

پزشک آموز

به من نگاه کن



پزشک آموز

اولین رسانه دیجیتال در حوزه علوم پزشکی

☎ 011-3335 5440

📍 0901 601 9192

📞 0901 601 9192

✉ pezeshkamooz.co@gmail.com

✉ poshtibani@pezeshkamooz.com

🌐 pezeshkamooz.com

Orthodontic Management of the Developing Dentition

An Evidence-Based Guide

Martyn T. Cobourne
Editor

 Springer

Orthodontic Management of the Developing Dentition

Martyn T. Cobourne
Editor

Orthodontic Management of the Developing Dentition

An Evidence-Based Guide

 Springer

Editor

Martyn T. Cobourne
Department of Orthodontics
Centre for Craniofacial Development and Regeneration
King's College London Dental Institute
London
United Kingdom

ISBN 978-3-319-54635-3 ISBN 978-3-319-54637-7 (eBook)
DOI 10.1007/978-3-319-54637-7

Library of Congress Control Number: 2017947876

© Springer International Publishing AG 2017

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

Management of the developing dentition has always been a fundamental role of the orthodontist. The transition from primary to secondary dentition is characterised by variation rather than conformity, and a multitude of local and more general problems can manifest during this period of development. However, in an era of evidence-based medicine, there is a surprising paucity of high-quality data to help inform decisions that often need to be made during this stage of development.

This textbook provides a rich source of information on the many aspects of dental development that an orthodontist might be engaged with. The text begins with an overview of the normal development of the dentition and the management of early space loss, including enforced extraction of first permanent molars. It then covers local problems associated with the mixed dentition, including tooth agenesis and supernumerary teeth, dental trauma and impacted teeth, including maxillary incisors and canines. Further chapters cover the interceptive management of class II and class III discrepancies and problems associated with the transverse dimension.

The chapters have been written by an international group of authors who have considerable expertise in the management of malocclusion and, in many cases, first-hand experience of conducting high-quality clinical trials investigating treatment interventions for these problems. The title of the book proclaims that it is evidence-based and in some areas it is. In particular, there have been recent advances in our knowledge of best practice for managing impacted maxillary canines and both class II and class III malocclusions. However, there are many common clinical problems that affect the developing dentition, which currently have only anecdote and retrospective clinician experience to inform them. Much work needs to be done in investigating many of these interventions with appropriate methodology. In the meantime, this textbook will provide you with the best current evidence that there is.

London, UK

Martyn T. Cobourne

Contents

1	Development of the Dentition	1
	Maisa Seppala and Martyn T. Cobourne	
2	Space Loss and Crowding	21
	Anthony J. Ireland, Fraser McDonald, Rebecca John, and Jonathan R. Sandy	
3	First Permanent Molars	33
	Gavin J. Mack	
4	Supernumerary Teeth	53
	Helen Tippett and Martyn T. Cobourne	
5	Tooth Agenesis	67
	Sirpa Arte, Wael Awadh, Pekka Nieminen, and David P. Rice	
6	Trauma to the Permanent Maxillary Incisors in the Mixed Dentition and Orthodontics	85
	Jadbinder Seehra and Serpil Djemal	
7	Impacted Maxillary Central Incisors	109
	Shruti Patel	
8	Early Management of the Palatally Displaced Maxillary Permanent Canine	131
	Philip E. Benson and Nicola A. Parkin	
9	Early Treatment of Class II Malocclusion	151
	Andrew DiBiase and Paul Jonathan Sandler	
10	Class III Malocclusion	169
	Simon J. Littlewood	
11	Early Management of Posterior Crossbites	185
	Jayne E. Harrison	

Maisa Seppala and Martyn T. Cobourne

Abstract

Respiration, swallowing, speech and mastication are the primary roles of the oral cavity. The human dentition has evolved to effectively carry out the latter function by having teeth with different sizes and shapes and by going through a transition from primary to secondary dentitions that ensure optimal space and occlusal relationships in the adult. Teeth start forming early during the sixth week of embryonic development and are governed by molecular signals that ensure the right teeth develop at the right time in the right place. The first primary (deciduous) teeth emerge during infancy around 6 months of age, and following many dynamic stages of dental development and facial growth, the final secondary (permanent) third molar teeth erupt around the age of 19 years to complete the permanent dentition. However, even after this event, occlusal changes continue to take place through late-stage facial growth, alveolar development, post-emergent eruption and occlusal forces.

Development of the Dentition

The human dentition begins formation in the embryo with postnatal development characterised by the transition from deciduous to permanent dentitions. The deciduous dentition consists of two incisors, one canine and two molars in each dental quadrant, whilst the permanent dentition consists of the successional incisors, canines and premolars and accessional molars (Fig. 1.1).

M. Seppala • M.T. Cobourne (✉)
Department of Orthodontics, Craniofacial Development and Stem Cell Biology,
King's College London Dental Institute, London SE1 9RT, UK
e-mail: martyn.cobourne@kcl.ac.uk

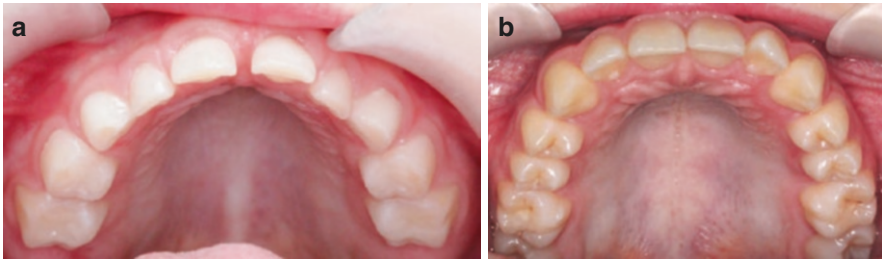


Fig. 1.1 The human dentition forms as a transition from deciduous to permanent dentition. The deciduous dentition consists of two incisors, one canine and two molars in each dental quadrant (a), whilst adult jaws accommodate an additional two premolars between the canine and first molar as well as a third molar (b)

Embryonic Dental Development

The first 3 months of embryonic development are crucial for formation of the facial structures that derive from five fundamental processes: the paired mandibular, paired maxillary and frontonasal processes [1]. The oral surfaces of these processes provide the platform for dental development as the lower dentition derives from tissue components originating in the mandibular processes and the upper incisors as well as the rest of the maxillary teeth develop within the frontonasal and maxillary processes, respectively. The appearance of the horseshoe-shaped epithelial thickenings that form in the early oral cavity around 6 weeks of gestation marks the start of dental development. Subsequently, this continuous epithelial band divides into an outer vestibular and inner dental lamina, the former giving rise to the lip and cheek vestibules and the latter to the enamel organs of the teeth [2, 3].

Molecular Basis of Dental Development in Brief

Teeth are epithelial appendages like hair, sweat glands and nails and share many similar morphological and molecular stages during their development. Their growth relies on epithelial-mesenchymal interactions mediated by secreted signalling molecules that, in turn, induce expression of multiple transcription factors. These signals are repeatedly used at different stages of dental development, and after first establishing oral-aboral and mesiodistal polarity in the jaws, then continue to regulate initiation, growth, morphogenesis, cell differentiation and cusp patterning of the teeth [4–6].

Humans are heterodonts, who have teeth with different sizes and shapes including two incisors, one canine, two premolars and three molars in each dental quadrant. The current developmental model for investigating tooth development is the mouse, which has a reduced dentition in comparison to humans. However, there is much commonality in the fundamental mechanisms underlying tooth development in mouse and human due to their genomic similarity and comparable stages of dental development [7].

In mice, teeth with different morphology develop depending on their mesiodistal position in either of the jaws, and the heterodont patterning is under control of at least

two well-studied signalling molecules, bone morphogenetic protein 4 (Bmp4) and fibroblast growth factor 8 (Fgf8) expressed by the oral epithelium. Bmp4 specifies the incisor region by inducing expression of homeobox-containing transcription factors *Msh homeobox 1 (Msx1)* and 2 (*Msx2*) in the underlying mesenchyme and in the molar field through Fgf8 initiating expression of *BarH-like homeobox 1 (Barx1)* and *Distal-less 2 (Dlx2)* [8, 9]. Significantly, murine studies have shown that inhibition of Bmp4 results in ectopic expression of *Barx1* in the presumptive incisor region, which can cause transformation of the incisors into teeth with more molariform characteristics, highlighting the importance of these homeobox genes in regulating heterodont patterning [9].

After mesiodistal polarity has been established, two signalling molecules, sonic hedgehog (Shh) and Wnt7b, become reciprocally expressed in the oral epithelium. Interestingly, the expression domain of *Shh* corresponds to the tooth-forming region and *Wnt7b* to the non-tooth-forming oral epithelium. Subsequently, their roles have been shown to delineate the regions that have potential for tooth formation [10]. At the time when tooth formation is initiated, Fgf8 also provides an inductive signal for formation of the localised thickenings in the oral epithelium that give rise to dental placodes. Fgf8 continues to induce proliferation in the dental placodes and together with Shh controls early cellular morphogenetic changes that result in progression of tooth development from a thickening to bud stage [11]. Following this early patterning, a whole host of molecules become dynamically expressed and take part in communication between the oral epithelium and underlying mesenchyme to ensure normal progression of dental development.

Histological Basis of Dental Development in Brief

The different stages of dental development are named after their resemblance to the shape of the invaginating epithelium that progress from thickening to bud, cap, bell and late bell stages. The surrounding mesenchyme condenses around the invaginating epithelium and at cap stage becomes partly encapsulated. At the bell stage, the enamel knots at the tip of the future cusps become visible. These are signalling centres that are important for morphogenesis and required for normal cusp formation in molars. At the late bell stage, histodifferentiation begins and derivatives of the oral ectoderm give rise to enamel-producing ameloblasts, whilst the rest of the tooth originates from cranial neural crest-derived mesenchyme, including dentin-producing odontoblasts, cementum-producing cementoblasts, periodontal ligaments and pulpal tissue [3, 4, 6]. Calcification of the deciduous dentition begins around 3–4 months of embryonic development [3, 12].

As diphyodonts, humans have two generations of teeth. Preparation for the transition from primary to secondary dentition begins during prenatal life. Successional secondary teeth develop from localised lingual proliferations from the dental lamina of their corresponding primary predecessors and give rise to two incisors, canine and two premolars in each dental quadrant. The rest of the secondary dentition are accessional teeth that include all three molars that form from a backward extension of the distal aspect of the primary second molar. The first sign of successional tooth development is seen around 3–4 months, whilst the accessional teeth start to form around 5 months of embryonic development [3] (Fig. 1.2).

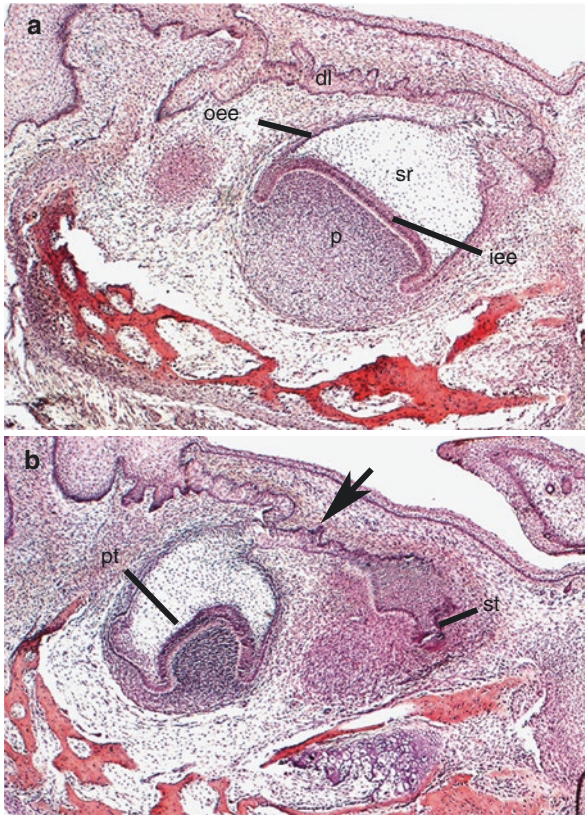


Fig. 1.2 Late cap stage tooth germ: developing mandibular incisor (a) and mandibular canine (b). During human embryonic development, the dental lamina (dl) connects the cap stage tooth germ to the oral epithelium. Outer enamel epithelium (oee) and inner enamel epithelium (iee) derive from the invaginated oral epithelium, and the iee gives rise to enamel-producing ameloblasts. Encapsulated neural crest-derived mesenchymal cells adjacent to the iee receive signals from the iee and differentiate into dentin-producing odontoblasts. Pulpal tissue (p) also originates from the mesenchyme. *Sr* stellate reticulum (a). The successional permanent tooth (st) is beginning to develop on the lingual side of the bud stage primary tooth (pt), and these are linked together by the successional lamina (arrow) (b)

Postnatal Development of the Dentition (Box 1.1)

At birth the head is nearly half of the body mass, and the mandible is strikingly small and retrognathic in relation to the maxilla [2]. The upper lip is short and the lower lip forms the majority of the anterior seal. Even when dental development has begun already early on during prenatal life, the infant's first smile is predominated by the presence of edentulous gum pads. However, inside the developing alveolar processes, dental development is well underway as the deciduous central incisor crowns have almost fully calcified, and the rest of the deciduous dentition has also begun this process.

Box 1.1 Key Stages of Development of the Deciduous Dentition

- 6 weeks of gestation: development of the deciduous dentition begins.
- 6 months: first deciduous teeth, mandibular central incisors, erupt.
- 2½ years: all 20 deciduous teeth have erupted.
- Root development of deciduous dentition is completed within 12–18 months after their eruption to oral cavity.
- Teeth normally erupt within a few weeks from eruption of the teeth in the contralateral side of the same jaw.

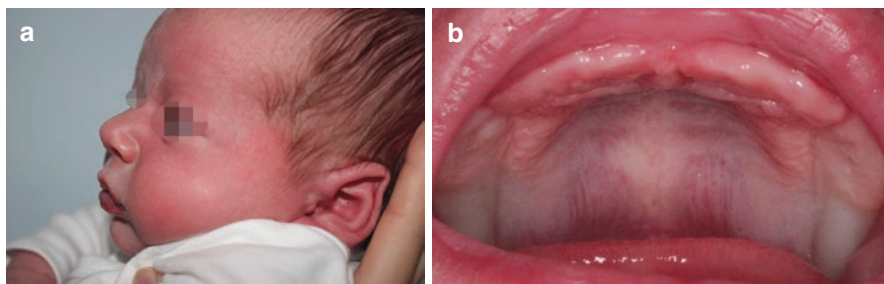


Fig. 1.3 Profile view of a 3-week-old baby boy and oral view of the maxilla. Newborn children have a small mandible in comparison to the maxilla (a), and their edentulous gum pads have small segmented elevations that mark the sites of the developing teeth (b)

The First 6 Months

In the newborn maxilla, the horseshoe-shaped gum pad surrounds the shallow palate and overlaps the U-shaped mandibular gum pad. The future positions of the developing teeth can be seen on the gum pads as small segmented elevations separated by small transverse grooves (Fig. 1.3). One of these grooves, the ‘lateral sulcus’, is prominent, extending vertically to the buccaneers sulcus and corresponding to the distal side of the future deciduous canine. The second molars are the last deciduous teeth to start developing, and subsequently the elevations in the prospective second molar regions do not become evident until around 5 months of age. Just palatal or lingual to these elevations, a dental groove represents a structure that is a remnant of the invaginated epithelial organs of the developing teeth. In addition, in the maxilla another shallow groove, the gingival groove, is located more palatally and anatomically separates the alveolar and palatal epithelium apart from each other [2, 3].

At birth, some of the infant’s vital physiological functions need to change abruptly, and the time of adaptation to a new environment can be associated with disruptions in enamel calcification resulting in formation of the so-called neonatal line. Normally this horizontal line is not visible by eye, but following more stressful or complicated births, it can become noticeable [13], and its location depends on the developmental stage of the relevant tooth crown at the time of birth. Although most

newborns are edentulous, 1:1000 to 1:30,000 depending on the racial group have natal teeth at birth or neonatal teeth that erupt within 30 days after birth. These are most frequently seen in the mandibular incisor region and can be either supernumerary or prematurely erupted deciduous teeth. It is difficult at this stage to determine if they are part of the normal complement of the deciduous dentition, and thereafter the decision on their removal depends on the presence of any possible symptoms such as disrupted feeding that can cause inadequate nutrient intake, ulcerations or increased mobility with risk of aspiration [14].

Infants get their nutrition up to the first 3–6 months exclusively from breast or substitute milk, and their ability to thrive depends on establishment of a successful feeding pattern. In the newborn, the tongue normally sits between the maxilla and mandible and during suckling forms a seal against the lower lip. Coordinated movements of the tongue, lips and cheeks during suckling activate the facial muscles and are considered to be important in stimulating facial growth [15]. During the first year, significant transverse growth takes place in both maxillary and mandibular sutures providing approximately 2 mm more space for eruption of the deciduous incisors [16].

From 6 Months to 5 Years of Age

The first deciduous teeth erupt around the same time as the infant adapts to a more complex swallowing pattern and is physiologically ready for weaning [17]. Teething can be a big event in the infant's life, as their eruption can cause multiple relatively minor symptoms such as general irritability, disturbed sleep, an increase in body temperature, drooling, gum-rubbing and increased biting [18].

The first deciduous teeth to emerge are the lower central incisors (median age 6.8 months), and contralateral teeth normally erupt only a few weeks apart from each other. Although some variation does take place in the eruption pattern, typically every following few months, a new pair of incisors erupts in the following sequence: maxillary central incisors (9.1 months), lateral maxillary incisors (9.8 months) and lower lateral incisor (11.4 months) [19]. By the end of the first year of life, around two thirds of deciduous incisor root development is completed. Variable degrees of root formation are also evident in the rest of the developing deciduous dentition excluding the second molars that are just completing their crown formation [12].

After a short while, eruption of the deciduous teeth resumes with eruption of the first molars (maxillary 14.8 and mandibular 15.4 months) slightly before the canines (maxillary 17.6 and mandibular 18.0 months). The last deciduous teeth to erupt are the second deciduous molars (mandibular at 26.2 months, maxillary at 26.6 months), following which, the full complement of five deciduous teeth is present in each dental quadrant [19]. Root development of the primary dentition is completed approximately 12–18 months after their eruption, and subsequently all deciduous incisors complete their root development by age of 2 years, first molars 2½ years and second molars 3 years, and deciduous canines are the last ones to complete their root development around 3¼ years of age [12].

Eruption sequence of the primary dentition is more important than chronological timing. Large variation in eruption times exists, and it is not unusual that some of the children do not get their first teeth until the age of one (Table 1.1). As a general rule, 6-month deviation from the average eruption times is considered normal. If a child at the age of 3 years has not yet attended any dental appointments, this is now a good time to visit dentist or dental hygienist in order to confirm normal dental development and good dental health. The primary dentition is usually established by the age of 3 years (Fig. 1.4).

Space and Occlusal Development in the Primary Dentition

From birth to 2 years of age, the intercanine width increases around 3.5 mm in the mandible and 5 mm in the maxilla [20]. Subsequently, even the primary incisors might erupt in crowded positions, transverse growth ideally results in spacing in the labial segments providing additional space for the wider permanent incisors to erupt. In addition to generalised upper and lower incisor spacing, ‘primate spaces’ mesial to the upper canines and distal to the lower canines provide further space for permanent dentition and are the most prevalent feature of the primary dentition [21].

Deciduous incisors are typically more upright, and the overbite tends to be transiently deep in the early deciduous dentition. Overbite reduces as a result of increase in the posterior lower facial height that is anteriorly compensated by post-emergent eruption and augmentation of the alveolar bone. Both jaws also grow in an

Table 1.1 Deciduous dentition: median eruption times

Deciduous	C incisor	L incisor	Canine	First molar	Second molar
Maxilla (months)					
Median	9.1	9.8	17.6	14.8	26.6
In brackets 5 and 95% percentiles	(6.8–12.7)	(7.2–15)	(13.6–23.8)	(11.8–18.5)	(20.1–34.4)
Mandible (months)					
Median	6.8	11.4	18.0	15.4	26.2
In brackets 5 and 95% percentiles	(4.3–10.6)	(7.9–16.7)	(14.0–24.6)	(11.8–18.8)	(20.2–33.1)

According to Nyström [19]

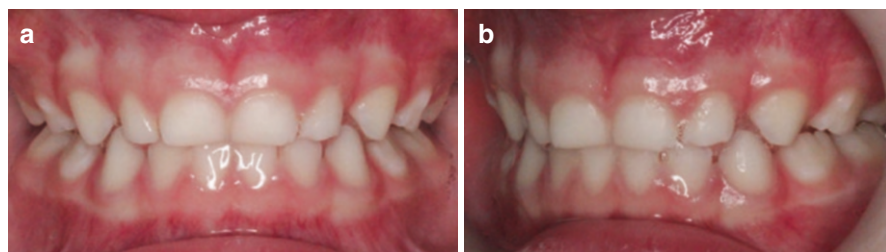


Fig. 1.4 Primary dentition. Complete primary dentition of a 3-year-old girl with the presence of primary spaces mesial to the maxillary canines and distal to the lower canines (a, b)

anterior-posterior direction, and, significantly, the mandible grows faster than the maxilla, resulting in more optimal jaw relationships, reduction in overjet and eruption of the teeth in better occlusion [2, 22]. Molar length increases with its highest rate up to the age of 3 years, providing posteriorly more space for eruption of the rest of the primary dentition [20]. In the deciduous dentition, the most common molar relationship is edge to edge, and the tendency for Class II malocclusion is much more common than Class III [21].

Development of the Permanent Dentition (Box 1.2)

Transition from deciduous dentition to permanent dentition is divided into two stages, early and late mixed dentition, as permanent teeth erupt in groups. These two stages of rapid dental development are separated by around a year and a half of more silent period of time, when no further deciduous teeth exfoliate, but progression of dental development can be assessed from radiographs based on the amount of crown and root development as well as the presence of root resorption in the deciduous dentition.

The Early Mixed Dentition (6–8 Years) (Fig. 1.5)

Transition to the permanent dentition commonly begins around the same time with the start of the juvenile growth spurt, at around 6–7 years of age, as the mandibular central incisors (6–7 years) or first molars (5.5–7 years) erupt. Only another year later, the mandibular lateral incisors (7–8 years) erupt around the same time with the

Box 1.2 Key Stages of Development of the Permanent Dentition

- 14 weeks of embryonic development: development of the permanent dentition begins.
- 6 years: first permanent teeth, mandibular central incisors or first molars, erupt.
- 9–10 years: permanent maxillary canines palpable bilaterally in the buccal sulcus.
- 11 years: permanent maxillary canines erupt.
- 12 years: permanent second molars erupt and complete development of the permanent dentition (excluding third molars).
- 19 years: if present, permanent third molars erupt; however, their timing has a lot of variation depending on the space availability.
- Root development of the permanent dentition completed within 2–3 years after their eruption to oral cavity.
- Eruption within 2 years from average eruption time is considered as normal variation.
- Teeth normally erupt within half a year from eruption of the teeth in the contralateral side of the same jaw.

Others

- Eruption sequence in both deciduous and permanent dentition is more important than correlation between eruption times and chronological age.
- Females are typically ahead of their male counterparts in terms of their dental development.

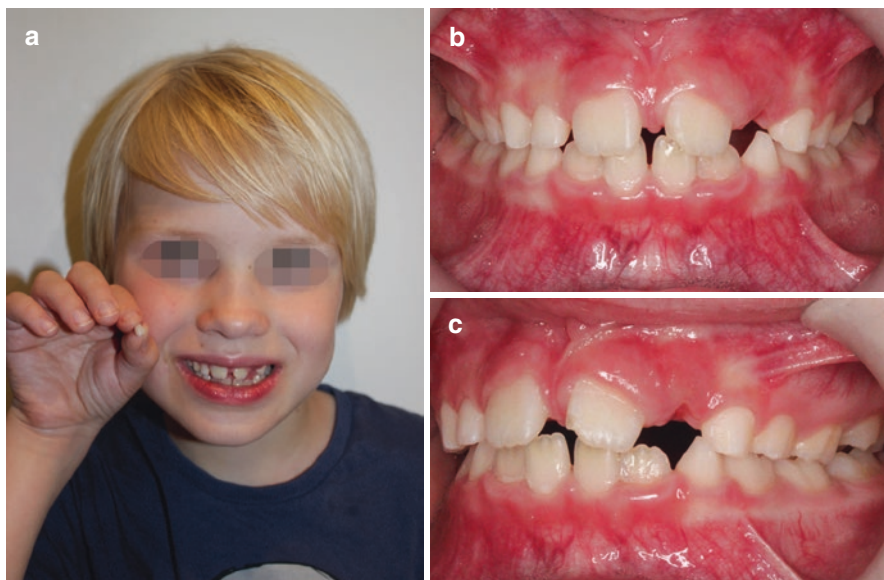


Fig. 1.5 Early mixed dentition. Transition to early mixed dentition usually begins around 6–7 years of age (a). Teeth exfoliate following root resorption of the deciduous teeth and after atraumatic loss good healing of the gingival tissue can already be seen a few hours after exfoliation (b, c)

upper central incisors (7–8 years). Lastly, the early mixed dentition stage is complete as the upper lateral incisors erupt at approximately 8–9 years of age [12]. As the deciduous teeth exfoliate, the permanent successors are expected to erupt within the following 6 months. The same time frame is applied when a permanent tooth has erupted on one side; the contralateral deciduous tooth can be expected to be lost again within the following 6 months. Similarly to the deciduous dentition, the correlation between chronological and dental age is also poor in the permanent dentition, and up to 2 year deviations from average eruption times are considered normal.

Occlusal Features of the Early Mixed Dentition (Fig. 1.6)

As the upper central incisors erupt, they can be flared distally leaving a space in the midline. This midline diastema can be present due to crowding inside the anterior maxillary bone where the unerupted lateral incisor crowns are still in their vertical



Fig. 1.6 Complete early mixed dentition. Class I early mixed dentition with very mild crowding in the lower labial segment in a 9-year-old boy. Deciduous dentition has thinner enamel and in the presence of acidic diet is more prone for erosion than permanent dentition (a–d)

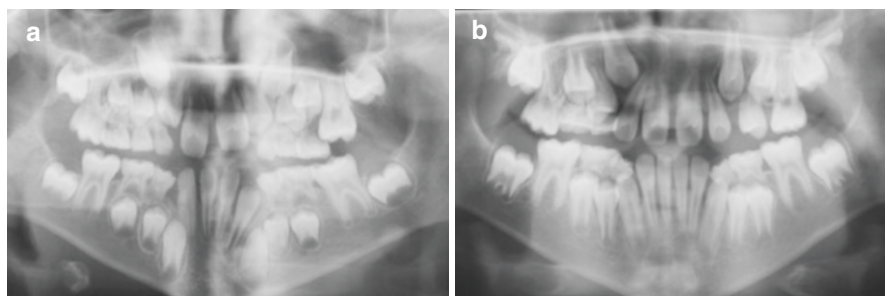


Fig. 1.7 Radiographic view of the 'ugly duckling stage'. Following their eruption, the upper central incisors can be flared due to unerupted upper lateral incisors causing pressure on the distal surface of the central incisor roots (a). After eruption of the lateral incisors, this same effect can move distally, resulting in high upper canines now causing distal angulation of the upper lateral incisor crowns and spacing (b)

position high and can apply pressure on the central incisor roots causing their crowns to tip laterally. Following eruption of the lateral incisors, this same effect can shift distally, and consequently the canine crowns can now compress the lateral incisor roots. Although there are multiple reasons for the presence of midline diastema, in the early mixed dentition, this phenomenon can be part of normal physiological development, and midline spaces up to around two millimetres can easily resolve spontaneously following the eruption of the maxillary canines. This transient period of physiological spacing and flaring of the maxillary incisors is often referred as 'ugly duckling stage' (Fig. 1.7).



Fig. 1.8 Occlusal features of the early mixed dentition. Up to 6 mm spacing in the deciduous dentition is required to provide enough space for larger permanent teeth. Developing anterior open bite present in this 3-year-old child with no family history of anterior open bite is likely to be caused by a prolonged dummy-sucking habit (a). Midline diastema and deep bite can be transient physiological features of the mixed dentition. Permanent central incisor is approximately 2 mm wider than deciduous central incisor (b). Permanent teeth develop lingually in relation to their permanent successors, and occasionally in the crowded dentition, lower incisors can erupt ectopically in the side of the tongue (c, d)

Permanent incisors are visibly larger and develop on the lingual/palatal side in relation to their deciduous predecessors. In the lower arch, four permanent incisors can take around six millimetres more space than the deciduous incisors. This space is obtained in variable ways such as by utilising the deciduous incisor spacing and permanent incisors erupting in more proclined inclination establishing a wider dental arch. Even the intercanine and molar widths are essentially already established at the age of 8 years; a small increase in the intercanine distance takes place at the time when the canines erupt [16]. In the mandible approximately 1 mm increase is obtained as the lower canines erupt taking up the primate spaces available distal to the lower deciduous canines. In contrast, in the maxilla lateral incisors take up the primate spaces that are present in the maxillary arch mesial to the deciduous canines. However, approximately 3 mm increase is gained partially because the maxillary canines erupt in more buccal positions in comparison to their deciduous predecessors. Lack of spacing between deciduous incisors is a strong predictor of future incisor crowding in the permanent dentition. Up to 6 mm space is required to reduce the risk of developing incisor crowding [22]. In the presence of crowding or retained deciduous incisors, the permanent incisors can erupt lingually/palatally due to their developmental position warranting removal of the retained deciduous teeth or interceptive orthodontic treatment if maxillary incisors erupt in crossbite (Fig. 1.8).

The Late Mixed Dentition (10–13 Years) (Fig. 1.9)

Eruption of the mandibular canines (9–11 years) marks the transition to late mixed dentition that happens around the same time as the start of the adolescent growth spurt. Lower canines erupt more buccally and distally than their predecessors that can result in increase in the mandibular intercanine width and provide small amount of additional space for lower incisors.

First mandibular premolars erupt almost at the same time (10–12 years) with the maxillary first premolars (10–11 years). At around 11–12 years of age, the maxillary canines (11–12 years) and all four second premolars (maxillary 10–12 years and mandibular 11–13 years) erupt. This is followed by eruption of the all four second molars around the age of 11–13 years in the mandible and 12–13 years in the maxilla that completes the late mixed dentition stage [12]. However, dental development still continues as formation of the third molars is now underway and undergoing crown calcification that is normally radiographically evident. Also, root development of the permanent teeth is not completed until around 2–3 years after their eruption [12] (Table 1.2).

Maxillary canine crown development is started at around 4 months of embryonic development, and root development is not completed until the age of around 13½ years. They also have a long eruption pathway that is guided by the lateral incisor roots [23]. In contrast to other successional teeth, maxillary canines erupt later than teeth immediately distal to them, making them more prone for localised crowding, and subsequently they often erupt in buccally displaced positions. These factors together with familial tendency [24] can all contribute to the fact that around 2% of the maxillary canines are impacted [25]. Majority of the unerupted canines are displaced palatally (61%) but can also be impacted aligned with the dental arch (34%) or in the buccal position (4.5%) [26]. If the maxillary canines are not palpable buccally at 10 years of age, it can be indicative of them being palatally ectopic. Early diagnosis of the palatally positioned maxillary canines can be beneficial as removal of the deciduous canines at the right time can sometimes normalise the eruption pathway of the maxillary canines depending on the severity of their displacement [27].

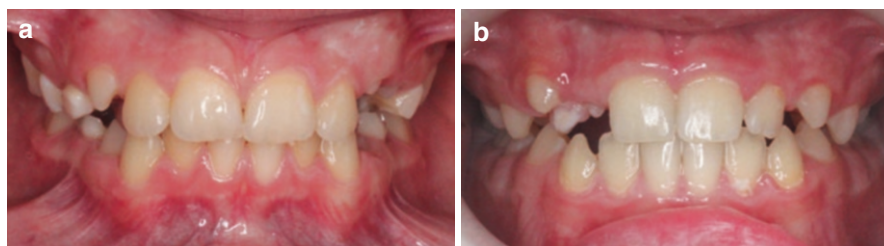


Fig. 1.9 Asymmetric dental development. Over 6 months delayed eruption of the contralateral tooth can be a sign of abnormal dental development or pathology. Normal dental development and symmetric eruption of the maxillary canines taking place in a 12-year-old boy (a) and delayed eruption of the upper right lateral incisor at the same time with upper canines seen in a same-age cousin whose father has a peg-shaped upper lateral incisor, that is, a dental feature with strong inheritance pattern (b)

Table 1.2 Permanent dentition: calcification of the crown begins and completed, eruption times and completion of root formation

Arch and tooth	Calcification begins	Crown completed (years)	Eruption (years)	Root completed (years)
Mx Ci	3–4 months (in utero)	4–5	7–8	9–10
Mx Li	10–12 months (in utero)	4–5	8–9	11
Mx canine	4–5 months (after birth)	6–7	11–12	12–15
Mx first premol	1.5–1.8 years (after birth)	5–6	10–11	12–13
Mx second premol	2–2.3 years (after birth)	6–7	10–12	12–14
Mx first molar	At birth	2.5–3	5.5–7	9–10
Mx second molar	2.5–3 years	7–8	12–14	14–16
Mx third molar	7–9 years (after birth)	13 ^a	17–30	18–25 ^a
Md Ci	3–4 months (in utero)	4–5	6–7	9–10
Md Li	3–4 months (in utero)	4–5	7–8	10
Md canine	4–5 months (after birth)	6–7	9–11	12–15
Md first premol	1.3–2 years (after birth)	5–6	10–12	12–13
Md second premol	2.3–2.5 years (after birth)	6–7	11–13	12–14
Md first molar	At birth	2.5–3	5.5–7	9–10
Md second molar	2.5–3 years (after birth)	7–8	12–14	14–16
Md third molar	8–10 years (after birth)	13.5 ^a	17–30	18–15 ^a

According to Logan and Kronfeld [12]

^aBased on data of Nyström [19]

Space and Occlusal Development in the Late Mixed Dentition (Box 1.3)

In contrast to incisors, the total mesiodistal width of the permanent canine and two premolars occupy less space than their deciduous predecessors. This space accounts for up to 2.5 mm space in the mandible and 1.5 mm in the maxilla and is called 'leeway space'. Majority of the leeway space is provided by wide deciduous second molars, and subsequently most of this space is used by first molars that following the loss of the second deciduous molars rapidly move mesially. Significantly, leeway space also contributes to formation of the Class I molar relationship as more space for mesial migration of the molars is available in the mandibular arch than in the maxilla [28]. Therefore, even the flush end of the first molars is the most common relationship in the early mixed dentition, the differential mesial movement as well as faster growth of the mandible in comparison to maxilla contributes all together for 3–4 mm more mesial movement of the mandibular than the maxillary

Box 1.3 Features Ensuring Adequate Space for Permanent Dentition

- Transverse and anterior-posterior growth of the jaws.
- Incisor spacing in the primary dentition.
- Primate spaces in the maxilla mesial and in the mandible distal to deciduous canines.
- Leeway space 1.5 mm in the maxilla and 2.5 mm in the mandible.
- Incisors erupt into more proclined positions than upright deciduous incisors.

Table 1.3 Mesiodistal widths of the deciduous and permanent teeth

	Deciduous mesiodistal width combined value for girls and boys (mm)	Permanent mesiodistal width girls (mm)	Permanent mesiodistal width boys (mm)
Mx Ci	6.4	8.6	8.9
Mx Li	5.2	6.6	6.9
Mx canine	6.8	7.7	8.0
Mx first premol		6.9	7.0
Mx second premol		6.6	6.7
Mx first molar	6.9	9.8	10.1
Mx second molar	8.5	9.3	9.6
Md Ci	4.0	5.4	5.5
Md Li	4.6	5.9	6.1
Md canine	5.8	6.6	7.0
Md first premol		6.9	7.0
Md second premol		6.9	7.0
Md first molar	7.5	10.3	10.7
Md second molar	9.4	9.9	10.2

According to Lysell and Myrberg [30]

molar, contributing to the establishment of Class I molar relationship. Similarly, if Class II molar relationship is found in the deciduous dentition, this is likely to improve and Class III is likely to get worse around the time when a child shifts from the deciduous to permanent dentition. Mainly because of the loss of the leeway space and mesial movement of the molars, the arch perimeter reduces during the transition to the permanent dentition approximately 3.5 mm in boys and 4.5 mm in girls [29] (Table 1.3; Fig. 1.10).

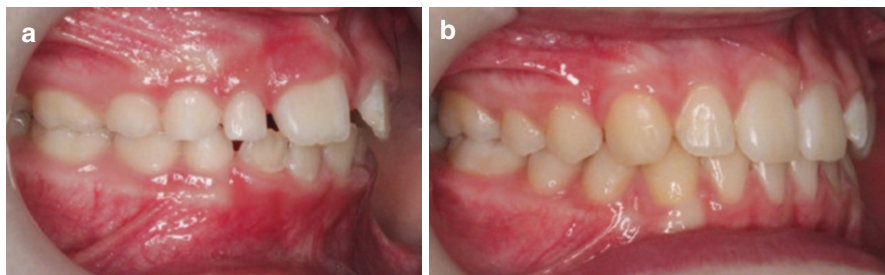


Fig. 1.10 Establishing Class I molar relationship. Deciduous molars are wider (a) than their successor premolars (b). In addition, the mandibular first and second deciduous molars are wider than the maxillary deciduous molars contributing to the flush vertical relationship of the distal ends of the deciduous second molars (a). This allows more mesial movement of the lower than upper permanent first molars at the time when deciduous teeth exfoliate subsequently facilitating to the development of Class I molar relationship (b)



Fig. 1.11 Permanent dentition. Full permanent Class I dentition in a 13-year-old boy (a–d)

Permanent Dentition (13 Years Onwards) (Fig. 1.11)

Dental development continues even after eruption of the second molars. Over 20% of the population have missing third molars [31, 32], and the rest develop one to four third molars that typically start crown calcification around the age of 9 years and complete by 14 years of age. Third molars erupt in on average around the age of 19 years, but due to posterior crowding and angular orientation, they can often be delayed or impacted.

After teeth have reached the occlusal level and completed root development, they still continue to erupt. This continued eruption together with alveolar growth compensates the increase in the ramus and condylar height and results in further increase in the lower anterior facial height during adolescence and adulthood [2]. Late alveolar growth and eruption is important to bear in mind when assessing the prognosis of the ankylosed teeth or planning the suitable time for implant placement. Neither ankylosed teeth nor implants have the ability to erupt and subsequently can become infraoccluded if the ankylosis has taken place during the childhood or adolescence or if the implants have been placed during the time of active growth.

Teeth are also exposed to occlusal forces that can cause attrition occlusally and interproximally resulting in changes in occlusion. Specifically, the mandibular teeth are subject to occlusal forces with an anterior force vector that together with anterior growth rotation of the mandible as well as eruption of the third molars are thought to contribute to the development of the late incisor crowding [33]. Although late incisor crowding typically occurs during late teen years, it can also appear later on during adulthood [34]. Eruption of the third molars has traditionally been thought to increase the anterior forces and likelihood of late incisor crowding. However, multiple studies have shown that individuals who have absent lower third molars also develop late incisor crowding providing evidence that third molars are not alone responsible for late incisor crowding, but the aetiology is more likely to be multifactorial [35, 36].

Eruption Is Divided in Five Different Stages

Eruption occurs in two distinct stages consisting of pre-emergent and post-emergent eruption. Post-emergent eruption can be further divided in four phases of pre-functional spurt that takes place until teeth meet the occlusal level and juvenile equilibrium, adolescent eruptive spurt and adult equilibrium that compensate for the vertical facial growth.

Pre-emergent eruption begins at the time when root formation is initiated, and two coordinated but independent processes are taking place: eruptive movement of the tooth and resorption of the surrounding bone. When successional teeth erupt, also root resorption of the deciduous predecessors is necessary before they can obtain their correct positions in the dental arch. Interactions between the follicle and surrounding bone consisting of osteoclasts and osteoblasts stimulate resorption that clears the eruption pathway and can subsequently act as a rate-limiting factor for eruption. Studies done on dogs and coincidental finding on patients who have experienced trauma show that resorption of the surrounding bone occurs even after teeth have been ligated to the lower border of the mandible [37, 38]. On the other hand, after removal of the root apex, teeth still have potential to erupt, providing evidence that eruptive movement does not only rely on root development [10]. These studies together indicate that eruptive movement and bone resorption are two distinct mechanisms that are differentially regulated.

Eruption continues even after completion of root development. Indeed, after molars have reached the occlusal level, they still continue to erupt approximately

one centimetre in order to keep up with vertical facial growth. Multiple mechanisms have been suggested to induce the post-emergent eruption. Interestingly, studies using video microscope revealed that majority of the juvenile equilibrium premolar eruption took place between 6 pm and 1 am. This time correlated with high release of the growth hormone rather than changes in occlusal forces. Other mechanisms that have been suggested to stimulate eruption are shrinking and cross-linking of maturing collagen fibres as well as vascular pressure created by blood flow in the periodontal ligaments [38]. In addition, a more recent theory suggests that patterns of compression and tension created by occlusal forces and mediated by soft tissues provide a lifting force to teeth towards the oral cavity as compression in the coronal part of the tooth causes resorption and tension in the apical end creates bone [39].

Conclusions

Understanding normal development and maturation of the dentition is essential for any dentist who treats children, and it forms the basis for appropriate diagnosis and treatment. Any considerable deviation from average dental development can be an indication of underlying pathology or malocclusion, and it is important to be able to identify when further investigation or referral to a specialist is necessary. Furthermore, a thorough knowledge of dental developmental stages can help to predict the consequences of environmental influences, such as maternal or childhood illnesses and dental trauma.

