

Multidisciplinary Management of Hypodontia in Adolescents: Case Report

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ABSTRACT

The purpose of this article is to describe a team approach to treatment of hypodontia in adolescent dentition. A case report of hypodontia with a microdontic lateral incisor in a Class I malocclusion illustrates the principles of case management. Multidisciplinary consultation during treatment planning and coordination and appropriate timing of subsequent interdisciplinary dental care enables the clinician to provide the optimum care. The scope of orthodontic and restorative management depends on the severity of the hypodontia.

MeSH Key Words: anodontia/therapy; dental prosthesis, implant-supported; dental restoration, permanent; orthodontic space closure

© J Can Dent Assoc 2006; 72(8):740–6
This article has been peer reviewed.

Hypodontia is the developmental absence of 1 or more teeth.¹ Oligodontia is the term conventionally used in cases where 6 or more teeth are missing and anodontia, a much more rare finding, describes the developmental absence of all teeth.² The prevalence of hypodontia in the primary dentition ranges from 0.08% to 1.55%.¹ In the permanent dentition, prevalence has been reported to range from 2.3% to 11.3% depending on the population investigated.^{3–5} Hypodontia of third molars has a prevalence of 9% to 37%.⁶ Hypodontia in the primary dentition has no significant sex distribution, but in the permanent dentition females are affected more frequently than males by a ratio of 3:2.⁵

The etiology of hypodontia is unknown⁷; however, a definite familial trend has been reported.^{8,9} Brook⁹ suggests that most cases of hypodontia have a polygenetic inheritance pattern and that the risk of relatives having hypodontia will depend on a combination of numerous genetic and environmental factors,

each with a small effect. Hypodontia may also occur with no hereditary history. An association between hypodontia and microdontia has been found and affects females more than males.^{3,9} Conversely, the incidence of supernumerary teeth is greater in males, with an association between hyperdontia and macrodontia.^{3,9,10} Hypodontia has been found in association with impaction of permanent canines, maxillary canine–first premolar transposition and taurodontism.⁵

Although not all reports are in agreement, it is generally accepted that, excluding third permanent molars, the second mandibular premolar is the most frequently missing permanent tooth representing 40% to 50% of the total number of developing missing teeth.^{11,12} Hypodontia affecting the maxillary lateral incisor is next in terms of frequency (25%), followed by the maxillary second premolar (20%) and the mandibular central incisor (6.5%).¹¹ These 4 teeth account for 90% of absent teeth in hypodontia studies.^{11,13}



Figure 1a: Clinical views of malocclusion at initial presentation.



Figure 1b: Radiographic views at initial presentation: orthopantomographic view and periapical views of incisor area.



Figure 1c: Lateral cephalogram at initial presentation.

In approximately 80% of reported cases of hypodontia, only 1 or 2 teeth are missing; in 10%, 4 or more teeth are missing, while in fewer than 1%, 6 or more teeth are absent.⁵ Permanent first premolars, first molars and canines are very rarely developmentally absent; their absence is usually associated with severe hypodontia or oligodontia.¹⁴ A meta-analysis by Polder and colleagues¹⁵ on reported data from 1936 to 2002 found that the prevalence of hypodontia in Europe and Australia was higher than in North America. Hypodontia may occur in isolation or in association with such syndromes as ectodermal dysplasia, Down's syndrome Ellis van Crevald syndrome and such conditions as cleft lip and palate.^{5,16-19}

Hypodontia presents significant challenges for the clinician.⁵ Treatment options will depend on the severity of the case. Simple adhesive bridges may resolve mild hypodontia cases or it may be appropriate to close the resultant spaces by orthodontic movement of adjacent teeth. In more severe cases, a combined orthodontic–restorative–surgical approach may be necessary with orthodontic treatment needed to relocate space in preparation for later conventional fixed prostheses or implants.²⁰⁻²²

This case report of an adolescent female who presented with significant hypodontia illustrates the importance of an accurate diagnosis and an effective treatment plan that relies on appropriate coordination among orthodontist,

an oral surgeon and prosthodontist in terms of timing of interventions. The timing of extraction of retained primary teeth is also critical to the final result. This case report shows that it is sometimes better to delay the removal of retained primary teeth to maintain the surrounding dento-alveolar bone until implants are feasible.

