delayed or delayed implants? A Cochrane systematic review. *Eur J Oral Implantol*. 2010;3:189–205.

23. Esposito M, Grusovin MG, Felice P, et al. The efficacy of horizontal and vertical bone augmentation procedures for dental implants—a Cochrane systematic review. *Eur J Oral Implantol*. 2009;2:167–184.

24. Vahid-Dastjerdi E, Borzabadi-Farahani A, Mahdian M, Amini N. Non-syndromic hypodontia in an Iranian orthodontic population. *J Oral Sci*. 2010;52:455–461.

25. Lidral AC, Reising BC. The role of MSX1 in human tooth agenesis. *J Dent Res*. 2002;81:274–278.

26. Symons AL, Stritzel F, Stamation J. Anomalies associated with hypodontia of the permanent lateral incisor and second premolar. *J Clin Pediatr Dent*. 1993;17:109–111.

27. Schalk-van der Weide Y, Steen WH, Bosman F. Distribution of missing teeth and tooth morphology in patients with oligodontia. *ASDC J Dent Child*. 1992;59:133–140.

28. Stockton DW, Das P, Goldenberg M, D'Souza RN, Patel PI. Mutation of PAX9 is associated with oligodontia. *Nat Genet*. 2000;24:18–19.

29. Bergendahl B, Bergendahl T, Hallonsteen A-L, et al. A multidisciplinary approach to oral rehabilitation with osseointegrated implants in children and adolescents with multiple aplasia. *Eur J Orthod*. 1996;18:119–129.

30. Rølling S, Poulsen S. Oligodontia in Danish schoolchildren. *Acta Odontol Scand*. 2001;59:111–112.

31. Worsaae N, Jensen BN, Holm B, Holsko J. Treatment of severe hypodontia–oligodontia—an interdisciplinary concept. *Int J Oral Maxillofac Surg*. 2007;36:473–480.

32. Scully C, Hobkirk J, Dios PD. Dental endosseous implants in the medically compromised patient. *J Oral Rehabil*. 2007;34:590–599.

33. Schalk-Van Der Weide Y, Beemer FA, Faber JAJ, Bosman F. Symptomatology of patients with oligodontia. *J Oral Rehabil*. 1994;21:247–261.

34. Cobourne MT. Familial human hypodontia—is it all in the genes? *Br Dent J*. 2007;25:203:203–208.

35. Garn SM, Lewis AB. The gradient and the pattern of crown-size reduction in simple hypodontia. *Angle Orthod*. 1970;40:51–58.

36. Schalk-van der Weide Y, Steen WH, Beemer FA, Bosman F. Reductions in size and left-right asymmetry of teeth in human oligodontia. *Arch Oral Biol*. 1994;39:935–939.

37. Pinho T, Maciel P, Pollmann C. Developmental disturbances associated with agenesis of the permanent maxillary lateral incisor. *Br Dent J*. 2009;207:E25.

38. Baccetti T. A controlled study of associated dental anomalies. *Angle Orthod*. 1998;68:267–274.

39. Garib DG, Peck S, Gomes SC. Increased occurrence of dental anomalies associated with second-premolar agenesis. *Angle Orthod*. 2009;79:436–441.

40. Seow WK, Lai PY. Association of taurodontism with hypodontia: a controlled study. *Pediatr Dent*. 1989;11:214–219.

41. Schalk-van der Weide Y, Steen WH, Bosman F. Taurodontism and length of teeth in patients with oligodontia. *J Oral Rehabil*. 1993;20:401–412.

42. Tunç EŞ, Bayrak S, Koyutürk AE. Dental development in children with mild-to-moderate hypodontia. *Am J Orthod Dentofacial Orthop*. 2011;139:334–338.

43. Bjerklin K, Bennett J. The long-term survival of lower second primary molars in subjects with agenesis of the premolars. *Eur J Orthod*. 2000;22:245–255.