References

1. Francis-West PH, Robson L, Evans DJ. Craniofacial development: the tissue and molecular interactions that control development of the head. *Adv Anat Embryol Cell Biol.* 2003;169:III–VI, 1–138.
2. Enlow DH, Hans MG. *Essentials of facial growth.* Philadelphia: Saunders; 1996.
3. Kumar GS, Bhaskar SN. *Orban's oral histology and embryology.* 13th ed. London: Elsevier; 2011.
4. Cobourne MT, Sharpe PT. Making up the numbers: the molecular control of mammalian dental formula. *Semin Cell Dev Biol.* 2010;21(3):314–24.
5. Thesleff I, Sharpe P. Signalling networks regulating dental development. *Mech Dev.* 1997;67(2):111–23.
6. Tucker A, Sharpe P. The cutting-edge of mammalian development; how the embryo makes teeth. *Nat Rev Genet.* 2004;5:499–508.
7. Seppala M, Zoupa M, Onyekwelu O, Cobourne MT. Tooth development: 1. Generating teeth in the embryo. *Dent Update.* 2006;33(10):582–4, 586–8, 590–1.
8. Thomas BL, Tucker AS, Qui M, Ferguson CA, Hardcastle Z, Rubenstein JL, Sharpe PT. Role of *Dlx-1* and *Dlx-2* genes in patterning of the murine dentition. *Development.* 1997;124(23):4811–8.
9. Tucker AS, Matthews KL, Sharpe PT. Transformation of tooth type induced by inhibition of BMP signalling. *Science.* 1998;282:1136–8.
10. Sarkar L, Cobourne M, Naylor S, Smalley M, Dale T, Sharpe PT. Wnt/Shh interactions regulate ectodermal boundary formation during mammalian tooth development. *Proc Natl Acad Sci U S A.* 2000;97(9):4520–4.

11. Li J, Chatzeli L, Panousopoulou E, Tucker AS, Green JB. Epithelial stratification and placode invagination are separable functions in early morphogenesis of the molar tooth. *Development*. 2016;143(4):670–81.
12. Logan W, Kronfeld R. Development of the human jaws and surrounding structures from birth through the age of fifteen years. *J Am Dent Assoc*. 1933;20:379–427.
13. Kurek M, Zadzińska E, Sitek A, Borowska-Struginska B, Rosset I, Lorkiewicz W. Prenatal factors associated with the neonatal line thickness in human deciduous incisors. *Homo*. 2015;66(3):251–63.
14. Newadkar UR, Chaudhari L, Khalekar YK. Natal and neonatal teeth: terminology with diverse superstitions!! *J Family Med Prim Care*. 2016;5(1):184–5.
15. Festila D, Ghergie M, Muntean A, Matiz D, Serb Nescu A. Suckling and non-nutritive sucking habit: what should we know? *Clujul Med*. 2014;87(1):11–4.
16. Bishara SE. Arch width changes from 6 weeks to 45 years of age. *Am J Orthod Dentofacial Orthop*. 1997;111:401–9.
17. Mahoney P. Dental fast track: prenatal enamel growth, incisor eruption, and weaning in human infants. *Am J Phys Anthropol*. 2015;156(3):407–21.
18. McIntyre GT, McIntyre GM. Teething troubles? *Br Dent J*. 2002;192:251–5.
19. Nyström M. Clinical eruption of deciduous teeth in a series of Finnish children. *Proc Finn Dent Soc*. 1977;73:155–61.
20. Sillman JH. Dimensional changes of the dental arches: longitudinal study from birth to 25 years. *Am J Orthod*. 1964;50:824–42.
21. Foster TD, Hamilton MC. Occlusion in the primary dentition. *Br Dent J*. 1969;126:76–9.
22. Leighton BC. The early signs of malocclusion. *Trans Eur Orthod Soc*. 1969;45:353–68.
23. Becker A. In defense of the guidance theory of palatal canine displacement. *Angle Orthod*. 1995;65(2):95–8.
24. Peck SM, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. *Angle Orthod*. 1994;64(4):249–56.
25. Ericson S, Kurol J. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbance. *Eur J Orthod*. 1986;8(3):133–40.
26. Stivaros N, Mandall NA. Radiographic factors affecting the management of impacted upper permanent canines. *J Orthod*. 2000;27(2):169–73.
27. Naoumova J, Kürol J, Kjellberg H. Extraction of the deciduous canine as an interceptive treatment in children with palatally displaced canines – part II: possible predictors of success and cut-off points for a spontaneous eruption. *Eur J Orthod*. 2015;37(2):219–29.
28. Moyers RE. *Handbook of orthodontics*. 4th ed. Ann Arbor: Year book Medical Publisher; 1988. p. 127, 238.
29. Moorrees CF, Reed RB. Changes in dental arch dimensions – expressed on the basis of tooth eruption as a measure of biological age. *J Dent Res*. 1965;44:129–41.
30. Lysell L, Myrberg N. Mesiodistal tooth size in the deciduous and permanent dentition. *Eur J Orthod*. 1982;4:113–22.
31. Mok YY, Ho KK. Congenitally absent third molars in 12 to 16 year old Singaporean Chinese patients: a retrospective radiographic study. *Ann Acad Med Singapore*. 1996;25(6):828–30.
32. Rozkovicova E, Markova M, Lanik J, Zvarova J. Development of third molar in the Czech population. *Prague Med Rep*. 2004;105(4):391–422.
33. Richardson ME. Late lower arch crowding: the aetiology reviewed. *Dent Update*. 2002;29(5):234–8.
34. Jonsson T, Arnlaugsson S, Saemundsson SR, Magnusson TE. Development of occlusal traits and dental arch space from adolescence to adulthood: a 25-year follow-up study of 245 untreated subjects. *Am J Orthod Dentofacial Orthop*. 2009;135(4):456–62.
35. Richardson ME. Late lower arch crowding: facial growth or forward drift? *Eur J Orthod*. 1979;1:219–25.

36. Zawawi KH, Melis M. The role of mandibular third molars on lower anterior teeth crowding and relapse after orthodontic treatment: systematic review. *ScientificWorldJournal*. 2014;2014(615429):1–6.
37. Cahill DR, Marks SC Jr. Tooth eruption: evidence for the central role of the dental follicle. *J Oral Pathol*. 1980;9:189–200.
38. Proffit WR, Frazier-Bowers SA. Mechanism and control of tooth eruption: overview and clinical implications. *Orthod Craniofac Res*. 2009;12:59–66.
39. Sarrafpour B, Swain M, Li Q, Zoellner H. Tooth eruption results from bone remodelling driven by bite forces sensed by soft tissue dental follicles: a finite element analysis. *PLoS One*. 2013;8(3):e58803.

Anthony J. Ireland, Fraser McDonald, Rebecca John,
and Jonathan R. Sandy

Abstract

Crowding and spacing within the dental arches is largely under genetic control but is affected by a number of local factors. This chapter describes these local factors, discusses how they can influence the development of the dentition and also describes the interceptive measures available to the orthodontist.

Introduction

Orthodontists are often expected to predict, prevent and treat the effects of crowding and space loss during development of the dentition. Although this may entail comprehensive treatment, more often than not, it comprises of short, interceptive measures. Before describing these measures, we should first look at the aetiology of crowding and space loss.

A.J. Ireland (✉) • R. John • J.R. Sandy
School of Oral and Dental Sciences, University of Bristol,
Lower Maudlin St., Bristol BS1 2LY, UK
e-mail: tony.ireland@bristol.ac.uk; Rebecca.John@uhbristol.nhs.uk;
jonathan.sandy@bristol.ac.uk

F. McDonald
Guys Hospital, Kings College London Dental Institute, London SE1 9RT, UK
e-mail: fraser.mcdonald@kcl.ac.uk

Aetiology of Crowding

For the teeth to fit perfectly within the dental arches and to be in the correct relationship with those in the same and opposing jaws, mesiodistal tooth widths should be a match with the jaws. Any discrepancy in the sizes of either the teeth or the jaws is likely to lead to spacing or, more commonly, crowding. Although the aetiology is most likely to be genetic in origin, other more local factors can have an effect on how this crowding or spacing may present within an individual patient. These include, for example, extra teeth, missing teeth, retained deciduous teeth and the early unscheduled loss of teeth.

It is worth at this point perhaps asking the question “*Has crowding and space loss always been an issue in the developing dentition?*”. Studies on pre-industrialised civilisations have shown that in most instances, there is little evidence that dental crowding was present to the same extent as is seen today [1, 2]. This has led to the theory that early civilisations ate a more abrasive diet, which resulted in the loss of tooth tissue, not only occlusal but also interdental. As a result, the mesiodistal tooth widths of the teeth were thought to gradually reduce over time, permitting all of the teeth to fit within the arches, including the third permanent molars [3, 4]. Therefore pre-industrialised occlusions were not without crowding, it was perhaps just less common.

Predictors of Crowding in the Developing Dentition

As has previously been described in Chap. 1, the presence of severe crowding in the deciduous dentition is relatively rare. More often than not, the teeth (in particular, the incisors) are slightly spaced. Indeed, it is thought the degree of crowding or spacing of the deciduous incisors can be used as a possible predictor of the likely crowding that may be initially seen in the early permanent dentition. It was Leighton who suggested that if the deciduous incisors were well aligned, with no spacing or crowding, then there were more than 2 in 3 chances that the permanent incisors would be crowded [5]. If the sum of the spaces was less than 3 mm, the chance of crowding was slightly better than 1 in 2, and if the sum of the spacing was between 3 and 6 mm, this improved to 1 in 5. Where the total was greater than 6 mm then there was little chance of permanent incisors being crowded. Other than this, there is little in the way of predicting crowding.

Local Factors Affecting Crowding and Space Loss

Local factors that may affect crowding and subsequent space loss include:

- Early loss of deciduous teeth
- Retained deciduous teeth
- Developmental absence of teeth
- Unscheduled loss of permanent teeth

- Extra teeth (supernumerary and supplemental)
- Anomalies in tooth form (microdont and megadont)
- Anomalies in tooth position

Of all of these local factors, it is relatively easy to understand how the presence of large, small or extra teeth will have a direct influence on the presence of spacing or crowding. What is not quite so easy to understand is the effect when teeth are lost prematurely through trauma or disease. The most important factor is most probably the presence or absence of crowding. If the arches are spaced, in both the mixed or permanent dentitions, then the effect of early loss on the remaining teeth within the same arch is likely to be minimal. However, in the presence of crowding, the loss of a tooth is likely to lead to drifting of the adjacent teeth towards the site of loss. This space loss, in turn, can affect occlusal relationships leading to a change in the molar relationship or a shift in the dental centreline. The earlier a tooth is lost, the greater the likely effect on the developing occlusion. Each of these local factors will now be described in turn.

Early Loss of Deciduous Teeth

The effect of early loss of deciduous teeth will, as previously described, depend largely on the underlying crowding within the permanent dentition. If there is no crowding, the effect will be minimal. However, in the presence of crowding, the effect will depend on which tooth is lost and the age at which this occurs. In general, the more anterior the tooth loss the greater the effect on the centreline, and the more posterior the tooth loss the greater the effect on the buccal segment tooth relationship, usually as a result of mesial movement of the first permanent molar. In order to try to prevent a shift in the dental centreline, it is sometimes useful to extract the same tooth on the opposite side of the same arch, known as a balancing extraction.

With this in mind, “*Should balancing extractions always be performed?*” The loss of a deciduous incisor is not usually balanced. However, whenever a deciduous canine is lost, due, for example, to resorption of its root by the permanent lateral incisor, or a first deciduous molar is lost prematurely due to caries, it is worth balancing the loss. This can be done either by the extraction of the opposite deciduous canine or first deciduous molar, in order to prevent a shift of the dental centreline. If a second deciduous molar is lost prematurely, the effect on the centreline is minimal and so it should not be balanced. The greatest effect of early loss of the second deciduous molar is mesial drifting of the first permanent molar, which is then likely to encroach on the space for the second premolar. This often results in the premolar being squeezed out of the arch (Fig. 2.1) and eventually erupting palatal to the arch. In all cases the earlier the loss of the deciduous tooth, the greater the effect it has on either the centreline or the buccal segment relationship.

“*Should we ever retain the space following early loss of a deciduous tooth?*” In most instances the answer is no and there are a number of reasons for this. Firstly, if the deciduous tooth has been lost prematurely due to caries, then such a patient is



Fig. 2.1 Panoramic radiograph showing an impacted upper second premolar following early loss of the deciduous second molar

Fig. 2.2 Fixed space maintainer. Notice how the lower first premolar is beginning to erupt and there is just sufficient space



unlikely to be a good candidate for the long-term wear of either a fixed or removable space maintainer. Secondly, as has already been mentioned, in the absence of crowding, there is no need to maintain the space, and, thirdly, if there is moderate to severe crowding, extractions may be required at a later date in any case. Only very occasionally is space maintenance the treatment option of choice, and an example would be the enforced extraction of an ankylosed and submerging deciduous tooth, where space maintenance might obviate the need for any future orthodontic treatment. In such a case the space can be maintained with a removable or a fixed space maintainer (Fig. 2.2).

“What about compensating extractions?” A compensating extraction is an extraction of a tooth in the opposing arch, and the aim is to preserve the buccal segment relationships of the teeth. In general compensating extractions in the deciduous dentition are less often performed than balancing extractions.

Retained Deciduous Teeth

Deciduous teeth are not infrequently retained beyond their normal age of eruption. This may be associated with the ectopic path of eruption or developmental absence of the permanent successor or the presence of chronic infection at the deciduous tooth root apex (Fig. 2.3), all of which may delay the normal process of root resorption that leads to the tooth being naturally shed. In some instances this failure of resorption can lead to ankylosis and the appearance of a submerging tooth. In reality, it is not the affected deciduous tooth that is submerging. What is in fact happening is that with continued facial growth the adjacent teeth erupt relative to the ankylosed tooth, which then appears to submerge. Submergence is most commonly seen in the deciduous molar regions and, if left unchecked, can lead to the adjacent teeth tipping over the occlusal surface of the submerging deciduous molar (Fig. 2.4). In extreme circumstances the deciduous tooth can submerge so far that it will not be visible in the mouth, only on a radiograph. Not only does this make removal of the submerged tooth somewhat difficult but it can also lead to space loss, with insufficient room left for the permanent successor to erupt.

This begs the question “*If and when should a submerging deciduous tooth be extracted?*” In reality a degree of submergence during the lifetime of a deciduous molar is a relatively common part of normal occlusal development. The eventual natural loss of a deciduous tooth is a dynamic process of root resorption and repair, and provided there is more resorption than repair, the tooth may submerge a little, re-erupt and then is ultimately shed. However, if there is more repair and little resorption, the tooth is likely to ankylose and continue to submerge. If a permanent successor is present and the deciduous tooth is only slightly submerged, being above the contact points of the adjacent teeth and with no signs of these teeth tipping over its occlusal surface, then the deciduous tooth can be kept under observation. If the tooth submerges below the contact points of the adjacent teeth and they begin tipping over the occlusal surface, then extraction of the deciduous tooth and space management might be required [6].



Fig. 2.3 Retained upper central deciduous incisors preventing the eruption of the permanent central incisor teeth

Fig. 2.4 Submerging upper left second deciduous molar. Notice how the adjacent teeth are tipping over the submerging tooth



Developmental Absence of Teeth

It is very rare that deciduous teeth are developmentally absent. However, the developmental absence of permanent teeth is relatively common. Excluding the third permanent molars, there are reports that it may affect between <math><0.1\%</math> [7] and 10.3% of children [8]. The most common missing tooth, apart from the third permanent molar, is the upper second premolar, followed by the upper lateral incisor, the lower second premolar and the lower central incisor. In the early permanent dentition, when it is discovered on a radiograph that a permanent tooth is developmentally absent, there are a number of possible treatment options for the retained deciduous tooth, including:

- Preservation of the deciduous tooth for as long as possible provided it is in good condition. It can then be replaced when naturally shed with a prosthetic tooth. There are reports of second deciduous molars being retained in the mouth until the fifth decade of life [9], which is longer than many intraoral prostheses are able to survive.
- To extract the deciduous tooth in order to encourage mesial movement on eruption of other, as yet unerupted permanent teeth. In this way the space created by the missing tooth is either closed or reduced, eliminating or reducing the need for later orthodontic treatment or a prosthetic replacement tooth. For this to work there should be some underlying crowding; otherwise the teeth may not spontaneously drift into the primary tooth extraction space.
- To preserve the deciduous tooth until a later date when it can be extracted as part of a comprehensive orthodontic treatment plan to relieve crowding, align the teeth and close the space or relocate the space prior to the provision of a definitive prosthetic replacement.

Whichever treatment option is chosen, it is important a full orthodontic and radiographic assessment is undertaken, being mindful that in the case of apparently missing second premolars, these may not become apparent radiographically until 9 years of age [10].

Unscheduled Loss of Permanent Teeth

As with the deciduous dentition, the effect of the loss of a permanent tooth will be dependent on a number of factors:

- *Presence or absence of crowding*—as with the loss of a deciduous tooth, the loss of a permanent tooth will have a greater effect within the same arch in the presence of crowding. This is because crowding will promote drifting of the adjacent teeth into the extraction site.
- *Position of the tooth within the arch*—the more anterior the tooth loss, the greater the effect on the centreline. Therefore the loss of a central incisor in a crowded arch will have a profound effect on the centreline (Fig. 2.5), whilst the loss of a second permanent molar will have minimal effect. Conversely the loss of a posterior tooth will have a greater effect on the buccal segment relationship than the loss of an incisor.
- *Patient age*—in general and in the presence of crowding, the earlier the tooth loss, the greater the effect, as the adjacent erupted and also unerupted teeth will drift towards the extraction site. Therefore the effect will be greater in the developing dentition than in the mature adult dentition.
- *The occlusion*—the angulations of the teeth adjacent to the extraction site and the interdigitation of remaining teeth within the arch with those in the opposing arch will both have an effect on space loss. Erupted teeth will more readily tip than bodily move into an extraction site. Therefore, if the crown of a tooth is angulated away from an extraction site, it is more likely to move into the extraction site than if it is angulated towards it (Fig. 2.6). The interdigitation of teeth, particularly in the buccal segments, may also have an effect on space loss. If the interdigitation in the buccal segments is very good, it may prevent the teeth adjacent to an extraction site from spontaneously drifting into it and closing the space. Indeed, such interdigitation can be sufficiently effective in this regard as to sometimes make space closure even with fixed appliances more difficult.



Fig. 2.5 Loss of the upper left central incisor tooth has resulted in space loss and a shift of the upper centreline to the left

Fig. 2.6 Notice how the permanent canine is mesially angulated in this crowded case. Loss of the first premolar during the eruption of the canine would have encouraged the canine to tip back into the extraction space



Fig. 2.7 Loss of the upper left central incisor in this crowded case has led to complete space loss as a result of drifting of the adjacent teeth



Previously we have described the various treatment options available when a permanent tooth is found to be developmentally absent during the developing dentition, including timely deciduous tooth extractions to encourage spontaneous space closure. When a permanent tooth is lost due to disease, e.g. caries or periodontal disease, the treatment options are often fewer and include either the maintenance of space for a prosthetic replacement or space closure as part of a more comprehensive orthodontic treatment plan involving usually fixed appliance. The treatment choice will depend on various factors including the presence of crowding and type of malocclusion, the skeletal pattern, overjet, overbite and buccal segment relationships. At this point it is worth perhaps considering the unscheduled loss of each permanent tooth in turn during the developing dentition:

Central incisor—the loss of a permanent central incisor due to trauma or caries can result in rapid space loss (Fig. 2.7). As a result, in the upper arch, it is usually worth fitting a space maintainer, not only from the point of view of the

immediate aesthetic improvement for the patient but also because orthodontic space closure and restoration of the lateral incisor to simulate the central incisor rarely gives a good long-term aesthetic result. In the lower arch the loss of a central incisor in the presence of crowding can be incorporated into an overall orthodontic treatment plan at a later date, and in most instances space should not be preserved whilst awaiting the development of the remaining occlusion. This is because it can lead to alveolar bone loss which can make later space closure more challenging.

Lateral incisor—when an upper lateral incisor is lost due to trauma or caries, once again the decision should be made whether to preserve the space or close the space. If the lateral incisor is lost prior to the eruption of the upper permanent canine, it is likely the canine will erupt into the upper lateral incisor position (Fig. 2.8). In which case the decision whether to close or reopen the space can only be made once the canine has erupted. This decision will depend on other features of the occlusion but principally the degree of crowding/spacing and also the shape and colour of the permanent canine as a possible substitute for the lateral incisor. Once again in the lower arch in the developing dentition, the loss of a lower permanent lateral incisor is usually accepted and the occlusion treated on its merits in the permanent dentition.

Permanent canine—the permanent canine is rarely lost due to trauma or caries but is more commonly absent due to an ectopic path of eruption. This will be dealt with in Chap. 7.

Premolars and molars—when a first premolar tooth is lost, usually due to caries, this can lead to spontaneous space closure and unwanted affects such as a shift in the centreline or buccal segment relationship. As a result when there is the enforced loss of a first premolar in a crowded arch, consideration should be given to the loss of the first premolar on the opposite side of the same arch, a balancing extraction. If the buccal segment relationship is to be preserved, then sometimes



Fig. 2.8 The absence of the upper lateral incisors in this crowded case has led to space loss with mesial eruption of the upper permanent canines

a compensating extraction is also required. However, such balancing and compensating extractions may not be necessary if a space maintainer is fitted to allow, for example, a crowded upper permanent canine to drop into the line of the arch. In the case of second premolars and first permanent molars in the developing dentition, it is not necessary to carry out a balancing extraction to preserve centrelines, but a compensating extraction may be required to once again preserve the anteroposterior buccal segment relationship. Other factors that will effect whether or not to compensate the loss of a first permanent molar include the presence of second and third permanent molars and whether or not the unopposed molar tooth is likely to overerupt. If all of the other molars are developing normally, then consideration should be given to the compensating extraction of the unopposed molar. Not only will this reduce the likelihood of trauma from biting on the gingivae, but it will also improve the likelihood the second molar will move into the correct anteroposterior position without hindrance from an overerupted first molar in the opposing arch.

Extra Teeth: Supernumerary and Supplemental Teeth

The extra teeth that most commonly disrupt the normal development of the dentition include the upper midline conical supernumerary tooth or mesiodens and the tuberculate supernumerary. The mesiodens can displace the path of eruption of the upper central incisors and lead to the development of a midline diastema, in which case it should be extracted. The mesiodens itself may or may not erupt and is obviously easy to remove if it does so (Fig. 2.9). The tuberculate supernumerary usually prevents the eruption of the central incisor tooth, as it lies directly over the cingulum of the tooth. Neither the supernumerary nor the central incisor will erupt. As a result the supernumerary tooth should be extracted, and the central incisor may then erupt



Fig. 2.9 This erupted mesiodens has displaced the upper central incisors from their normal path of eruption

spontaneously. If it doesn't it may then require exposure and bonding to bring it into the line of the arch [11]. Occasionally additional teeth of similar form to the normal series develop and may erupt into the arch, and these are known as supplemental teeth. Sometimes there is sufficient space to accommodate such a tooth within what would otherwise be a spaced dentition. However, in most cases it leads to localised crowding, in which case a decision has to be made as to the best tooth to remove, the supplemental or the one of the normal series. Sometimes it can be very difficult to tell which is the supplemental tooth, and the extraction decision will be dependent on factors such as the condition of the tooth/teeth, the position within the arch and which extraction will promote the best spontaneous improvement in the alignment of the remaining teeth.

Anomalies in Tooth Form (Microdont/Megadont)

Large or small teeth within the arch can lead to either crowding or spacing and where they are very obviously of a different size to the normal series. Extraction may be the best option. The decision whether or not to maintain or perhaps recreate some of the space will be dependent on the position of the tooth in question (see section “[Unscheduled Loss of Permanent Teeth](#)”) and other features of the malocclusion.

Anomalies in Tooth Position

The most common ectopically positioned teeth are the permanent upper central incisor and the maxillary permanent canine. The sequelae and management will be described in Chaps. 7 and 8.

References

1. Evensen JP, Øgaard B. Are malocclusions more prevalent and severe now? A comparative study of medieval skulls from Norway. *Am J Orthod Dentofacial Orthop.* 2007;131:710–6.
2. Helm S, Prydsö U. Prevalence of malocclusion in medieval and modern Danes contrasted. *Scand J Dent Res.* 1979;87:91–7.
3. Begg PR. Stone age man's dentition. *Am J Orthod.* 1954;40:298–312, 373–83, 462–75, 517–31.
4. Kaifu Y, Kasai K, Townsend GC, Richards LC. Tooth wear and the “design” of the human dentition: a perspective from evolutionary medicine. *Am J Phys Anthropol.* 2003;122:47–61.
5. Leighton BC. The early signs of malocclusion. *Trans Eur Orthod Soc.* 1969;45:353–65.
6. Kennedy DB. Treatment strategies for ankylosed primary molars. *Eur Arch Paediatr Dent.* 2009;10:201–10.
7. Byrd ED. Incidence of supernumerary and congenitally missing teeth. *J Dent Child.* 1943;10:84–6.
8. Hunstadbraten K. Hypodontia in the permanent dentition. *J Dent Child.* 1973;40:115–7.

9. Sletten DW, Smith BM, Southard KA, Casco JS, Southard TE. Retained deciduous mandibular molars in adults: a radiographic study of long-term changes. *Am J Orthod Dentofacial Orthop.* 2003;124:625–30.
10. Houston WJB, Stephens CD, Tulley WJ. *A textbook of orthodontics.* Oxford: Wright; 1993.
11. Yaqoob O, O’Neill J, Gregg T, Noar J, Cobourne MT, Morris D. Management of unerupted maxillary incisors. 2010. https://www.rcseng.ac.uk/fds/publications-clinical_guidelines/clinical_guidelines/documents/ManMaxIncisors2010.pdf. Accessed 08/09/15.

Gavin J. Mack

Abstract

Complications with the formation and eruption of first permanent molars can result in patients in the mixed dentition stage of development seeking orthodontic advice. The prognosis of first permanent molars can also be compromised by dental decay, and this can mean decisions have to be made with regard to the optimum timing of extractions in relation to the developing occlusion.

A structured assessment includes consideration of patient compliance with dental treatment, prognosis of the teeth, presence or absence of crowding and the underlying skeletal pattern.

Advice provided regarding the timing of the extraction of first permanent molars will reflect any future need for orthodontic treatment, and the impact first permanent molar extractions will have on the anchorage management during future orthodontic treatment.

Normal Development of the First Permanent Molar

Development of the First Permanent Molars

The first permanent molar (FPM) is rarely absent from the dentition, and when they do fail to develop, this is usually associated with severe hypodontia. Along with the upper central incisors, FPMs have been reported as the teeth least likely to be developmentally absent [1]. Morphological evidence of the formation of FPMs is usually present at 17 weeks in utero, and calcification of the crown commences at birth [2].

G.J. Mack

King's College Hospital Dental Institute, King's College Hospital NHS Foundation Trust,
London SE5 9RS, UK

e-mail: gavin.mack@nhs.net

Table 3.1 Average dimensions of FPMs

	Crown height (mm)	Length of root (mm)	Mesiodistal crown diameter (mm)	Labiolingual crown diameter (mm)
Maxillary FPM	7.5	12.5	10.5	11.0
Mandibular FPM	7.5	14.0	11.0	10.0

FPMs usually erupt into the mouth at the age of 6–7 years, and root formation is completed by the age of 9–10 years [3, 4]. The mandibular FPMs will typically erupt into the oral cavity before the maxillary FPMs. As these teeth erupt, they are guided into a position in the arch that is distal to and in contact with the distal aspect of the second primary molar.

Morphology of the FPM

The FPMs are usually the largest tooth in each jaw quadrant. The upper FPMs are rhomboid in outline when viewed occlusally and generally have four major cusps separated by an irregular H-shaped occlusal fissure. The upper FPMs typically have three diverging roots, with one relatively large palatal root and two smaller buccal roots. The lower FPMs are pentagonal in outline when viewed occlusally and tend to have five cusps separated into three buccal and two lingual cusps by a mesiodistal occlusal fissure. The lower FPMs have two roots, one mesial and one distal, and both are flattened mesiodistally and curved distally. Average dimensions of the FPMs are shown in Table 3.1.

Ideal Occlusion

FPMs erupt as root formation progresses until contact is made with an opposing tooth, and the opposing tooth will typically be the FPM in the opposing arch, but some contact with primary teeth is also possible. The relationship between upper and lower FPMs forms the basis of the occlusal classifications described by Edward Angle and Lawrence Andrews. In the developing dentition of patients with an underlying class I skeletal base, it is typical for the FPMs to occlude in a one-half unit class II molar relationship, with the FPMs having ‘flush terminal planes’.

As the second primary molars exfoliate, there is an increased potential for the mandibular FPM to migrate mesially, and this allows for a class I molar relationship to become established.

Complications Associated with FPMs

Hypomineralization

As the incidence of caries has reduced, the developmental anomaly of molar-incisor hypomineralisation (MIH) has been increasingly recognised as a clinical condition that affects young patients [5]. MIH is the hypomineralisation of systemic origin of one to

four FPMs and is frequently associated with affected incisors. The severity of the extent of the hypomineralisation can vary significantly between patients and between teeth in an individual mouth. The affected molar teeth may present with a small hypomineralised area, or more severely affected teeth may have complete breakdown of the occlusal surface of the tooth. The destruction of the crown of affected teeth can commence during the eruption process, and patients can initially present complaining of sensitivity that further impairs effective toothbrushing around the erupting teeth.

Management of FPMs presenting with a mild degree of hypomineralisation can range from the use of desensitising agents, such as the repeated application of fluoride varnish and the daily use of 0.4% stannous fluoride gel, to the restoration of the localised defects with adhesive restorations such as composite. FPMs that are more significantly compromised and presenting with a greater extent of enamel hypomineralisation can be either restored with occlusal coverage restorations, such as a cast adhesive coping or a preformed stainless steel crown. FPMs that are severely affected can be considered as unrestorable and require extraction.

Assessing the prognosis of FPMs affected by MIH can be difficult. The early presentation of affected teeth can allow for good-quality, relatively small restorations to be placed in young patients. However, if the quality of the enamel adjacent to the restoration margins is affected, the progressive breakdown of the enamel of the tooth can be difficult to prevent over time, meaning the prognosis of the tooth is inherently compromised. Similarly, the option of temporising FPMs affected by MIH, to allow for the teeth to be extracted at a later stage in development, can also be compromised by the affected teeth being symptomatic and difficult to restore (Fig. 3.1).



Fig. 3.1 A 9-year-old girl presented with MIH and FPMs of poor prognosis. The *upper left panel* shows the DPT taken at age 9, which resulted in a decision being made to extract all four FPMs. The remaining panels show the same patient as a young adult with composite restorations on the anterior teeth and favourable space closure in the buccal segments without the need for orthodontic treatment

Impaired Eruption of FPMs

As the upper FPMs erupt, they can impact into, and cause resorption of, the distal root of the upper second primary molar. The severity of the impaction influences the treatment options and management. Milder impactions can spontaneously improve, and this has been reported to occur in 66% of cases [6]. More significant impactions can be corrected with treatment using either a separating elastic or an appliance or can resolve following exfoliation of the primary molar. Severe impactions causing resorption to the root of the primary molar can involve the pulp chamber and may be painful for the patient. In such situations the second primary molars can be extracted, and a space maintainer can be used once the FPM has erupted (Fig. 3.2).

The incidence of first and second permanent molar teeth failing to erupt is relatively low and has been reported as 0.06% [7]. The clinical implications of FPMs failing to erupt can be significant and can include posterior open bites and altered tongue function. In patients presenting in the mixed dentition, the failure of FPMs to establish occlusal contact with the opposing arch can be considered as a diagnostic indicator that normal dental development is not occurring. Similarly, in an older patient, if the occlusal surfaces of the second molars are superior to the occlusal surfaces of the FPMs, this can be an indication that the FPM is affected by primary failure of eruption. The clinical implication being that the prescription of alternative teeth for orthodontic extraction should be avoided if at all possible as orthodontic treatment to reposition the FPM is likely to be unpredictable, and the FPM may not respond to the application of an orthodontic force (Fig. 3.3).

Caries

FPMs have a deep complex fissure patterns on the occlusal surface, and buccal pits are also frequently present in lower FPMs. FPMs erupt into the mouth at an age when children are not particularly dextrous or diligent enough to thoroughly clean the most posterior teeth in their mouths, and if dietary habits are not ideal, these teeth are prone to decay. The incidence of caries in the UK has been reported to have reduced over the last 30 years [8]. However, recent surveys have suggested that approximately one third of 15-year-olds in the UK still have carious lesions extending into dentine, and the majority of FPMs are extracted as a consequence of decay [9].

Fig. 3.2 The upper FPMs are impacted against the distal aspect of the primary second molars (*upper panel*). Following the extraction of these teeth (*middle panel*), a removable space maintainer was provided to prevent unwanted mesial drift of the upper FPMs (*lower panel*). The patient was instructed to wear this appliance on a nocturnal-only basis





Fig. 3.3 DPT of a patient in the late mixed dentition with delayed eruption of the lower right FPM. There is an asymmetry in the eruptive progress of both the lower FPMs, with the lower left FPM being in a more ideal position. There is also a disordered eruption sequence in the lower right quadrant, with the second permanent molar and second premolar being vertically positioned above the occlusal surface of the FPM. These are clinical indicators of the lower right FPM failing to erupt and could potentially be overlooked in a developing dentition

Extracting FPMs

The extraction of FPMs is rarely requested for patients who present with a caries-free, healthy dentition, irrespective of the presence of dental crowding or an underlying skeletal discrepancy. This is because the position of the FPMs within the dental arches means that extraction spaces are not ideally positioned to relieve crowding in the labial segments or assist with the correction of a positive or reverse overjet. In addition, the size of the residual extraction spaces can be excessive in relation to mild or moderate degrees of crowding, and closure of these spaces can extend orthodontic treatment times. The extraction of FPMs is typically undertaken when the tooth is severely broken down and beyond restoring in the short term. FPMs are also extracted in young patients when the teeth are less severely broken down but are considered to have a poor long-term prognosis in relation to unrestored premolars and the developing second and possibly third molars.

Irrespective of the age of a patient, or the presence of an underlying or developing malocclusion, a symptomatic, compromised FPM is considered unrestorable if the margins of the potential restoration are likely to be positioned below the level of the supporting periodontium and alveolar bone. Similarly, once carious enamel and dentine have been removed, the remaining tooth tissue should be sufficient to provide viable support for a definitive restoration.

In addition to the status of the FPM, patient factors can also influence extraction decisions. Patient co-operation should be taken into account and assessed. Complex restorative treatment, such as lengthy endodontic therapy, is not routinely prescribed for young patients, either under local anaesthesia or sedation in a dental clinic or under general anaesthesia in an operating theatre. The presenting oral health status of the individual patient should also be assessed. Multiple decayed but potentially restorable FPMs in the mouth of a dental phobic patient with uncontrolled caries and poor plaque control are likely to be extracted as a part of a pragmatic treatment plan to improve the patient's dental health. The converse of this would be the

complex restorative management of an individual FPM that is compromised due to localised hypoplasia in the mouth of a cooperative patient with an otherwise caries-free, unrestored dentition and excellent oral health.

Orthodontic Considerations When Extracting FPMs

When a patient presents in the mixed dentition with one or more FPMs that are considered to have a poor short- or long-term prognosis, the implication of elective extractions on the developing dentition and future occlusion would be considered. This is typically achieved by requesting an opinion from an orthodontic specialist.

Extra-Oral Assessment of the Underlying Pattern of Facial Growth

Skeletal growth occurs in all three dimensions, and in young patients, it is not possible to accurately predict exactly how growth will be expressed over future years. However, distinct growth patterns, particularly in the anteroposterior dimension, can be determined, and these can suggest if growth modification or orthodontic camouflage treatment will be required as part of the future orthodontic treatment of the individual. Young patients presenting with an underlying skeletal I base will typically maintain this skeletal relationship throughout their growth, but late mandibular growth may result in a class III tendency. Young patients presenting with an underlying skeletal II base are also likely to maintain the underlying skeletal II base throughout growth, with the potential for favourable mandibular growth to reduce the skeletal II discrepancy to a limited extent. Young patients presenting with an underlying skeletal III base will typically grow progressively and unfavourably so that the skeletal III relationship becomes more pronounced.

Intra-Oral Assessment of the Presence/Absence and Status of the Erupted Teeth

The clinical assessment of the erupted teeth is essential to ensure that the full complement of primary and permanent teeth is present for the patient's stage of dental development. Any teeth that are absent due to previous extractions, trauma or hypodontia should be noted. In addition, the caries status and prognosis of the erupted teeth, and particularly the erupted permanent teeth, should be assessed and considered as part of the consideration to extract FPMs.

Intra-Oral Assessment of Existing and Potential Crowding in the Upper and Lower Arches

As a general rule, mild spacing in the mixed dentition stage of dental development can suggest that as dental development progresses, the permanent teeth will be well

aligned within the arch. An excess of spacing around the erupted teeth in the mixed dentition is likely to result in spacing around the permanent teeth in the established adult mouth. The absence of spacing in the mixed dentition, or even the presence of crowding in the mixed dentition, suggests that as dental development progresses, the indicated dentoalveolar disproportion will become more pronounced, and the patient will develop progressively crowded arches. Careful consideration should be given to the effects of premature loss of the first and second primary molars. This can allow the FPMs to migrate mesially. On clinical examination, in the mixed dentition, the erupted teeth do not necessarily appear crowded, and spacing may be present, but the effect of the mesial migration of the FPMs can increase the potential for the premolar teeth to be crowded or even impacted at a later stage in development.

An Intra-Oral Assessment of the Occlusion

An occlusal assessment of a young child can be complicated by the young patient's tendency to posture their mandible anteriorly when asked to 'bite together'. Care should be taken to assess the occlusion from a position where the condyles are posteriorly, superiorly and symmetrically seated in the glenoid fossae. In patients with a class I skeletal pattern of growth, in the mixed dentition, the molar relationship between the upper and lower FPMs can be described as having 'flush terminal planes', and as dental development progresses, the future loss of the relatively large lower second primary molar allows for the mesial migration of the lower FPM, and this allows the class I molar relationship to become established.

In patients presenting in the mixed dentition with variation from FPMs having flush terminal planes, the molar relationship between the upper and lower FPMs can represent the underlying anteroposterior skeletal pattern of growth, with molar relationships tending towards either class II or class III as a consequence of the relative size and shape of the underlying maxilla and mandible.

When assessing the molar relationship between FPMs, consideration should again be given to the possibility of space being lost prematurely within the arch. FPMs can migrate mesially as a consequence of either the premature extraction of primary teeth or interproximal caries reducing the mesiodistal dimension of the primary teeth. If space has been prematurely lost in one of the arches but not the other, the anteroposterior relationship between the FPMs in occlusion can be affected in a way that does not indicate an underlying skeletal discrepancy.

In relation to the anterior occlusion, patients presenting with an overjet and overbite are unlikely to be affected by the premature loss of primary teeth, but other factors, such as the existence of an ongoing thumb-sucking habit, would be relevant. Patients presenting in the mixed dentition with class I incisors, with the overjet and overbite assessed as being within normal limits, are unlikely to grow unfavourably and develop an underlying skeletal discrepancy in the future. A possible exception to this would be when a family history of having a significant skeletal III discrepancy exists and there is a potential for late, pronounced mandibular growth to occur. For this reason, a discussion with the patient and their parents or guardian can be helpful to explore this aspect of the family history. In the absence of a digit-sucking

habit, patients in the mixed dentition presenting with a significantly increased overjet are likely to have an underlying skeletal II pattern of growth that would not self-correct with future growth. Similarly, patients with an edge-to-edge bite or reverse overjet will typically have an underlying skeletal III pattern of facial growth, and this is likely to become more pronounced with future growth and development.

Radiographic Assessment of the Presence/Absence of Unerupted Permanent Teeth

A DPT is indicated to review the presence and position of all the developing permanent teeth. The prevalence of hypodontia affecting the second premolar teeth and the second permanent molar teeth has been reported as 1–3% and <1%, respectively, and the absence of these teeth would influence the decision to extract FPMs [10]. The presence or absence of the third molar teeth is also of relevance. The incidence of hypodontia affecting the third molars is relatively high, with at least one third molar being absent in 20–30% of European populations [11–13]. Also, calcification of the third molars has been reported to commence between the age of 7 and 10 years [3]. If FPMs are to be extracted, ideally the third molars would be present and reasonably positioned. However, late developing third molars may not be identified on a DPT until a chronological age of 10 years, and this can mean patients presenting in the mixed dentitions have extraction decision made regarding their FPMs without the clinician knowing for certain whether all the third molars are present or absent.

Extraction Decisions in Patients with a Low Need for Orthodontic Treatment

Timing of Extractions

When a decision is made to extract FPMs in a patient with little or no need for future orthodontic treatment, a major consideration is how to avoid residual extraction spaces remaining in the buccal segments of the patient's dentition. FPM extraction spaces are primarily closed by mesial migration of the second permanent molars. In the upper arch, extraction spaces are more likely to close spontaneously, and the timing of extraction is considered to be less sensitive in allowing the space to close. This space closure occurs through the upper second permanent molars drifting mesially and rotating mesiopalatally. The spontaneous closure of extraction spaces in the lower arch is less predictable, and the lower second permanent molars have a tendency to tip mesially, with significant space remaining between the second permanent molars and the second premolars. There is a paucity of scientific evidence to allow for the accurate prediction of the effects of FPM extractions. The majority of the published literature in this area was presented in the 1960s and 1970s. Such studies tended to focus on the general development of the occlusion post-extractions and assessed changes in overjet, overbite and soft tissue profile, as opposed to changes in the buccal occlusion [14].