Case Report

A girl, aged 12 years 9 months, was referred by her family dentist for orthodontic care to the Graduate Orthodontic Clinic, University of Western Ontario. The patient's presenting complaint was "the missing grown-up teeth and what happens next." Her general medical and dental histories were nonsignificant and she had no family history of any oral or dental anomaly. The patient was a regular attendee at dental appointments and had no history of extractions. Extraoral examination revealed a well-balanced face with normal facial profile and normal skeletal dental base relations. Intraoral examination revealed a Class I malocclusion in the late mixed dentition (**Figs. 1a to 1c, Table 1**). A 2-mm maxillary median diastema was present and the maxillary left lateral incisor (tooth 22) was microdontic. Oral hygiene and gingival status were good and no caries was found. Radiographic examination confirmed that 12 teeth were developmentally missing: teeth 18, 17, 12, 25, 27, 28, 38, 35, 31, 41, 45 and 48. Significant external root resorption was found in the retained primary teeth 75 and 85 (**Fig. 1b**).

Table 1 Cephalometric analysis at initial presentation and at debonding

| Cephalometric analysis | Normative values | Initial presentation (age 12 years, 9 months) | Debonding (age 15 years, 5 months) |
|---------------------------------------|------------------|-----------------------------------------------|------------------------------------|
| Skeletal | | | |
| <i>Anteroposterior</i> | | | |
| SNA angle; ° | 80 ± 2 | 79 | 77 |
| SNB angle; ° | 78 ± 2 | 77 | 76 |
| ANB angle; ° | 2 ± 2 | 2 | 1 |
| Facial angle; ° | 88 ± 5 | 85 | 86 |
| Maxillary convexity; mm | 2 ± 1 | 1 | 0 |
| A point perpendicular to nasion; mm | 0 ± 0.5 | -3 | -4 |
| Pogonion perpendicular to nasion; mm | -4 ± 3 | -8 | -7 |
| Wits; mm | -1 | -3 | -1 |
| <i>Vertical</i> | | | |
| Mandibular plane angle; ° | 26 ± 4 | 31 | 31 |
| Facial axis; ° | 90 ± 3 | 89 | 90 |
| Y axis to SN; ° | 64-68 | 69 | 69 |
| SN/GoGn; ° | 32 ± 4 | 38 | 39 |
| Maxillary/mandibular plane; ° | 28 ± 4 | 32 | 32 |
| Lower vertical face height; % | 55 | 57 | 57 |
| Upper vertical face height; % | 45 | 43 | 43 |
| Dental | | | |
| Maxillary incisor to sella-nasion; ° | 103 ± 5 | 98 | 104 |
| Maxillary incisor to palatal plane; ° | 110 ± 5 | 104 | 112 |
| Maxillary incisor to A vertical; mm | 5 ± 1 | 4 | 5 |
| Mandibular incisor to Md plane; ° | 90 ± 5 | 86 | 85 |
| Mandibular incisor to APg line; mm | 1 ± 2 | 1 | 2 |
| Mandibular incisor to NB line; mm | 3-4 | 3 | 4 |
| Interincisal angle; ° | 130 ± 5 | 140 | 131 |
| Maxillary incisal edge display; mm | 2-3 | 6 | 4 |
| Jarabak analysis | | | |
| Saddle angle (N-S-Ar); ° | 123 ± 5 | 123 | 125 |
| Articular angle (S-Ar-Go); ° | 143 ± 5 | 148 | 144 |
| Gonial angle (Ar-Go-Me); ° | 128 ± 7 | 126 | 128 |
| Upper gonial angle (Ar-Go-N); ° | 52-55 | 51 | 52 |
| Lower gonial angle (N-Go-Me); ° | 70-75 | 75 | 76 |
| Anterior cranial base (S-N); mm | 68-74 | 72 | 73 |
| Mandibular corpus (Go-Me); mm | 64-76 | 75 | 77 |
| ACB: corpus ratio | 1 : 1 | 1 : 1.04 | 1 : 1.05 |
| Posterior cranial base (S-Ar); mm | 30-36 | 34 | 35 |
| Ramus (Ar-Go); mm | 39-49 | 40 | 42 |