44. Winter GB, Gelbier MJ, Goodman JR. Severe Infra-occlusion and failed eruption of deciduous molars associated with eruptive and developmental disturbances in the permanent dentition: a report of 28 selected cases. *Br J Orthod*. 1997;24:149–157.

45. Endo T, Yoshino S, Ozoe R, Kojima K, Shimooka S. Association of advanced hypodontia and craniofacial morphology in Japanese orthodontic patients. *Odontology*. 2004;92:48–53.

46. Woodworth DA, Sinclair PM, Alexander RG. Bilateral congenital absence of maxillary lateral incisors: a craniofacial and dental cast analysis. *Am J Orthod*. 1985;87:280–293.

47. Ogaard B, Krogstad O. Craniofacial structure and soft tissue profile in patients with severe hypodontia. *Am J Orthod Dentofacial Orthop*. 1995;108:472–477.

48. Sarnas K-V, Rune B. The facial profile in advanced hypodontia: a mixed longitudinal study of 141 children. *Eur J Orthod*. 1983;5:133–143.

49. Wisth PJ, Thunold K, Boe OE. The craniofacial morphology of individuals with hypodontia. *Acta Odontol Scand*. 1974;32:281–290.

50. Ben-Bassat Y, Brin I. Skeletodental patterns in patients with multiple congenitally missing teeth. *Am J Orthod Dentofacial Orthop*. 2003;124:521–525.

51. Tavajohi-Kermani H, Kapur R, Sciote JJ. Tooth agenesis and craniofacial morphology in an orthodontic population. *Am J Orthod Dentofacial Orthop*. 2002;122:39–47.

52. Nodal M, Kjar I, Solow B. Craniofacial morphology in patients with multiple congenitally missing permanent teeth. *Eur J Orthod*. 1994;16:104–109.

53. Hobkirk JA, Brook AH. The management of patients with severe hypodontia. *J Oral Rehabil*. 1980;7:289–298.

54. Bishop K, Addy L, Knox J. Modern restorative management of patients with congenitally missing teeth: 4. The role of implants. *Dent Update*. 2007;34:79–80, 82–84.

55. Williams P, Travess HC, Sandy J. The use of osseointegrated implants in orthodontic patients: I. Implants and their use in children. *Dent Update*. 2004;31:287–290.

56. Thilander B, Odman J, Gröndahl K, Lekholm U. Aspects on osseointegrated implants inserted in growing jaws. A biometric and radiographic study in the young pig. *Eur J Orthod*. 1992;14:99–109.

57. Thilander B, Odman J, Grondahl K, Friberg B. Osseointegrated implants in adolescents. An alternative in replacing missing teeth? *Eur J Orthod*. 1994;16:84–95.

58. Brugnolo E, Mazzocco C, Cordioll G, Majzoub Z. Clinical and radiographic findings following placement of single-tooth implants in young patients—case reports. *Int J Periodont Restor Dent*. 1996;16:421–433.

59. Cronin RJ Jr, Oesterle LJ. Implant use in growing patients. Treatment planning concerns. *Dent Clin North Am*. 1998;42:1–34.

60. Fudalej P, Kokich VG, Leroux B. Determining the cessation of vertical growth of the craniofacial structures to facilitate placement of single-tooth implants. *Am J Orthod Dentofacial Orthop*. 2007;131:(4 suppl):S59–S67.

61. Oesterle LJ, Cronin RJ Jr, Ranly DM. Maxillary implants and the growing patient. *Int J Oral Maxillofac Implants*. 1993;8:377–387.

62. Cronin RJ Jr, Oesterle LJ, Ranly DM. Mandibular implants and the growing patient. *Int J Oral Maxillofac Implants*. 1994;9:55–62.

63. Björk A, Skieller V. Normal and abnormal growth of the mandible. A synthesis of longitudinal cephalometric implant studies over a period of 25 years. *Eur J Orthod*. 1983;5:1–46.