However, in the absence of more robust clinical investigations, these publications have influenced clinical practice. The ideal time to extract FPMs to encourage complete space closure through the mesial migration of the second permanent molars is generally considered to be represented by the onset of the calcification of the furcation of the roots of the unerupted lower second permanent molars [12]. This stage of dental development tends to correlate with a chronological age of 8–10 years. Other factors to consider when predicting the potential for the second permanent molar to spontaneously drift mesially are the presence of the developing third molar and the mesial angulation of the second permanent molar.

When spontaneous space closure is desirable for a patient requiring the extraction of FPMs, the other consideration for spontaneous space closure is the presence or absence of crowding in the buccal segments. Space closure is more likely to occur if some of the FPM extraction space is closed by the premolars drifting distally (Figs. 3.4 and 3.5). When lower FPMs are to be extracted, consideration should also be given to the position of the developing second premolar. Ideally, this tooth should be contained within the roots of the overlying second primary molar. If this is not the case, then the potential for the second premolar to drift distally and impact against the second permanent molar exists, and this results in significant spacing between the lower premolars and reduced mesial migration of the lower second permanent molars (Fig. 3.6).

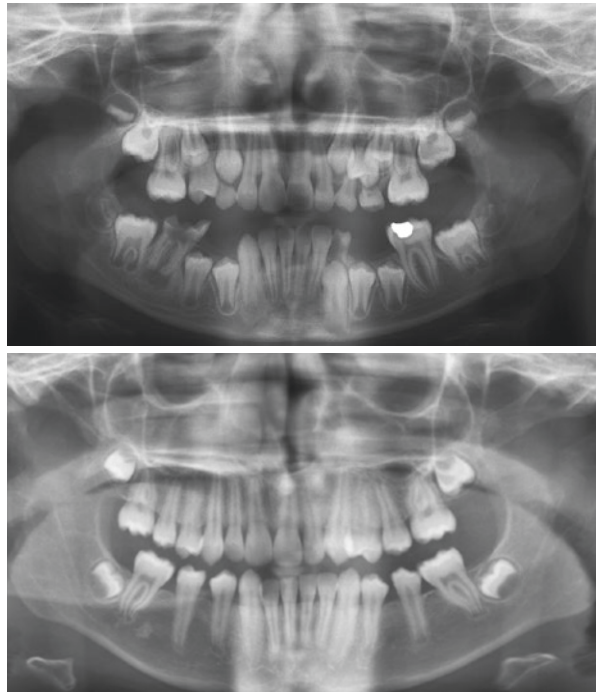


Fig. 3.4 Favourable space closure subsequent to the extraction of poor prognosis FPMs. The DPTs were taken when the patient was age 10 and 13 years, respectively. When the FPMs were extracted, there was a lack of space for the developing premolars to erupt

Fig. 3.5 Unfavourable space closure subsequent to the extraction of poor prognosis FPMs. Residual extraction spaces remain in the lower arch. The FPM extractions were undertaken after calcification of the bifurcation of the roots of the second permanent molars had commenced. Also of note is the premolar crowding in the upper arch, whereas the lower arch was relatively uncrowded prior to the extractions

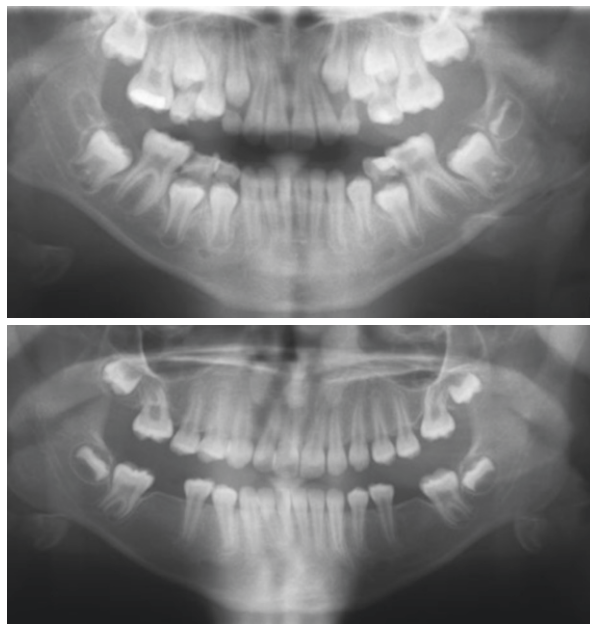


Fig. 3.6 DPT showing an impacted lower right second premolar following extraction of the lower right FPM



Compensating Extractions

Compensating extractions can be considered when FPMs of poor prognosis are going to be extracted. A compensating extraction is undertaken when the opposing tooth is extracted from the same side of the patient's mouth. This practice has been suggested to aid the process of spontaneous space closure.

An example would be the necessary extraction of an unrestorable lower right FPM being accompanied by the elective extraction of the opposing upper right FPM. The proposed benefit of the compensating extraction is that spontaneous space closure can more predictably occur on this side of the patient's mouth and both the second permanent molars can drift mesially, erupt and oppose each other in a reasonable occlusal relationship, essentially assuming the position of the FPMs.

Compensating extractions are requested due to the concern that single FPM extractions can lead to occlusal disturbances. This can theoretically occur if only the unrestorable lower right FPM is extracted and the upper right FPM is left unopposed. There is the potential for the unopposed upper right FPM to progressively super erupt into the extraction space, assuming a nonideal functional relationship with the lower arch and preventing the lower second permanent molar from drifting mesially and spontaneously closing the extraction space.

The current guidelines for the extraction of FPMs recommend, as a general rule, the compensating extraction of the upper FPM when the extraction of the lower FPM is required. This is to avoid the overeruption of the upper FPM. However, if an upper FPM has to be extracted and the opposing lower FPM has a reasonable prognosis, the compensating extraction of the lower FPM is not recommended as the lower FPM is considered to have a negligible propensity to overerupt [15].

Balancing Extractions

A balancing extraction occurs when the contralateral tooth in the same dental arch is electively extracted to accompany the necessary extraction of a tooth. Balancing extractions have been suggested as a useful way of maintaining arch symmetry. An example would be the necessary extraction of an unrestorable lower right FPM accompanied with the elective extraction of a sound lower left FPM. However, there is a lack of research presenting the benefits of balancing FPM extractions, and the extraction of an otherwise healthy FPM is a significant procedure to prescribe in the absence of clear clinical evidence. In addition, the possibility of correcting a resulting dental line asymmetry as part of a future course of fixed appliance orthodontic treatment exists if necessary (Fig. 3.7). For these reasons, the routine practice of extracting sound FPMs as balancing extractions to preserve dental centrelines has not been recommended [15].

FPM Extraction Decisions in Patients with a Need for Orthodontic Treatment

Class I Crowding

An assessment of the severity of the likely crowding for an individual patient is possible in the mixed dentition. This assessment can be made through a clinical examination and the review of a recently taken DPT. Both the extent and the location of the crowding are important to assess, as is the aetiology. It is generally accepted that the extraction of FPMs can provide spontaneous relief of crowding in the buccal segments. This presentation of crowding is typically due to the early loss of primary teeth allowing the FPMs to migrate mesially, reducing the available space for the premolars to erupt. However, the extraction of FPMs is unlikely to allow significant relief of the crowding that presents in the labial segments. This presentation of

Fig. 3.7 DPT of a patient with dentinogenesis imperfecta and a lower right FPM that has a poor prognosis (*upper panel*). Extraction of upper and lower right FPMs was prescribed, and these extractions were not balanced. The *middle panel* shows a DPT from the same patient taken 5 years later, and reasonable spontaneous space closure has occurred. A frontal occlusal clinical photograph of the same patient indicates the mild centreline shift that has occurred (*lower panel*). No orthodontic treatment was provided for this patient



crowding is primarily due to dentoalveolar disproportion and occurs when the alveolar bone volume is reduced relative to the size and shape of the developing teeth.

When it is evident that crowding is present in the developing dentition, and particularly if crowding is present in the labial segments, a key consideration relates to the future prescription of orthodontic extractions. If the presenting crowding appears to be relatively mild, to the extent that a non-extraction approach to treatment is likely to be undertaken in the future, then the extraction of FPMs of poor prognosis should be prescribed at the stage of dental development when spontaneous space closure can most predictably be achieved. This will allow the restorative problem of the poor prognosis FPMs to be addressed, as the FPMs will ultimately be replaced by the sound second permanent molars, and future orthodontic treatment to relieve the mild to moderate crowding is relatively predictable to deliver.

However, if the presenting crowding is more severe, to the extent that premolar extractions would likely be prescribed as part of the future orthodontic treatment, then the extraction of FPMs in the young patient should be avoided if at all possible. In patients with severe crowding, temporising the FPMs of poor prognosis can allow for progressive dental development to occur, and typically the crowding may also worsen during this time. When the second permanent molars have erupted, the orthodontic treatment plan can then include the extraction of the FPMs and the use of upper and lower fixed appliances. The resulting extraction spaces can be used to relieve the crowding in the labial segments, and the extraction of the FPMs obviates the need for the extraction of premolar teeth. When FPMs are extracted to relieve crowding, orthodontic treatment can be relatively lengthy as the extraction spaces are larger than those created when premolar teeth are extracted. Also, the extraction spaces are located more remotely to the crowded labial segments, and careful treatment mechanics are required to ensure that:

- Anchorage is controlled during treatment. The upper second permanent molars will migrate mesially more readily than the lowers. This means the extraction spaces in the upper arch will close by mesial movement of the upper second permanent molar, and consideration should be given to reinforcing upper arch anchorage throughout treatment. This can be through the use of a trans-palatal arch with or without the addition of a Nance acrylic button contacting the palatal mucosa (Fig. 3.8).
- The position of the second permanent molars is controlled during treatment. The lower second permanent molars will have a tendency to tip mesially and roll lingually as they are protracted during treatment. These unwanted tooth movements can be controlled by using rectangular stainless steel archwires during the space closure stage of treatment. In addition, the use of class II elastics from the lingual aspect of the lower second permanent molars can be considered, as can the introduction of additional progressive buccal crown torque, as required to the archwire (Fig. 3.9).
- The unnecessary advancement of teeth in the labial segments during the early stages of treatment. A 'round-tripping' effect can result in the crowded incisors being unduly proclined and advanced during the alignment stages of treatment. This can potentially lead to gingival recession around the incisors. To avoid this,



Fig. 3.8 Trans-palatal arch (*left panel*) and trans-palatal arch with a Nance acrylic button (*right panel*)

Fig. 3.9 Class II elastic traction being applied from the lingual aspect of the lower second permanent molars. This will support mesial movement of the lower second permanent molars and control the tendency for these teeth to tip lingually during the space closure stage of treatment



the anterior teeth can be selectively included in the fixed appliance, allowing arch form to be controlled to the extent that archwires of 0.017×0.025 in. stainless steel can be used to allow the controlled retraction of premolars and canines into the FPM extraction spaces. When the extraction spaces have been redistributed to the extent that spacing is present anteriorly, the remaining anterior teeth can then be included in the full fixed appliance (Fig. 3.10).

Class II Malocclusion

The decision regarding extractions in relation to the extent of the future crowding is similar for patients presenting with class I malocclusions and class II malocclusions, but certain other orthodontic considerations are required in the presence of an anteroposterior discrepancy. These primarily relate to the relative difficulty of



Fig. 3.10 Selective bonding and partial ligation of crowded lower incisors during the alignment stage of treatment can avoid the excessive proclination of the lower incisors. The teeth can be included in the fixed appliance after the premolars and canines have been partially retracted and the extraction space has been relocated

closing FPM spaces in the lower arch and the use of functional appliances during orthodontic treatment.

Class II Division 1 Incisor Relationship

When patients present with an increased overjet, the orthodontic management of FPM extraction cases can have specific considerations when using fixed appliances and these include:

- Are upper arch extractions required to retract proclined upper incisors? If this is the case, the timing of upper FPM extractions would ideally be delayed until the second permanent molars have erupted. This allows for anchorage reinforcement to be used in the upper arch to ensure the extraction spaces are at least partially closed by the retraction of the upper labial segments and the spontaneous mesial migration of the upper FPMs is controlled. Anchorage reinforcement techniques include the use of a trans-palatal arch possibly accompanied with a headgear appliance, a Nance palatal arch or the use of temporary anchorage screws.
- Can lower FPM extraction spaces be minimised to limit the possible retraction of the lower incisors? This consideration particularly applies in patients presenting with increased overjets and uncrowded lower arches. As it is relatively difficult to move lower second permanent molars mesially without reciprocally retracting the lower labial segment, and increasing the overjet, the extraction of lower FPMs at a time when spontaneous mesial movement of the lower second permanent molars is optimal is indicated. Should only partial space closure be achieved in the lower arch, this can allow for the use of class II elastic traction during the subsequent treatment with fixed appliances.

The use of functional appliances is typically indicated for growing patients presenting with increased overjets, increased overbites and a degree of mandibular

retrognathia. Patients suitable for treatment with functional appliances may also present with FPMs of poor prognosis. In such cases, the possible treatment approaches include:

- Deferring extractions until after the functional appliance stage of treatment. This can be useful in the short term as the FPMs are available to aid retention during the treatment with the removable functional appliances. This approach can also be useful in crowded cases in the longer term as the subsequent extraction of the poor prognosis FPMs at the start of the transition stage from functional appliances to fixed appliances can ensure the extraction spaces are available at the ideal time to start aligning the arches.
- Modifying the design of a Twin Block appliance. As the most commonly used functional appliance the Twin Block is recognised as being a versatile appliance. Modifying the design of a Twin Block appliance to allow for retention to be gained from premolars and/or retained primary molars can allow for the extraction of FPMs of poor prognosis to be undertaken at the ideal stage of dental development to allow for spontaneous space closure through the mesial migration of the second permanent molars whilst allowing class II correction to commence with a Twin Block before the second permanent molars erupt.
- Using an alternative functional appliance design. One-piece functional appliances such as a Frankel Functional Regulator appliance can be relatively more challenging for patients to tolerate than a two-piece appliance such as a Twin Block. However, for patients requiring the extraction of FPMs at a stage in dental development when primary molars are exfoliating and premolars are not fully erupted, the use of a one-piece tissue-borne appliance can allow for class II correction to commence at a time when there is a relative lack of posterior teeth available for tooth-borne retention.

Class II Division 2 Incisor Relationship

The complication of poor prognosis FPMs in patients presenting with class II division 2 incisor relationships primarily impacts on the treatment mechanics required to control the overbite and relieve the crowding. In patients with a severe class II division 2 incisor relationship, the overbite is typically increased, and at least some of the teeth in both the upper and lower labial segments are crowded and retroclined. The typical approach to treatment will involve using fixed appliances to level the arches and normalise the inclination of the incisors. These treatment changes improve the inter-incisal angle and reduce the overbite. If a patient presents with a class II division 2 incisor relationship and the arches are either mildly or moderately crowded, the challenge of closing FPM extraction spaces can significantly complicate treatment mechanics and compromise the end result of treatment. This is because the mesial protraction of the second permanent molars, particularly the lower second permanent molars, is technically difficult to deliver without retracting the lower labial segment. This is relevant as any retraction of the lower labial segment will potentially retract the incisors and deepen the overbite. Therefore, the early extraction of FPMs is indicated to allow for spontaneous extraction space closure when possible.

If in addition to a significant class II division 2 incisor relationship there is also severe crowding, the extraction of the FPMs should be delayed until the second permanent molars erupt, allowing the extraction spaces to be available for the relief of the crowding. Typically, it is challenging to close the extraction spaces in patients presenting with increased overbites and reduced vertical facial proportions, so often anchorage reinforcement is not required, and the use of class II elastic traction can aid closing the space in the lower arch whilst also contributing to overbite reduction.

Class III Malocclusion

When assessing patients in the mixed dentition, an underlying class III skeletal pattern of growth complicates the treatment planning process as the extent of future facial growth, which typically enhances the class III tendency, is often unknown. The uncertainty surrounding the severity of the skeleton III pattern is focused on the possibility of the class III malocclusion being satisfactorily corrected by orthodontic camouflage treatment alone as opposed to orthognathic surgery. The key issue in this treatment planning decision relates to the management of the lower labial segment.

In patients with mild to moderate skeletal III patterns, if orthodontic camouflage treatment is anticipated, the scope to retract the lower incisors further may well be desirable and required to correct a reverse overjet and establish a satisfactory anterior occlusion. It is therefore advisable to avoid the early loss of poor prognosis FPMs if possible. This means that the temporised FPMs can be incorporated into a future orthodontic extraction pattern and the scope to close the lower FPM extraction spaces through the partial protraction of the second permanent molars, and the retraction of the lower anterior teeth can aid the class III correction. In the upper arch, the upper second permanent molars have a tendency to readily migrate mesially, so in the absence of any crowding, the upper arch extraction spaces will close predictably without appreciably retracting the upper labial segment.

A different approach to patient management is required if future orthognathic treatment is anticipated. In this situation, decompensation of the lower incisors is often required to ensure orthognathic surgery can correct the true extent of the underlying skeletal discrepancy. The implication of this is that in uncrowded arches, poor prognosis lower FPMs should be extracted at the stage of dental development to allow as much spontaneous space closure as possible. This is preferable to delaying the extractions until the pre-surgical orthodontic treatment commences as it can be challenging to decompensate retroclined lower incisors at the same time as closing lower FPM extraction spaces in the absence of crowding.

In the upper arch, the management of poor prognosis FPMs can be considered in relation to the extent of any possible crowding and the need for upper incisor decompensation. In uncrowded, well-aligned arches, the extraction of the upper FPMs will allow for predictable mesial migration of the upper second permanent molars. In crowded upper arches, or arches where the decompensation of proclined upper incisors is required, the possibility of delaying the extraction of the FPMs to allow for the upper second permanent molars to erupt should be considered. This is because

allowing for the upper second permanent molars to erupt before requesting the extraction of the upper FPMs will allow for upper arch anchorage reinforcement to be used, and the need to additionally extract upper premolars as part of the pre-surgical management can be avoided. Often patients presenting with crowded upper arches and severe skeletal III discrepancies do not want to delay improving the appearance of the upper incisors until the pre-surgical orthodontic treatment commences in their late teenage years. It can therefore be timely and beneficial to the patient to align the upper arch only in the early teenage years. This relatively simple treatment can allow for the incorporation of the extraction of poor prognosis FPMs. This means the restorative burden of temporising and stabilising the poor prognosis FPMs is removed, whilst growth is ongoing and the patient considers whether they want to embark on the orthognathic process in the future.

Summary When assessing young patients presenting with FPMs of poor prognosis, a number of considerations have to be taken into account. These include the prognosis of the individual teeth, the developing occlusion and the likely mechanics of possible future orthodontic treatment.

In summary the key principles that can be applied to individual cases include:

- In young patients, FPMs may have to be extracted even if this complicates future orthodontic treatment. This can be due to the individual teeth being unrestorable or the patient not being compliant with the restorative treatment required to stabilise the teeth.
- In young patients with developing, significant malocclusions that are likely to require permanent tooth extractions as part of their future orthodontic treatment, delaying the extraction of the FPMs until the second permanent molars erupt can complicate the anchorage management of the future orthodontic treatment but will reduce the need for the additional extraction of permanent teeth.
- After the extraction of an upper FPM, space closure through the mesial migration of the upper second permanent molar is relatively predictable. If the upper FPM extraction space is required to correct a malocclusion, then anchorage reinforcement is required.
- After the extraction of a lower FPM, space closure through the mesial migration of the lower SPM is less predictable. If the relief of anterior crowding or the retraction of the lower labial segment is not required during future orthodontic treatment, it is advisable to extract the lower FPM at an optimum stage of dental development to allow for spontaneous closure of the extraction space.
- The optimum stage of dental development to allow spontaneous space closure to occur has been associated with the onset of the calcification of the bifurcation of the lower second permanent molar. However, this can be considered as a guide, and other factors such as the angulation of the lower Second permanent molar and the presence of the developing third molar are also useful indicators of the likelihood of spontaneous space closure occurring.
- The compensating extraction of a sound upper FPM can allow for more predictable space closure in the lower arch when a poor prognosis lower FPM requires

extraction and spontaneous space closure is desirable. The compensating extraction of an unrestored lower FPM is not recommended to facilitate space closure if an upper FPM is extracted.

- The balancing extraction of unrestored FPMs to maintain arch symmetry is not routinely recommended.
- With appropriate orthodontic advice provided in the mixed dentition and contemporary orthodontic treatment with fixed appliances and planned anchorage reinforcement in the permanent dentition, the need to extract permanent teeth in addition to the extraction of poor prognosis FPMs can be avoided in the majority of clinical cases.

References

1. 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.
2. Ooë T. Human tooth and dental arch development. St. Louis: Ishiyaku EuroAmerica; 1981.
3. Berkovitz BKB, Holland GR, Moxham BJ. Oral anatomy, embryology and histology. New York: Mosby; 2002.
4. Nanci A. Ten Cate's Oral Histology Development, structure and Functions, 8/e. Elsevier india; 2012. Ten Cate AR. Oral anatomy: development, structure and function; 2014.
5. Crombie F, Manton D, Kilpatrick N. Aetiology of molar–incisor hypomineralization: a critical review. *Int J Paediatr Dent.* 2009;19(2):73–83.
6. Young DH. Ectopic eruption of the first permanent molar. *J Dent Child.* 1957;24:153–62.
7. Prece JW. The incidence of unerupted permanent teeth and related clinical cases. *Oral Surg.* 1985;29:420–5.
8. Pitts NB, Chestnutt IG, Evans D, White D, Chadwick B, Steele JG. 1 Verifiable CPD Paper: the dental caries experience of children in the United Kingdom, 2003. *Br Dent J.* 2006;200(6):313–20.
9. Albadri S, Zaitoun H, McDonnell ST, Davidson LE. Extraction of first permanent molar teeth: results from three dental hospitals. *Br Dent J.* 2007;203(7):E14.
10. 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.
11. Grahnen H. Hypodontia in the permanent dentition: a clinical and genetical investigation, vol. 3. Lund: Gleerup; 1956.
12. Haavikko K. Hypodontia of permanent teeth. An orthopantomographic study. *Suom Hammaslaak Toim.* 1970;67(4):219–25.
13. Neal JJ, Bowden DE. The diagnostic value of panoramic radiographs in children aged nine to ten years. *Br J Orthod.* 1988;15(3):193–7.
14. Thunold K. Early loss of the first molars 25 years after. In: Report of the congress. European Orthodontic Society; 1970. p. 349.
15. Cobourne MT, Williams A, Harrison M. National clinical guidelines for the extraction of first permanent molars in children. *Br Dent J.* 2014;217:643–8.

Helen Tippett and Martyn T. Cobourne

Abstract

A supernumerary tooth is defined as one that has developed in addition to the normal complement of teeth within the dentition. Supernumerary teeth are most commonly seen in the permanent dentition and are rare in the deciduous dentition. Males are affected twice as often as females, with the most frequently affected site being the anterior maxilla (by a reported ratio ranging between 5 and 10:1 compared to the mandible). Supernumerary teeth can be found in almost any region of the dental arch. The anomaly is generally classified according to its morphology and site but, like tooth agenesis, can occur as an isolated trait or coexist with a syndrome. Management is dependent on the type, location, number and complications arising from their presence.

Introduction

A supernumerary tooth is defined as one that has developed in addition to the normal complement of teeth within the dentition. These teeth usually occur in isolation but can more rarely be associated with a number of developmental disorders. They can cause local problems during development of the dentition and may require removal in these circumstances.

H. Tippett (✉) • M.T. Cobourne
Orthodontic Department, King's College Dental Institute, London, UK

Department of Orthodontics, Craniofacial Development and Stem Cell Biology,
King's College London Dental Institute, London SE1 9RT, UK
e-mail: helen.tippett@nhs.net

Table 4.1 Disorders associated with supernumerary teeth

Disorder	OMIM
Cleft lip and/or palate	
Cleidocranial dysplasia	119600
Familial adenomatous polyposis	175100
Opitz GBBB syndrome	300000
Rubinstein-Taybi syndrome (RSTS1)	180849
Rubinstein-Taybi syndrome (RSTS2)	613684
Robinow syndrome (dominant form)	180700
Kreiborg-Pakistani syndrome	614188
Insulin-resistant diabetes mellitus with acanthosis nigricans	610549
Ehlers-Danlos syndrome (classic)	130000
Ehlers-Danlos syndrome (hypermobility)	130020
Ellis-van Creveld	225500
Incontinentia pigmenti	308300
Fabry disease	301500
Trichorhinophalangeal syndrome	190350
Nance-Horan syndrome	302350
Neurofibromatosis type 1	162200
Orofaciodigital syndrome	311200

Prevalence

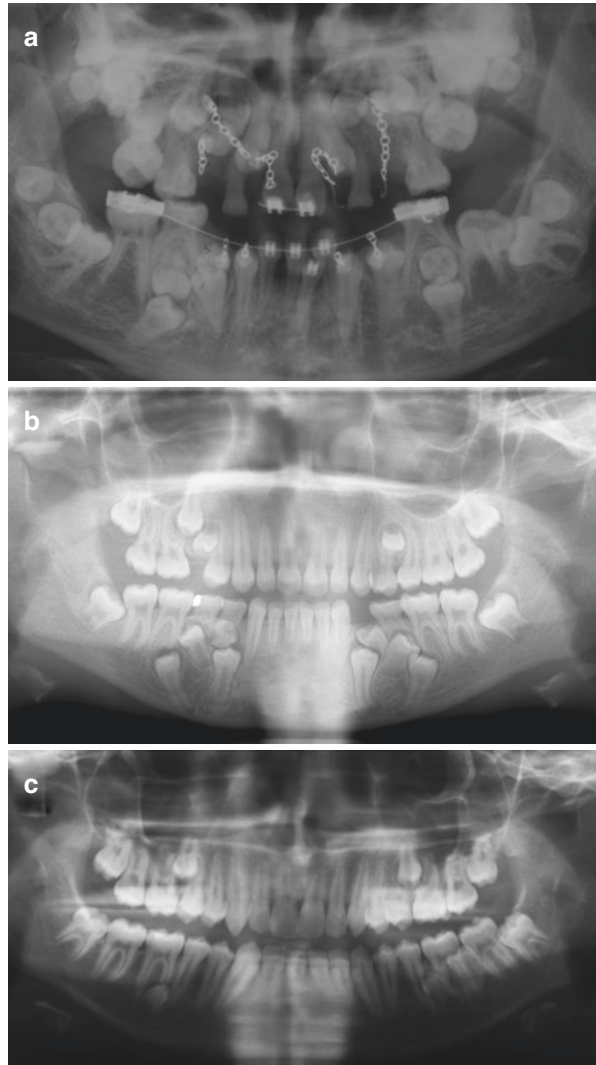
The prevalence of supernumerary teeth in the deciduous dentition is reported as 0.3–0.8%, and in the permanent dentition as 1.2–3.5% ([1, 2]; and reviewed by [3]). This wide variation in prevalence is most likely a result of the methodology utilised and a reflection of the age ranges and populations studied. Those individuals who present with a supernumerary in the deciduous dentition demonstrate a higher prevalence of supernumerary development in the permanent dentition. However, it has also been suggested that this is not necessarily a significant risk factor [4].

A wealth of studies have suggested that males are twice as likely as females to present with a supernumerary in the adult dentition, although this is dependent on the population studied and there is considerable variability in these ratios (1.3:1 to 2.64:1) [2, 5, 6]. This sexual dimorphism is not displayed in the primary dentition.

Supernumerary teeth can occur singly, in multiples, unilaterally or bilaterally and in either the maxilla or mandible. Numerous studies have found that the majority of patients present with one or two supernumerary teeth [5]. Multiple supernumerary teeth are more commonly seen in patients with an associated syndrome or systemic condition (Table 4.1). The syndromes most frequently associated with supernumerary teeth are cleidocranial dysplasia (OMIM 119600) and familial adenomatous polyposis (OMIM 175100).

Cleidocranial dysplasia (CCD) is a rare congenital defect with an autosomal dominant inheritance. Individuals are characterised by persistent open cranial

Fig. 4.1 (a) DPT of patient with cleidocranial dysplasia undergoing orthodontic traction with fixed appliances. There are multiple unerupted supernumerary teeth in the premolar and molar regions. (b) DPT of patient with neurofibromatosis type 2 associated with multiple unerupted supernumerary teeth in the premolar regions. (c) DPT of patient with five late-forming supernumerary teeth. The patient had no underlying medical history



sutures, hypoplasia or aplasia of the clavicles and dental abnormalities (Fig. 4.1), including multiple unerupted supernumerary teeth. Loss-of-function mutations of the *RUNX2* gene, involving transcription factor *CBFA1*, are the cause of CCD [7, 8]. The affected gene is located on chromosome 6p21 [9].

Familial adenomatous polyposis (FAP) is an autosomal dominant condition with variable expression. FAP is characterised by the development of innumerable adenomatous polyps of the colon and rectum, which may become malignant. The responsible gene, adenomatous polyposis coli (*APC*), is located on the long arm of chromosome 5 [10]. Gardner's syndrome is an accepted variant of FAP and was originally distinguished from FAP by the presence of extra-colonic findings,

particularly oral and maxillofacial abnormalities. It is reported that up to 30% of patients can present with supernumerary teeth, which is far higher than in the normal population [11].

Although the presence of supernumerary teeth has been linked to many disorders (Table 4.1), a recent review of the literature argued that conditions where just one or two patients demonstrated the presence of supernumerary teeth could be coincidental rather than a true association [12].

The prevalence of supernumeraries in patients with cleft lip and palate has also been reported at between 1.9 and 10% [13] and is thought to be a consequence of disruption of the dental lamina during cleft formation. Supernumerary teeth are the second-most common anomaly found in the cleft area [14, 15].

Although rare, cases have been observed where multiple supernumerary teeth have been located in patients in the absence of any underlying disorder [16, 17] (Fig. 4.1).

Aetiology

The aetiology of supernumerary teeth has yet to be fully established, although various hypotheses have been proposed. Hyperactivity of the dental lamina has been suggested [6], with this hypothesis assuming that the lingual extension of an additional tooth bud leads to development of a tooth with normal morphology and the supernumerary tooth arises from proliferation of epithelial remnants of the dental lamina. Others have considered a genetic predisposition or even a multifactorial aetiology [18]. Support for genetic involvement has been derived from numerous familial studies; offspring of parents with supernumerary teeth have been shown to have an increased risk of their development by a factor of six [19]. Furthermore, a sex-linked transmission, or variability in penetrance, could explain why supernumerary teeth are more commonly found in males. Despite the strong hereditary tendency, the pattern of inheritance does not entirely fit the Mendelian model. Moreover, mouse models have also demonstrated a genetic basis for supernumerary tooth formation, with both loss and gain-of-function mutant mice demonstrating a capacity to generate supernumerary teeth [20].

Supernumerary teeth are an aberration of tooth development, and as such, a greater understanding of the molecular mechanisms underlying this complex process has contributed to current thinking. Although a genetic component is the aetiological factor most strongly linked with supernumerary teeth, it does not act in isolation and different molecular signalling pathways, together with transcription factors, also play an important role [21–23].

Classification

Supernumerary teeth are generally classified according to their location (Fig. 4.2) or morphology (Table 4.2).

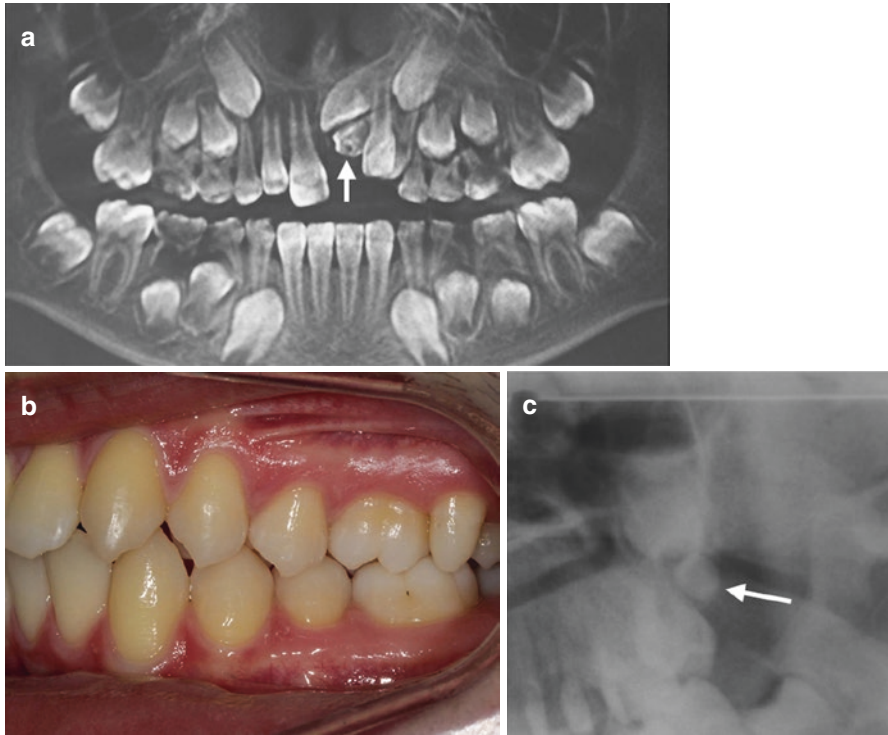


Fig. 4.2 (a) DPT showing a midline supernumerary tooth (tuberculate) (*arrowed*) preventing eruption of the UL1 (see also Fig. 4.4b). (b) Erupted paramolar between the upper left first and second molar. (c) Section of a bimolar radiograph showing a distomolar in the upper left buccal segment (*arrowed*)

Location

- Mesiodens: occurs in the midline
- Paramolar: buccal or lingual to a maxillary molar or located in the space buccal to the second and third molar
- Distomolar: lies distal to the third molar

In individuals who present with just one or two supernumerary teeth, they are most frequently found in the anterior region of the maxilla [2, 5, 6, 16], followed by the mandibular premolar area. It is rare for a supernumerary tooth to be found in the mandibular incisor region and, rarer still, for it to be erupted [5, 6, 24, 25] (Fig. 4.3). In non-syndromic patients with multiple supernumeraries, these are more frequently located in the mandibular premolar area.

Table 4.2 Morphological classification of supernumerary teeth in the permanent dentition

	Typical morphology	Frequency (%)	Number	Site	Eruption
Conical	Small	74.8 ^a	Usually isolated	Anterior maxilla	May erupt palatally but rarely labially
	Peg-shaped	83.5 ^b		Frequently between central incisors	
	Normal root development			May be inverted	
Tuberculate	Multicuspid	11.9 ^a	Frequently occur in pairs	Palatal to maxillary incisors	Rarely erupt but commonly prevents eruption of maxillary incisors
	Aberrant or absent root formation				
Supplemental	Similar to normal tooth in series	6.9 ^a	Usually isolated	End of series	Frequently erupts
				Most commonly maxillary lateral incisor	
Odontomes	Small group of malformations which contain calcified dental tissues of epithelial and mesenchymal origin	6.4 ^a	Can give appearance of multiple supernumeraries within one structure	Complex type more frequently located in posterior jaw	
	Compound—discrete structures resembling fully developed teeth				
	Complex—poor level of organisation and little resemblance to normal tooth				

^aRajab and Hamdan [6]^bLiu et al. [5]



Fig. 4.3 Rare finding of an erupted conical supernumerary preventing eruption of a LR2. (a) Supernumerary situated between the LR1 and LR3. (b) Corresponding long-cone periapical radiograph

Morphology

Supernumerary teeth in the deciduous dentition are frequently of either normal morphology or conical. In the adult dentition, there is more variability and four morphological classes have been described: conical, tuberculate, supplemental and odontomes (Fig. 4.4). Each group is characterised by particular features (Table 4.2). The conical type is the most frequently observed class [5, 6, 16], followed by tuberculate and supplemental. The frequency of odontomes is less clear, as this form is not universally accepted as a supernumerary and some studies have not included them in their sample [16]. As indicated in Table 4.2, morphology is also related to the likelihood of a supernumerary to erupt. Studies have shown that the supplemental type is most likely to erupt followed by conical and tuberculate.

Clinical Features

The presence of a supernumerary tooth may have no effect on the developing dentition and can be a chance finding when a patient first presents for orthodontic treatment (Fig. 4.5). However, in other instances, a variety of effects may be seen including displacement, crowding (Fig. 4.6), dilaceration, root resorption, cystic change and nasal cavity eruption. However, pathology is rare and the complication seen most frequently is delayed eruption of an incisor (Fig. 4.7).

Management

In the first instance, a careful clinical and radiographic examination is required to detect and localise supernumerary teeth. Treatment is dependent on the type and position of the supernumerary and its effect on adjacent teeth.

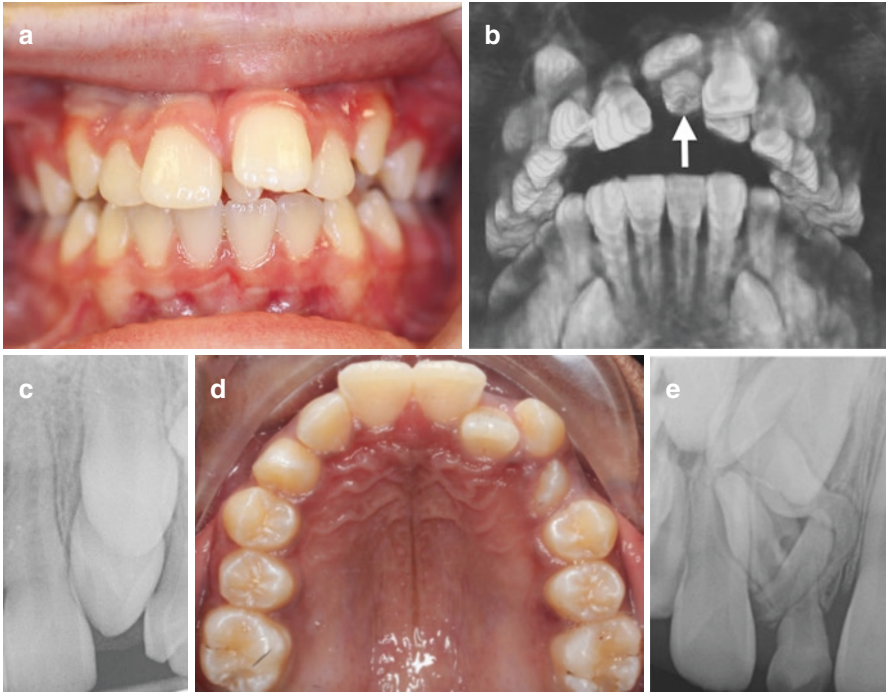


Fig. 4.4 In the permanent dentition, four morphological classes of supernumerary have been described. (a) Conical midline supernumerary tooth, which has erupted and displaced the UL1. (b) Cone beam CT image of an unerupted tuberculate supernumerary (see also Fig. 4.2a). (c) Long-cone periapical radiograph of a supplemental maxillary canine and a maxillary canine in the upper left quadrant. (d) Occlusal view of the patient from (c), the supplemental canine is erupting distal to the normal UL3, which is buccally placed. (e) Upper standard occlusal radiograph demonstrating a large odontome in the anterior maxilla composed of four denticles fused into two pairs preventing eruption of the UR1

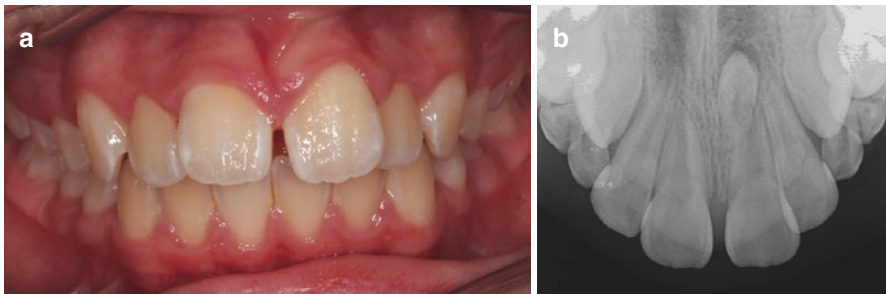


Fig. 4.5 (a) There is no obvious reason to suspect the presence of a supernumerary tooth on clinical examination of this dentition. (b) An earlier upper standard occlusal radiograph demonstrates the presence of an unerupted midline conical supernumerary

Fig. 4.6 (a) DPT showing a supplemental supernumerary tooth adjacent to the UR1 (*arrowed*), which has caused displacement of the UR2 and crowding in the region of the UR3. (b) The supplemental tooth was extracted to allow for mesial movement of the UR2 with an upper removable appliance and space creation to facilitate eruption of the UR3. Note that the UR5 and UR5 are both absent

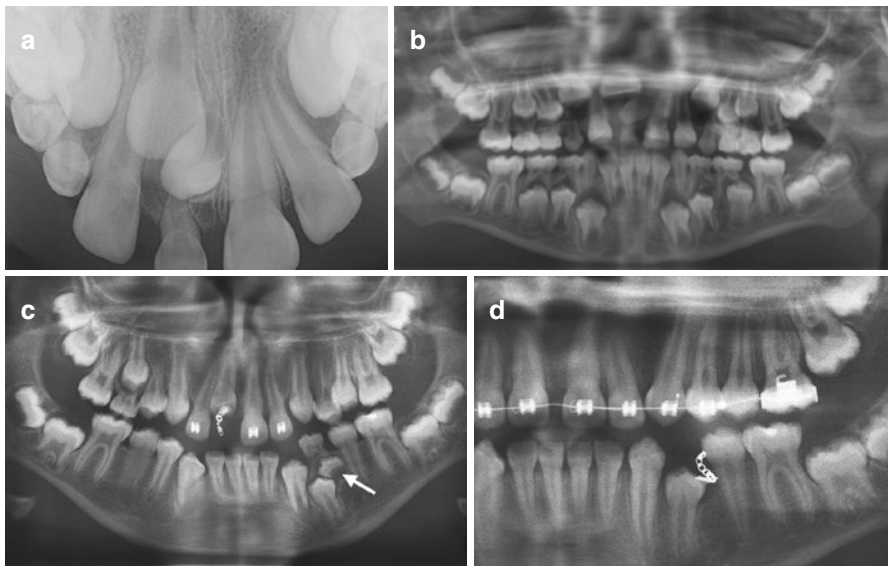
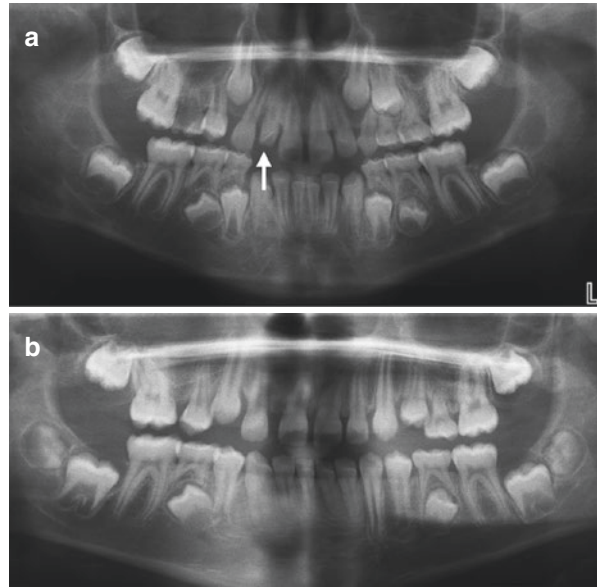


Fig. 4.7 (a) Upper standard occlusal radiograph and (b) DPT showing an unerupted midline conical supernumerary in the anterior maxilla with delayed exfoliation of the URA and failure of eruption of the UR1. The URA and supernumerary were removed and the UR1 exposed and bonded. As the UR1 was aligned, it was noted that there was delayed eruption of the LL4 and a further DPT was taken (c) which clearly demonstrated a late-forming supernumerary premolar tooth superior to the LL4 (*arrowed*) and preventing its eruption. Closer inspection of the earlier DPT (b) shows the first sign of this supernumerary is visible between the roots of the LLD. Surgical removal of the premolar supernumerary was undertaken and the LL4 exposed and bonded. (d) Sectional DPT shows eruption of the LL4 without active traction to the tooth



Fig. 4.8 Cone beam CT showing a conical supernumerary (*arrowed*) situated palatal to the UL1 that has not interfered with eruption of the maxillary incisors (see also Fig. 4.5a). Planned retraction of the upper labial segment was required to reduce an increased overjet. The CBCT revealed that the proximity of the supernumerary to the root of the UL1 would interfere with orthodontic tooth movement and it was surgically removed

Routine radiographs such as a dental panoramic radiograph, upper standard occlusal and long-cone periapical can be used in combination to localise a supernumerary tooth using the parallax technique (horizontal or vertical tube shift) and may give sufficient information to allow safe removal. However, if the proximity to developing roots is a concern, assessment of resorption is required or indeed a decision is needed as to whether orthodontic tooth movement can go ahead without removal of a supernumerary, then cone beam computed tomography may be indicated (Fig. 4.8). This additional imaging technique has been shown to enhance the ability to carefully localise a supernumerary [5]. Indeed, a system has been proposed to classify the complex location of supernumeraries in the maxillary anterior arch based on evaluation with cone beam computed tomography [5].