continued

| Cephalometric analysis | Normative values | Initial presentation (age 12 years, 9 months) | Debonding (age 15 years, 5 months) |
|----------------------------------------------------------------|---------------------------------------------|-----------------------------------------------|------------------------------------|
| PCB: ramus ratio | 3 : 4 | 3 : 3.5 | 3 : 3.6 |
| Posterior face height as % of anterior face height (S-Go/N-Me) | 54–59 clockwise; 65–75 counter-clockwise | 59.5 | 59 |
| Soft tissue analysis | | | |
| Frankfort plane to glabella; ° | 90 ± 3 | 89 | 87.5 |
| Subnasale to glabella vertical; mm | 6 | 4 | 4 |
| Pogonion to glabella vertical; mm | 0 | -4 | -3 |
| Lower lip to E plane; mm | -2 ± 2 | -2 | -3.5 |
| Facial contour angle (G-Sn-Pg); ° | 12 | 13 | 11 |

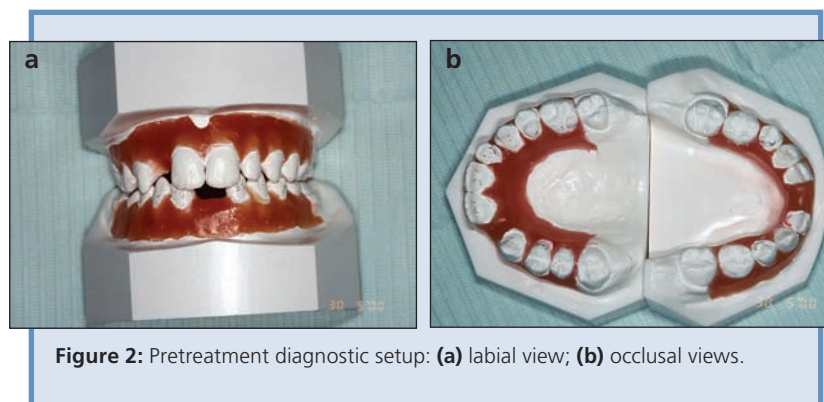


Figure 2: Pretreatment diagnostic setup: (a) labial view; (b) occlusal views.

Given the patient's presenting malocclusion, a multi-disciplinary team approach involving orthodontics, oral surgery and advanced restorative dentistry was essential in the consultation process, treatment planning and later clinical management of this case. The various treatment options open to the patient were considered. Nonintervention was not an option. Selective extraction of the retained primary teeth or their retention with buildups were also inappropriate options, given the severity of the hypodontia, the resultant poor esthetics and the malocclusion and poor root formation in 2 of the 3 retained primary molars. Limiting treatment to just 1 dental specialty, such as orthodontics, was also unrealistic. Orthodontics alone could not close the spaces or deal appropriately with the anterior occlusal asymmetry arising from the absent tooth 12.

In consultation with the patient, a combined orthodontic–restorative–surgical team approach to care was adopted. The objectives of orthodontic treatment were to correct the malocclusion and align the teeth in preparation for later prosthodontic care. To assist the multidisciplinary consultation process, a diagnostic setup was prepared and, with the patient's consent, a

treatment plan was agreed to involving all 3 specialties (Fig. 2). The orthodontic treatment took a non-extraction approach using a pre-adjusted fixed appliance system (Figs. 3a, 3b). Treatment commenced when the patient was 13 years and 1 month old. The patient was reviewed regarding her prosthodontic–restorative needs during orthodontic treatment and before debonding (Fig. 3c). Debonding was completed when the patient was 15 years and 5 months old (Figs. 4a and 4b, Table 1). Due to poor esthetics, teeth 71 and 81 were extracted following debonding (Fig. 4a). Conventional orthodontic retainers with replacement dental units were fitted initially with a view to the long-term insertion of implants and placement of final suprastructure fixtures (Fig. 5). The orthodontic goals during both the active and retentive phases were achieved with good treatment outcome.

The patient was followed in the Graduate Orthodontic Clinic until maturation of her gingival unit and completion of her skeletal growth. Two years after debonding, the patient was assessed in the fixed prosthodontic and oral surgery departments for final management of the edentulous spaces. When she was 19 years old, 3 implants



Figure 3a: Clinical view of orthodontic appliance in situ before debonding.



Figure 3b: Orthopantomographic view before debonding.



Figure 3c: Lateral cephalogram at debonding.



Figure 4a: Clinical views of occlusion following debonding and before buildup of the microdontic maxillary left lateral incisor.



Figure 4b: Radiographic views following debonding: orthopantomographic view and periapical views of incisor area.