64. Björk A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. *J Dent Res*. 1963;42:400–411.

65. Björk A, Skieller V. Growth of the maxilla in three dimensions as revealed radiographically by the implant method. *Br J Orthod*. 1977;4:53–64.

66. Guckes AD, McCarthy GR, Brahim J. Use of endosseous implants in a 3-year-old child with ectodermal dysplasia: case report and 5-year follow-up. *Pediatr Dent*. 1997;19:282–285.

67. Johnson EL, Roberts MW, Guckes AD, et al. Analysis of craniofacial development in children with hypohidrotic ectodermal dysplasia. *Am J Med Genet*. 2002;112:327–334.

68. Kearns G, Sharma A, Perrott D, et al. Placement of endosseous implants in children and adolescents with hereditary ectodermal dysplasia. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1999;88:5–10.

69. Gill DS, Jones S, Hobkirk J, et al. Counselling patients with hypodontia. *Dent Update*. 2008;35:344–346, 348–350, 352.

70. Kokich VG, Kokich VO. Congenitally missing mandibular second premolars: clinical options. *Am J Orthod Dentofacial Orthop*. 2006;130:437–444.

71. Bernard JP, Schatz JP, Christou P, Belser U, Kiliaridis S. Long-term vertical changes of the anterior maxillary teeth adjacent to single implants in young and mature adults. A retrospective study. *J Clin Periodontol*. 2004;31:1024–1028.

72. Carmichael RP, Sándor GK. Dental implants, growth of the jaws, and determination of skeletal maturity. *Atlas Oral Maxillofac Surg Clin North Am*. 2008;16:1–9.

73. Hedegård B. The traumatised front tooth. Some prosthetic aspects on therapeutic procedures. *Rep Congr Eur Orthod Soc*. 1965; 41:347–357.

74. Bergendal B. When should we extract primary teeth and place implants in young individuals with tooth agenesis?. *J Oral Rehabil*. 2008;35(suppl 1):55–63.

75. Bergendal B, Norderyd J, Bagesund M, Holst A. Signs and symptoms from ectodermal organs in young Swedish individuals with oligodontia. *Int J Paediatr Dent*. 2006;16:320–326.

76. Becker A, Gillis I, Shpack N. The etiology of palatal displacement of maxillary canines. *Clin Orthod Res*. 1999;2:62–66.

77. Polder BJ, Van't Hof MA, Van der Linden FP, Kuijpers-Jagtman AM. A meta-analysis of the prevalence of dental agenesis of permanent teeth. *Commun Dent Oral Epidemiol*. 2004;32:217–226.

78. Altug-Atac AT, Erdem D. Prevalence and distribution of dental anomalies in orthodontic patients. *Am J Orthod Dentofacial Orthop*. 2007;131:510–514.

79. Bjerklin K, Al-Najjar M, Karestedt H, Andren A. Agenesis of mandibular second premolars with retained primary molars: a

longitudinal radiographic study of 99 subjects from 12 years of age to adulthood. *Eur J Orthod*. 2008;30:254–261.

80. Aktan AM, Kara S, Akgunlu F, Malkoc S. The incidence of canine transmigration and tooth impaction in a Turkish subpopulation. *Eur J Orthod*. 2010;32:575–581.

81. Aktan AM, Kara I, Sener I, et al. An evaluation of factors associated with persistent primary teeth. *Eur J Orthod*. 2012;34:208–221.

82. Joshi MR. Transmigrant mandibular canines: a record of 28 cases and a retrospective review of the literature. *Angle Orthod*. 2001;71:12–22.

83. Shapira Y, Kuftinec MM. Intrabony migration of impacted teeth. *Angle Orthod*. 2003;73:738–743.

84. Kurol J, Koch G. The effect of extraction of infraoccluded deciduous molars: a longitudinal study. *Am J Orthod*. 1985;87:46–55.