In some instances, no intervention may be indicated, particularly when the position of the supernumerary is unlikely to interfere with potential orthodontic treatment or if its removal poses a significant risk to the roots of adjacent teeth [26] (Fig. 4.9). Many authors suggest that the early diagnosis and treatment of supernumerary teeth can limit complications, but early removal is not without controversy. If the supernumerary lies adjacent to the maxillary incisors, delaying removal until root development is complete has been advised [6]. This is because the risk of damage to developing roots is viewed as too great to warrant early removal. This rationale may be similarly applied to supernumerary teeth in the premolar region.

In situations where further dental development is awaited, or a decision made to leave a supernumerary in situ, the patient may be reviewed at an appropriate interval and further radiographs taken to reassess (Fig. 4.9). Tyrologou et al. [27] reviewed 43 patients with mesiodens and no complications were reported. However, if the

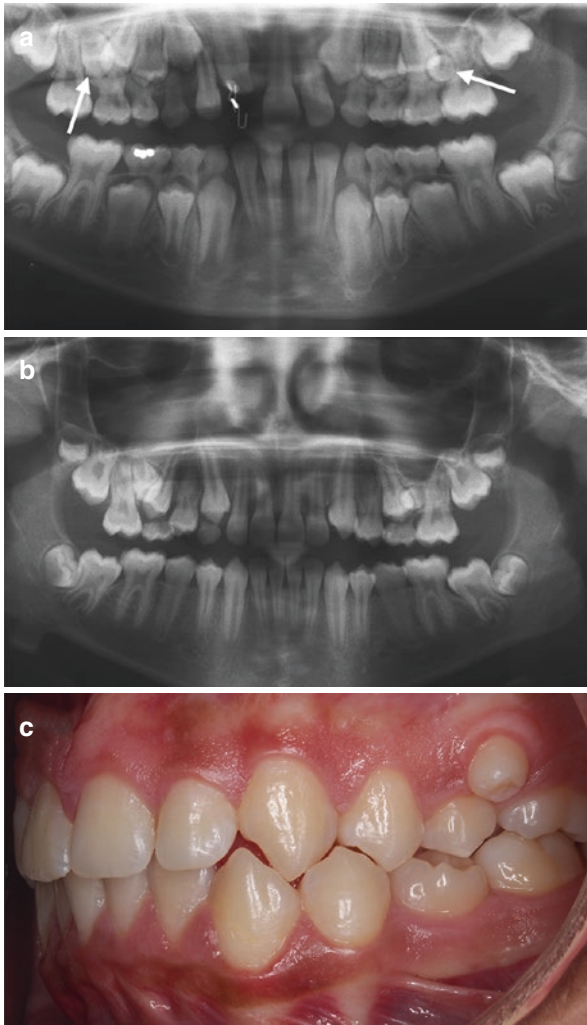


Fig. 4.9 A patient who initially presented at the age of 9 years with retained URAB, unerupted UR1 and two unerupted supernumeraries in the midline. These teeth were removed and the UR1 bonded with a gold chain. (a) DPT taken at review 8 months post-surgery, where a further two supernumeraries were identified developing in both upper quadrants (*arrowed*). These teeth did not interfere with dental development in these regions (b) and the patient underwent simple alignment of the upper arch. (c) At the age of 17, the patient presented for review and arrangements were made for extraction of the supernumeraries. In the upper right quadrant, the supernumerary had erupted palatally between the UR5 and UR6. In the upper left quadrant, it had erupted buccally between the UR5 and UR6 (shown here)

supernumerary has interfered with normal dental development or will impede orthodontic tooth movement or there is evidence of pathology, then its removal should be planned. On rare occasions, removal of a supernumerary tooth may be required if its presence would otherwise compromise alveolar bone grafting in patients with cleft lip and palate. Similarly, removal is indicated if a supernumerary is positioned at a potential implant site. In these complex cases, surgical removal of the supernumerary can compromise the amount of bone available and bone grafting may also be required prior to implant placement.

Patients presenting with supernumerary teeth in the anterior maxilla associated with failure of permanent maxillary incisor eruption and who require a general anaesthetic for removal should, in general, also have the unerupted incisor exposed and bonded with an orthodontic attachment to facilitate guided traction. The time frame within which the tooth will erupt is influenced by the degree of displacement and the space available within the arch (for more details, refer to Chap. 7).

Where the supernumerary tooth is of the supplemental type, its extraction is often indicated due to crowding or displacement of adjacent teeth and the difficulties associated with tooth-size discrepancies between the arches if it is retained. The choice of tooth for removal is usually based on the crown and root morphology and the degree of displacement.

Supernumerary teeth may also develop late [28, 29], and it has been reported in the literature that those patients with a midline supernumerary have an increased risk of developing supplemental premolars [30]. Figure 4.7 shows radiographs of a patient who initially presented with an unerupted central incisor. Following surgical removal of the supernumerary and exposure and bonding of the incisor, the patient underwent a course of sectional fixed appliance treatment to align the incisor. However, during this phase of treatment, they were regularly monitored and it was noted that there was delayed eruption of the lower left first premolar. A radiograph revealed a further late-developing supernumerary, which was subsequently removed, and the first premolar exposed and bonded to facilitate eruption. This illustrates the need to follow up patients who have undergone removal of supernumerary teeth to determine if further ones develop.

References

1. Brook AH. Dental anomalies of number, form and size: their prevalence in British schoolchildren. *J Int Assoc Dent Child*. 1974;5:37–53.
2. Mahabob MN, Anbuselvan GJ, Kumar BS, Raja S, Kothari S. Prevalence rate of supernumerary teeth among non-syndromic South Indian population: an analysis. *J Pharm Bioallied Sci*. 2012;4(Suppl S2):373–5.
3. Ata-Ali F, Ata-Ali J, Penarrocha-Oltra D, Penarrocha-Diago M. Prevalence, etiology, diagnosis, treatment and complications of supernumerary teeth. *J Clin Exp Dent*. 2014;6(4):e414–8.
4. Marinelli A, Giuntini V, Franchi L, Tollaro I, Baccetti T, Defraia E. Dental anomalies in the primary dentition and their repetition in the permanent dentition: a diagnostic performance study. *Odontology*. 2012;100:22–7.
5. Liu DG, Zhang WL, Zhang ZY, Wu YT, Ma XC. Three-dimensional evaluations of supernumerary teeth using cone-beam computed tomography for 487 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2007;103:403–11.

6. Rajab LD, Hamdan MA. Supernumerary teeth: review of the literature and a survey of 152 cases. *Int J Paediatr Dent.* 2002;12:244–54.
7. Lee B, Thirunavukkarasu K, Zhou L, et al. Missense mutations abolishing DNA binding of the osteoblast-specific transcription factor OSF2/CBFA1 in cleidocranial dysplasia. *Nat Genet.* 1997;16:307–10.
8. Mundlos S, Otto F, Mundlos C, et al. Mutations involving the transcription factor CBFA1 cause cleidocranial dysplasia. *Cell.* 1997;89:773–9.
9. Mundlos S, Mulliken JB, Abramson DL, et al. Genetic mapping of cleidocranial dysplasia and evidence of a microdeletion in one family. *Hum Mol Genet.* 1995;4:71–5.
10. Bodmer WF, Bailey CJ, Bodmer J, et al. Localization of the gene for familial adenomatous polyposis on chromosome 5. *Nature.* 1987;328:614–6.
11. Wijn MA, Keller JJ, Giardiello FM, Brand HS. Oral and maxillofacial manifestations of familial adenomatous polyposis. *Oral Dis.* 2007;13:360–5.
12. Lubinsky M, Kantaputra PN. Syndromes with supernumerary teeth. *Am J Med Genet A.* 2016;170:2611–6.
13. Akcam MO, Evirgen S, Uslu O, Memikoglu UT. Dental anomalies in individuals with cleft lip and/or palate. *Eur J Orthod.* 2010;32:207–13.
14. Tereza GP, Carrara CF, Costa B. Tooth abnormalities of number and position in the permanent dentition of patients with complete bilateral cleft lip and palate. *Cleft Palate Craniofac J.* 2010;47:247–52.
15. Tsai TP, Huang CS, Huang CC, See LC. Distribution patterns of primary and permanent dentition in children with unilateral complete cleft lip and palate. *Cleft Palate Craniofac J.* 1998;35:154–60.
16. De Oliveira Gomes C, Drummond SN, Jham BC, Abdo EN, Mesquita RA. A survey of 460 supernumerary teeth in Brazilian children and adolescents. *Int J Paediatr Dent.* 2008;18:98–106.
17. Orhan AI, Ozer A, Orhan K. Familial occurrence of nonsyndromal multiple supernumerary teeth. A rare condition. *Angle Orthod.* 2006;76:891–7.
18. Brook AH. A unifying aetiological explanation for anomalies of human tooth number and size. *Arch Oral Biol.* 1984;29:373–8.
19. Kawashima A, Nomura Y, Aoyagi Y, Asada Y. Heredity may be one of the etiologies of supernumerary teeth. *Pediatr Dent J.* 2006;16:115–7.
20. Cobourne MT, Sharpe PT. Making up the numbers: The molecular control of mammalian dental formula. *Semin Cell Dev Biol.* 2010;21:314–24.
21. Anthonappa RP, King NM, Rabie AB. Aetiology of supernumerary teeth: a literature review. *Eur Arch Paediatr Dent.* 2013;14:279–88.
22. Fleming PS, Xavier GM, DiBiase AT, Cobourne MT. Revisiting the supernumerary: the epidemiological and molecular basis of extra teeth. *Br Dent J.* 2010;208:25–30.
23. Xavier GM, Patist AL, Healy C, et al. Activated WNT signalling in postnatal SOX2-positive dental stem cells can drive odontoma formation. *Sci Rep.* 2015;5:14479.
24. Fukuta Y, Totsuka M, Takeda Y, Yamamoto H. Supernumerary teeth with eumorphism in the lower incisor region: a report of five cases and a review of the literature. *J Oral Sci.* 1999;41:199–202.
25. Tanaka S, Murakami Y, Fukami M, Nakano K, et al. A rare case of bilateral supernumerary teeth in the mandibular incisors. *Br Dent J.* 1998;185:386–8.
26. Kurol J. Impacted and ankylosed teeth: why, when, and how to intervene. *Am J Orthod Dentofacial Orthop.* 2006;129:S86–90.
27. Tyrologou S, Koch G, Kurol J. Location, complications and treatment of mesiodentes – a retrospective study in children. *Swed Dent J.* 2005;29:1–9.
28. Breckon JJ, Jones SP. Late forming supernumeraries in the mandibular premolar region. *Br J Orthod.* 1991;18:329–31.
29. Chadwick SM, Kilpatrick NM. Late development of supernumerary teeth: a report of two cases. *Int J Paediatr Dent.* 1993;3:205–10.
30. Solares R, Romero MI. Supernumerary premolars: a literature review. *Paediatr Dent.* 2004;26:450–8.

Sirpa Arte, Wael Awadh, Pekka Nieminen, and David P. Rice

Abstract

This chapter describes tooth agenesis in the developing dentition. Firstly, it describes the prevalence and aetiology of this condition, focussing on genetic and environmental factors. The associated anomalies are discussed and syndromic forms of tooth agenesis are described in detail, including those associated with cleft lip and palate. Finally, the clinical management of patients affected by tooth agenesis is described in the developing dentition.

Definition and Diagnosis

Failure to develop the normal number of 20 primary teeth or 32 permanent teeth, tooth agenesis (hypodontia), is one of the most common developmental anomalies. A tooth is defined to be congenitally missing if it has not erupted in the oral cavity and is not visible in a radiograph at an age when it would be detected.

Usually, all primary teeth have erupted by the age of 3 years and all permanent teeth except the third molars between the ages of 12 and 14 years. Therefore, 3–4-year-old children are suitable for diagnosis of agenesis of primary teeth by clinical examination and 12–14-year-old children for diagnosis of permanent teeth, excluding the third molars. Radiographic diagnosis can be made at a younger age depending on the

S. Arte (✉) • W. Awadh • P. Nieminen

Department of Oral and Maxillofacial Diseases, University of Helsinki, Helsinki, Finland
e-mail: Sirpa.Arte@Helsinki.fi; wael.awadh@helsinki.fi; pekka.nieminen@Helsinki.fi

D.P. Rice

Department of Oral and Maxillofacial Diseases, University of Helsinki, Helsinki, Finland

Department of Oral and Maxillofacial Diseases, Helsinki University Hospital,
Helsinki, Finland

e-mail: David.Rice@Helsinki.fi

timetable of the development in different tooth groups. For that reason it is important to pay attention to the age of the child and also the stage when a tooth is expected to erupt. The use of panoramic radiography is recommended, together with clinical examination in detecting or confirming a diagnosis of tooth agenesis.

The development of the dentition starts during the second month of embryogenesis and continues for many years, until in adolescence the third molars erupt. Tooth germs are visible in radiographs depending on their stage of development. The mineralization of the primary dentition starts early in the prenatal period, while first permanent molars start their mineralization perinatally and the other permanent teeth except third molars before the age of 6 years. However, their crypts become visible earlier. It should be noted that differences in mineralization stages and dental age exist among individuals of the same chronological age depending on ethnic background, on gender and even within a family and in an individual. Therefore a developing tooth germ with late onset of mineralization like second premolars can result in false-positive diagnosis of tooth agenesis even at the age of 6, and later confirmation is necessary.

Terminology

Tooth agenesis, dental agenesis and congenitally missing tooth or hypodontia are terms used commonly when describing failure of tooth development. More specific terms hypodontia (one to five teeth absent, excluding third molars), oligodontia (six or more teeth absent, excluding third molars) and anodontia (complete absence of teeth) are in common use according to the severity of phenomenon (Fig. 5.1).

Prevalence of Tooth Agenesis

Primary Dentition

The prevalence of tooth agenesis in the primary dentition is relatively rare and no significant difference exists in prevalence by gender. The prevalence varies from 0.4

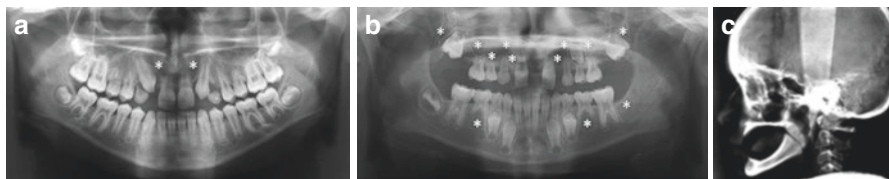


Fig. 5.1 Terminology of tooth agenesis. (a) Hypodontia 1–5 teeth absent (third molars excluded). Dental panoramic tomograph shows the absence of upper permanent lateral incisors (*asterisks*). (b) Oligodontia—six or more teeth absent (third molars excluded). Dental panoramic tomograph shows the absence of 12 permanent teeth (*asterisks*). (c) Anodontia—absence of all teeth (primary and/or permanent dentitions). Lateral skull radiograph shows a complete absence of teeth

to 0.9% in the European population [1] but is reported to be higher, 2.4%, in Japan [2]. Typically one or two primary teeth are missing and the incisor region seems to be affected most often. In Europe the upper lateral incisors whereas in Asian population the lower incisors are the teeth most frequently missing in the primary dentition. Tooth agenesis of the primary tooth is a sign of the absence of the successor tooth.

Permanent Dentition

One or a few permanent teeth are missing in 3–10% of the humans (excluding third molars), and in more than 20% of the subjects, at least one third molar (wisdom teeth) fail to develop. The absence of one or two permanent teeth is most frequent, observed in 83% of the subjects with hypodontia. Most commonly, one or a few permanent upper lateral incisors and second premolars are missing; therefore, this mild phenotype is alternatively called incisor-premolar hypodontia. The prevalence of more severe tooth agenesis phenotypes becomes gradually more rare so that the prevalence of oligodontia (six or more permanent teeth missing) is approximately 0.1% [3]. Non-syndromic anodontia is extremely rare. If anodontia is diagnosed, it points to a syndromic form of tooth agenesis such as an ectodermal dysplasia syndrome [3].

The prevalence of permanent tooth agenesis has differences between populations and between females and males. For females the prevalence is 1.37 times higher than for males. Prevalence of tooth agenesis appears to be lower in North America than in Europe and Australia as well as in Asia. In a meta-analysis, it was shown that in European countries (Caucasians, white) the prevalence of tooth agenesis is approximately 5.5% but 6% or higher in most studies of Scandinavian countries, 3.9% in North America (white and African American), 6.3% in Australia (white), 6.9% in Asia (Chinese Mongoloid) and 2.5% in the Middle East (Saudi Arabia, white) [3].

Most Frequently Missing Permanent Teeth

The frequency of tooth agenesis is similar in the maxilla and mandible as well as in the left and right sides of the jaws. For most teeth bilateral agenesis is noticed in about half of the cases. It is most likely that the last developing tooth within its dental group is congenitally missing: third molars, second premolars and lateral incisors.

Apart from the third molars, the most commonly missing teeth in permanent dentition of Caucasian populations are clearly mandibular second premolars (more than 40% of the missing teeth) followed equally by maxillary lateral incisors and maxillary second premolars and then the mandibular incisors [3]. Among the Japanese, Chinese and Korean populations, the most commonly missing tooth is in the mandibular incisor region [4]. The most stable permanent teeth are maxillary central incisors, canines and mandibular first molars (Fig. 5.2).

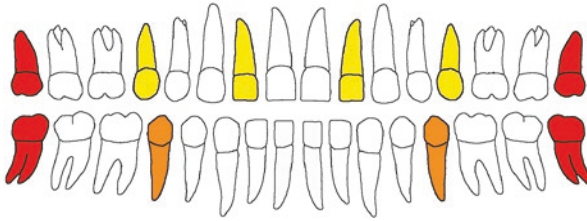


Fig. 5.2 Schematic of most frequently missing permanent teeth. In Caucasian populations the most commonly missing are the third molars (*red*), followed by the mandibular second premolars (*orange*) and followed equally by the maxillary lateral incisors and maxillary second premolars (*yellow*). Less frequently missing teeth are the mandibular incisors, maxillary canines, first premolars and mandibular second molars. The maxillary central incisors, mandibular canines, maxillary and mandibular first molars and maxillary second molar teeth are very rarely missing [3]

Aetiology of Tooth Agenesis

The long time span and complexity of human dental development means that abnormalities may arise from multiple genetic and environmental factors which may affect the teeth at different stages of development.

The important role of genetics as a cause of tooth agenesis stems from the facts that tooth agenesis is usually observed without any obvious environmental cause and it is more common in monozygotic than in dizygotic twins and among relatives than in the general population. Many gene mutations have been discovered to cause isolated and syndromic tooth agenesis. However, both twin and family studies indicate that the relationship of the genotype and phenotype is not straightforward but shows variation presumably caused by genetic background, that is to say additional genetic factors, as well as epigenetic and external factors. Thus, family members affected by the same mutation typically show variation in the phenotype, and even monozygotic twins often do not have identical phenotypes [5].

Environmental Causes of Tooth Agenesis

The most dramatic known external effect on tooth development is caused by treatment of cancer during early childhood. The effects include tooth agenesis, extreme microdontia and hypoplasia of tooth roots. The effect is especially strong after radiological treatments but also chemotherapy can cause microdontia and agenesis [6].

Tooth germs may sometimes be destroyed by external trauma. It has been suggested that agenesis of mandibular third molars may be related to application of local anaesthetics during childhood dental care [7].

Tooth agenesis has been described in children whose mothers' have suffered rubella infection during pregnancy [8].

Both experimental animal and population studies have shown that certain pollutants, especially dioxins, are harmful for normal tooth development. As shown by

animal studies, dioxins affect both tooth morphogenesis and cell differentiation, and predisposition to dioxins after an industrial accident in Seveso, Italy, was associated with increased prevalence of tooth agenesis [9]. Confirmed environmental causes of tooth agenesis are rare and do not have a significant contribution to tooth agenesis at a population level. However, factors that affect the size of tooth germs may also play a role in determining the outcome of genetic predisposition.

Genetic Causes of Tooth Agenesis

Both case reports and systematic studies of cohorts of patients and their families indicate that genetic factors cause tooth agenesis by different modes of inheritance. Thus autosomal dominant, autosomal recessive and X-linked modes of inheritance as well as suggested complex inheritance have been reported in families with oligodontia. In his pioneering study of hypodontia, Grahnen studied Swedish families and observed hypodontia in at least one of the parents of 43 of the 55 cases (78%), suggesting predominantly dominant inheritance [10].

Underlying genes have been identified in all common syndromes featuring tooth agenesis and a significant part of nonsyndromic oligodontia [11, 12].

Identified human mutations, complemented by experimental mutations in mice, show that disturbances in any of the important signalling pathways (WNT, Hedgehog, FGF and TGF β /BMP) or their intracellular effectors affect normal tooth development. Furthermore, mutation identification in hypohidrotic (also named anhidrotic) ectodermal dysplasia (HED, EDA), the most common ectodermal dysplasia syndrome, led to a discovery of a whole new signalling pathway, EDA signalling. In this pathway, an identical disease can be caused by inactivating mutations in the genes coding for the extracellular signal protein, its cell surface receptor or an intracellular mediator of signalling (EDA, EDAR and EDARADD, respectively). Similarly, identified causative mutations in other signalling pathways may affect the extracellular signals or their receptors or intracellular mediators or effectors. Several genes for transcription factors critical for BMP, FGF and WNT signalling and cell fate decisions are mutated in isolated or syndromic forms of tooth agenesis. In addition, tooth agenesis may be caused by mutations in molecules mediating cell adhesion and/or even by defects in extracellular matrix molecules [12].

The first identified causative genes for isolated tooth agenesis were *MSX1* and *PAX9*, which code for transcription factor proteins critical for tooth development [13, 14]. Heterozygous mutations segregate with severe tooth agenesis in multi-generation families. In the family segregating an *MSX1* missense mutation in the homeobox, all affected family members lacked second premolars and third molars and to variable extent other teeth like first molars, first premolars and some incisors [14], whereas a frameshift mutation in *PAX9* affected predominantly all molars, second premolars and some incisors [13]. Numerous other mutations, each unique, have since been identified, and phenotypes of the patients largely conform to the patterns described above [11]. Data from patient cohorts suggests that less than 10% of families with oligodontia have a mutation in *MSX1* or *PAX9*; however, due to the

dominant inheritance, an overall contribution of each of these genes to oligodontia is larger [11, 15].

MSX1 and *PAX9* are expressed in the mesenchymal tissue during early tooth development and mediate effects of epithelial-mesenchymal signalling especially by BMPs and FGFs. Selective tooth agenesis as a consequence of a heterozygous inactivating mutation indicates a haploinsufficiency of these genes in human tooth development, presumably by defective mesenchymal condensation and signalling. Haploinsufficiency, i.e. insufficiency of a single normal copy, as a cause of tooth agenesis is a more general mechanism present in numerous syndromes and isolated tooth agenesis with dominant inheritance.

Two genes involved in WNT signalling have been identified to harbour dominant mutations causing oligodontia. Heterozygous truncating mutations in *AXIN2*, coding for an intracellular antagonist of WNT signalling, have been identified in severe oligodontia of permanent teeth [16]. Interestingly, the patients were also predisposed to colorectal cancer. Recently, a WNT cell surface co-receptor *LRP6* has been shown to harbour heterozygous inactivating mutations causing dominantly inherited tooth agenesis, in most cases expressed as a severe phenotype [17, our unpublished data].

The important role of WNT signalling in human tooth development is emphasised by unravelling the role of *WNT10A* in tooth agenesis. Recessive mutations affecting this extracellular signal protein were first identified in an ectodermal dysplasia syndrome, odonto-onycho-dermal dysplasia. Subsequently they have been identified in a spectrum of patients with ectodermal defects, from an allelic Schöpf-Schulz-Passarge syndrome (additional symptoms eyelid cysts and telangiectasias) to ectodermal dysplasia with hypo- or hyperhidrosis to tooth agenesis with minor or no other ectodermal defects. Mutations in *WNT10A* are by far the most common known cause of isolated tooth agenesis: in different samples, biallelic or heterozygous genotypes have been found in 26–56% of the non-syndromic oligodontia patients (Table 5.1) [11, 15, 18].

According to the verifiable data, despite strong effects on permanent dentition, *WNT10A*, *AXIN2* or *LRP6* mutations very seldom affect deciduous teeth [11, 16] implying that abnormalities of WNT signalling tend to affect mechanisms involved in development of successional teeth. Unlike mutations in *MSX1* and *PAX9*, mutations in *WNT10A* or *LRP6* show variable phenotypes sometimes affecting mostly anterior and sometimes posterior teeth. This extensive variation may depend on other mutations that affect *WNT10A* expression or are located in other genes. Indeed, examples of co-detection of *WNT10A* and *EDA* signalling mutations have been presented [11].

Like *WNT10A*, genes of EDA signalling are associated with both syndromic and isolated tooth agenesis. While completely inactivating genotypes cause an ectodermal dysplasia syndrome (see below), specific hypomorphic mutations in *EDA* are expressed as isolated tooth agenesis affecting especially anterior deciduous and permanent dentition. Similar defects can arise due to heterozygosity of *EDA* and *EDAR* mutations in carriers [11, 19].

Table 5.1 Selected genetic causes of tooth agensis

<i>Non-syndromic/isolated causes</i>			
Gene mutated/ chromosomal change	Estimated percentage of non-syndromic oligodontia families (%)	Type of molecule encoded	Associated non-dental defects
<i>WNT10A</i>	26–56	Growth factor	Minor ectodermal features
<i>PAX9</i>	5	Transcription factor	
<i>MSX1</i>	3	Transcription factor	Cleft lip/palate, nail dysplasia
<i>AXIN2</i>	2	Signal regulator	Colorectal cancer
<i>LRP6</i>	5	Co-receptor	
<i>EDA</i> (ectodysplasin)	8–10	Growth factor	Minor ectodermal features
<i>EDAR</i>		TNF receptor	
<i>EDARADD</i>		Signal transducer	
<i>Syndromic causes</i>			
Condition	Gene mutated/ chromosomal change	Type of molecule encoded	Associated non-dental defects
Hypohidrotic (anhidrotic) ectodermal dysplasia (HED, ED)	<i>EDA</i> (encodes ectodysplasin)	Growth factor	Ectodermal dysplasia, hypoplastic hair/glands
	<i>EDAR</i>	TNF receptor	
	<i>EDARADD</i>	Signal transducer	
Ectrodactyly, ectodermal dysplasia and cleft lip/palate syndrome (EEC)	<i>TP63</i>	Transcription factor	Ectodermal dysplasia, cleft palate, split hands
Odonto-onycho- dermal dysplasia (OODD)	<i>WNT10A</i>	Growth factor	Ectodermal dysplasia
Cleft lip/palate- ectodermal dysplasia syndrome (CLPED1)	<i>PVRL1</i> (encodes nectin 1)	Adhesion molecule	Ectodermal dysplasia, cleft lip/palate, cutaneous syndactyly
Axenfeld-Rieger syndrome (ARS)	<i>PITX2</i>	Transcription factors	Eye defects, umbilical anomalies
	<i>FOXC1</i>		
Diastrophic dysplasia	<i>DTDST</i>	Sulphate transporter	Osteochondrodysplasia
Van der Woude syndrome 1 (VWS1)	<i>IRF6</i>	Transcription factor	Cleft lip/palate, pits in the lower lip
Van der Woude syndrome 2 (VWS2)	<i>GRHL3</i>	Transcription factor	Cleft lip/palate, pits in the lower lip
Incontinentia pigmenti (IP)	<i>NEMO (IKBK)</i>	Kinase subunit	Ectodermal dysplasia, neurological problems
Down syndrome	Trisomy 21		Dysmorphic craniofacial features, mental retardation

The mutations described above have been mainly identified in oligodontia. However, many heterozygous carriers of *WNT10A* or *EDAR* mutations, that is, family members of oligodontia patients, present with hypodontia, lacking one or a few permanent teeth. These variants behave as dominant mutations but with reduced penetrance and variable expression, presumably explained by genetic background and other factors affecting the developmental outcome. It is probable that similar variants in other genes will be discovered in hypodontia.

Anomalies Associated with Tooth Agenesis

Dental Anomalies

Tooth agenesis is commonly associated with different kinds of dental anomalies in other teeth (Fig. 5.3). These anomalies can occur even in the dentition with mild hypodontia but are more frequent in cases of oligodontia. In addition, tooth agenesis contributes to the development of abnormal occlusion, malfunctions and aesthetic problems especially in subjects with severe oligodontia.

The typical dental anomaly associated with tooth agenesis is a peg-shaped upper lateral incisor which is often noticed when the contralateral incisor is missing. It belongs to the spectrum of anomalies that are associated with tooth agenesis: reduction of tooth crown size (mesiodistal and bucco-lingual dimension of the crown), abnormal morphology (peg-shaped tooth, conical tooth, tapered- or shovel-shaped tooth, reduction of the cusp number and form) and shortened roots and taurodontism [20–22].

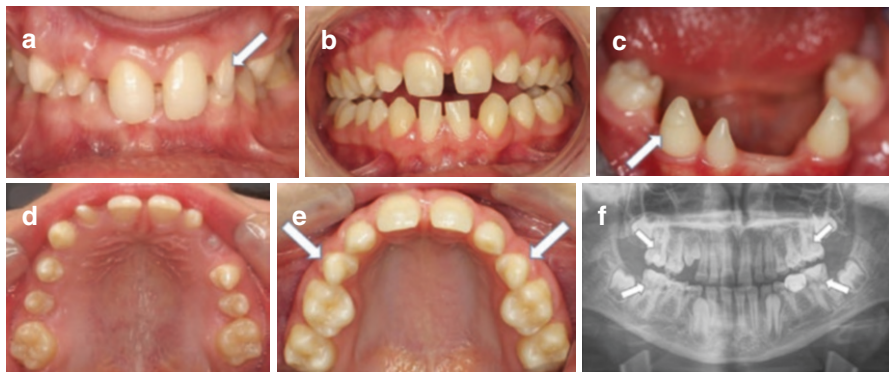


Fig. 5.3 Anomalies associated with tooth agenesis. (a, d) are images from the same individual. The patient has oligodontia mainly in the mandible and has a *WNT10A* mutation. The images show reduced crown size, tapered teeth and a peg-shaped upper right lateral incisor (arrow). (b, e) are images from the same individual. The images show tapered maxillary central incisors and rotated maxillary first premolars (arrows). (c) Conical-shaped mandibular primary teeth in a patient with extensive oligodontia (arrow). (d) in the text pointing to the radiograph should be the same as in the picture. (f) Taurodontism in all four first permanent molars, vertically enlarged and misshaped pulp chambers (arrows)

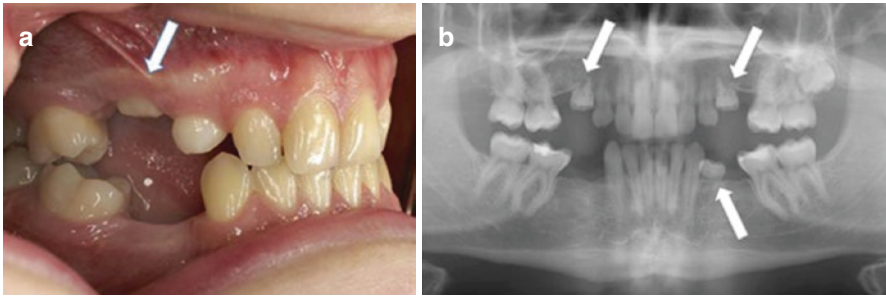


Fig. 5.4 Infraocclusion of primary molar. (a, b) are images from the same individual. The patient has agenesis of all permanent premolars and maxillary canines and infraocclusion of the remaining primary first molars (arrows)

It has been shown that, as the severity of tooth agenesis increases, the delay of dental age also increases. A mean delay of up to 2 years compared to the chronological age has been reported. From a clinical perspective, it must be taken into consideration that a tendency to developmental delay is possible in teeth contralateral or adjacent to the missing tooth [23].

Ectopic teeth, especially ectopic canines (palatal or labial maxillary canines) but also other teeth such as first premolars and molars, as well as transpositions of teeth (canine-premolar, incisor-canine transpositions), show association with tooth agenesis [22, 24, 25].

An association exists between infraocclusion of primary molars and agenesis of premolars. In about 20% of the subjects with agenesis of second premolars, infraocclusion of primary second molars has been noted (Fig. 5.4).

In subjects with tooth agenesis, the prevalence of abnormal tooth positions such as rotations (especially premolars) as well as enamel defects (hypoplasia, hypocalcification) is higher than in the control groups [22].

General Signs and Symptoms

Approximately half of young individuals with oligodontia have one or more signs or symptoms from ectodermal origin in a Swedish study. One in three individuals with oligodontia has low salivary secretion, but only one in ten individuals has self-reported symptoms from the hair, nails or sweat glands. Therefore, measurement of salivary secretion is indicated in subjects with oligodontia [26].

Tooth Agenesis and Cancer

It has been shown that tooth agenesis and cancer development share common molecular pathways. The connection between tooth agenesis and colorectal cancer predisposition as a consequence of *AXIN2* mutations was found in a large Finnish

family [16]. An increased prevalence of tooth agenesis has been reported among patients with epithelial ovarian cancer [27]. However, more studies on this subject are needed to understand genetic mutations and networks which together contribute both to tooth agenesis and to cancer.

Tooth Agenesis in Orofacial Clefting and Syndromes

Cleft Lip/Palate

Anomalies in the number of teeth (tooth agenesis, supernumerary teeth), in the morphology of teeth (shape and size) and delayed development and eruption of teeth are common in patients with clefts. The prevalence of hypodontia increases with the severity of the cleft and has been reported to be 10–68% in different cleft types, being 10% in cleft lip, 16% in submucous cleft, 33% in cleft palate and 49% in unilateral and 68% in bilateral cleft lip and palate. The teeth most commonly affected are in the cleft area (upper permanent lateral incisor), but tooth agenesis is more common than in general population also outside the cleft region [28].

Syndromic Tooth Agenesis

Tooth agenesis with varying severity is associated with more than 80 malformation syndromes [28, 29].

Down Syndrome (Trisomy 21)

Down syndrome, the most common chromosomal abnormality in man, is caused by trisomy of all or a critical portion of chromosome 21. Together with typical dysmorphic craniofacial features, mental retardation and structural anomalies, tooth agenesis and other dental aberrations are very common. The prevalence of tooth agenesis is about 50% in the patients if the third molars are excluded and about 90% if they are considered. The upper lateral incisor is most commonly missing in Down syndrome, and peg-shaped upper lateral incisors are frequent. Other dental anomalies in the permanent dentition of patients with Down syndrome include taurodontism, ectopic eruption, impaction, delayed eruption, transposition of teeth and microdontia [30].

Axenfeld-Rieger (Rieger) Syndrome

Axenfeld-Rieger syndrome (ARS) is an autosomal dominant disorder with malformations of the anterior chamber of the eye, umbilical anomalies and tooth agenesis.

The prevalence of ARS is approximately 1 in 200,000. ARS Type 1 is caused by mutation in a homeobox transcription factor gene *PITX2*; Type 2 maps to chromosome 13q14; and Type 3 is caused by mutation in the *FOXC1* gene.

The maxillary primary and permanent incisors and second premolars are most commonly missing and conical and misshapen teeth and microdontia have been reported. Maxillary hypoplasia is a typical craniofacial finding, which is caused, in part, by missing teeth in the region [28].

Ectodermal Dysplasias

Ectodermal dysplasias (EDs) include a large clinically and genetically heterogeneous group of rare conditions where at least two of the ectodermal derivatives such as hair, nails, glands and teeth are affected. There are about 200 EDs and ED syndromes with 11 subgroups [31].

Hypohidrotic (also named anhidrotic) ectodermal dysplasia (HED, EDA) is the most common ectodermal dysplasia. HED is genetically heterogeneous and caused by mutations in three different genes which all disrupt the same signalling pathway. Mutations in the X-chromosomal *EDA* gene coding for the tumour necrosis factor-like signal called ectodysplasin cause X-linked EDA, whereas autosomal dominant and recessive HED are caused by mutations in the TNF receptor *EDAR* and its intracellular regulator *EDARADD*. Autosomal HED is clinically indistinguishable from X-linked form, but in autosomal HED both males and females can be similarly affected. Recently, mutations in *WNT10A* have been shown to cause hypohidrotic/anhidrotic ectodermal dysplasia with distinctive clinical features including marked dental phenotype without facial dysmorphism.

The patients with EDA show frontal bossing, nasal and maxillary hypoplasia, low face height, sparse or absent hair and dry skin with periorbital hyperkeratosis which contribute to a typical appearance. The most characteristic features in EDA are delayed eruption of primary teeth and the reduced number and abnormal shape of teeth which can alert the clinician to making diagnosis in early childhood. In X-linked HED affected males have severe oligodontia or anodontia in both the primary and permanent dentitions with on average only six permanent teeth developing. The teeth which most commonly do develop are the maxillary central incisors and first molars. There is also a delay in dental development. The teeth which develop often have abnormal conical crowns especially in the incisor region, and other teeth may be microdont or may have otherwise altered morphology. Taurodontism is also more common in the molars of the patients with HED. The sweat glands are hypoplastic or aplastic resulting in an inability to sweat normally which can be life threatening especially in early infancy. Female carriers in X-linked HED are affected with milder phenotype than male patients [19].

Odonto-onycho-dermal dysplasia (OODD) is a recessive ectodermal dysplasia characterised by severe oligodontia in the permanent dentition with less affected primary dentition, conical teeth, lack of taste buds of the tongue, hypoplastic nails and thin, dry hair. Several different mutations in *WNT10A* have been identified in

patients with OODD or the similar Schöpf-Schulz-Passarge syndrome where cysts of the eyelids are additional manifestations.

Incontinentia pigmenti (IP, Bloch-Sulzberger syndrome) is a rare X-chromosomal dominant ectodermal dysplasia syndrome caused by mutation in the *IKK-gamma* gene also called *NEMO*. Due to X-chromosomal dominant inheritance, IP affects females and is usually lethal prenatally in males. The patients have variable abnormalities of the skin, hair, nails, eyes, teeth and central nervous system. Dental anomalies have been diagnosed in more than 50% of the patients. Tooth agenesis, mostly oligodontia, is most frequent of the anomalies, but dental shape abnormalities (conical, peg-shaped teeth, generalised microdontia), macrodontia (extra cusps), taurodontism and delayed eruption of teeth have been reported [32].

Clinical Problems and Management of Patients with Tooth Agenesis

Tooth agenesis creates special functional and aesthetic problems, and several treatment phases starting in childhood and continuing to adulthood are necessary. An ideal management from diagnosis to treatment planning and treatment requires a multidisciplinary approach, and many specialities of dentistry are needed. Nearly all patients, 90%, with hypodontia or oligodontia need orthodontic treatment. In some cases, medical and genetic consultation should be considered. A repetitive communication between the members of the team is also essential [33, 34].