Figure 5: Orthodontic Hawley retainers with dental units used in the interim period between debonding and placement of final crowns.

were placed at teeth 12 and 45 and in the mandibular midline area. Due to the lack of dentoalveolar bone in the mandibular midline area, an augmentation bone graft was necessary and was carried out 6 months before implant insertion using bone harvested from the right external oblique ridge. No surgical complications arose and subsequently coronal fixtures were placed in teeth 12, 31, 41 and 45 (Figs. 6a and 6b). The microdontic tooth 22 was built up to ensure symmetry with 12 (Fig. 6a). For now, the mandibular left primary second molar, which has good function, is being retained. In the long term, this tooth will be replaced with an implant as well. The patient, now aged 23 years, continues to be reviewed annually.

Discussion

Hypodontia, microdontia, supernumerary teeth and megadontia tend to be associated, and a number of researchers have proposed explanations for these associations.^{3,9,14} Brook⁹ attempted to unify the etiologic explanation for these associated dental features, proposing a multifactorial hypothesis with genetic and environmental components. Brook's model suggests that hypodontia and microdontia form one extreme on a scale, with megadontia and supernumerary teeth at the other end. His model explains the previously reported finding that males with hypodontia have more significant microdontia than females. Kjaer and co-workers¹⁷ suggest that the wide variation in the presentation of hypodontia



Figure 6a: Clinical views of occlusion, patient aged 21 years, 2 years following placement of implants and final crowns.



Figure 6b: Radiographic orthopantomographic view, 2 years following placement of implants and final crowns.

imply that the etiology is different for each case. They demonstrate that teeth located near the ends of peripheral nerve branches are most often affected by agenesis. They report hypodontia cases in which, on orthopantomographic examination, tooth agenesis is associated with the absence of the mandibular canal. They propose that, in cases of ectodermal dysplasia and Ellis van Crevald syndrome, the oral mucosa and supporting structures have a role in the etiology of hypodontia. In our case report, microdontia was found in association with the hypodontia but no family history of hypodontia could be found to support a genetic basis for this patient's presentation. No mucosal or bony abnormalities, which would have supported Kjaer's model, were found during clinical and radiographic examination.

This case report illustrates the need for a multidisciplinary team approach to care, not only at the treatment planning stage, but also throughout the entire course of treatment. The main objectives in the management of any hypodontia case are to improve esthetics and restore masticatory function; both were achieved in this case. Given that the patient presented in early adolescence, the timing of treatment and the coordination of care were additional critical components. Orthodontic treatment involved significant time, as implants and final prosthodontic restorations had to be delayed until gingival maturation and skeletal development were complete. Good coordination was achieved among all 3 dental specialties throughout

the treatment. In cooperation with the patient, a combined treatment plan was agreed to and the patient was reviewed at planned intervals both during active orthodontic treatment and later during retention. All pretreatment orthodontic goals were achieved without complication. Both arches were correctly aligned, with coincident midlines. Normal buccal and incisor relations were restored. The result was both occlusally and periodontally stable, while allowing appropriate access for later implant insertion. Restorative and oral surgeries were timed appropriately and both were carried out without complication.

One complication arose in the management of this case. Pretreatment bone levels in the mandibular midline area were low (**Fig. 1b**). Further bone loss occurred following extraction of the primary central incisors and before implant insertion, resulting in the definitive need for dentoalveolar bone augmentation. Carrying out the 2 mandibular primary central incisor extractions nearer the time of implant insertion may have been a better option. However, for patient esthetics, these teeth were removed and esthetic replacements placed on retainers.

Conclusions

Marked hypodontia demands coordinated treatment planning and appropriate timing of the delivery of care by various dental specialties. Management of hypodontia in adolescent patients permits optimum orthodontic control of the developing occlusion. This literature review and case report of hypodontia illustrate the principles of case management.

Multidisciplinary referral or consultation is important in treatment planning. Planning for space management is best carried out before initiating orthodontic treatment. A diagnostic setup is an essential adjunct to the treatment planning process. Tooth size measurements provide valuable data for evaluating the final tooth position and morphology. Careful consideration should be given to the timing of extraction of primary teeth and, if possible, extraction should coincide with implant insertion. ✦

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The authors have no declared financial interests.