85. Dias C, Closs LQ, Fontanella V, de Araujo FB. Vertical alveolar growth in subjects with infraoccluded mandibular deciduous molars. *Am J Orthod Dentofacial Orthop*. 2012;141:81–86.

86. Robinson S, Chan MF. New teeth from old: treatment options for retained primary teeth. *Br Dent J*. 2009;207:315–320.

87. Kokich V. Early management of congenitally missing teeth. *Semin Orthod*. 2005;11:146–151.

88. Biggerstaff RH. The orthodontic management of congenitally absent maxillary lateral incisors and second premolars: a case report. *Am J Orthod Dentofacial Orthop*. 1992;102:537–545.

89. Spear F, Mathews D, Kokich VG. Interdisciplinary management of single-tooth implants. *Semin Orthod*. 1997;3:45–72.

90. Sabri R. Management of congenitally missing second premolars with orthodontics and single tooth implants. *Am J Orthod Dentofacial Orthop*. 2004;125:634–642.

91. Sletten DW, Smith BM, Southard KA, Casko JS, Southard TE. Retained deciduous mandibular molars in adults: a radiographic study of long-term changes. *Am J Orthod Dentofacial Orthop*. 2003;124:625–630.

92. Haselden K, Hobkirk JA, Goodman JR, Jones SP, Hemmings KW. Root resorption in retained deciduous canine and molar teeth without permanent successors in patients with severe hypodontia. *Int J Paediatr Dent*. 2001;11:171–178.

93. Stanford CM. Issues and considerations in dental implant occlusion: what do we know, and what do we need to find out? *J Calif Dent Assoc*. 2005;33:329–336.

94. Misch CE, Suzuki JB, Misch-Dietsh FM, Bidez MW. A positive correlation between occlusal trauma and peri-implant bone loss: literature support. *Implant Dent*. 2005;14:108–116.

95. Schulte W. Implants and the periodontium. *Int Dent J*. 1995;45:16–26.

96. Davies SJ. Occlusal considerations in implantology: good occlusal practice in implantology. *Dent Update*. 2010;37:610–612. 615–616, 619–620.

97. Spray JR, Black CG, Morris HF, Ochi S. The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering. *Ann Periodontol*. 2000;5:119–128.

98. Lops D, Chiapasco M, Rossi A, Bressan E, Romeo E. Incidence of inter-proximal papilla between a tooth and an adjacent immediate implant placed into a fresh extraction socket: 1-year prospective study. *Clin Oral Implants Res*. 2008;19:1135–1140.

99. Romeo E, Lops D, Rossi A, Storelli S, Rozza R, Chiapasco M. Surgical and prosthetic management of interproximal region with single-implant restorations: 1-year prospective study. *J Periodontol*. 2008;79:1048–1055.

100. Araujo M, Lindhe J. The edentulous alveolar ridge. In: Lindhe J, Karring T, Lang NP, eds. *Clinical Periodontology and Implant Dentistry*. 5th ed. Oxford, UK: Blackwell Munksgaard; 2003:53–63.

101. Nowzari H, Molayem S, Chiu CH, Rich SK. Cone beam computed tomographic measurement of maxillary central incisors to determine prevalence of facial alveolar bone width $\geq$2 mm. *Clin Implant Dent Relat Res*. May 11, 2010. doi:10.1111/j.1708–8208.2010.00287.x.

102. Ghassemian M, Nowzari H, Lajolo C, et al. The thickness of facial alveolar bone overlying healthy maxillary anterior teeth. *J Periodontol*. 2012;83:187–197.

103. Katranji A, Misch K, Wang H. Cortical bone thickness in dentate and edentulous human cadavers. *J Periodontol*. 2007;78:874–878.

104. Botticelli D, Berglundh T, Lindhe J. Hard-tissue alterations following immediate implant placement in extraction sites. *J Clin Periodontol*. 2004;31:820–828.