Giving sufficient information about tooth agenesis to the patient and the family as well as support during the different phases of the treatment is important. It is recommended that siblings and children of the patient should be examined for tooth agenesis and associated anomalies at appropriate age, firstly in the early mixed dentition and then at approximately 9–10 years of age.

During the growth period, it is important to follow and to evaluate the development of occlusion and the growth pattern of the jaws (sagittal, vertical, transversal growth). Patients with tooth agenesis often have typical dentofacial features, and the severity and location of missing teeth have a significant effect on them. Cephalometric analyses have shown bimaxillary retrognathism, reduced vertical facial dimensions, concave profile, decreased mandibular plan angle, incisors which are upright and overerupted in oligodontia patients [35].

In tooth agenesis cases, in order to optimise long-term success, forethought should be given into maintaining alveolar bone, for example, around a deciduous tooth where no permanent successor is present. Depending on the malocclusion, maintaining alveolar bone will improve the success of possible dental implants later and may facilitate possible orthodontic tooth movement into the space. The alveolar process grows with teeth and each tooth creates its own bone while erupting into the oral cavity. If there are no teeth developed in the jaws, there are no alveolar processes either. On the other hand, after losing a tooth, alveolar resorption continues for years which results in shortage of bone for later reconstruction.

The diagnosis and treatment of tooth agenesis and the diagnosis and treatment of orthodontic malocclusion should not be planned in isolation but with an understanding of both entities. That said, it can be helpful to assess the patient from a malocclusion perspective first and then add the complexities of the tooth agenesis. In this way the missing teeth can be incorporated into the overall plan for the patient. For example, in a class I severe crowding case with a favourable skeletal pattern, premolar extractions may be planned. If the patient is missing premolar teeth, then this hypodontia can easily be integrated into the plan. Conversely, if the patient presents with a deep bite and closing-type growth pattern, then one would be more inclined to treat on a non-extraction basis, and it may be advisable to maintain the lower deciduous molars for as long as possible if lower premolars are missing.

Primary and Early Mixed Dentition

Both functional and aesthetic problems can arise early in childhood. The preliminary evaluation of the facial growth pattern and type of occlusion is made, and interceptive orthodontics (elimination of crossbites, scissors bites) can be carried out to promote favourable orofacial functions and growth of the jaws.

Before a child starts school, it is advisable to evaluate orofacial functions (speaking, mastication, smiling) together with aesthetics (conical, malformed teeth, spacing). This is because children with tooth agenesis can be exposed to bullying at school. Closure of diastema (midline maxillary diastema), composite build-ups of malformed primary and permanent teeth (incisors, canines) and removable dentures are possible in severe oligodontia or anodontia. In children with missing incisors, fixed constructions like banded molars with acrylic incisors fixed to lingual or palatal arches can be considered. However, all these early constructions will require regular follow-up adjustments during the growth period.

If the premolar is missing, ankylosis and developing of infraocclusion of the predecessor tooth is possible. This can result in tipping of the neighbouring teeth and overeruption of the opposing tooth. Ankylosis can be diagnosed by evaluating the alveolar bone levels between the primary molar and the adjacent teeth. A flat level of the alveolar bone indicates that the primary tooth is erupting evenly with the permanent ones. If the alveolar bone level becomes oblique and the bone level around the primary tooth is more apically, this confirms ankyloses [36]. In a Swedish study, 20% of the primary molars without successors were submerged 1 mm or more relative to the adjacent teeth at the age of 12 years and 55% of them between 0.5 and 4.5 mm at the age of 20 years. Whenever infraocclusion of primary molar is noted, a build-up of the occlusal surface can be made to improve occlusal contacts. However, if the infraocclusion worsens, extraction of the ankylosed tooth can be considered [33, 37].

In addition to ankylosis, root resorption of the primary molars must be assessed and monitored. Large individual variation is seen in an amount of root resorption. Lower primary molars without a successor have a good prognosis, and in more than 90% of the patients, these teeth survived up to late adulthood [38].

Palatally or labially impacted permanent canines are common in patients with tooth agenesis. The maxillary permanent canines should be palpated labially at the age of 9–10 years, and if they are not, further radiological examination must be carried out to confirm their position. The positions of permanent canines have particular significance in cases of tooth agenesis, especially if the upper lateral incisor(s) are missing. The position and eruption pattern of permanent canines will influence the management of space of primary lateral incisors and canines.

Late Mixed and Permanent Dentition

At approximately 8–10 years of age, making of a preliminary long-term treatment plan including orthodontic and future prosthodontic therapy is recommended. Orthodontic treatment includes both management of general features of malocclusion and special problems linked to tooth agenesis. During the growth period, orthodontic treatment with functional jaw orthopaedics to improve skeletal discrepancies and using of fixed appliances to arrange dental arches is often indicated. A proper positioning of the incisors, canines and molars, treatment of deep bite and a decision on maintaining or closing spaces of missing teeth are advised. When considering the treatment options, an evaluation of the facial components including hard and soft tissues and dental show in the face is essential. If the number of teeth is low, the arrangement of anchorage during orthodontic treatment can be challenging. However, using of temporary skeletal anchorage devices has brought many new possibilities for the orthodontic therapy of tooth agenesis patients [33].

There are two options for treating patients with missing maxillary lateral incisors: to close the space or to open the space. In addition, a symmetrical appearance of the incisor region must be included as a treatment aim. If the space is planned to be closed, permanent canines are allowed to erupt next to the central incisors, and extractions of primary laterals and canines can be considered before the permanent canines erupt. Posterior teeth are later protracted mesially to substitute the maxillary first premolars for canines. The second option is to open the space for prosthetic replacement of upper laterals.

The desired occlusion at the end of treatment must be considered. If the patient has a Class I relationship in both the molars and canines, normal overbite and overjet, it is ideal for a prosthetic replacement of lateral incisors. The ideal situation for closing the space is Class II occlusal intercuspatation, or maxillary posterior teeth have to be brought mesially into a proper Class II relationship. Treatment is often required to make the canines look and function more like lateral incisors, and subsequently that the first premolars, now in the canine position, look and function more like canine teeth. This may involve reshaping the teeth in three planes of space: vertically, mesiodistally and palato-labially. With regard to the first premolar, it may be advisable to grinding the palatal cusp, so that it does not interfere with the occlusion and also rotate the tooth mesio-palatally so that the tooth presents a wider crown and improved aesthetic result. In some cases, reshaping of the teeth may not

produce an acceptable aesthetic result and the placement of prosthetic replacements can be considered.

If the maxilla and the mandible have different number of teeth, it may be impossible to achieve a normal Class I molar and canine relationship without compensatory procedures such as an extraction of a lower permanent incisor or a reduction of the width of second primary molar. It must be noted that due to generalised microdont teeth, it is not always possible to close all spaces in the dental arches with orthodontics. Instead, composite build-ups or extra prosthetic teeth can be considered.

Extractions of upper primary second molars without successors at appropriate time are beneficial because permanent molars tend to drift mesially closing the space. Mandibular molars do not drift as favourably and orthodontics may be challenging. Especially in skeletal deep bite, it is not recommended to extract lower primary molars because of an unfavourable worsening of the deep bite. Maintaining of the space, autotransplantation or later implants are the other alternatives.

Before the orthodontic appliances are removed, it is essential to examine the patient for correct amount of space for tooth replacement and the placement of implants. In addition to dental study models, 3D radiography gives the most exact measurements for spaces. The roots of the teeth must be parallel and adequately separated for proper placement of an implant. For instance, the minimum space between the root and an implant should be approximately 1–1.4 mm, the minimum interradicular space for a maxillary lateral is 6 mm and the vertical space should be 7 mm [39].

It should be remembered that the placement of dental implants should be delayed until growth of the jaws is complete. This is because dental implants, and the bone around them, do not ‘grow’ with the patient. If implants are placed too early, a vertical discrepancy between the prosthetic tooth and the neighbouring teeth can develop [40]. Even if implant treatment is not used in growing patients, children with ectodermal dysplasia syndromes and anodontia in the mandible have been treated with implants. However, treatment of ectodermal dysplasia patient is very demanding and should be centralised [33, 41].

Patients should be provided with retainers which maintain the spaces created for replacing the missing teeth. Removable retainers with acrylic teeth replacing missing teeth can be used to improve dental appearance and oral function as well as act as a retainer. Resin-retained bridges are also possible as a permanent or temporary solution.

Established Dentition

If the skeletal discrepancy is severe and camouflage treatment alone cannot provide the facial harmony, a combination of orthodontics and orthognathic surgery should be considered before prosthetic restoration. In addition, minor surgical procedures are often needed such as alveolar ridge and sinus floor augmentations and inferior dental nerve transpositions to ensure proper circumstances for the implants.

Estimation of the craniofacial growth is essential in planning an appropriate time for placing implants in order to prevent later submergence of an implant tooth. Following of the general growth is not always sufficient because cessation of craniofacial growth exhibits a great individual variation. However, in most cases craniofacial growth can be considered to have ceased at approximately 18–19 years of age in males and 17–18 years of age in females. It must be kept in mind that patients can show continued craniofacial vertical growth late into adulthood and even into an old age which can create aesthetic and functional disturbances in the implant region. It is widely recommended, however, that implants into an incisor area are not placed earlier than over 20 years of age to ensure better function and aesthetics in the future.

References

1. Jarvinen S, Lehtinen L. Supernumerary and congenitally missing primary teeth in Finnish children. An epidemiologic study. *Acta Odontol Scand.* 1981;39(2):83–6.
2. Yonezu T, Hayashi Y, Sasaki J, Machida Y. Prevalence of congenital dental anomalies of the deciduous dentition in Japanese children. *Bull Tokyo Dent Coll.* 1997;38(1):27–32.
3. 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(3):217–26.
4. Chung CJ, Han JH, Kim KH. The pattern and prevalence of hypodontia in Koreans. *Oral Dis.* 2008;14(7):620–5.
5. Townsend GC, Richards L, Hughes T, Pinkerton S, Schwerdt W. Epigenetic influences may explain dental differences in monozygotic twin pairs. *Aust Dent J.* 2005;50(2):95–100.
6. Pedersen LB, Clausen N, Schroder H, Schmidt M, Poulsen S. Microdontia and hypodontia of premolars and permanent molars in childhood cancer survivors after chemotherapy. *Int J Paediatr Dent.* 2012;22(4):239–43.
7. Swee J, Silvestri AR Jr, Finkelman MD, Rich AP, Alexander SA, Loo CY. Inferior alveolar nerve block and third-molar agenesis: a retrospective clinical study. *J Am Dent Assoc.* 2013;144(4):389–95.
8. Kraus BS, Ames MD, Clark GR. Effects of maternal rubella on dental crown development. *Clin Pediatr.* 1969;8(4):204–15.
9. Alaluusua S, Calderara P, Gerthoux PM, Lukinmaa PL, Kovero O, Needham L, Patterson DG Jr, Tuomisto J, Mocarelli P. Developmental dental aberrations after the dioxin accident in Seveso. *Environ Health Perspect.* 2004;112(13):1313–8.
10. Grahnen H. Hypodontia in the permanent dentition. A clinical and genetical investigation. *Odont Revy.* 1956;7(Suppl 3):1–100.
11. Arte S, Parmanen S, Pirinen S, Alaluusua S, Nieminen P. Candidate gene analysis of tooth agenesis identifies novel mutations in six genes and suggests significant role for WNT and EDA signaling and allele combinations. *PLoS One.* 2013;8(8):e73705.
12. Nieminen P. Genetic basis of tooth agenesis. *J Exp Zool Part B Mol Dev Evol.* 2009;312B(4):320–42.
13. Stockton DW, Das P, Goldenberg M, D'Souza RN, Patel PI. Mutation of PAX9 is associated with oligodontia. *Nat Genet.* 2000;24(1):18–9.
14. Vastardis H, Karimbox N, Guthua SW, Seidman JG, Seidman CE. A human MSX1 homeodomain missense mutation causes selective tooth agenesis. *Nat Genet.* 1996;13(4):417–21.
15. van den Boogaard MJ, Creton M, Bronkhorst Y, van der Hout A, Hennekam E, Lindhout D, Cune M, Ploos Van Amstel HK. Mutations in WNT10A are present in more than half of isolated hypodontia cases. *J Med Genet.* 2012;49(5):327–31.

16. Lammi L, Arte S, Somer M, Jarvinen H, Lahermo P, Thesleff I, Pirinen S, Nieminen P. Mutations in AXIN2 cause familial tooth agenesis and predispose to colorectal cancer. *Am J Hum Genet.* 2004;74(5):1043–50.
17. Massink MP, Creton MA, Spanevello F, Fennis WM, Cune MS, Savelberg SM, Nijman IJ, Maurice MM, van den Boogaard MJ, van Haaften G. Loss-of-function mutations in the WNT co-receptor LRP6 cause autosomal-dominant oligodontia. *Am J Hum Genet.* 2015;97(4):621–6.
18. Bohring A, Stamm T, Spaich C, Haase C, Spree K, Hehr U, Hoffmann M, Ledig S, Sel S, Wieacker P, Ropke A. WNT10A mutations are a frequent cause of a broad spectrum of ectodermal dysplasias with sex-biased manifestation pattern in heterozygotes. *Am J Hum Genet.* 2009;85(1):97–105.
19. Lexner MO, Bardow A, Hertz JM, Nielsen LA, Kreiborg S. Anomalies of tooth formation in hypohidrotic ectodermal dysplasia. *Int J Paediatr Dent.* 2007;17(1):10–8.
20. Alvesalo L, Portin P. The inheritance pattern of missing, peg-shaped, and strongly mesio-distally reduced upper lateral incisors. *Acta Odontol Scand.* 1969;27(6):563–75.
21. Apajalahti S, Arte S, Pirinen S. Short root anomaly in families and its association with other dental anomalies. *Eur J Oral Sci.* 1999;107(2):97–101.
22. Baccetti T. A controlled study of associated dental anomalies. *Angle Orthod.* 1998;68(3):267–74.
23. Ruiz-Mealin EV, Parekh S, Jones SP, Moles DR, Gill DS. Radiographic study of delayed tooth development in patients with dental agenesis. *Am J Orthodont Dentofacial Orthop.* 2012;141(3):307–14.
24. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. *Angle Orthod.* 1981;51(1):24–9.
25. Peck S, Peck L, Kataja M. Mandibular lateral incisor-canine transposition, concomitant dental anomalies, and genetic control. *Angle Orthodont.* 1998;68(5):455–66.
26. Bergendal B. Oligodontia ectodermal dysplasia—on signs, symptoms, genetics, and outcomes of dental treatment. *Swed Dent J Suppl.* 2010;205:13–78, 7–8.
27. Fekonja A, Cretnik A, Zerdoner D, Takac I. Hypodontia phenotype in patients with epithelial ovarian cancer. *Radiol Oncol.* 2015;49(1):65–70.
28. Klein OD, Oberoi S, Huysseune A, Hovorakova M, Peterka M, Peterkova R. Developmental disorders of the dentition: an update. *Am J Med Genet C Semin Med Genet.* 2013;163C(4):318–32.
29. Online Mendelian Inheritance in Man, OMIM. McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University (Baltimore, MD). Available from: <http://omim.org/>.
30. Sekerci AE, Cantekin K, Aydinbelge M, Ucar FI. Prevalence of dental anomalies in the permanent dentition of children with Down syndrome. *J Dent Children.* 2014;81(2):78–83.
31. Freire-Maia N, Lisboa-Costa T, Pagnan NA. Ectodermal dysplasias: how many? *Am J Med Genet.* 2001;104(1):84.
32. Minic S, Trpinac D, Gabriel H, Gencik M, Obradovic M. Dental and oral anomalies in *incontinentia pigmenti*: a systematic review. *Clin Oral Investig.* 2013;17(1):1–8.
33. Bergendal B, Bergendal T, Hallonsten AL, Koch G, Kurol J, Kvint S. A multidisciplinary approach to oral rehabilitation with osseointegrated implants in children and adolescents with multiple aplasia. *Eur J Orthodont.* 1996;18(2):119–29.
34. Gill DS, Barker CS. The multidisciplinary management of hypodontia: a team approach. *Br Dent J.* 2015;218(3):143–9.
35. Gungor AY, Turkkahraman H. Effects of severity and location of nonsyndromic hypodontia on craniofacial morphology. *Angle Orthodont.* 2013;83(4):584–90.
36. Kokich VG, Kokich VO. Congenitally missing mandibular second premolars: clinical options. *Am J Orthodont Dentofacial Orthop.* 2006;130(4):437–44.
37. Kurol J. Impacted and ankylosed teeth: why, when, and how to intervene. *Am J Orthodont Dentofacial Orthop.* 2006;129(4 Suppl):S86–90.
38. 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 Orthodont.* 2008;30(3):254–61.

39. Kokich VG. Maxillary lateral incisor implants: planning with the aid of orthodontics. *J Oral Maxillofac Surg.* 2004;62(9 Suppl 2):48–56.
40. Thilander B, Odman J, Lekholm U. Orthodontic aspects of the use of oral implants in adolescents: a 10-year follow-up study. *Eur J Orthodont.* 2001;23(6):715–31.
41. Bergendal B, Bjerklin K, Bergendal T, Koch G. Dental implant therapy for a child with X-linked hypohidrotic ectodermal dysplasia--three decades of managed care. *Int J Prosthodont.* 2015;28(4):348–56.

Trauma to the Permanent Maxillary Incisors in the Mixed Dentition and Orthodontics

6

Jadbinder Seehra and Serpil Djemal

Abstract

Dentoalveolar trauma to the permanent maxillary incisors is a relatively common occurrence in young children. The extent of the injury can be variable but can have significant emotional, social and financial implications to both the child and their parents in both the short and long term. Management of traumatised permanent maxillary incisors in the mixed dentition requires long-term monitoring of both pulp vitality and root development. Although comprehensive orthodontic treatment may not be immediately required, early interceptive management of a malocclusion characterised by an increased overjet may be indicated to reduce the risk of trauma or further trauma to the permanent maxillary incisors beyond the mixed dentition stage of development. If orthodontic treatment is to be considered in these cases, then a team approach involving general dental practitioners, restorative, orthodontic specialists and paediatric dentists is advocated. To prevent trauma to the permanent maxillary incisors in young children, education of children and their parents is vital. The use of custom mouthguards in patients at risk of trauma to the permanent maxillary incisors is recommended.

J. Seehra (✉)

King's College London Dental Institute, Floor 22, Guy's Hospital, Guy's and St Thomas NHS Foundation Trust, London, WC2R 2LS, UK

Department of Orthodontics, London, UK

e-mail: jadbinder.seehra@nhs.net

S. Djemal

King's College London Dental Institute, London, UK

King's College Hospital NHS Foundation Trust, London, UK

e-mail: serpil.djemal@nhs.net

Introduction

Within the literature, a wide variation in the prevalence of dental trauma has been reported ranging between 10.7 and 37.6% [1–4]. This has been attributed to local, environmental, socio-economic causes, behavioural and cultural diversities and a lack in consistency in the age of samples examined as well as standardisation in the classification of traumatic dental injuries [5, 6]. Both social deprivation [2] and more recently binge drinking [7] in adolescence are associated with a higher prevalence of dental traumatic injuries. Within the United Kingdom, up to 17% of children aged between 11 and 14 years of age will experience some form of traumatic injury to the anterior permanent teeth prior to leaving school [8]. The most commonly affected teeth are the permanent maxillary central and lateral incisors [6, 7, 9]. The prevalence of traumatic dental injuries appears to increase with increasing age [3, 9], with a peak incidence up to 12 years of age reported [1].

Aetiology of Dental Trauma

Physical trauma to the permanent maxillary incisor teeth remains the commonest cause of traumatic dental injuries. Types of physical trauma include falls, collisions, trauma whilst playing with others, during physical or sporting activities and traffic accidents [3, 4, 9]. In approximately 40% of cases, this results in an enamel-dentine fracture of the crown of the tooth without pulpal involvement. Enamel fractures are the next most common type of injury (33.8%), followed by subluxation (8.4%) and luxation (6.7%) injuries [9, 10].

Risk Factors for Dental Trauma

Differences in the incidence of dental trauma between males and females suggest gender is an aetiological factor. Based on retrospective studies, males tend to incur trauma to the permanent maxillary incisors more frequently compared to females [2, 9, 11] with the overall risk quantified as 1.88 times more likely [4]. However, the number of activities that an individual performs rather than their gender may predispose them to a higher risk of traumatic dental injuries [6].

Certain traits of a malocclusion have been reported to increase the risk of trauma to the permanent maxillary incisors [2, 9, 12]. An overjet is defined as the horizontal distance between the labial surface of the mandibular incisors and the labial aspect of the incisal edge of the maxillary incisors (Fig. 6.1). Retrospective analysis suggests the presence of an overjet above 3.5 mm increases the risk of trauma [5] and that traumatic dental injuries are 1.6 times more likely to occur if the overjet is greater than 5 mm [4]. Within an orthodontic sample compared to an untreated control group, the presence of an overjet greater than 6 mm increases the risk of trauma to the maxillary incisors [11]. Despite methodological weakness, a systematic review of observational studies suggests a direct proportional relationship with the presence of an increased overjet and an increased risk of trauma [13].

The soft tissues, in particular lip coverage, have a role in the aetiology of dental trauma. Previous studies have reported an association with a history of dental trauma

Fig. 6.1 An increased overjet present in a 9-year-old patient in the mixed dentition



Fig. 6.2 Incompetent lips



and the presence of lip incompetency [14], leading to the conclusion that the presence of inadequate lip coverage is one of the most important predictors of traumatic injury to the permanent maxillary incisors [5] (Fig. 6.2). It has been reported that the presence of both an increased overjet and inadequate lip coverage acts synergistically and substantially increases the risk of trauma [12]. It appears male adolescents, with

both an increased overjet and inadequate lip coverage, are at a higher risk of trauma to the permanent maxillary incisor teeth. However, it would be prudent for clinicians to refer children of both genders in the mixed dentition with a malocclusion characterised by an increased overjet for an early orthodontic assessment.

Impact of Traumatic Dental Injuries to Permanent Maxillary Incisor Teeth

Traumatic dental injuries in a young child can be an emotional and anxious experience for both the individual and his/her parents. Once the immediate trauma has been managed and the child is pain-free and both dental aesthetics and function have been restored, long-term monitoring is required in cases where the prognosis of the traumatised teeth is uncertain. However, the extent of this traumatic incident can also affect an individual's quality of life and result in both immediate and future direct and indirect costs.

Compared to an untreated control group, adolescents aged between 12 and 14 years of age who had a history of dental trauma without pulpal involvement were 20 times more likely to report an impairment on their daily living resulting in an impact on their ability to smile, eat, their social contacts and their emotional state [15]. The psychosocial implications are further highlighted by Porritt et al. [16] who reported higher functional limitations and impact on school-related activities in a sample of patients aged between 7 and 17 years of age who had a history of traumatic dental injuries.

Costs can be classified as direct (transport, health service and medicine costs) and indirect (loss of income and time of parents/guardians) [17]. Prospective analysis suggests direct costs generally outweigh indirect costs in the management of traumatic dental injuries [17]. However, indirect costs may increase with complicated dental injuries which require a multidisciplinary approach resulting in numerous treatment appointments and a lack of access to treatment from health-care providers [17, 18]. The latter may be further compounded by the perceived lack of remuneration available for the management of dental trauma cases [19]. The increased cost to health service providers in the management of these cases has been highlighted [20]. To prevent the long-term burden and costs to patients, their parents, primary care givers and healthcare providers, strategies to prevent dental trauma in young children should be stressed and reinforced at an early age.

Prevention of Trauma

Despite the majority of traumatic dental injuries occurring as a result of physical trauma such as sports-related situations, the predication of such events is difficult. Strategies to prevent trauma to the dentition include the development of life skills

aimed at a personal and social level, anti-bullying and health policies within schools, the school provision of mouthguards and maintaining links with health services [21]. As highlighted, the presence of an increased overjet and inadequate lip coverage can predispose a young individual to potential traumatic dental injury to the permanent maxillary incisor teeth. Approaches to protect the teeth or early correction of the underlying malocclusion could be considered to reduce this risk of trauma.

Use of Mouthguards

Based on low-quality evidence, the use of mouthguards has been advocated to reduce the risk and severity of orofacial trauma [22, 23]. Quantitative analysis of several studies suggests the overall risk of trauma is 1.6–1.9 times more likely when mouthguards are not worn [22]. Intraorally, the material properties of mouthguards such as thickness and resilience are thought to absorb traumatic forces and reduce their transmission to the dentoalveolar complex [24]. In relation to this, variation in the performance of different mouthguards has been reported with non-laboratory-constructed mouthguards achieving poor results [25]. However, this dissipating “shock absorber” effect may also result in increased mobility and dental injuries of adjacent teeth as the forces are distributed over a wider surface area within the mouthguard [10]. Three types of mouthguards are commonly available: stock, mouth-formed and custom-made. Custom-made types made from ethylene vinyl acetate (EVA) co-polymer are recommended as they are better tolerated, allow for normal function such as breathing and speaking, are better retained within the mouth and reported to afford the most protection to the dentition [26]. The latter is supported by the findings of *in vitro* analysis [10].

Mouthguards can be worn when a child is either in the mixed or permanent dentition (Fig. 6.3). In a cohort of 7–8-year-olds, custom-made mouthguards were reported to be better tolerated compared to both stock and mouth-formed types [27]. Despite this, the retention of mouthguards and hence its overall effectiveness may be compromised during the mixed dentition due to growth and development of the jaws and further tooth eruption [28]. Anticipation of these changes should be considered and can be incorporated in the design of custom-made mouthguards [28].

Dentists have a unique role in educating young children and their parents who participate in contact sports regarding the importance of wearing mouthguards [29]. In addition, highlighting situations where trauma may likely occur may further encourage parents to encourage their children to wear mouthguards [27]. Despite the lack of prospective clinical evaluation of the effectiveness of mouthguards to reduce dental trauma [23], the use of mouthguards by young children participating in sporting activities is recommended.

Fig. 6.3 Custom-made mouthguard worn in the mixed dentition



Early Orthodontic Treatment

Previous authors have suggested early interceptive orthodontic treatment to reduce an increased overjet and hence reduce the potential risk of future trauma to the upper permanent incisor teeth [9, 11]. Early orthodontic treatment is potentially considered between 7 and 11 years of age (early adolescence). Reduction of an increased overjet forms the primary reason for early treatment as the highest prevalence of trauma is reported to occur between 11 and 15 years of age [9]. Both a simple upper removable appliance (Fig. 6.4) and a functional appliance (Fig. 6.5) can be utilised to reduce an increased overjet in the mixed dentition. Favourable compliance and wear of a functional appliance can result in efficient reduction of both overjet and overbite (Fig. 6.6). However, as these patients grow and continued dental development occurs, relapse of the corrected overjet is anticipated and a possible further course of treatment in adolescence warranted. In a Cochrane systematic review and meta-analysis, the effects of one-phase (adolescence) versus two-phase (early adolescence and adolescence) treatment of children with prominent teeth were compared. Although the overall quality of evidence was deemed to be low, there were no differences in the final occlusal result, but there was a significant reduction in the incidence of trauma to the upper permanent incisors in patients who underwent two-phase treatment [30]. Individual case selection is imperative when considering the early correction of an increased overjet. Patients should be well motivated with high levels of oral hygiene, and compliance as a negative experience of early treatment may preclude the success of future treatment [31]. The known risks of orthodontic treatment are well documented and may increase with long durations or multiple phases of treatment. However, in a young patient who is at risk of dental trauma, the potential benefits of early orthodontic treatment from a dental health and psychosocial development aspect may outweigh these apparent risks.

Fig. 6.4 An upper removable appliance used to retract the permanent maxillary incisors. The design incorporates an anterior bite plane to disclude the occlusion, an activated labial bow and Adam's cribs to retain the appliance

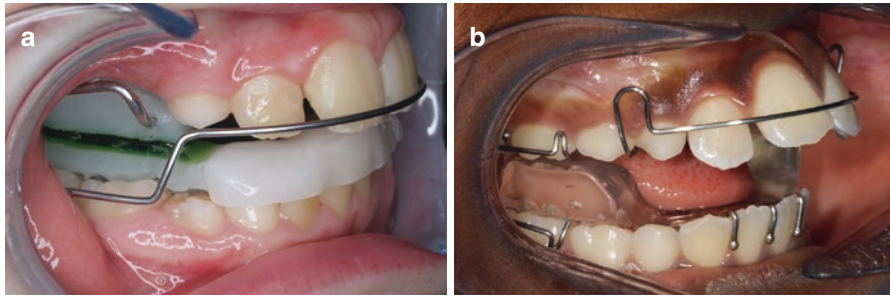
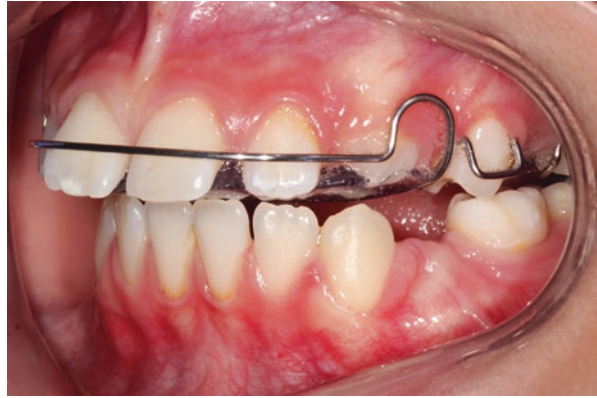


Fig. 6.5 Functional appliances that can be used in the mixed dentition to reduce an overjet: Balters bionator (a) and modified Clark Twin Block (b)

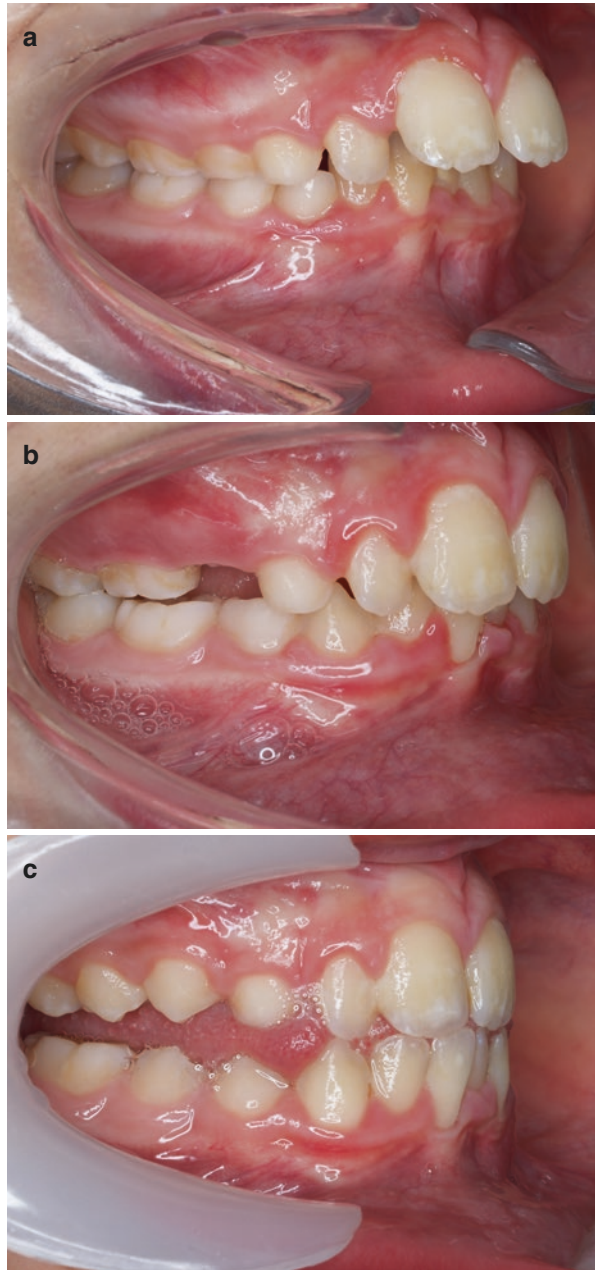
Management of Acute Traumatic Injuries in the Mixed Dentition

The mixed dentition stage is generally between 6 and 13 years of age. The permanent incisor teeth will be erupting or fully erupted with varying degrees of root development. Hertwig epithelial root sheath is present in immature teeth and plays an important role in root development. Odontoblasts on the pulpal side of the sheath are involved in the formation of dentine, and cementoblasts, fibroblasts and osteoblasts are involved in the formation of the periodontal membrane.

The aim in managing acute dental trauma in the mixed dentition is to:

1. Restore form.
2. Restore function.
3. Preserve pulp vitality.
4. Support continued root formation.
5. Improve self-esteem.
6. Promote long-term sustainable biological outcomes for the patient.

Fig. 6.6 Occlusal effects of a functional appliance worn in the mixed dentition. Pretreatment clinical appearance (a), progressive reduction of the increased overjet following full-time wear (b) and reduction of the increased overjet and establishment of lateral open bites (c)



A combination of the direction and force of an impact as well as the point of impact can result in two traumatic dental injuries that can affect the teeth: fractures and luxation injuries.

Fracture injuries can be categorised as:

- Uncomplicated crown fractures—enamel; enamel-dentine
- Complicated crown fractures—enamel-dentine with pulp exposure
- Crown-root fractures—with or without pulp exposure
- Root fractures—involving the cementum; can be horizontal or oblique

Luxation injuries can be categorised as:

- Concussion
- Subluxation
- Extrusion
- Lateral luxation
- Intrusion
- Avulsion

Injuries rarely occur in isolation, and it is possible for more than one type of injury to affect an individual tooth. Multiple injuries per tooth increase the risk of long-term sequelae.

The following account of the assessment and management of traumatic dental injuries in the young patient is an overview, and for a more comprehensive account, the reader is referred to the dental trauma guide: www.dentaltraumaguide.org.

History of the Traumatic Incident

It is important to obtain a detailed history of the event detailing how and when it took place. This will allow a picture to be built on what to expect when examining the patient but will also provide important information when discussing the prognosis with the patient and their parent(s). Where the injury has occurred is important in assessing whether a tetanus booster is necessary, e.g. near soil. It may be prudent to ascertain the tetanus vaccination history of the patient at this stage. Any loss of consciousness with signs of a head injury such as headache, amnesia, nausea or vomiting need to be taken seriously and referral to the accident and emergency unit arranged as a priority. Enquiring about previous traumas can be helpful to explain signs seen during the clinical examination.

Relevant medical history such as allergies, blood disorders, medication being taken and any degree of immunosuppression need to be noted before embarking on treatment. If the patient has arrived with an avulsed tooth, this should be placed in Hank's Balanced Salt Solution (HBSS) e.g. Save-a-tooth™ or milk whilst the history is being taken.

Sensibility testing can be useful in the long-term monitoring of traumatised teeth when attempting to assess pulp vitality. Bearing in mind that immature teeth can give

transient negative results, and there may be false-negative responses for up to 3 months after a traumatic dental injury, it is probably sensible to obtain these at the subsequent appointment when the patient is less emotional and anxious about their predicament.

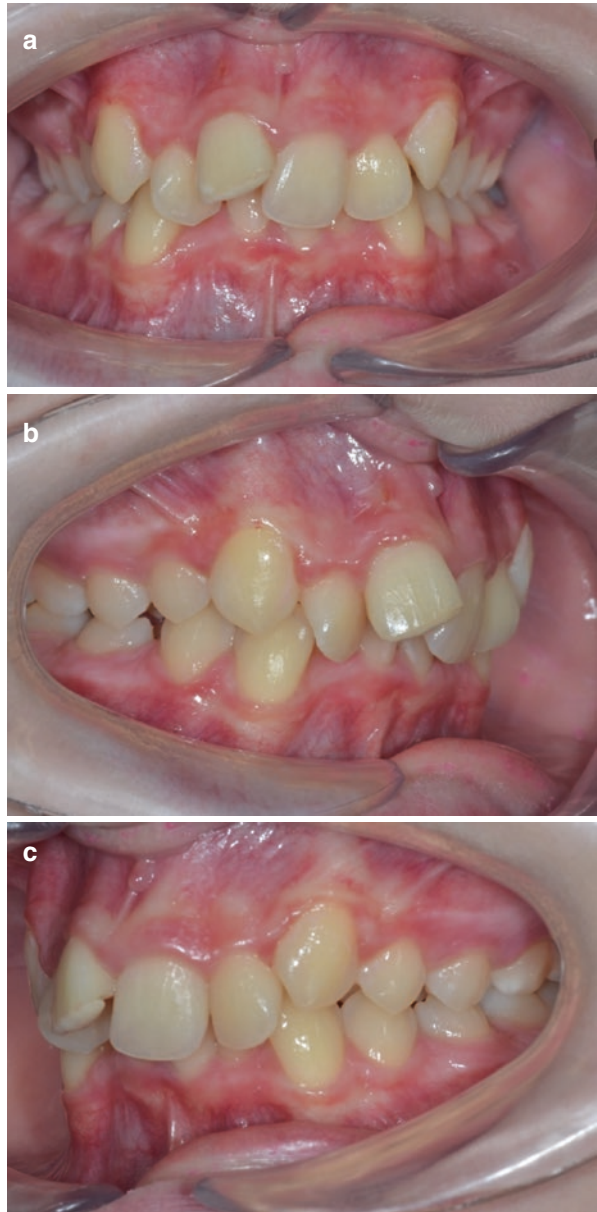
Radiographs should be obtained as required by the clinical presentation. Periapical radiographs are standard, but if there is a suspicion of a root fracture, an upper occlusal radiograph would help diagnose this. In the presence of a lip laceration and a fractured tooth where the fragment has not been identified, a soft tissue view should be obtained as well to eliminate the possibility of the fragment being embedded in the lip.

Photographs are an important and easy way of documenting the presentation of the patient. If a camera is not available, a line diagram outlining all of the soft tissue injuries can also help.

Fractures

- Uncomplicated crown fractures—enamel; enamel-dentine
Small fractures may be smoothed down. If restoration is necessary, then direct build up with composite resin as soon as possible is indicated. If time is a problem, placing a temporary ‘bandage’ with glass ionomer is acceptable (Fig. 6.7).
- Complicated crown fractures—enamel-dentine with pulp exposure
When the pulp is exposed, the aim of management is to try and preserve the vitality of the pulp to support continued root formation. A Cvek pulpotomy, carried out under local anaesthesia and rubber dam, is indicated (Fig. 6.8). Once pulp therapy has been completed, the tooth can be built up with composite, or if the tooth fragment is available and can be relocated with ease, it should be reattached (Fig. 6.9). Again, if time is a problem, placing a temporary ‘bandage’ with glass ionomer is acceptable whilst a longer appointment is arranged.
- Crown-root fractures—with or without pulp exposure
These may be difficult to restore and have a guarded prognosis. However, the long-term consequences of the loss of such teeth should be considered before committing them to be extracted. Treatment options for uncomplicated crown-root fractures include reattachment of the coronal fragment, build up with composite resin, surgical extrusion or orthodontic extrusion. Treatment options for complicated crown-root fractures are as above with consideration of pulp management either with a Cvek pulpotomy or root canal treatment before restoration of the tooth. The limiting factor for root canal treatment of such teeth will be whether isolation with rubber dam is possible or not.
- Root fractures—involving cementum; can be horizontal or oblique
These may be located in the apical, mid or cervical third of the root with the latter having the poorest prognosis. Depending on the presence of displacement of the

Fig. 6.7 Uncomplicated enamel-dentine fracture of the UR1 restored with a glass ionomer “bandage” (a–c)



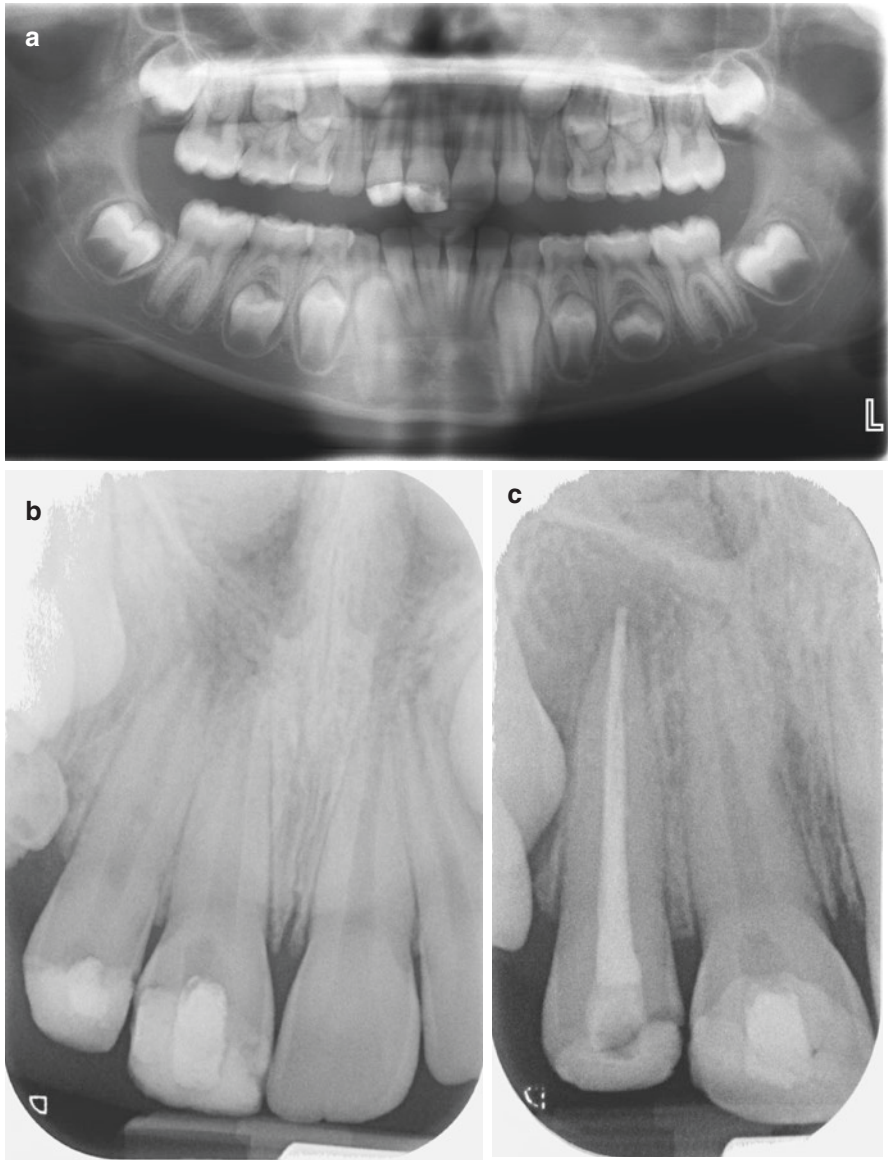


Fig. 6.8 Complicated enamel-dentine fracture involving the UR1 and UR2 in an 8-year-old. Pretreatment radiographic appearance (a), Cvek pulpotomy of the UR1 (b) and apexification using mineral trioxide aggregate (MTA), backfill obturation using gutta percha and coronal seal of the UR2 (c)

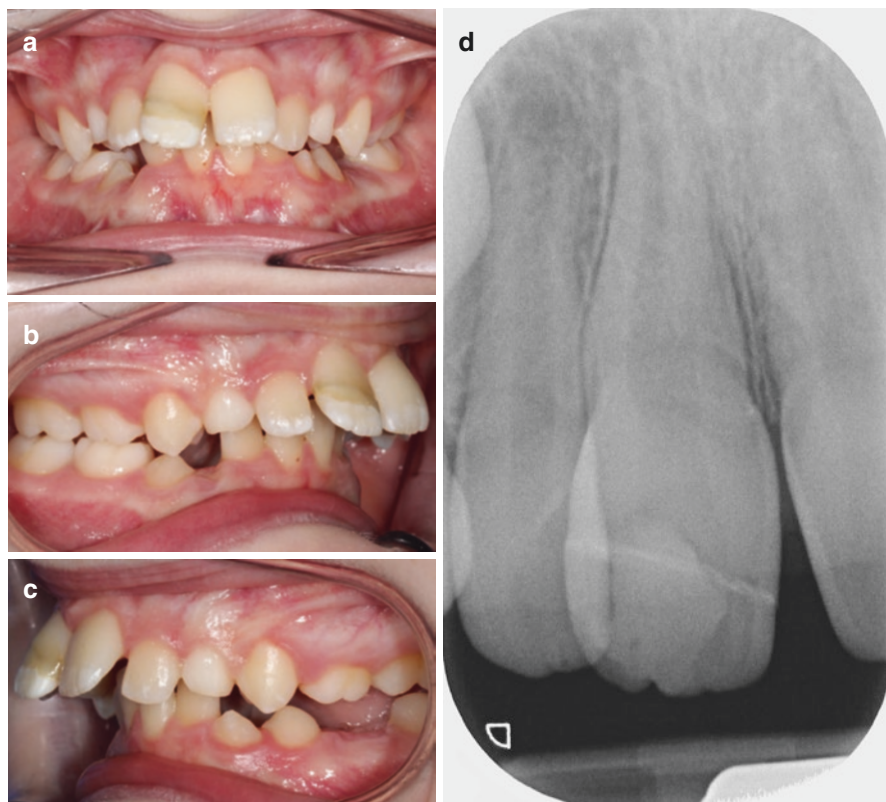


Fig. 6.9 Complicated enamel-dentine fracture involving the UR1. Post-treatment clinical (a–c) and radiographic appearance (d) following Cvek pulpotomy and reattachment of the tooth fragment using composite

coronal fragment, the crown may be longer than the adjacent tooth, be mobile and interfere with the occlusion. Where possible, the coronal fragment should be repositioned under local anaesthesia and splinted flexibly for 4 weeks in the apical and mid-third root fractures and for 4 months in cervical third root fractures. As already indicated, the latter have a poor prognosis and incorporation into an orthodontic plan, or retaining the root as a space maintainer should be considered.