References

- Whittington BR, Durward CS. Survey of anomalies in primary teeth and their correlation with the permanent dentition. *N Z Dent J* 1996; 92(407):4–8.
- Silva Meza R. Radiographic assessment of congenitally missing teeth in orthodontic patients. *Int J Paediatr Dent* 2003; 13(2):112–6.
- McKeown HF, Robinson DL, Elcock C, al Sharood M, Brook AH. Tooth dimensions in hypodontia patients, their unaffected relatives and a control group measured by a new image analysis system. *Eur J Orthod* 2002; 24(2):131–41.
- O'Dowling IB, McNamara TG. Congenital absence of permanent teeth among Irish school-children. *J Ir Dent Assoc* 1990; 36(4):136–8.
- Larmour CJ, Mossey PA, Thind BS, Forgie AH, Stirrups DR. Hypodontia — a retrospective review of prevalence and etiology. Part 1. *Quintessence Int* 2005; 36(4):263–70.
- Egermark-Eriksson I, Lind V. Congenital numerical variation in the permanent dentition. D. Sex distribution of hypodontia and hyperodontia. *Odontol Revy* 1971; 22(3):309–15.
- Goodman JR, Jones SP, Hobkirk JA, King PA. Hypodontia: 1. Clinical features and the management of mild to moderate hypodontia. *Dent Update* 1994; 21(9):381–4.
- Arte S, Nieminen P, Pirinen S, Thesleff I, Peltonen L. Gene defect in hypodontia: exclusion of EGF, EGFR, and FGF-3 as candidate genes. *J Dent Res* 1996; 75(6):1346–52.
- Brook AH. A unifying aetiological explanation for anomalies of human tooth number and size. *Arch Oral Biol* 1984; 29(5):373–8.
- Symons AL, Stritzel F, Stamation J. Anomalies associated with hypodontia and the permanent lateral incisor and second premolar. *J Clin Pediatr Dent* 1993; 17(2):109–11.
- Rose JS. A survey of congenitally missing teeth, excluding third molars in 6000 orthodontic patients. *Dent Pract Dent Rec* 1966; 17(3):107–14.
- Aasheim B, Ogaard B. Hypodontia in 9-year-old Norwegians related to need of orthodontic treatment. *Scand J Dent Res* 1993; 101(5):257–60.
- Rolling S. Hypodontia of permanent teeth in Danish schoolchildren. *Scan J Dent Res* 1980; 88(5):365–9.
- Hobkirk JA, Brook AH. The management of patients with severe hypodontia. *J Oral Rehabil* 1980; 7(4):289–98.
- Polder BJ, Van't Hof MA, Van der Linden FP, Kuijpers-Jagtman AM. A meta-analysis of the prevalence of dental agenesis of permanent teeth. *Community Dent Oral Epidemiol* 2004; 32(3):217–26.
- Kjaer I. Prenatal development of the maxillary primary incisors related to maturation of the surrounding bone and to postnatal eruption. In: Davidovitch Z, editor. Biological mechanisms of tooth eruption and root resorption. Birmingham (AL): EBSCO Media; 1988. p. 233–6.
- Kjaer I, Kocsis G, Nodal M, Christensen LR. Aetiological aspects of mandibular tooth agenesis — focusing on the role of nerve, oral mucosa, and supporting tissues. *Eur J Orthod* 1994; 16(5):371–5.
- Ribeiro LL, Das Neves LT, Costa B, Ribeiro Gomide M. Dental anomalies of the permanent lateral incisors and prevalence of hypodontia outside the cleft area in complete unilateral cleft lip and palate. *Cleft Palate Craniofac J* 2003; 40:172–5.
- Kumasaka S, Miyagi A, Sakai N, Shindo J, Kashima I. Oligodontia: a radiographic comparison of subjects with Down syndrome and normal subjects. *Spec Care Dent* 1997; 17(4):137–41.
- Thind BS, Stirrups DR, Forgie AH, Larmour CJ, Mossey PA. Management of hypodontia: orthodontic considerations. Part II. *Quintessence Int* 2005; 36(5):345–53.
- Forgie AH, Thind BS, Larmour CJ, Mossey PA, Stirrups DR. Management of hypodontia: restorative considerations. Part III. *Quintessence Int* 2005; 36(6):437–45.
- Huang LH, Shotwell JL, Wang HL. Dental implants for orthodontic anchorage. *Am J Orthod Dentofacial Orthop* 2005; 127(6):713–22.