105. Johnson K. A study of the dimensional changes occurring in the maxilla following tooth extraction. *Aust Dent J*. 1969;14:241–244.

106. Fu JH, Yeh CY, Chan HL, et al. Tissue biotype and its relation to the underlying bone morphology. *J Periodontol*. 2010;81:569–574.

107. Le BT, Borzabadi-Farahani A. Labial bone thickness in area of anterior maxillary implants associated with crestal labial soft-tissue thickness. *Implant Dent*. 2012;21:401–406.

108. Horowitz R, Holtzclaw D, Rosen PS. A review on alveolar ridge preservation following tooth extraction. *J Evid Based Det Pract*. 2012;12(3 Suppl):149–160.

109. Gholami GA, Najafi B, Mashhadiabbas F, Goetz W, Najafi S. Clinical, histologic and histomorphometric evaluation of socket preservation using a synthetic nanocrystalline hydroxyapatite in comparison with a bovine xenograft: a randomized clinical trial. *Clin Oral Implants Res*. 2012;23:1198–1204.

110. Sisti A, Canullo L, Mottola MP, et al. Clinical evaluation of a ridge augmentation procedure for the severely resorbed alveolar socket: multicenter randomized controlled trial, preliminary results. *Clin Oral Implants Res*. 2012;23:526–535.

111. Kanomi R. Mini-implant for orthodontic anchorage. *J Clin Orthod*. 1997;31:763–767.

112. Melo AC, Jawonski ME, Largura LZ, et al. Upper molar intrusion in rehabilitation patients with the aid of microscrews. *Aust Orthod J*. 2008;24:50–53.

113. Oliveira DD, de Oliveira BF, de Araújo Brito HH, de Souza MM, Medeiros PJ. Selective alveolar corticotomy to intrude over-erupted molars. *Am J Orthod Dentofacial Orthop*. 2008;133:902–908.

114. Lee W, Karapetyan G, Moats R, et al. Corticotomy-/osteotomy-assisted tooth movement microCTs differ. *J Dent Res*. 2008;87:861–867.

115. Roblee RD, Bolding SL, Landers JM. Surgically facilitated orthodontic therapy: a new tool for optimal interdisciplinary results. *Compend Contin Educ Dent*. 2009;30:264–275.

116. Mantzikos T, Shamus I. Forced eruption and implant site development: soft tissue response. *Am J Orthod Dentofacial Orthop*. 1997;112:596–606.

117. Salama H, Salama M. The role of orthodontic extrusive remodeling in the enhancement of soft and hard tissue profiles prior to implant placement: a systematic approach to the management of extraction site defects. *Int J Periodont Restor Dent*. 1993;13:312–333.

118. Zuccati G, Bocchieri A. Implant site development by orthodontic extrusion of teeth with poor prognosis. *J Clin Orthod*. 2003;37:307–311.

119. Uribe F, Taylor T, Shafer D, Nanda R. A novel approach for implant site development through root tipping. *Am J Orthod Dentofacial Orthop*. 2010;138:649–655.

120. Tonetti MS, Hammerle CH. Advances in bone augmentation to enable dental implant placement: Consensus Report of the Sixth European Workshop on Periodontology. *J Clin Periodontol*. 2008;35(8 suppl):168–172.

121. Kokich VG, Kokich VO. Interrelationship of orthodontics with periodontics and restorative dentistry. In: Nanda R, ed.

Biomechanics and Esthetic Strategies in Clinical Orthodontics. St Louis, Mo: Elsevier; 2005:348–372.

122. Salama H, Salama M, Kelly J. The orthodontic-periodontal connection in implant site development. *Pract Periodont Aesthet Dent*. 1996;8:923–932.

123. Pietrokovski J, Massler M. Alveolar ridge resorption following tooth extraction. *J Prosthet Dent*. 1967;17:21–27.

124. Johnson K. A study of the dimensional changes occurring in the maxilla following closed face immediate denture treatment. *Aust Dent J*. 1969;14:370–376.