Luxation Injuries

- Concussion
Teeth with concussion will be slightly tender to touch but demonstrate no increase in mobility. Patients should be reassured that they do not require any active treatment.

- **Subluxation**
Teeth with subluxation are tender to touch and exhibit slightly increased mobility. As above, patient reassurance is indicated, but flexible splinting can sometimes be reassuring for anxious patients and may be used if patients demonstrate considerable distress.
- **Extrusion**
Extruded teeth appear slightly longer than the adjacent teeth and are mobile and there may be an occlusal interference. Digital repositioning under local anaesthetic and flexible splinting for 2 weeks is indicated.
- **Lateral luxation**
Teeth may be displaced palatally (most common) or labially and are locked into position due to the presence of a dentoalveolar fracture. Palatally displaced teeth may result in an occlusal interference. Digital repositioning under local anaesthetic and flexible splinting for 4 weeks is indicated.
- **Intrusion**
Intruded teeth are displaced apically into the socket, and the crown appears shorter than the adjacent teeth. In such situations it is possible to think that the crown has been fractured. As a result, the tooth is locked into position, and if tapped, a high-pitched ankylotic sound is elicited. Depending on the degree of intrusion, the tooth may be monitored for spontaneous eruption, be orthodontically or digitally repositioned. Teeth repositioned digitally should be splinted flexibly for 4 weeks. The following tables are the guidance provided by the International Association for Dental Traumatology (www.dentaltraumaguide.org) (Tables 6.1 and 6.2).

Table 6.1 Recommended management of intrusion injuries with teeth with an open apex

	Degree of intrusion	Repositioning		
		Spontaneous	Orthodontic	Digital
Open apex	Up to 7 mm	✓		
	More than 7 mm		✓	✓

Table 6.2 Recommended management of intrusion injuries with teeth with a closed apex

	Degree of intrusion	Repositioning		
		Spontaneous	Orthodontic	Digital
Closed Apex	Up to 3 mm	✓		
	3–7 mm		✓	✓
	>7 mm			✓

Table 6.3 Summary of recommended management of avulsed teeth with differing degrees of root formation and extraoral dry time

Root development	Extra oral dry time	Root canal treatment	Splinting time	Prognosis
Open apex	<60 min	Aim for revascularisation	2 weeks	Fair
Open apex	>60 min	Root canal treatment (extraoral or asap)	4 weeks	Poor
Closed apex	<60 min	Root canal treatment within 7–10 days	2 weeks	Fair
Closed apex	>60 min	Root canal treatment (extraoral or asap)	4 weeks	Poor

- Avulsion

This is the total displacement of a tooth out of its socket. Immediate replantation of the tooth confers the best prognosis in the long term. If a tooth with an immature root is replanted within 5 min, monitoring for revascularisation should be adopted. In the same scenario for a mature tooth, root canal treatment is indicated within 7–10 days. If this is not possible, the ideal storage medium is Hank's Balanced Salt Solution (HBSS) or, if this is not available, milk. The following table summarises the different treatment strategies for delayed replantation of teeth with open and closed apices (Table 6.3).

Complications

The following complications may arise following traumatic dental injuries in the mixed dentition:

- Pulp necrosis ± discolouration
- Pulp canal obliteration ± discolouration
- Root resorption
- Ankylosis
- Infraocclusion (Fig. 6.10)

Fig. 6.10 Intrusion injury of the UR1 in an 8-year-old. Failed orthodontic extrusion was attempted 8–9 months post-injury. Radiographically, the UR1 has undergone pulp canal obliteration and clinically is ankylosed as demonstrated by the lack of vertical development in relation to the UL1



Follow-Up

Long-term monitoring of all traumatised teeth is essential to diagnose problems early to allow interception to reduce the sequelae above.

Root canal treatment should be carried out if there are two signs or symptoms of pulp necrosis such as:

- Pain
- Swelling
- Sinus

- Discolouration
- Increased mobility
- Negative sensibility tests (weak sign)
- Periapical radiolucency on radiograph

Sensibility test results should not be relied upon in the absence of other compelling signs or symptoms. For more information, visit www.dentaltraumaguide.org.

Orthodontic Movement of Traumatized Teeth

Depending on the severity of the dentoalveolar injury, future orthodontic movement of previously traumatized permanent maxillary incisors is not without possible risk. It is thought that the application of orthodontic forces from either removable or fixed appliances may increase the susceptibility to root resorption and non-vitality. The evidence to support these assumptions is based on low-quality studies lacking standardised outcomes and heterogeneous trauma samples. However, the incidence of loss of vitality of permanent maxillary incisors with a history of trauma following application of orthodontic forces from fixed appliances is reported to range between 7.3 and 10.4% [12, 32]. The risk of pulpal necrosis is higher in lateral incisors and incisors with a history of intrusion, lateral luxation and extrusion type injuries [12]. It is well reported that comprehensive orthodontic treatment may result in root resorption. However, whether a history of trauma increases this risk of root resorption in permanent maxillary incisors is subject to debate. Retrospective analysis suggests that a history of trauma is a risk factor of root resorption in permanent maxillary incisors [33]. In contrast, compared to an uninjured maxillary incisor in the same patient, the contralateral traumatized permanent maxillary incisor appears not to have a greater tendency for root resorption [34]. Systematic review of the available evidence suggests there is little evidence that a history of previous trauma to the incisors increases the risk of root resorption during orthodontic treatment [35]. Importantly, if there is evidence of pre-existing root resorption in traumatized permanent maxillary incisors prior to orthodontic treatment, then these teeth appear to be more prone to root resorption during treatment [34].

Orthodontic treatment is often performed in patients with a history of trauma to the dentition (Fig. 6.11). A period of observation/monitoring is often recommended prior to the application of orthodontic forces. The duration of this observation period varies in relation to the severity of the traumatic injury. The following observation periods prior to orthodontic tooth movement have been proposed [31, 36]:

- Crown and crown-root fractures without pulpal involvement (3 months)
- Crown and crown-root fractures with pulpal involvement (3 months after coronal pulpotomy following radiographic signs of a hard tissue barrier)
- Root fractures (12–24 months)

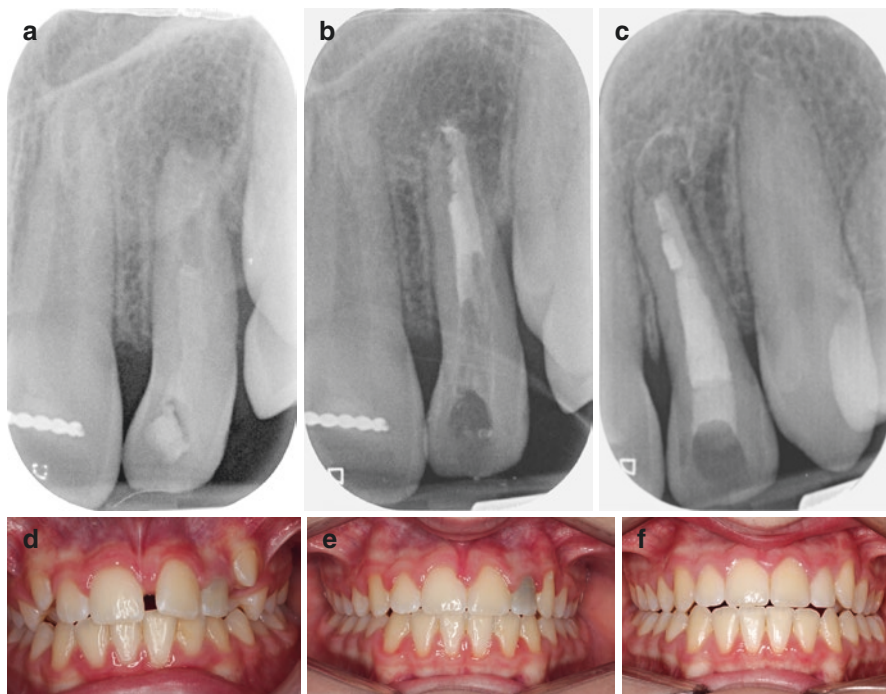


Fig. 6.11 Comprehensive orthodontic treatment in a 12-year-old with a mild Class III malocclusion complicated by the previously traumatised UL2 and crowding. At age 10, the UL2 was traumatised (a). Apexification of the immature UL2 was undertaken using Mineral Trioxide Aggregate (MTA) (b), followed by backfill obturation using gutta percha and placement of a coronal seal (c). Pretreatment clinical appearance of the malocclusion (d). Post-treatment clinical appearance following orthodontic alignment using upper and lower fixed appliances (e). The appearance of the discoloured UL2 was improved with inside-outside (nonvital) bleaching technique (f)

- Minor injuries including concussion, subluxation, extrusion, minor lateral luxation (3 months)
- Moderate/severe injuries including avulsion and replantation and moderate/severe lateral luxation (12 months if ankylosis not present)
- Immature traumatised teeth (await radiographic signs of continued root development)

Orthodontic treatment may incorporate traumatised teeth with a poor long-term prognosis in the extraction pattern [37] (Fig. 6.12), or they may be maintained within the dental arches aligned and spacing optimised for a possible future prosthetic replacement (Fig. 6.13). The former obviates the need for prosthetic replacement teeth. Ideally, light forces of short duration should be applied and, if possible, avoidance of bonding the tooth, hence limiting exposure to orthodontic forces which could accelerate its loss. During orthodontic treatment, permanent maxillary incisors with a history of trauma should be closely monitored. Regular clinical and

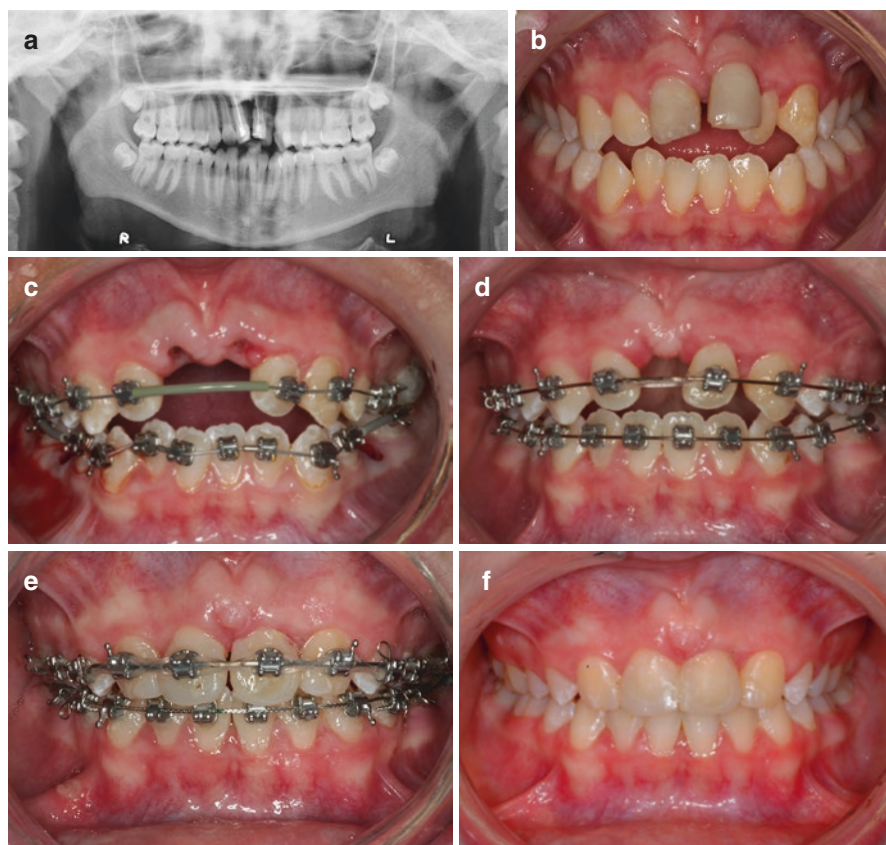


Fig. 6.12 Comprehensive orthodontic treatment in a 13-year-old with a Class I malocclusion complicated by an increased overjet, previously traumatised UL1 and UL1 and crowding. Pretreatment radiographic (a) and clinical (b) appearance. The treatment plan involved the extraction of the UR1, UL1 and loss of two lower premolar units. As part of the treatment, the UR2, UL2, UR3 and UL3 were to be substituted into the position of the UR1, UL1, UL2 and UR2, respectively, and aesthetically modified with composite material. Extraction of the UR1, UL1 and placement of the upper and lower fixed appliances (c). Elastomeric chain used to mesialise both the UR1 and UL1 (d). Composite modification of the UR3, UR2, UL2, UL3 and continued space closure (e). Post-treatment clinical appearance (f)

radiographic assessment of pulpal health and root status including signs of root resorption should be undertaken. Prior to any form of orthodontic treatment, it is essential that an accurate history of the traumatic incident is taken, supported by clinical examination and appropriate special investigations. It is imperative that any evidence of pre-existing root resorption is identified and recorded (Fig. 6.14). A multidisciplinary opinion may also be beneficial in these cases. Patients and their parents should be consented and the possible risks associated with orthodontic movement of traumatised permanent maxillary incisor teeth explained [31].

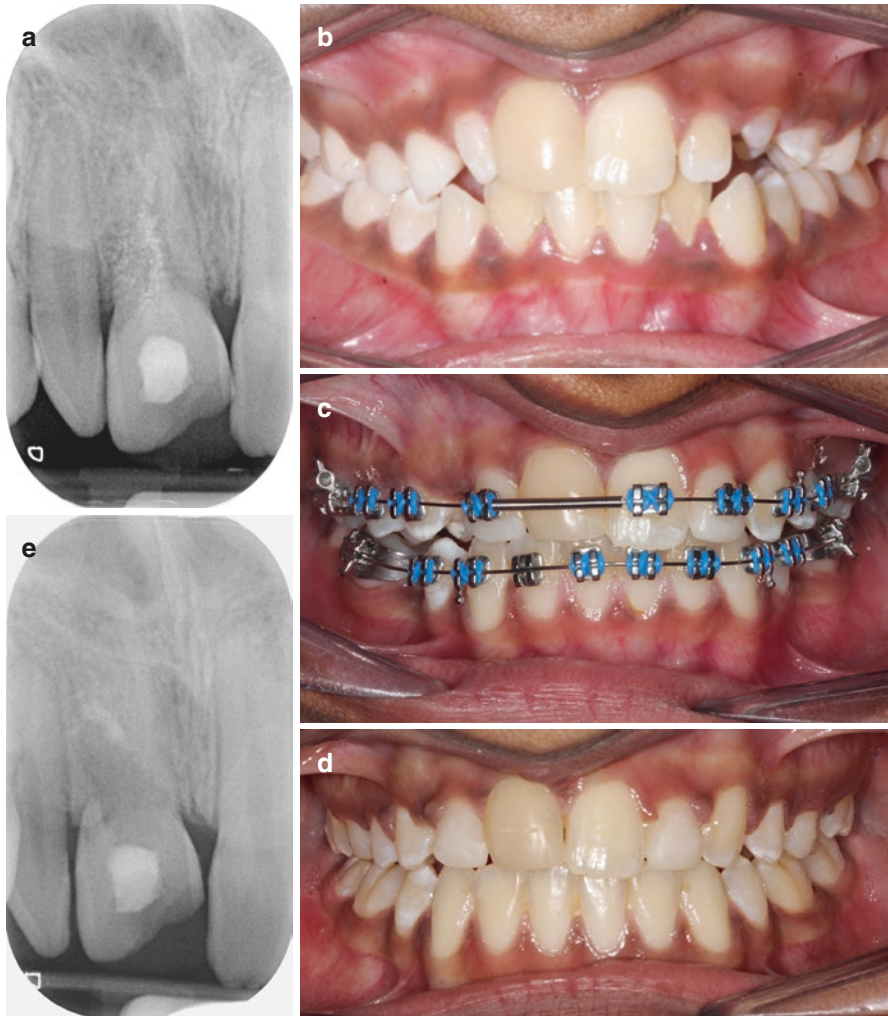


Fig. 6.13 Comprehensive orthodontic treatment in a 12-year-old with a Class I malocclusion complicated by the previously avulsed and reimplanted UR1, ectopic UR3, diminutive UR2 and UL2 and crowding. Pretreatment radiographic (a) and clinical appearance (b). The reimplanted UR1 had undergone external root resorption and ankylosis. The enamel-dentine fracture was restored with a composite veneer. The treatment plan involved the extraction of the unfavourably positioned UR3, UL5, LL5 and LR5. As part of the treatment, the UR1 was to be maintained, and the UR2 and UL2 built up with composite material. As part of the fixed appliance mechanics, the ankylosed UR1 was not bonded as it was fairly well aligned and to avoid unwanted reciprocal anchorage loss (c). Post-treatment clinical (d) and radiographic appearance (e). The UR2 and UL2 were built up with composite to improve their aesthetic appearance

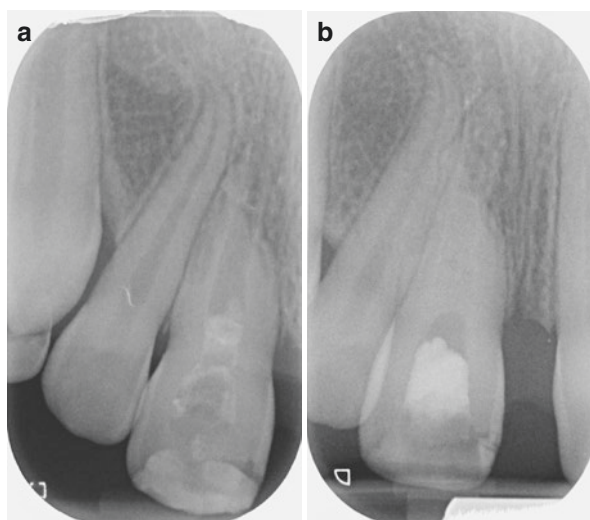


Fig. 6.14 Enamel-dentine fracture and avulsion injury of the UR1 in a 9-year-old. Periapical assessment of the UR1 which has undergone progressive external root resorption following reimplantation (a) and at 12-month follow-up (b)

Discussion

Early orthodontic treatment to reduce an increased overjet and subsequently reduce the risk of trauma to the permanent maxillary incisors may be beneficial. All potential risks such as possible non-vitality of teeth, root resorption and future prosthetic replacement of teeth should be outlined and documented during the consenting process. Patients and parents should be informed of the possible need of further orthodontic treatment in the permanent dentition. To ensure an optimal treatment outcome, a multidisciplinary approach should be adopted. Additional preventative measures to reduce the risk of dentoalveolar trauma to the permanent maxillary incisors in young children should be promoted by clinicians and dentists. This includes educating parents and children regarding environments or situations where trauma is likely to occur and to stress the importance of wearing mouthguards when participating in activities, which may predispose the permanent maxillary incisors to trauma.

Acknowledgments The authors would like to express their gratitude to Dr Padhraig Fleming, Reader/Honorary Consultant, Queen Mary University of London and Mrs Shruti Patel, Consultant Orthodontist, King's College Hospital NHS Foundation Trust for providing the images used in Figure 6.12 and Figure 6.13, respectively. We would also like to thank our consultant colleagues in the Paediatric Dental department at King's College Hospital NHS Foundation Trust.

References

1. Andersson L. Epidemiology of traumatic dental injuries. *J Endod.* 2013;39:S2–5.
2. Marcenes W, Murray S. Social deprivation and traumatic dental injuries among 14-year-old schoolchildren in Newham, London. *Dent Traumatol.* 2001;17:17–21.
3. Oldin A, Lundgren J, Nilsson M, Norén JG, Robertson A. Traumatic dental injuries among children aged 0-17 years in the BITA study – a longitudinal Swedish multicenter study. *Dent Traumatol.* 2015;31:9–17.
4. Traebert J, Almeida IC, Marcenes W. Etiology of traumatic dental injuries in 11 to 13-year-old schoolchildren. *Oral Health Prev Dent.* 2003;1:317–23.
5. Burden DJ. An investigation of the association between overjet size, lip coverage, and traumatic injury to maxillary incisors. *Eur J Orthod.* 1995;17:513–7.
6. Glendor U. Epidemiology of traumatic dental injuries—a 12 year review of the literature. *Dent Traumatol.* 2008;24:603–11.
7. Paiva PC, Paiva HN, Oliveira Filho PM, Lamounier JA, Ferreira RC, Ferreira EF, Zarzar PM. Prevalence of traumatic dental injuries and its association with binge drinking among 12-year-olds: a population-based study. *Int J Paediatr Dent.* 2015;25:239–47.
8. O'Brien M. Children's dental health in the United Kingdom 1993. In Report of Dental Survey, Office of Population Censuses and Surveys. London: Her Majesty's Stationery Office; 1994.
9. Bauss O, Röbling J, Schweska-Polly R. Prevalence of traumatic injuries to the permanent incisors in candidates for orthodontic treatment. *Dent Traumatol.* 2004;20:61–6.
10. Johnston T, Messer LB. An in vitro study of the efficacy of mouthguard protection for dento-alveolar injuries in deciduous and mixed dentitions. *Endod Dent Traumatol.* 1996;12:277–85.
11. Brin I, Ben-Bassat Y, Heling I, Brezniak N. Profile of an orthodontic patient at risk of dental trauma. *Endod Dent Traumatol.* 2000;16:111–5.
12. Bauss O, Freitag S, Röbling J, Rahman A. Influence of overjet and lip coverage on the prevalence and severity of incisor trauma. *J Orofac Orthop.* 2008;69:402–10.
13. Nguyen QV, Bezemer PD, Habets L, Pahl-Andersen B. A systematic review of the relationship between overjet size and traumatic dental injuries. *Eur J Orthod.* 1999;21:503–15.
14. Ghose LJ, Baghdady VS, Enke H. Relation of traumatized permanent anterior teeth to occlusion and lip condition. *Community Dent Oral Epidemiol.* 1980;8:381–4.
15. Cortes MI, Marcenes W, Sheiham A. Impact of traumatic injuries to the permanent teeth on the oral health-related quality of life in 12-14-year-old children. *Community Dent Oral Epidemiol.* 2002;30:193–8.
16. Porritt JM, Rodd HD, Ruth Baker S. Quality of life impacts following childhood dento-alveolar trauma. *Dent Traumatol.* 2011;27:2–9.
17. Glendor U, Jonsson D, Halling A, Lindqvist K. Direct and indirect costs of dental trauma in Sweden: a 2-year prospective study of children and adolescents. *Community Dent Oral Epidemiol.* 2001;29:150–60.
18. Glendor U, Halling A, Bodin L, Andersson L, Nygren A, Karlsson G, Kouckeki B. Direct and indirect time spent on care of dental trauma: a 2-year prospective study of children and adolescents. *Endod Dent Traumatol.* 2000;16:16–23.
19. Jackson NG, Waterhouse PJ, Maguire A. Management of dental trauma in primary care: a postal survey of general dental practitioners. *Br Dent J.* 2005;198:293–7.
20. Borum MK, Andreassen JO. Therapeutic and economic implications of traumatic dental injuries in Denmark: an estimate based on 7549 patients treated at a major trauma centre. *Int J Paediatr Dent.* 2001;11:249–58.
21. Sheiham A, Watt RG. The common risk factor approach: a rational basis for promoting oral health. *Community Dent Oral Epidemiol.* 2000;28:399–406.
22. Knapik JJ, Marshall SW, Lee RB, Darakjy SS, Jones SB, Mitchener TA, delaCruz GG, Jones BH. Mouthguards in sport activities : history, physical properties and injury prevention effectiveness. *Sports Med.* 2007;37:117–44.

23. Maeda Y, Kumamoto D, Yagi K, Ikebe K. Effectiveness and fabrication of mouthguards. *Dent Traumatol.* 2009;25:556–64.
24. Oikarinen KS, Salonen MA, Korhonen J. Comparison of the guarding capacities of mouth protectors. *Endod Dent Traumatol.* 1993;9:115–9.
25. Hoffmann J, Alfter G, Rudolph NK, Göz G. Experimental comparative study of various mouthguards. *Endod Dent Traumatol.* 1999;15:157–63.
26. Newsome PR, Tran DC, Cooke MS. The role of the mouthguard in the prevention of sports-related dental injuries: a review. *Int J Paediatr Dent.* 2001;11:396–404.
27. Walker J, Jakobsen J, Brown S. Attitudes concerning mouthguard use in 7- to 8-year-old children. *ASDC J Dent Child.* 2002;69:207–11.
28. Croll TP, Castaldi CR. Custom sports mouthguard modified for orthodontic patients and children in the transitional dentition. *Pediatr Dent.* 2004;26:417–20.
29. Ferrari CH, Ferreria de Medeiros JM. Dental trauma and level of information: mouthguard use in different contact sports. *Dent Traumatol.* 2002;18:144–7.
30. Thiruvengkatachari B, Harrison JE, Worthington HV, O'Brien KD. Orthodontic treatment for prominent upper front teeth (Class II malocclusion) in children. *Cochrane Database Syst Rev.* 2013;(11):CD003452.
31. Kindelan SA, Day PF, Kindelan JD, Spencer JR, Duggal MS. Dental trauma: an overview of its influence on the management of orthodontic treatment. Part 1. *J Orthod.* 2008;35:68–78.
32. Brin I, Ben-Bassat Y, Heling I, Engelberg A. The influence of orthodontic treatment on previously traumatized permanent incisors. *Eur J Orthod.* 1991;13:372–7.
33. Linge L, Linge BO. Patient characteristics and treatment variables associated with apical root resorption during orthodontic treatment. *Am J Orthod Dentofacial Orthop.* 1991;99:35–43.
34. Malmgren O, Goldson L, Hill C, Orwin A, Petrini L, Lundberg M. Root resorption after orthodontic treatment of traumatized teeth. *Am J Orthod.* 1982;82:487–91.
35. Weltman B, Vig KW, Fields HW, Shanker S, Kaizar EE. Root resorption associated with orthodontic tooth movement: a systematic review. *Am J Orthod Dentofacial Orthop.* 2010;137:462–76.
36. Malmgren O, Malmgren B, Goldson L. Orthodontic management of the traumatised dentition. In: Andreasen JO, Andreasen FM, Andersson L, editors. *Textbook and color atlas of traumatic injuries to the teeth.* 4th ed. Oxford: Blackwell Munksgaard; 2007. p. 669–715.
37. Fleming PS, Seehra J, Dibiase AT. Combined orthodontic-restorative management of maxillary central incisors lost following traumatic injury: a case report. *Orthodontics (Chic.).* 2011;12:242–51.

Shruti Patel

Abstract

Any significant delay in eruption of the maxillary central incisor teeth needs careful management as presenting patients are usually young and may not have had any previous dental experience. Treatment will depend on many variables including the age and compliance of the patient, aetiology of impaction, position of the impacted tooth, the potential need to recreate space, appropriate surgical intervention and possible guided tooth eruption with orthodontic traction. Following clinical and radiographic assessment, an accurate diagnosis of the problem is required. A favourable outcome is achieved in the majority of cases, although alignment of dilacerated incisors and those impacted high in the alveolus are more challenging. Ideally, by the time an individual starts secondary school, both upper central and lateral incisors should have erupted, affording dental, functional, aesthetic and psychosocial benefits to the patient. Specific clinical guidelines for the management of impacted maxillary incisors are available in the UK (https://www.rcseng.ac.uk/fds/publications-clinical_guidelines/clinical_guidelines/documents/ManMaxIncisors2010.pdf).

Introduction

The maxillary central incisors are the most prominent teeth in a patient's smile and are usually the most visible during smiling and speech (Fig. 7.1). Hence, their eruption and position can have a major impact on dental and facial aesthetics in patients. Failure of eruption can be considered to be unattractive and may influence self-esteem and confidence in developing social relationships with others [1]. Due to the

S. Patel
King's College Hospital NHS Foundation Trust, London, UK



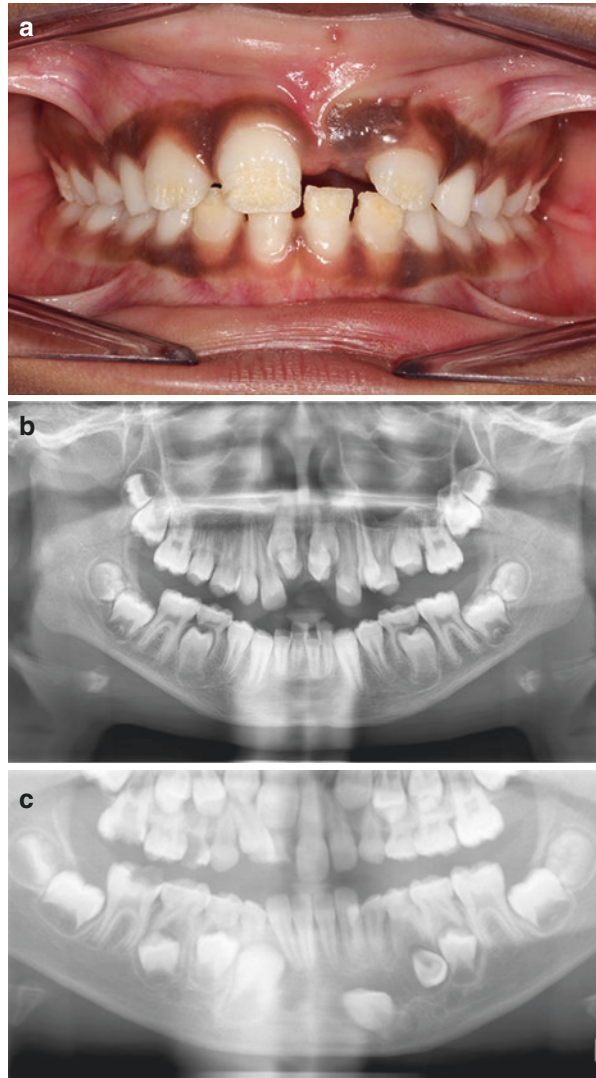
Fig. 7.1 The importance of maxillary central incisor teeth in facial aesthetics

increasing use of the Internet and social media, young patients have greater awareness of facial attractiveness and dental appearance. Children of this age are often targets for teasing and bullying by their peers. A prevalence of bullying in 12.8% of adolescents with a malocclusion aged between 10 and 14 has been reported [2]. As many patients presenting with this problem are relatively young, early identification and management is recommended to optimise a favourable outcome.

Aetiology and Prevalence

The maxillary central incisor is the third-most commonly impacted tooth in Caucasians, after third molars and maxillary canine teeth. Unerupted incisors present more frequently in males than females and are more common when there are other inherited dental anomalies, such as enamel hypoplasia, hyperdontia and other ectopic teeth [3] (Fig. 7.2). Failure of eruption of maxillary central incisors can be grouped according to whether hereditary or environmental factors are causative. By far the most common hereditary factor is obstruction secondary to supernumerary teeth, especially if tuberculate in morphology. Other hereditary, less frequent causes of failure of eruption include the presence of odontomes, abnormal tooth/tissue ratio, generalised delayed eruption, gingival fibromatosis, cleft lip and palate and cleidocranial dysostosis. Environmental aetiological factors include trauma, early extraction or loss of deciduous teeth with associated loss of space, retained deciduous teeth, cystic formation,

Fig. 7.2 Aetiology. Unerupted teeth present more frequently when other dental anomalies are present. (a) Enamel hypoplasia. (b) Supernumerary teeth. (c) Other ectopic teeth



endocrine abnormalities and bone pathology [4]. As calcification of the maxillary central incisors commences at 3–4 months after birth, and calcification of the crown continues until on average, 4.5 years of age, any significant trauma to the deciduous central incisors may disturb normal crown and root development of the permanent successor, which is positioned in close proximity. The extent of disturbance to the developing tooth will depend on the age of the child at the time of the trauma and the severity and direction of the traumatic impact. As a result, dilaceration may develop, which is defined as a developmental disturbance in the shape of a tooth, such as an angulation, curve or sharp bend in the crown or root. Any significant dilaceration will potentially disturb the normal eruption pathway of the maxillary central incisor.

Diagnosis

Provisional diagnosis of impaction and failure of eruption of the maxillary central incisor tooth can be made when:

- The contralateral central incisor tooth has erupted more than 6 months previously.
- The upper central incisor teeth remain unerupted when the upper lateral incisors have erupted.
- Both maxillary central incisors remain unerupted when the lower central incisors have erupted more than 12 months previously.

Clinical Examination

As most impactions are asymptomatic, the patient may not be aware of the problem. Referral will result from either a chance finding following a routine appointment or as a result of parental concern. For younger patients, this may be their first visit to see a specialist, and hence it is important that they have a positive experience and remain relaxed. Future treatment can be complex and uncomfortable to a degree, and patient compliance will be needed.

A comprehensive medical history should be taken as part of the clinical examination because a number of systemic conditions including prematurity, low birth weight, endocrine disorders, vitamin D deficiency, anaemia or renal disease can all lead to delayed tooth eruption. A detailed trauma history is also required. Trauma to the teeth at a young age is common, and fortunately, in the majority of cases these injuries are minor. Parents may not remember specific accidents as they are generally accepted as part of the normal scrapes and bumps that occur whilst growing up. Mentioning bicycle accidents, falling from chairs, trees or slipping by the side of the swimming pool can often help jog a memory. Should there be any concerns regarding the reported cause of traumatic injury, child safeguarding protocols should be followed. Relevant answers should be carefully documented with the approximate dates of the traumatic incidences.

A thorough intraoral examination should then be undertaken. This should include assessment of teeth present, with reference to any retained deciduous teeth which should have normally exfoliated, palpation of the buccal and palatal tissues, angulation and inclination of the adjacent teeth and measurement of the available space present for potential eruption of the unerupted incisor (Fig. 7.3). The amount of keratinised gingivae in the anterior region should be assessed, as well as the oral hygiene and general dental condition. Such a comprehensive examination may give an insight into how cooperative the young patient will be to allow further investigations, such as radiographs, intraoral photographs and impressions.

Assessing the angulation and inclination of adjacent teeth is generally helpful in determining the position of the unerupted incisor. If the impaction occurs close to eruption, the adjacent teeth may be affected and tip mesially. However, if the impaction is very high, the adjacent teeth may not be affected at all. Space available for the unerupted tooth should be measured. If space has been lost as a result of crowding,

Fig. 7.3 Varying degrees of space loss at presentation



or mesial drift of the lateral incisors, spontaneous eruption of the impacted central incisor is unlikely to occur, and any treatment plan will need to recreate space first.

Radiographic Examination

Radiographic examination is necessary to accurately locate the impacted tooth and assist in confirming the aetiology of the failure of eruption. Each case should be evaluated on an individual basis when considering the indications for standard radiographs and/or three-dimensional imaging.

A request for any radiographic imaging must be clinically justified for every patient, but more so for younger patients, where the radiation dosage should be kept as low as reasonably practicable. As single radiographs can only offer a two-dimensional view, more than one radiograph will be needed to locate the unerupted tooth. A range of radiographic views are available, which include the following:

- Upper standard occlusal radiograph
- Periapical radiograph
- Dental panoramic radiograph
- Lateral cephalometric radiograph
- Cone beam computed tomography

Upper Standard Occlusal Radiograph

This radiograph shows the maxillary incisor region clearly and is taken with the X-ray tube angled at 60° to the occlusal plane. It is relatively comfortable for the young patient to tolerate the position of the intraoral film and offers good clarity of the unerupted incisor. However, due to the angulation of the X-ray beam, the roots of the upper central incisor teeth will appear ‘shortened’, and accurate diagnosis of the vertical position of the impacted tooth in the alveolus will not be possible from this view alone (Fig. 7.4a).

Periapical Radiograph

Periapical radiographs are taken with the X-ray tube angled so that the X-rays pass through the minimum of surrounding tissue, which results in a clear image of the unerupted incisor with minimal distortion. The tooth follicle, cystic change, angulation of the incisor, morphology of adjacent roots and any possible obstruction will be visible. If the obstruction is due to the presence of a supernumerary tooth, then it may be possible to identify the type from this view. As with the upper standard occlusal radiograph, it is not possible to accurately locate the unerupted incisor in the vertical plane or buccolingual position. However, due to the improved clarity of view, and a lower radiation dose, it may be beneficial to request this radiograph if a patient can tolerate the positioning of the intraoral film (Fig. 7.4b).

Dental Panoramic Radiograph

This is the most commonly requested radiograph when undertaking a full orthodontic assessment. Whilst it lacks detail in the clarity of image, it supplements the clinical examination and provides an excellent overview of the presence, position and morphology of all the teeth, both erupted and unerupted (Fig. 7.2b, c). The height of the impaction will be evident, although due to the fact that the focal trough is relatively narrow in the incisor region, the anterior region has the least clarity. In addition, superimposition of the cervical spine also causes some blurring in this area.

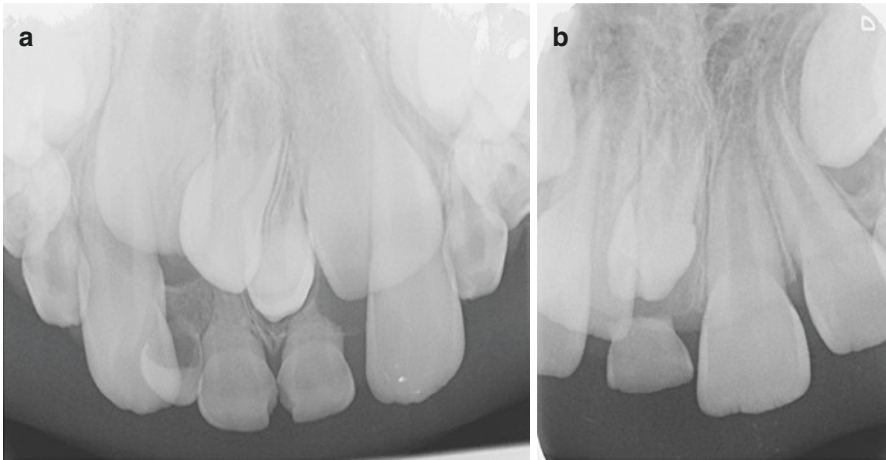
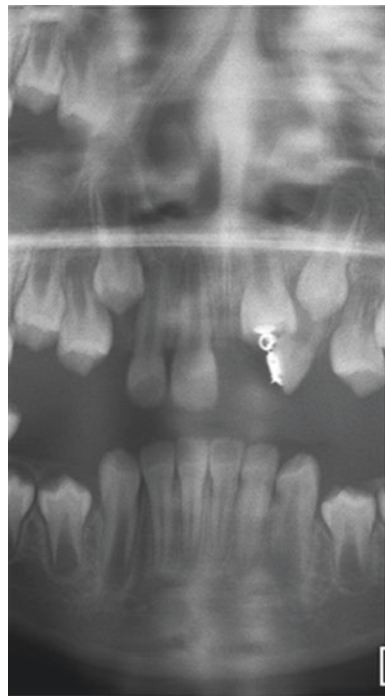


Fig. 7.4 Radiographic appearance of unerupted maxillary incisors. (a) Upper anterior occlusal view showing two impeding supernumerary teeth. (b) Periapical view

Fig. 7.5 Anterior section panoramic view of an impacted UL1 at age 7 years and 8 months approximately 6 months following exposure and bonding (see Fig. 7.6 for imaging relating to the original position of this tooth)



Newer panoramic machines have the advantage of reducing radiation dosage as settings allow field limitation and appropriate collimation of the images. ‘Dentition only’ views can result in a reduction in radiation dosage by up to 50%. Anterior section panoramic views extending from canine to canine can also be requested, further reducing the radiation dose (Fig. 7.5).

Lateral Cephalometric Radiograph

Cephalometric images may be requested in conjunction with a dental panoramic radiograph and upper standard occlusal view if information is needed to view the unerupted tooth in the sagittal plane (Fig. 7.6a). Using a combination of radiographs taken from different angles will allow a three-dimensional image of the unerupted tooth to be visualised and give further information on the height of the impaction and buccolingual inclination of the crown and root of the tooth.

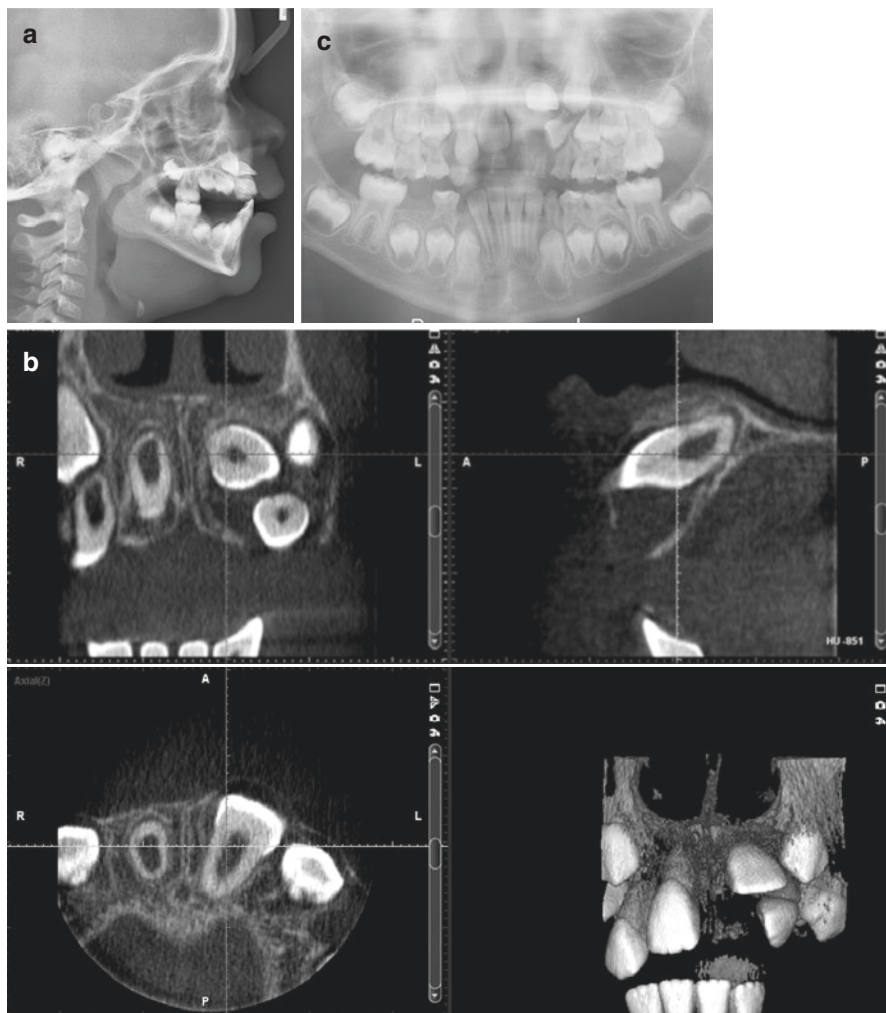


Fig. 7.6 Pretreatment imaging for the unerupted maxillary incisor (shown in Fig. 7.5 following exposure and bonding). (a) Cephalometric lateral skull radiograph. (b) CBCT. (c) Panoramic radiograph

These combined views may be sufficient for accurate localisation, diagnosis and planning of further treatment in most cases, but if dilacerations or other pathology is suspected, then cone beam computed tomography may be indicated in selected cases.

Cone Beam Computed Tomography

In complex cases, further radiographic investigation with cone beam computed tomography may be indicated, which can often be requested in conjunction with surgical colleagues. If multiple supernumerary teeth are present or a severe dilaceration of the root is suspected, cone beam computed tomography will have the advantage of giving a clear three-dimensional view, which can be rotated and viewed from all angles and supplements the panoramic view (Fig. 7.6b, c). It is vital to share any additional information with surgical colleagues and ideally patients should be seen together on combined multidisciplinary clinics to allow definitive treatment planning decisions to be discussed collectively.

When requesting cone beam computed tomography, the risks of the additional radiation exposure delivered must be considered and weighed against the benefits of allowing improved visualisation and potential improved clinical outcome. The volume of cone beam computed tomography needs to be specified and should be kept as minimal as possible to reduce the radiation dosage. The size of the volume is referred to as the field of view (FOV).

Potential advantages of cone beam computed tomography include the following:

- The images can be viewed from all angles, allowing a complete three-dimensional view of the unerupted tooth. This is helpful when explaining the nature of the problem to parents.
- Neighbouring structures can be viewed in all planes of space, allowing mechanics to be planned when guiding the incisor tooth through the least obstruction.
- Adjacent roots can be assessed for resorption.
- Optimum position of attachment on the incisor can be discussed and agreed with surgical colleagues.
- Ideal surgical access can be planned to minimise bone removal.
- Degree of dilaceration, if present, can be discussed as traction to severely dilacerated central incisors can be particularly challenging. Any application of force to the crown of the tooth may result in the root moving in an unfavourable direction. For successful outcome, both crown and dilacerated root have to be moved whilst completely surrounded in alveolar bone.

Due to the considerable increase in radiation dosage, the routine use of cone beam computed tomography cannot be justified. Recent radiographic guidelines published in the United Kingdom [5] and European guidelines [6] do not support the use of cone beam computed tomography as a routine part of orthodontic practice. Requests for these investigations should be reserved for when the orthodontist

and/or surgeon consider that the additional information gained will contribute to an improved treatment outcome. Future advances in medical physics may result in the development of cone beam computed tomography machines with a radiation dosage equal to or less than standard radiographs. This will most likely lead to greater use for radiographic assessment and treatment planning.

Timing of Treatment

The majority of unerupted central incisors are diagnosed at 7–8 years of age. Unless diagnosis is made at a much younger age, treatment should be commenced if the orthodontist is confident that the patient will be able to cope and understands sufficiently what treatment will involve. There is no conclusive evidence to suggest that the younger the patient, the more effectively the tooth will erupt, but there are considerable psychosocial benefits from early alignment. Several different approaches have been proposed for treatment, but all share the common theme that the less that normal eruption is delayed, the more favourable the outcome.

Management

Management of the impacted maxillary central incisor tooth involves a number of processes, which includes obtaining appropriate consent, creating sufficient space in the dental arch for the impacted tooth, planning the surgical intervention, removing any associated obstruction, and facilitating appropriate guided eruption.

Informed Consent

Parents will naturally wish to fully understand the risks and benefits of any orthodontic and surgical treatment, together with the prognosis for successful outcome. Ideally, treatment duration should be kept to a minimum, and it should be stressed that the aim of this early phase is primarily to bring the unerupted tooth into the mouth and maintain alveolar bone in the anterior region of the maxilla. Younger patients will require longer appointment times and considerable encouragement throughout treatment. If fixed appliances are to be used, there will be increased demands on oral hygiene and diet control. It is difficult to discontinue treatment whilst traction is being applied. Before commencing treatment with fixed appliances, time must be spent with the younger patient and parents, so that all are fully aware of the commitment required. Risks of non-completion of treatment should be explained. Photographs of appliances during the stages of treatment are useful for explanation to a younger patient who may find long discussions daunting. The more understanding the patient has, the better prepared they will be, potentially improving their chance of optimal outcome. Parents should be warned that there is an increased risk of further impacted teeth occurring with dental development and, in

particular, maxillary canine impaction on the same side as the unerupted incisor [7, 8]. This reinforces the need to keep this phase of interceptive treatment as short as possible, as future treatment may be necessary. In some cases, minor malalignment of the incisors may have to be accepted. Other risks of treatment include root resorption, alveolar bone resorption, loss of vitality and ankylosis. Fortunately, all are rare complications. Occasionally surgery may be necessary on more than one occasion. A recent study has reported a success rate of 90% for patients presenting with unerupted incisors who had surgical exposure with orthodontic guided tooth eruption. Of the six incisors which failed to erupt, five had dilacerations. Overall treatment time was longer for the dilacerated incisors and those impacted high in the alveolus [8].

Following successful outcome with eruption of the tooth, oral hygiene instruction is essential as many teeth do not have optimal gingival condition and contour. This usually improves with time, but in some cases, differing gingival margin levels and recession may remain after treatment.

Create Sufficient Space

Definitive management will ultimately depend on the final diagnosis and confirmation of the aetiological basis of the unerupted incisor. If there is no obvious cause, the primary incisor should be extracted if present and space created for the permanent incisor. Retrospective evaluation has suggested that up to 75% of unerupted incisors will erupt spontaneously if more than sufficient space is created with orthodontic treatment and possible extraction of the primary canines in the presence of moderate to severe crowding [9].

Prior to commencing any active appliance treatment, a full orthodontic examination should be undertaken, as the patient may present with other anomalies, such as anterior or posterior crossbites, deep overbite or class II malocclusion. It may be possible to correct some or all of these, but generally, treatment priority should be given to the unerupted incisor. Other occlusal anomalies are usually accepted until definitive orthodontic treatment can be planned in the full permanent dentition. The choice of appliance will depend on patient cooperation. If there are any concerns about how a young patient may cope with treatment, it is favourable to start with a simple upper removable appliance with midline expansion screw and finger springs (Fig. 7.7). However, its use should be limited to the short term, as space is created by tipping the crowns of the central and lateral incisor teeth distally. This may result in the roots of the adjacent teeth being tipped in a mesial direction and potentially into the path of the unerupted central incisor crown. Despite their theoretical limitations, an upper removable appliance can be very helpful in encouraging a nervous child cope with treatment. Fixed appliances are more challenging to use, but they have many advantages provided the patient can manage the increased demands placed on diet and oral hygiene. They allow controlled bodily movement of adjacent teeth, and once sufficient space is created, this is retained easily during and after the surgical procedure.

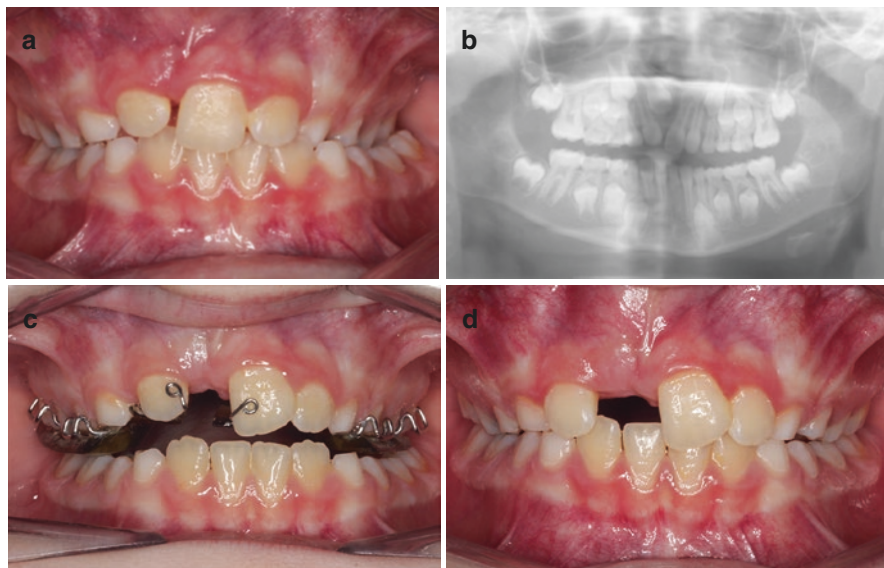


Fig. 7.7 Space creation for impacted UR1 using an upper removable appliance with a combination of expansion and tipping of the adjacent incisors. (a) Pretreatment clinical and (b) radiographic appearance, (c) 3 months and (d) 10 months following fitting of the removable appliance

Plan Surgical Intervention

If surgery is planned, it is best undertaken when sufficient space has been created for the impacted tooth. A combined decision should be made with surgical colleagues on which type of exposure is best for the patient. Generally, there are two basic approaches to exposing impacted teeth:

- *Open eruption technique.* The simplest and most conservative method of exposing a maxillary central incisor is to carry out a simple gingivectomy, with a small incision to expose the crown. This method is rarely suitable, as the crown of the tooth has to be palpable immediately under the oral mucosa comprising of keratinised gingivae. The long-term periodontal outcome will depend on the quality of the mucogingival attachment around the labial surface of the tooth and following exposure, at least 3 mm of attached gingivae needs to remain around the tooth (Fig. 7.8). The apically repositioned flap is an alternative way of performing an open exposure. A labial flap taken from the crest of the alveolar ridge is raised carefully, keeping the attached mucosa intact and repositioned higher up on the crown of the newly exposed tooth. This can pose problems with post-operative hygiene and the exposure may have to be maintained with a non-eugenol-based periodontal dressing. Whilst open exposures provide the orthodontist with the advantage of clearly being able to bond the attachment under a dry field and direct the tractional force,



Fig. 7.8 Simple open exposure for a labial impacted URI. (a) Pretreatment appearance. (b) Immediately following the gingivectomy. (c) Two weeks post-gingivectomy. (d) Six months post-gingivectomy

there is some evidence of a greater risk of poor aesthetic and periodontal outcome with this technique [10].

- *Closed eruption technique.* Most surgeons prefer this method for surgical exposure of an unerupted maxillary central incisor if traction is planned to help guide the tooth into its correct functional position. A flap is raised and a bracket is bonded to the surface of the exposed incisor with composite resin. The flap is then replaced and sutured with the gold chain protruding from the alveolar ridge through keratinised mucosa (Fig. 7.9). If practical, the bracket should be bonded to the palatal surface, which minimises the risk of the attachment fenestrating through the alveolar mucosa during eruption (Fig. 7.10), but this can be challenging and preservation of bone around the exposed tooth should be priority. This method allows conservative bone removal with rapid healing and the ability to apply traction if needed. As the orthodontist is unable to view the exposed incisor crown, it is helpful to request that photographs are taken in theatre of the exposed crown and bonded attachment, prior to replacing the buccal flap. This information is especially helpful when planning the direction of traction. If multiple supernumerary teeth are present, both central incisors can become impacted, and photographs may be helpful if a decision has to be made to apply traction to one incisor before the other (see Fig. 7.9c). Better aesthetics and bone support have been recorded when the closed eruption technique is followed [11].

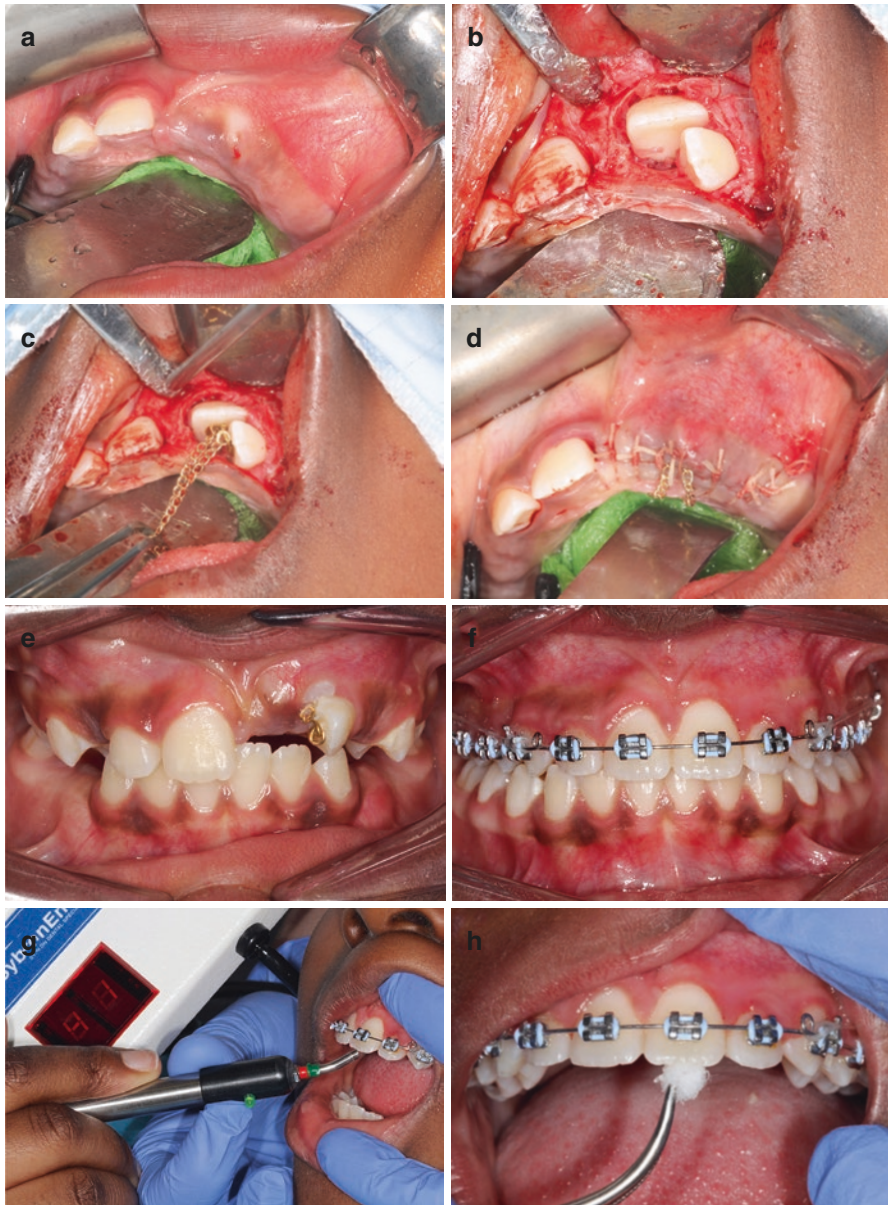


Fig. 7.9 Closed exposure and orthodontic traction for the impacted UL1 shown in Figs. 7.5 and 7.6. The tooth remained vital and achieved an excellent occlusal position

Fig. 7.10 Labial bonded attachment visible through alveolar mucosa with risk of fenestration



Fig. 7.11 (a) Tuberculate supernumerary teeth and odontomes are more likely to obstruct incisors than conical supernumerary teeth (b, c)

Remove Any Physical Obstruction with Evidence of Delayed Incisor Eruption

The presence of a supernumerary tooth or odontome does not necessarily delay eruption of incisors. Tuberculate supernumerary teeth rarely erupt and often obstruct the eruption of the permanent incisor, whereas conical supernumerary teeth frequently erupt and do not usually delay eruption [12] (Fig. 7.11). In an analysis of compound and complex odontomes, complex odontomes obstructed eruption in half of cases and compound odontomes in a third of cases [13]. Once there is evidence to confirm that the supernumerary tooth or odontome is obstructing or interfering with normal eruption, surgical removal is recommended. Usually these develop palatal to the crown of the incisor tooth and rarely cause any significant dilacerations of the roots. Hence, if removed early, the prognosis for eruption of the maxillary central incisor tooth is favourable. Provided the obstruction is removed keeping the entire dental follicle intact, the submerged tooth should erupt spontaneously without the need for orthodontic traction (Fig. 7.12). However, in many cases,



Fig. 7.12 Eruption of impacted upper central incisors following the removal of tuberculate supernumerary teeth

the proximity of the supernumerary tooth and the palatal surface of the incisor is so close that damage to the dental follicle is inevitable, and hence it is advisable to bond an attachment to the incisor, which can be used to apply traction.

Exposure of Dilacerated Incisors

Developmental disturbances in the shape of the crown and root of teeth can occur due to a variety of causes. Although significant trauma is often cited as the main cause, the majority of dilacerations are developmental in origin and most likely the result of ectopic development of the tooth germ [14]. The extent of dilaceration and the inclination of the crown and root will determine whether it is possible to bring the crown into alignment (Fig. 7.13). Cone beam computed tomography will allow the orthodontist to view the dilaceration from all angles and also provide information on the quality and quantity of alveolar bone at the site where the tooth will be moved. Traction can then be planned to maintain both the crown and root in alveolar

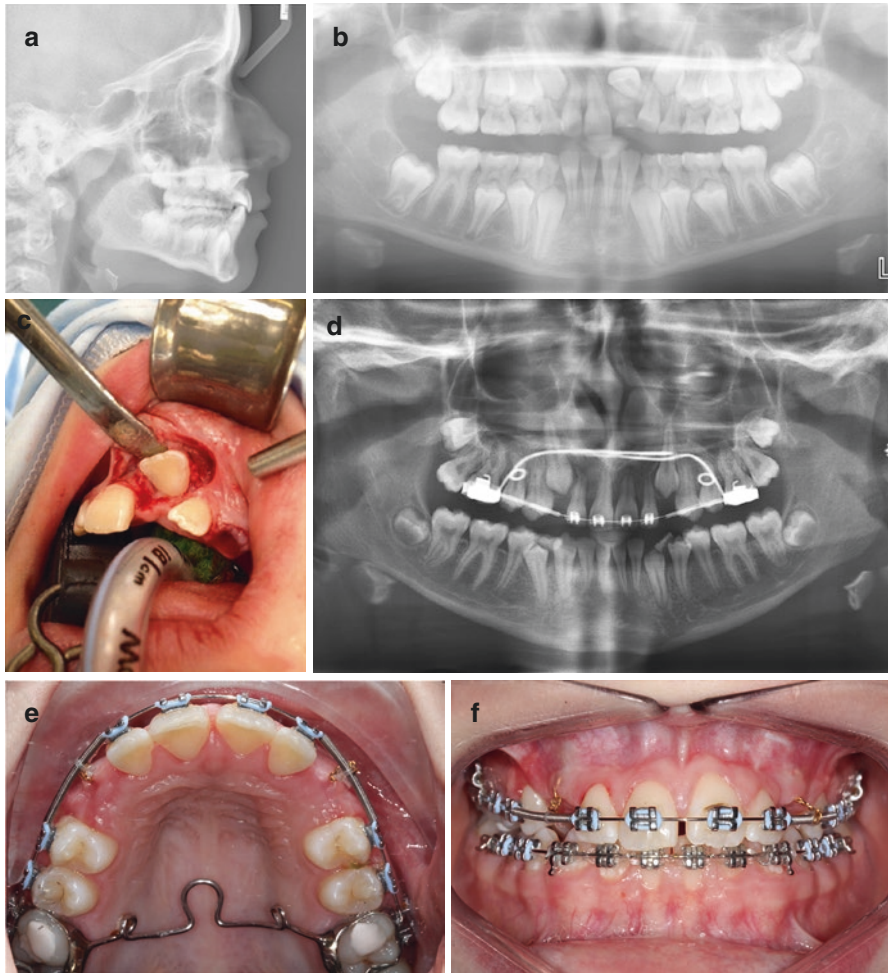


Fig. 7.13 Management of a dilacerated UL1

bone if alignment is to be successful [15]. If the crown can be aligned and the apex becomes prominent through the labial plate of bone, amputation of the apex can be considered with root canal treatment of the remaining incisor tooth [16]. Alignment of dilacerated incisors will have the benefit of maintaining alveolar bone, even if optimal alignment and gingival margins cannot be achieved (Fig. 7.14). Severe dilacerations may necessitate surgical removal.

Orthodontic Guided Tooth Eruption

The biomechanical principles of orthodontic tooth movement must be adhered to when guiding the unerupted incisor to its functional position. Planning sufficient anchorage can be challenging as the first permanent molars, and possibly three

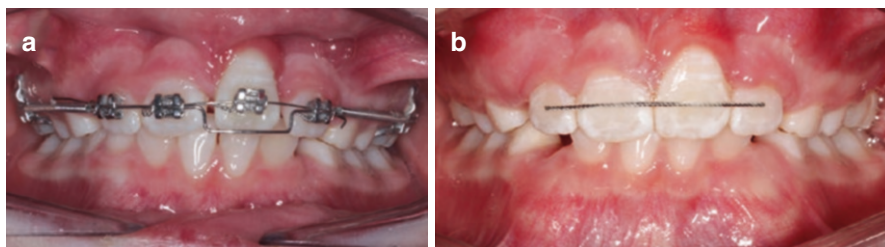


Fig. 7.14 Poor gingival margin differences following alignment of dilacerated UL1 (a) During treatment (b) Minimal improvement 6 months following debond. Labial bonded retainer made of multistranded stainless steel wire

Fig. 7.15 '2 × 4' appliance



incisor teeth are often the only erupted permanent teeth at the time of guided eruption. To maximise potential anchorage, molar bands soldered to a transpalatal arch can be cemented to the first permanent molars, and brackets should be bonded to both maxillary lateral incisors and the erupted central incisor. This is referred to as a '2 × 4 fixed appliance' [17] (Fig. 7.15). The long spans of archwire between the molars and lateral incisor teeth should be supported by housing them in closed stainless steel tubing, cut precisely to fit the exact length of the unsupported base archwire. In addition, it is useful to cinch the archwire distal to the first permanent molar teeth. Occasionally, it may be necessary to bond attachments to the primary canines or molars if there is insufficient support for the archwire. Wide bore eyelet attachments can be used as their primary purpose is to support the archwire and not to move the primary teeth (Fig. 7.16). Gentle traction can be applied when a stiff stainless steel archwire is in place, and as progress is made, a light nickel titanium wire can be used as an overlay wire tied to the rigid steel archwire (Fig. 7.17). Rectangular steel wires should be avoided to minimise torquing of roots which may impede the unerupted tooth. The direction of traction must be planned especially if dilaceration is present, as the tooth should be guided carefully through the

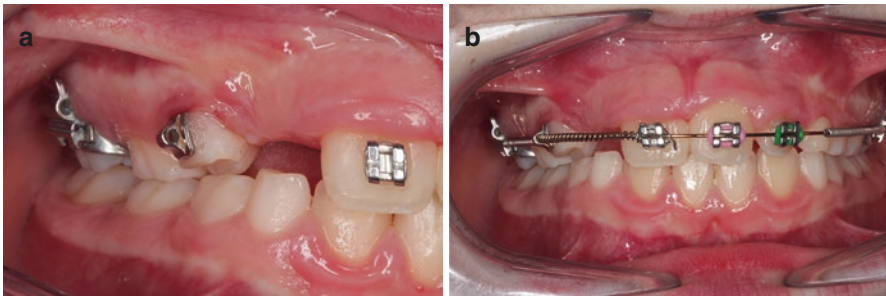


Fig. 7.16 ‘Wide-bore’ attachment bonded to deciduous molar tooth to support archwire as the lateral incisor is congenitally absent

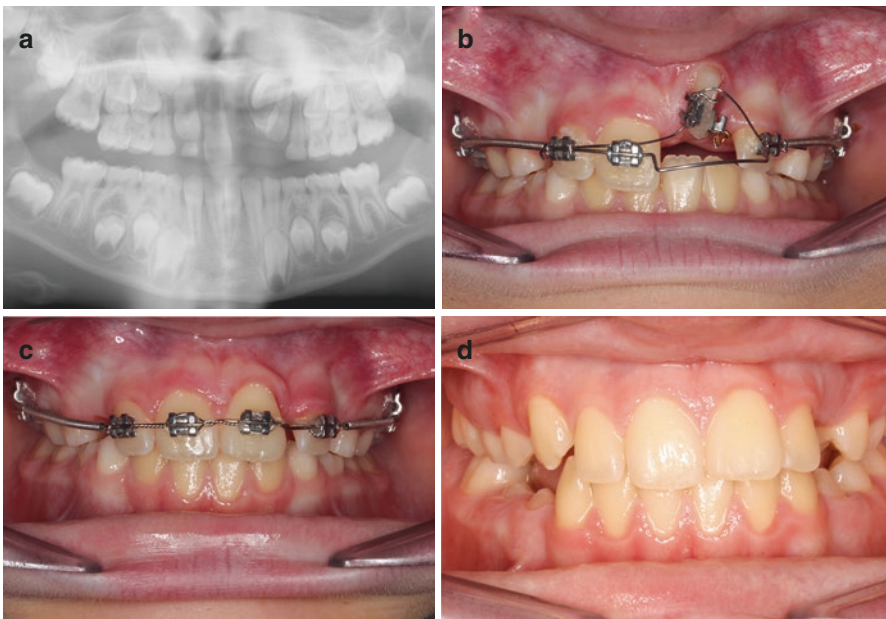


Fig. 7.17 0.012-in. Nickel titanium overlay wire tied to rigid stainless steel base wire

thick attached keratinised mucosa to optimise the periodontal support. Once the tooth is visible, its vitality should be checked and monitored until alignment is achieved (see Fig. 7.9).

Cystic Change

As with any unerupted teeth, cystic formation can occur. If follicular enlargement is suspected, with possible associated displacement of the unerupted tooth, cone beam computed tomography may be indicated to identify the full extent of the

lesion. A conservative surgical approach is recommended to optimise the chances for eruption of the incisor. Depending on the size of the lesion, enucleation or marsupialisation may be the treatment of choice. A period of healing is recommended, and if the incisor does not improve in position, a further exposure with traction may be considered at a later date.

Surgical Removal of Unfavourable Incisors

Orthodontic traction may not be successful if the unerupted incisor has become ankylosed. This can occur to any tooth during the eruption process when replacement resorption occurs at the innermost layer of the periodontal ligament. True ankylosis is difficult to diagnose but should be suspected if there is no progress with continued light tractional forces, as evident by measuring the gold chain, and adjacent teeth included in the appliance become intruded.

Surgical removal of the incisor must be considered as a last resort, as removal will result in considerable loss of alveolar bone. Replacement with an implant prosthesis in the future will require bone grafting, and this may compromise success. If possible, the unerupted tooth should be left in situ and monitored radiographically over time, as it will then help maintain alveolar bone thickness until a permanent prosthesis can be placed when the patient has stopped growing. In many cases, this will not be possible if orthodontic treatment of adjacent teeth is required to prepare space for a replacement tooth. In crowded cases, surgical removal of the impacted incisor, closure of space and alignment of the lateral incisor in place of the central incisor is a possible medium term interim solution. If aesthetics are compromised, replacement of the lateral incisor with a prosthetic implant can be considered in the future.

Surgical Replantation

In rare circumstances, the surgical removal and replantation of an impacted maxillary incisor can be considered if there is a severe dilaceration or arrested root development and guided tooth eruption is not considered to be a viable option. The unerupted tooth is removed with minimum trauma, and the transplant site is prepared by creating a greenstick fracture of the labial plate to expand it to allow positioning of the incisor. The crown is splinted to adjacent teeth. Orthodontic movement may be attempted after 4 months [18].

Retention

Interceptive treatment should cease as soon as the incisor is aligned. Fitting a removable retainer to maintain alignment can be problematic, as retention of the appliance is challenging due to the normal exfoliation of deciduous teeth and their

replacement with the permanent dentition. A simple fixed retainer bonded to the labial surfaces of the teeth is an ideal choice, as it will be easy to clean, help prevent vertical relapse and allow normal dental development (Fig. 7.14b). This can be easily removed after 6–9 months.

Discussion

When managing impacted maxillary central incisor teeth, each individual case must be considered independently. The clinician must diagnose the problem with accuracy and communicate with surgical colleagues to plan treatment with the best potential outcome. Multidisciplinary teamworking allows the ideal environment for patient examination, group discussion and definitive planning. With favourable patient compliance and operative skills, the majority of unerupted maxillary central incisor teeth will successfully erupt into a functional occlusion with ideal aesthetics, resulting in significant long-term dental and psychosocial benefit to the patient.

Acknowledgements The author would like to acknowledge the expert surgical skills and teamworking of her colleagues, Dr Cathy Bryant, consultant paediatric oral surgeon and the paediatric dental consultants at King's College Hospital, London.

References

1. Shaw WC, O'Brien KD, Richmond S, Brook P. Quality control in orthodontics: risk/benefit considerations. *Br Dent J.* 1991;170:33–7.
2. Seehra J, Fleming PS, Newton T, DiBiase AT. Bullying in orthodontic patients and its relationship to malocclusion, self-esteem and oral health-related quality of life. *J Orthod.* 2011;38:247–56.
3. Bartolo A, Camilleri A, Camilleri S. Unerupted incisors-characteristic features and associated anomalies. *Eur J Orthod.* 2010;32:297–301.
4. Yaqoob O, O'Neill J, Gregg T, Noar J, Cobourne MT, Morris D. Management of unerupted maxillary incisors. 2010. https://www.rcseng.ac.uk/fds/publications-clinicalguidelines/clinical_guidelines/documents/ManMaxIncisors2010.pdf. Accessed 22/03/16.
5. Isaacson KG, Thom AR, Atack NE, Horner K, Whaites E. Orthodontic radiographs guidelines. 4th ed. London: British Orthodontic Society; 2015.
6. European Commission. Cone beam CT for dental and maxillofacial radiology (Evidence based guidelines). Radiation Protection no. 172. Luxembourg: Publications office; 2012. http://www.sedentext.eu/files/radiation_protection_172.pdf.
7. Chaushu S, Zilberman Y, Becker A. Maxillary incisor impaction and its relationship to canine displacement. *Am J Orthod Dentofacial Orthop.* 2003;124:144–50.
8. Chaushu S, Becker T, Becker A. Impacted central incisors: factors affecting prognosis and treatment duration. *Am J Orthod Dentofacial Orthop.* 2015;147:355–62.
9. DiBiase DD. The effects of variations in tooth morphology and position on eruption. *Dent Pract Dent Rec.* 1971;22:95–108.
10. Chaushu S, Brin I, Ben-Bassat Y, Zilberman Y, Becker A. Periodontal status following surgical-orthodontic alignment of impacted central incisors with an open-eruption technique. *Eur J Orthod.* 2003;25:579–84.

11. Becker A, Brin I, Ben-Bassat Y, Zilberman Y, Chaushu S. Closed eruption surgical technique for impacted maxillary incisors: a post-orthodontic periodontal evaluation. *Am J Orthod Dentofacial Orthop.* 2002;122:9–14.
12. Foster TD, Taylor GS. Characteristics of supernumerary teeth in the upper central incisor region. *Dent Pract Dent Rec.* 1969;20:8–12.
13. Katz RW. An analysis of compound and complex odontomas. *J Dent Child.* 1989;56:445–9.
14. Stewart DJ. Dilacerate unerupted maxillary central incisors. *Br Dent J.* 1978;145:229–33.
15. Kapila SD. Cone beam computed tomography in orthodontics. Indications, insights and innovations: Wiley-Blackwell; 2014.
16. Becker A. Orthodontic treatment of impacted teeth. 3rd ed. Oxford: Wiley-Blackwell; 2012.
17. McKeown HF, Sandler PJ. The two by four appliance: a versatile appliance. *Dent Update.* 2001;28:496–500.
18. Kokich VG, Mathews DP. Orthodontic and surgical management of impacted teeth. Chicago: Quintessence; 2014.

Early Management of the Palatally Displaced Maxillary Permanent Canine

8

Philip E. Benson and Nicola A. Parkin

Abstract

This chapter describes the current evidence concerning the early management of the palatally displaced canine (PDC).

To practice effective interceptive treatment, normal development must be understood. Key papers are summarized, which help in the diagnosis of a PDC, and some thought is given to the timing of interceptive treatment. Any intervention should be undertaken early enough to allow time for self-correction, and it appears the optimum time is when a child is aged 10–13 years.

Justification for extracting the primary canine often stems from one, extensively cited, study that had no control group. The study methodology was not robust, and it is possible that many of the canines included might have erupted without intervention. Little knowledge is gained from this investigation; however, it has evoked much interest, and many studies have been published subsequently exploring the same concept. These will be described, as well as alternative interceptive techniques, such as space creation, using maxillary expansion devices, headgear and/or fixed appliances.

We conclude that most published trials in this field have inadequate detail or inconsistencies in reporting, dictating the need for further well-designed clinical trials. After studying the available literature, it appears that the horizontal sector is the best predictor for outcome, with success defined as eruption of the permanent canine without the need for surgical uncovering. We have developed guidelines to help the practitioner decide when interceptive treatment might be appropriate. These guidelines are based on conventional radiography as, currently, this is most commonly used in clinical practice.

P.E. Benson (✉) • N.A. Parkin
University of Sheffield, Sheffield, UK
e-mail: p.benson@sheffield.ac.uk; nparkin@nhs.net

Normal Development and Eruption

In order to determine when a child has a palatally displaced canine (PDC), it is important to know how a normally erupting canine should appear clinically and radiographically at a given age. Although there can be considerable variation in chronological age versus biological age, regarding the degree of inclination, height and medial position of the canine, it is important to understand what happens during normal development in the average child. Early detection of an eruptive disorder is undoubtedly crucial if interceptive prevention is planned.

The crown of the maxillary permanent canine starts to calcify at 4–5 months after birth, is fully formed at approximately 6–7 years of age and erupts between the ages of 11 and 12 years [1]. The tooth has a long path of eruption, moving about 22 mm between the ages of 5 and 15 years [2]. This might explain why the maxillary canine is the tooth most frequently associated with displacement. Ericson and Kurol examined a large community-based sample of 505 schoolchildren aged 8–12 years over 3 years and determined that both the maxillary permanent canines are palpable in the buccal mucosa of 67 out of 94 or 71% of 10-year-olds [3]. This proportion increased to 95% in 11-year-olds (104 out of 109 young people) (Fig. 8.1).

Coulter and Richardson [2] examined the longitudinal radiographic records of 30 children from the Belfast growth study. The participants had lateral and depressed postero-anterior radiographs taken annually from the age of 5 to 15 years, and the authors found that between the ages of 5 and 9 years, lateral movement of the upper permanent canine was small and mainly in a palatal direction. After 9 years the canines moved buccally, with the greatest amount occurring between 10 and 12 years. This suggests that little is to be gained from a radiographic investigation of the canine position prior to the age of 9 years, before the canine starts to move in a buccal direction. This observation is substantiated by Ericson and Kurol who, in their study of 3000 schoolchildren, confirmed that examination before the age of 10 years did not prove a reliable basis for the prognosis of a future unfavourable path of canine eruption [4].

Fernandez and colleagues examined the inclination of the upper canine on panoramic radiographs and concluded that they become more mesially inclined until

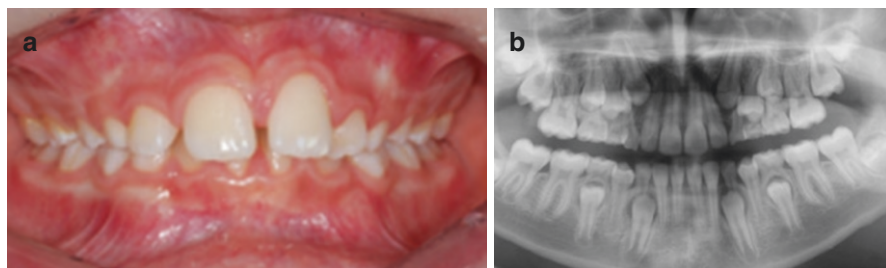


Fig. 8.1 (a) According to the work of Ericson and Kurol [3], maxillary permanent canines are palpable in the buccal mucosa of 71% of 10-year-olds and 95% of 11-year-olds. (b) Radiograph of a normally developing dentition in a 9-year-old child

the age of 9 years, after which they progressively upright; however, they also found considerable individual variation and state that ‘the capacity to predict inclination at a given age is limited.’

Disturbances of Development and Eruption

In a large community-based sample of Swedish children examined at 7, 10 and 13 years of age, the prevalence of maxillary canine displacement was found to be 2.2% (123 out of 5459) [5]. In a smaller school-based sample, Ericson and Kuroi determined that 41 out of 505 (8%) children had clinical signs of a displaced maxillary canine requiring a radiographic examination and 1.7% of their canines had a disturbance in their eruption path following a radiographic examination [3]. In a larger sample of 3000 school children aged 10–15 years, 201 (7%) had clinical signs suggesting a displaced canine [4], and following a radiographic examination, 84 children (2.8%) were diagnosed with 93 displaced permanent canines, of which 69 (74%) canines were palatally displaced (PDC).

McSherry and Richardson, again using records from the longitudinal Belfast growth study, concluded that PDC, unlike those that erupted normally, never moved buccally between the ages of 10 and 12 years, but always moved palatally [6]. The annual differences in the amount of palatal tooth movement between displaced and undisplaced maxillary canines ranged from 1.0 to 2.2 mm between the ages of 5 and 9 years and 1.3 to 3.0 mm after 9 years.