125. Pietrokovski J, Sorin S, Hirschfeld Z. The residual ridge in partially edentulous patients. *J Prosthet Dent*. 1976;36:150–158.

126. Schropp L, Wenzel A, Kostopoulos L, Karring T. Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study. *Int J Periodont Restor Dent*. 2003;23:313–323.

127. Van der Weijden F, Dell'Acqua F, Slot DE. Alveolar bone dimensional changes of post-extraction sockets in humans: a systematic review. *J Clin Periodontol*. 2009;36:1048–1058.

128. Ostler M, Kokich V. Alveolar ridge changes in patients congenitally missing mandibular second molars. *J Prosthet Dent*. 1994;71:144–149.

129. Uribe F, Chau V, Padala S, et al. Alveolar ridge width and height changes after orthodontic space opening in patients congenitally missing maxillary lateral incisors. *Eur J Orthod*. July 12, 2011. doi:10.1093/ejo/cjr072.

130. Nováčková S, Marek I, Kamínek M. Orthodontic tooth movement: bone formation and its stability over time. *Am J Orthod Dentofacial Orthop*. 2011;139:37–43.

131. Beyer A, Tausche E, Boening K, Harzer W. Orthodontic space opening in patients with congenitally missing lateral incisors. *Angle Orthod*. 2007;77:404–409.

132. Carmichael RP, Sándor GK. Dental implants in the management of nonsyndromal oligodontia. *Atlas Oral Maxillofac Surg Clin North Am*. 2008;16:11–31.

133. Gündüz E, Rodríguez-Torres C, Gahleitner A, Heissenberger G, Bantleon HP. Bone regeneration by bodily tooth movement: dental computed tomography examination of a patient. *Am J Orthod Dentofacial Orthop*. 2004;125:100–106.

134. Lindskog-Stokland B, Hansen K, Ekestubbe A, Wennström JL. Orthodontic tooth movement into edentulous ridge areas—a case series. *Eur J Orthod*. March 2, 2011. doi:10.1093/ejo/cjr029.

135. Petridis HP, Tsiggos N, Michail A, et al. Three-dimensional positional changes of teeth adjacent to posterior edentulous spaces in relation to age at time of tooth loss and elapsed time. *Eur J Prosthodont Restor Dent*. 2010;18:78–83.

136. Lindskog-Stokland B, Hansen K, Tomasi C, Hakeberg M, Wennström JL. Changes in molar position associated with missing opposed and/or adjacent tooth: a 12-year study in women. *J Oral Rehabil*. 2012;39:136–143.

137. Dickinson G. Space for missing maxillary lateral incisors—orthodontic perceptions. *Ann R Australas Coll Dent Surg*. 2000;15:127–131.

138. Olsen TM, Kokich VG Sr. Postorthodontic root approximation after opening space for maxillary lateral incisor implants. *Am J Orthod Dentofacial Orthop*. 2010;137:158.e1–158.e8.

139. Sharma A, Vargervik K. Using implants in the growing child. *J Calif Dent Assoc*. 2006;34:719–724.

140. Salinas TJ, Sheridan PJ, Castellon P, Block MS. Treatment planning for multiunit restoration—the use of diagnostic planning to predict implant and esthetic results in patients with congenitally missing teeth. *J Oral Maxillofac Surg*. 2005;63(9 suppl):45–58.

141. Carmichael RP, Sandor GKB. Dental implants in children, adolescents, and young adults. *Atlas Oral Maxillofac Surg Clin North Am*. 2008;16:vii–viii.

142. Sclar AG, Kannikal J, Ferreira CF, Kaltman SI, Parker WB. Treatment planning and surgical considerations in implant therapy for patients with agenesis, oligodontia, and ectodermal dysplasia: review and case presentation. *J Oral Maxillofac Surg*. 2009;67(11 suppl):2–12.