There is much speculation in the literature about whether PDCs are caused by a disturbance in the local environment as the tooth erupts, for example, a diminutive or developmentally absent lateral incisor (the guidance theory) or whether it is a genetic condition [7, 8]. Whatever the cause, a PDC can lead to resorption of adjacent teeth (Fig. 8.2), particularly in females with enlarged dental follicles [9] and very occasionally unerupted teeth develop cysts [10].

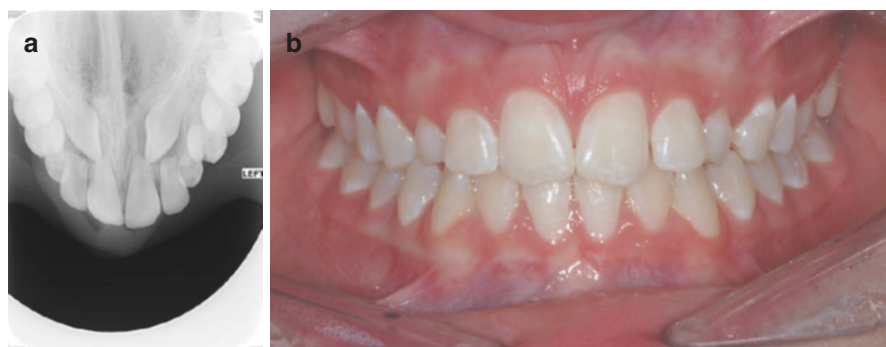


Fig. 8.2 (a) Standard maxillary occlusal radiograph of a 14-year-old girl with resorbed lateral and central incisors. (b) There were no clinical signs of resorption, no discoloration or mobility of any of the incisors

Diagnosis of a Palatally Displaced Maxillary Permanent Canine

Clinical Features

Ericson and Kuroi outline three clinical features that indicate a radiographic examination to determine the position of the unerupted permanent canine is necessary [11]:

- Asymmetry on palpation or a pronounced difference in eruption of canines between the left and right side.
- The canines cannot be palpated in the normal positions, and the occlusal development is advanced.
- The lateral incisor is late in eruption or shows a pronounced buccal displacement or proclination (Fig. 8.3).

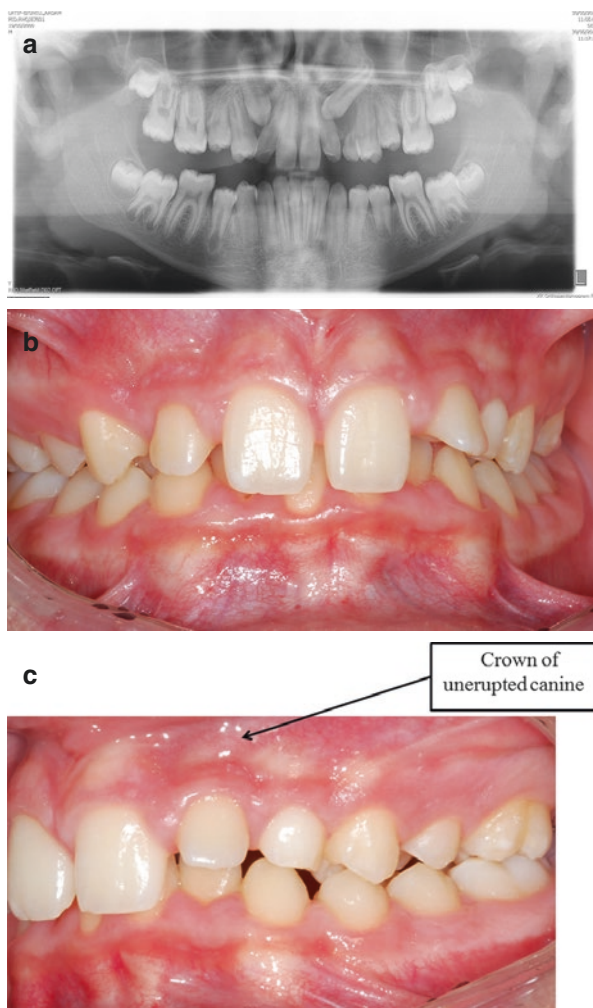


Fig. 8.3 (a) The upper left canine is overlapping the root of the lateral incisor on the dental pantomogram and the root of the incisor is fully developed. (b and c) The angulation of the lateral incisor suggests that the canine is buccally positioned

Radiographic Features

Ericson and Kurol then went on to outline a method of assessing the displacement of the maxillary canine on a radiograph using three criteria [12]:

- The medial position of the canine crown classified into five sectors (Fig. 8.4)
- The angulation of the long axis of canine to a vertical line drawn between the central incisors (Fig. 8.5)
- The vertical distance of the canine cusp tip to the occlusal plane (Fig. 8.5)

Fig. 8.4 Classification of the horizontal position of the unerupted maxillary permanent canine (With permissions from Ericson and Kurol [12])

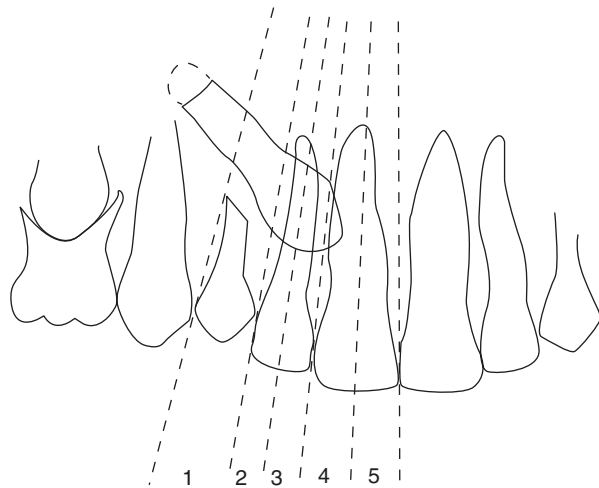
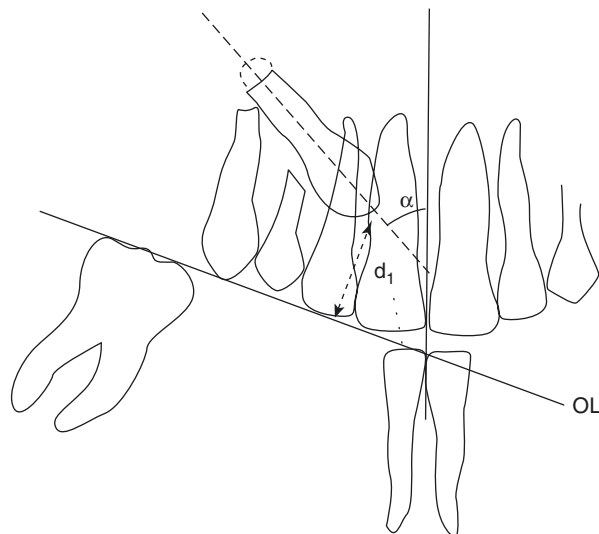


Fig. 8.5 Methods of measuring the angulation and vertical position of the unerupted maxillary permanent canine (With permissions from Ericson and Kurol [12])



Lindauer and colleagues [13] undertook a retrospective examination of panoramic radiographs and concluded that at the age of 9 years 92% of non-PDC teeth were positioned in horizontal sector 1. By the age of 12 years, if the upper canine was in sector 1, then the proportion that were eventually diagnosed as displaced was 12%, but if in sector 2, then the proportion was 83% and if in sectors 3 and 4, the proportion that were eventually diagnosed as displaced was 100%; however, the sample size was small (28 young people with PDC and 28 without a PDC).

Warford and colleagues examined the dental pantomograms of patients diagnosed with and without PDCs from one orthodontic practice. They concluded that the horizontal sector was a better predictor of future displacement than angle [14]. The odds of a maxillary canine being eventually diagnosed were 0.05 if the tooth was in sector 1, 0.53 in sector 2, 0.80 in sector 3 and 0.99 in sector 4; however, the numbers of teeth in each sector were very small and no confidence intervals were quoted.

Fernandez and colleagues observed that the extent of root development of the lateral incisor was also important when determining if the permanent canine was displaced or not [15]. They found that some overlap of the canine and adjacent lateral incisor was commonly found on a panoramic radiographs when the root of the lateral incisor is not fully developed. This important finding has been overlooked in some of the trials published over the last decade, leading to some readers doubting whether all the canines in the sample were actually displaced at the outset.

Some authors have suggested that radiographs can be used to predict that the maxillary canine is displaced earlier than 10 years of age. Sambataro and colleagues used a PA radiograph at the age of 8 years, but their formula for predicting a PDC was based on the radiographs of just 12 individuals with PDC [16]. The authors state that two children were incorrectly diagnosed, but do not indicate whether these were children with a PDC, who were not diagnosed (false negative) or more worryingly children without a PDC who were incorrectly diagnosed with a PDC (false positive). Sajjani and King used measurements from panoramic radiographs taken as early as 8 years of age, but again this was based on just 14 individuals who were less than 9 years old and had follow-up radiographs [17]. The study included individuals with both buccal and palatal displacement, and it is not clear how many false positives (and hence unnecessary interventions) would result. Others have disputed whether a displaced maxillary canine can be diagnosed before the age of 10 years [11, 18].

Early Interventions

Treatment for a PDC, after the secondary dentition has been established, frequently involves the young person undergoing a surgical operation to uncover or expose the displaced tooth or teeth, often with a general anaesthetic. This is followed by a prolonged course of orthodontic treatment to pull the tooth or teeth into the correct position [19–21]. To avoid the need for this potentially lengthy and expensive treatment, several interventions, designed to correct the eruption direction of a PDC, have been proposed.

Extraction of the Primary Canine

The extraction of the primary canine when the clinician suspects that the permanent canine is displaced was described in a case report by Buchner in 1936 [22]; however, the justification for extracting the primary canine as an interceptive technique often stems from one cohort study, with no control group, reported by Ericson and Kurol [12], possibly one of the most quoted articles in the orthodontic literature. Ericson and Kurol published the outcomes from a case series of 35 children, aged 10–13 years. All the young people in their article had their primary canines removed, and there was no untreated control group, and the data should be interpreted with caution. Following removal of the primary canine 38 out of 46 (83%) suspected PDCs permanent canines erupted successfully. This appears an impressive success rate; however, on closer examination of the data, 13 of the unerupted canines were in sector 1, which might be considered a normal position and a low risk of displacement. Of the 11 teeth diagnosed in sector 2, 10 erupted satisfactorily; however, Ericson and Kurol do not explain how many of these young people had a fully developed root of the lateral incisor on the radiograph (see section on [15]). The success rate for the more severely displaced teeth was 14 out of 22 teeth (64%); however, it is not clear how many were in the more severely displaced sector 4, as the authors combined the numbers for sectors 3 and 4 (none was in the most severely displaced sector 5).

Power also reported a case series of 39 patients, aged 9.3–14.5 years, diagnosed with 47 PDCs [23]. The young people were all consecutively treated with extraction of the primary canine, and there was no untreated control group. In 8 of the 47 permanent canines that were treated, there was no horizontal overlap between the permanent canine and the adjacent incisor (sector 1), which many would consider to be a normal developmental position. In 16 of the 47 canines, there was some overlap of the canine with the lateral incisor, but again, the authors do not describe the development of the lateral incisor root in these patients, and therefore it is not clear how many of these teeth might be considered to be in an abnormal position. The authors did conclude that the more the permanent canine was displaced horizontally, then the lower the probability that extraction of the primary canine would be successful. Out of nine unerupted canines that overlapped the nearest incisor by more than half the tooth (sector 3), only 4 or 44% erupted successfully without surgical intervention, and the figures were even worse (one erupted out of eight teeth, or 13%) when the unerupted canine was even further towards the midline (sector 4 or 5).

The first reported prospective clinical trial of this intervention, including an untreated control group, was by Leonardi and colleagues [24]. They compared the successful eruption of the permanent canine (defined as ‘permitting bracket positioning for final arch alignment when needed’) in the untreated, control patients, with participants who had undergone two treatments (extraction of the primary canine only and extraction of the primary canine with the use of cervical pull headgear). They concluded that there were similar proportions of successfully erupting canines between participants who had the primary canine extracted (50%) and those

in the untreated control group (success rate not reported); however, there were just 11 young people in this intervention group and 3 dropped out or withdrew. The proportion of canine teeth that successfully erupted when the primary canine was extracted and headgear fitted was 80%, but this group was much larger than the other intervention group, with 21 young people. All the groups included canines that were judged to be in sector 1 (undisplaced), and it is not clear how horizontal displacement effected the success rates. Unfortunately there were numerous confusions and inconsistencies in the reporting of the trial as outlined in two systematic reviews [21, 25], and all attempts to obtain further clarification from the authors failed.

Baccetti and colleagues reported a study containing a larger number of participants, but with the same interventions as Leonardi and colleagues [26]. Interestingly the introduction to the article states that there was ‘no study in the literature’ that had reported using a randomized, prospective study design, with untreated controls and a ‘statistically appropriate number’ of participants in this area, even though three of the authors were common to the Leonardi et al. article. Baccetti and colleagues found a 36% successful eruption of the permanent canine with no intervention, compared with 66% when the primary canines alone were extracted. The proportion of successfully erupting canines increased to 88% with the extraction of the primary canine and the use of cervical headgear. Again serious deficiencies in the reporting of the study have been identified, including whether outcome assessors were masked, if there was pretreatment equivalence regarding the inclination, distance from the occlusal plane and the midline location of the displaced canine and missing data for the successful eruption of teeth in the control and experimental groups [21]. In addition the sample included young people with a ‘dental age older than 8’, which is younger than many clinicians would consider it possible to determine that the permanent canine is displaced [11, 18]. Most importantly the authors did not report the success rates according to the initial horizontal displacement (sector) and again all efforts to obtain further data from the authors failed.

Bonetti and colleagues proposed the extraction of both the primary canine and the primary first molar in young people with a suspected PDC [27, 28]. In the first article, they report the results of a clinical trial involving 60 young people aged 9–12 years and 7 months. Participants with suspected palatally displaced maxillary permanent canines were randomly allocated to either have the primary canine only removed or the primary canine and the primary first molar. They defined a successful outcome as ‘when surgical uncovering was not required, as the [*sic*] complete eruption of the PMCs into the dental arch within 48 months’; however, there were no data in the article about the proportions of participants with successful eruption of the permanent canine, only improvements in the position of the permanent canine.

In a second article (which does not cite the first article), Bonetti and colleagues report the results of the same interventions, but with 40 young people. The upper age limit in this trial was also 13 years; however, the lower age limit was reported as 8 years, which as previously explained, many clinicians believe is too young to confidently predict that a maxillary canine is palatally displaced. The participants

were randomly allocated to one of the two interventions outlined in the previous report. Success was also judged according to the criteria of Leonardi and colleagues; however, the sample size (which was not justified in the first article) was calculated on the basis of improvement in the angulation of the unerupted canine and cited for the number of teeth, not the number of participants. This does not take into account the potential clustering effect of multiple teeth within the same individual [29]. The authors report successful eruptions of 22 out of 28 canines (79%) when the primary canine alone was extracted and 36 of 37 canines (97%) when both the primary canine and molar were removed; however, again there were no data on the success rates according to the initial horizontal displacement (sector), which several studies have identified as an important criterion for a successful outcome.

Bazargani and colleagues undertook a randomized trial involving young people (aged 10–14 years), with bilateral PDC, that were in horizontal sectors 2–5 [30]. The sample size was based on improvement in the angulation of the canine over an 18-month observation period. They recruited 24 young people and randomized one upper quadrant, in each participant, for extraction of the primary canine and the contralateral upper quadrant as an untreated control. The reported proportions for successful eruption of teeth were 16 out of 24 (67%) for the side where the extraction was undertaken and 10 out of 24 (42%) for the control side. The authors conclude that the intervention was more successful in younger participants, aged 10–11 years, compared with older participants, aged 12–14 years; however, there are no details about the numbers of participants or the proportions of successfully erupted teeth (the ultimate aim of the intervention) in each group. The authors also noticed that the intervention was more successful when the canine was closer to the normal horizontal position at the start. An improvement in the horizontal position of canines was seen over 18 months in 15 out of 19 teeth that were initially in sectors 2 or 3 (2 out of 19 teeth showing no change and 2 teeth migrated to a worse position). This compares with improvement in the horizontal position of only one out of five teeth that were initially in sectors 4 and 5 (two showed no change and in two the position worsened); however, the numbers of teeth (let alone participants) was clearly very small. In addition, the authors do not state what proportion of the teeth that improved their horizontal position also successfully erupted without surgical intervention. Interestingly the authors observed no midline shift towards the side where the primary canine was removed in any of the participants.

Naoumova and colleagues undertook a randomized controlled trial involving 67 young people with both unilateral and bilateral PDCs [31]. Participants, who were aged 10–13 years, were randomized to either an intervention group (extraction of one or both primary canines) or to an untreated control group. Although the declared primary outcome of the study was the proportion of canines that successfully erupted within a 24-month observation period, the primary canines were extracted in the control group if there were no signs of mobility at 12 months. In addition, surgical exposure was undertaken if there were no signs of improvement in the position of the canine on radiographs taken at 12 months, regardless of which group the participant was allocated to. The sample size was estimated on the basis of improvement

in the angulation of the canine, which was a secondary outcome, not the primary outcome. Despite these inconsistencies, the study did show a clear difference in the proportions of successfully erupted canines between the two groups (69% in the extraction group and 39% in the untreated control). Twenty-seven canines in the control group required surgical exposure compared with only 14 in the extraction group. The mean eruption time was slightly longer in the control group (18.3 months, SD 5.8 vs. 15.6 months, SD 5.6), but no differences were found in the eruption times between younger (aged 10–11 years) and older participants (12–13 years). In regard to the displacement severity of the unerupted canines, this was assessed from cone beam computed tomography (CBCT), rather than plain films; therefore, the horizontal displacement towards the midline was expressed in millimetres rather than sectors. In a second publication, the authors did find that the distance from the canine cusp tip to the midline demonstrated a very good discriminatory power and could be used as a prognostic indicator for whether the PDC would spontaneously erupt or not [32]. They concluded that when the canine cusp tip was 11 mm from the midline then the probability of spontaneous eruption was high, whereas canines that were 6 mm or less from the midline would probably need surgical exposure, even following extraction of the primary canine.

There are now a number of published studies that have examined the effectiveness of extracting the primary canine when the clinician suspects that a maxillary canine is palatally displaced. All the studies have irregularities in study design and inconsistencies in their reporting; however, they all suggest that extracting the primary canine is an effective method of increasing the chances that a palatally displaced permanent maxillary canine will erupt without surgical intervention; however, the procedure can lead to unpredictable results (Fig. 8.6), and none of the studies provide sufficient information concerning at what age or what horizontal displacement of the permanent canine this intervention is likely to be most effective. These are areas for future research.

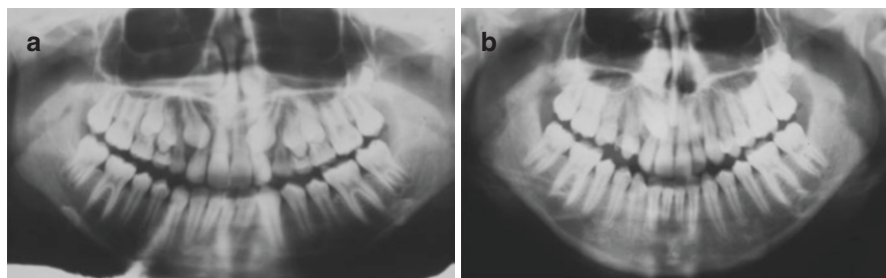


Fig. 8.6 (a) Dental pantomogram of an 11-year-old girl showing both upper canines overlapping the upper lateral incisors, which have fully developed roots. The upper left canine looks to be more mesial than the right canine. Both upper primary canines were removed. (b) A follow-up radiograph of the same patient 18 months later. The upper left canine has erupted, but the upper right canine (which was in a more favourable position on the first radiograph) has continued to move mesially and was surgicaly exposed (the retained crown of the upper right primary first molar probably did not help)

Space Creation

Some have advocated creating space when the canine is potentially displaced. This might be achieved using transverse expansion of the maxillary arch using RME or anterior-posterior expansion using headgear or a bonded orthodontic appliance.

Below is an example of a young child presenting at the age of 10.1 years. He has a skeletal transverse maxillary deficiency and crowding, the canine on the right side is excluded and in a more medial position to that on the left side. Maxillary expansion was carried using a Haas type maxillary expansion device together with space creation using fixed appliances with the result that both canines erupted spontaneously in a favourable position.

Rapid Maxillary Expansion (RME, Fig. 8.7)

Baccetti and colleagues reported the results of a clinical trial to investigate the effect of rapid maxillary expansion on the eruption patterns of maxillary canines [33]. The study presented data from 60 young people, aged 7.6 and 9.6 years, from two centres in Italy, who were apparently diagnosed with palatally displaced canines from PA radiographs, using the mathematical technique described by Sambataro and colleagues. As stated earlier this method was based on the radiographs of just 12 individuals [16]. The authors state that participants were randomly allocated to either receive the intervention (RME) or to be observed as an untreated control, but contrary to CONSORT guidelines, neither the method of randomization nor allocation concealment were explained, and there were unequal numbers in the two groups (35 in the intervention group vs. 25 controls). The authors admit that the participants in this study showed no evidence of skeletal transverse maxillary deficiency, but there was evidence of constriction at the dentoalveolar level. Expansion was continued until 'the palatal cusps of the maxillary posterior teeth were in contact with the buccal cusps of the mandibular posterior teeth'. No sample size calculation was reported. The participants were re-evaluated in the early permanent dentition at an average age of 13.1 years (SD 6.8 months) after a mean observation time of 4.4 years, at which point the canines had successfully erupted in 21 out of 32 participants (66%) in the intervention group, but only 3 out of 22 participants (14%) in the control group. There were numerous deficiencies in the reporting of this trial according to the CONSORT guidelines; however, many would consider the main limitation was the early age at which the authors claimed to have diagnosed a PDC. Notwithstanding these limitations, a surprising number of canines in the control group failed to erupt.

In a further article, written by some of the same authors as the previous article, they report the findings from 120 young people, aged 9.5–13 years, recruited in Italy [34]. Although the study design was described as 'randomized, prospective and longitudinal', the recruitment period was extremely long (1991–2009), and the authors explain that it was 'standard practice that orthodontic patients in the University clinic are given a serial number as they are enrolled for orthodontic treatment or monitoring'. This, as well as the numerous missing details from the CONSORT checklist, suggests that the sample was a longitudinal cohort. There were no descriptions of the

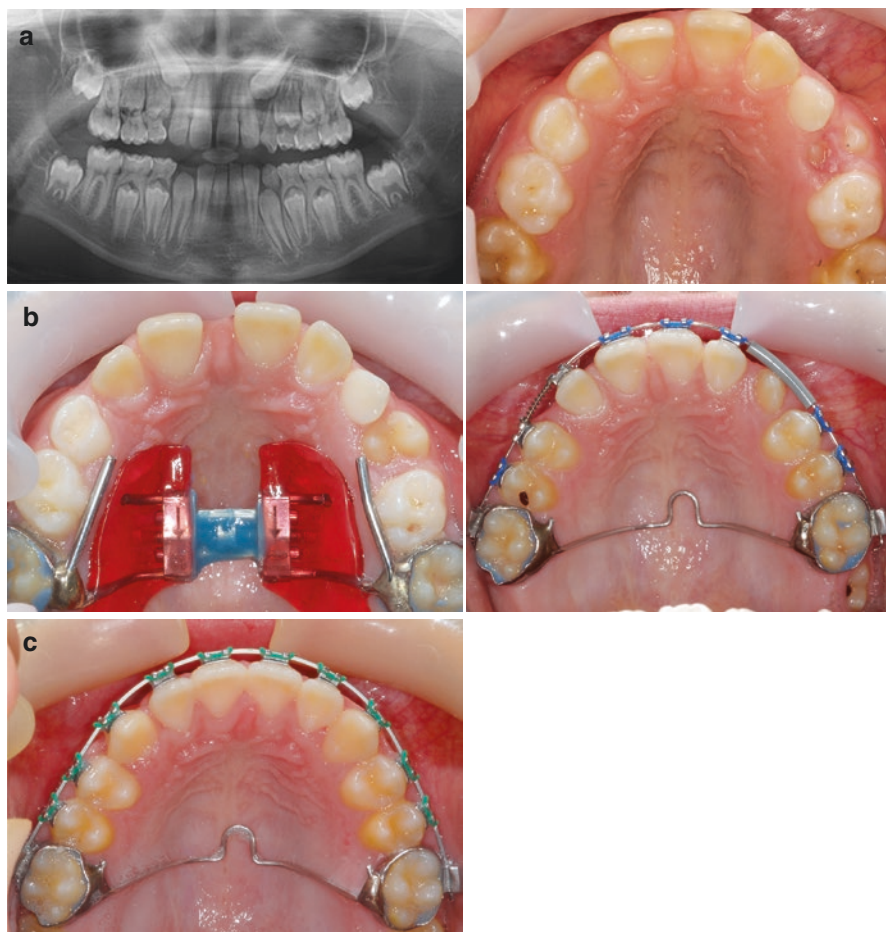


Fig. 8.7 (a) Pretreatment records of a patient aged 10 years 5 months with insufficient space for the maxillary canines and a displaced upper right permanent canine. (b) The patient was treated with RME initially to widen the maxillary arch and create space. The canine on the left erupted spontaneously 7 months after expansion was complete. (c) Appearance 25 months after expansion. The canine on the right erupted after 20 months of treatment with fixed appliances. Surgical exposure was not required

method of randomization and allocation concealment, no details of the primary or secondary outcomes and when they were to be assessed and the sample size calculation were incomplete. There was no flow diagram, no indication that the outcome assessor was masked as to group allocation and the number of withdrawals and drop-outs was small. Data from three intervention groups, including RME followed by placement of a transpalatal arch (TPA) and extraction of the primary canine; placement of a TPA and extraction of the primary canines and finally extraction of primary

canine only, were compared with data from young people in an untreated control group. The numbers of individuals in each group were different, as well as the length of the observation periods, and there was a high proportion diagnosed with bilaterally displaced canines (58 out of 88 or 66% in the intervention groups). The authors report that successful eruption of permanent canines occurred in 32 out of 40 (80%) of the young people in the RME group, which was no different to those who had just received a TPA and extraction of the primary canine (19 out of 24 individuals, 79%). Successful eruption occurred in 15 out of 24 (63%) of those who had extraction of the primary canine only, compared with 8 out of 29 (28%) in the untreated control group. The authors do helpfully provide an indication of success rates according to the initial horizontal displacement of the permanent canine, with 13 out of 14 individuals (93%) demonstrating a successful eruption of the canine when the canine was initially positioned in sector 1 (no overlap with the lateral incisor), compared with two out of seven individuals (29%) where the canine was in sector 4 (none were in sector 5, overlapping the central incisor by more than half of a line through the long axis).

A final article, from the same group, compared one intervention group (RME, placement of a TPA and extraction of the primary canine) with an untreated control group and included young people treated in Italy and the USA [35]. This study was only described as prospective, and there is no mention of random allocation in the article. The mean length of the observation period was 3 years 7 months in the treatment group and 3 years 1 month in the control group. The prevalence rates for successful eruption of the canines were 31 out of 39 (80%) in the treated group and 8 out of 29 (28%) in the untreated controls. Again the authors reported useful data about the success rates according to the initial horizontal position of the unerupted permanent canine. Five out of five young people (100%) with a canine in sector 1 had a successful outcome compared with one out of three (33%) in sector 4. The majority of the canines were initially diagnosed in either sector 2 (19 out of 21 or 90% successful eruptions) or sector 3 (six out of ten or 60% successful eruptions). In the control group, all the successful eruptions were with canines that were initially diagnosed in either sector 1 (six out of eight or 75% successful) or sector 2 (2 out of 14 or 14% successful).

Headgear

Silvola and colleagues undertook an investigation into the effects of cervical headgear treatment on the eruption pattern of maxillary canines in young people, with a mean age of 7.6 years (SD 0.3) [36]. The inclusion criteria were simply described in this article as moderate crowding and a Class II tendency, and it was not apparent that there was any clinical or radiographic diagnosis of PDC. The participants were randomly allocated to either a headgear or an untreated control group, although they do state that the controls received 'any necessary interceptive procedures'. A final examination was carried out after 8 years, although the data presented in this article were for changes in the canine angulation one or 2 years after recruitment. The authors do report the horizontal placement of the canines, but state that all the canines were in sectors 3, 4 or 5, with none in sectors 1 and 2. They confirm that there were no

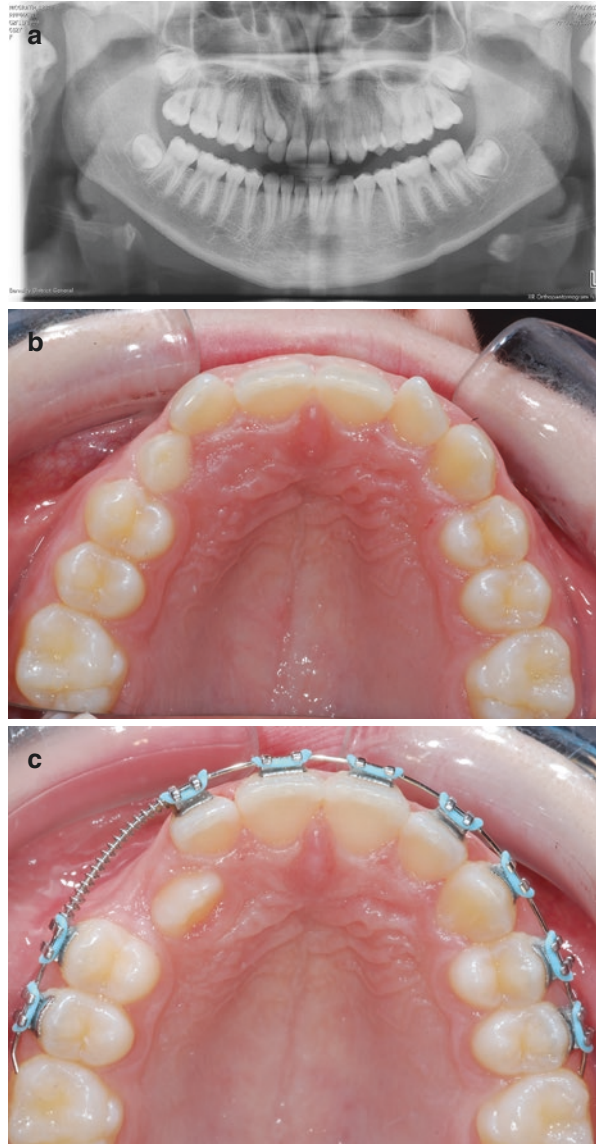
differences in the horizontal position of any of the permanent canines between the control and headgear groups; however, as the young people were still only about 9.5 years old, many clinicians and researchers in this area would consider that it is too early to diagnose that the maxillary permanent canine is displaced.

Armi and colleagues, from the same Italian group as several previous articles, compared two interventions (cervical pull headgear vs. RME and cervical pull headgear) with an untreated control group [37]. Once again the exact design of the study is not clear. The title suggests that allocation was random, whereas the methods initially mentions that the study was prospective, but later that the participants 'were assigned randomly'; however, few, if any requirements from the CONSORT guidelines, are included. The authors report a success rate (full eruption of the tooth permitting bracket placement) of 86% in the RME/headgear group compared with 82% in the headgear alone group. Few details (except the mesial movement of the upper molars) are available in this report, including the success rate of permanent canine eruption in the untreated controls.

Bonded Orthodontic Appliance

Olive advocates the extraction of the primary canine in patients with a suspected PDC, followed by creation of up to 1 cm more space than the width of the unerupted permanent canine [38, 39]. As in the patient illustrated (Fig. 8.8) space creation can be achieved with bonded appliances and/or rapid maxillary expansion by accepting an increased overjet and perhaps a shift of the upper centre line. In the first article, Olive summarizes the outcomes in 28 young people aged between 11.4 and 16.1 years diagnosed with PDC and treated with orthodontic appliances alone. All the patients had canines that were diagnosed in sectors 2 to 4 before treatment and Olive states that those in sectors 2 and 3 (19 out of the 28 patients) had been consecutively treated (although it is not clear if they were consecutively started or consecutively finished). Fifteen of the patients had had the primary canine extracted between 6 and 42 months (mean 15 months) prior to the start of active orthodontic treatment, and in the remaining 13 patients, the primary canines were removed within 4 months of starting active orthodontic treatment. In this sample 8 out of the 28 patients required surgical exposure, a success rate of 71%. Initial horizontal location was a strong predictor of success with only 1 out of 11 (9%) individuals with a canine in sector 2 requiring surgery, compared with 2 out of 12 (17%) in sector 3, but five out of eight (63%) when the canine was in sector 4. The treatment times for those who had successful eruption of their canines were between 2 and 27 months. Olive suggests that this approach can be used for young people under 16 years of age with unerupted permanent canines in sectors 1, 2 and 3, whereas the young people with an unerupted canine in sector 4 treatment should be started before the age of 13 years. He concluded that surgical exposure should be undertaken after 9 months of appliance treatment if the canine has still not erupted. In the follow-up article [39], Olive also confirms that the greater the horizontal impaction, the longer the canine takes to erupt; however, the samples from both articles appear to have been highly selected, so definitive conclusions are hard to produce.

Fig.8.8 An example of an adolescent aged 11 years with an impacted upper right canine. After 1 cm of space was created using an upper fixed appliance, the permanent canine took 5 months to start erupting. Total treatment time was 14 months and no surgery was required



Implications for Research

Published studies investigating early interventions to correct the position of a PDC frequently have absent details or inconsistencies in reporting. None claiming to have used random allocation are fully compliant with the CONSORT guidelines [40]. This has been reported previously by two systematic reviews summarizing the evidence for the extraction of the primary canine [21, 25]. It is essential that

future investigators design and report studies appropriately. Our summary of the current literature would suggest the following recommendations for further research in this area:

- The inclusion criteria should include young people in an appropriate age range, when the clinician can be reasonably certain that palatal displacement of the upper permanent canine can be diagnosed. We would suggest a lower age limit of 10 years and an upper age limit of 13 or 14 years depending upon the stage of dental development.
- Young people with both unilateral and bilateral PDCs can be included in trials; however, participants with bilateral displaced canines should be allocated according to the participant, not the teeth and both sides should receive the same treatment (or no treatment). Sample sizes should be estimated on the basis of participant numbers, not the number of displaced teeth.
- A clear and stated definition of when the upper canine is considered displaced should be provided. The work of Ericson and Kurol described earlier is useful for this [11, 12]. Angulation has been used by some authors, but in the opinion of the current authors, it is not such a clear indicator of displacement as medial horizontal position (sector).
- The exclusion criteria of Bazargani et al. seem reasonable: patients with previous or ongoing orthodontic treatment; aplasia or microdontia of the upper lateral incisors; moderate to severe crowding in the upper arch (>3 mm) and craniofacial syndromes, odontomas, cysts, or cleft lip or palate [30].
- The required sample size for any trial should be estimated on the basis of a clinically relevant primary outcome. This should be whether or not the canine erupts sufficiently to permit bracket placement without the need for any surgical exposure. This is surely the aim of the intervention. If the position of the canine improves, but the patient still has to undergo a surgical exposure, then in the view of the authors the intervention has failed to show any clinical effectiveness. A clinically relevant outcome should be the difference in the proportions of canines that successfully erupt with the intervention (or interventions in a factorial study design) compared with an untreated control group.
- A reasonable and predetermined follow-up period should be used (between 18 months and 2 years, unless the patient is at the older end of the ideal age range).
- There is evidence in the literature that the success of an intervention depends upon the initial horizontal displacement of the canine, i.e. the further towards the midline the canine is at the start, the less successful the intervention. The initial horizontal displacement (sector) of the canines should be fully described and success rates per initial sector should be reported. In addition, investigators should consider stratifying the randomization of participants according to the initial horizontal sector to ensure that each intervention group has equal numbers of severely displaced canines.

Implications for Clinical Practice

Taking into account the findings in the current literature, we recommend the following guidelines for the management of unerupted maxillary permanent canines in children, with a dental age between 10 and 13 years, using conventional radiographic examination:

Canines in sector 1 with an angle of less than 21° to the midline (coloured green in Fig. 8.9)

These are unlikely to be displaced and are of little concern. They should erupt in time provided that space is available.

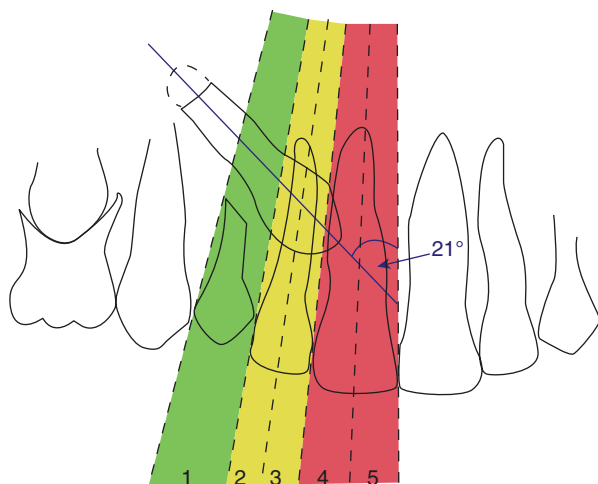
Canines in sectors 2 and 3 with an angle of less than 21° to the midline (coloured yellow Fig. 8.9)

Interceptive treatment should be considered, either in the form of creating space or extraction of the primary canine (or both). The outcome of this treatment is questionable; however, there is little to lose if the root of the primary canine is already resorbing and the patient is willing.

Canines in sectors 4 and 5 (coloured red in Fig. 8.9)

These canines will require surgical exposure and alignment. There is therefore no advantage to extracting the primary canine and risking the patient not being suitable for fixed appliances when the eruption of the permanent canine fails to normalize. It is better to leave the patient with a primary canine than an unsightly gap. In these cases, where the canine is considerably displaced, the primary canine root is often of a reasonable length and has a good medium-to-long-term prognosis.

Fig. 8.9 Classification of the horizontal position of the unerupted maxillary permanent canine according to Ericson and Kuroi [12]. The colour coding suggests that unerupted canines in the *green area* are unlikely to be displaced and are of little concern. Canines in the *yellow area* might respond to intervention, which should be considered. Canines in the *red zone* are unlikely to respond to intervention



References

1. Berkovitz BKB, Holland GR, Moxham BJ. Oral anatomy, histology and embryology. 4th ed. Edinburgh: Mosby Elsevier; 2009.
2. Coulter J, Richardson A. Normal eruption of the maxillary canine quantified in three dimensions. *Eur J Orthod.* 1997;19(2):171–83.
3. Ericson S, Kurol J. Longitudinal study and analysis of clinical supervision of maxillary canine eruption. *Community Dent Oral Epidemiol.* 1986a;14(3):172–6.
4. Ericson S, Kurol J. Radiographic examination of ectopically erupting maxillary canines. *Am J Orthod Dentofac Orthop.* 1987;91(6):483–92.
5. Thilander B, Myrberg N. The prevalence of malocclusion in Swedish schoolchildren. *Scand J Dent Res.* 1973;81(1):12–21.
6. McSherry P, Richardson A. Ectopic eruption of the maxillary canine quantified in three dimensions on cephalometric radiographs between the ages of 5 and 15 years. *Eur J Orthod.* 1999;21(1):41–8.
7. Becker A, Chaushu S. Etiology of maxillary canine impaction: a review. *Am J Orthod Dentofac Orthop.* 2015;148(4):557–67. doi:10.1016/j.ajodo.2015.06.013.
8. Rutledge MS, Hartsfield JK. Genetic factors in the etiology of palatally displaced canines. *Semin Orthod.* 2010;16(3):165–71.
9. Chaushu S, Kaczor-Urbanowicz K, Zadurska M, Becker A. Predisposing factors for severe incisor root resorption associated with impacted maxillary canines. *Am J Orthod Dentofac Orthop.* 2015;147(1):52–60. doi:10.1016/j.ajodo.2014.09.012.
10. Shetty R, Sandler PJ. Keeping your eye on the ball. *Dent Update.* 2004;31(7):398–402.
11. Ericson S, Kurol J. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbance. *Eur J Orthod.* 1986b;8(3):133–40.
12. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. *Eur J Orthod.* 1988;10(4):283–95.
13. Lindauer SJ, Rubenstein LK, Hang WM, Andersen WC, Isaacson RJ. Canine impaction identified early with panoramic radiographs. *J Am Dent Assoc.* 1992;123(3):91–2. 95–97
14. Warford JH Jr, Grandhi RK, Tira DE. Prediction of maxillary canine impaction using sectors and angular measurement. *Am J Orthod Dentofac Orthop.* 2003;124(6):651–5. doi:10.1016/S0889540603006218.
15. Fernandez E, Bravo LA, Canteras M. Eruption of the permanent upper canine: a radiologic study. *Am J Orthod Dentofac Orthop.* 1998;113(4):414–20.
16. Sambataro S, Baccetti T, Franchi L, Antonini F. Early predictive variables for upper canine impaction as derived from posteroanterior cephalograms. *Angle Orthod.* 2005;75(1):28–34. doi:10.1043/0003-3219(2005)075<0028:EPVFUC>2.0.CO;2.
17. Sajnani AK, King NM. Early prediction of maxillary canine impaction from panoramic radiographs. *Am J Orthod Dentofac Orthop.* 2012;142(1):45–51. doi:10.1016/j.ajodo.2012.02.021.
18. Peck S (2011) Problematic sample in the study of interception of palatally displaced canines. *Am J Orthod Dentofac Orthop.* 140(1):2–3; author reply 3–4. doi:10.1016/j.ajodo.2011.05.004
19. Bazargani F, Magnuson A, Dolati A, Lennartsson B. Palatally displaced maxillary canines: factors influencing duration and cost of treatment. *Eur J Orthod.* 2013;35(3):310–6. doi:10.1093/ejo/cjr143.
20. Iramaneerat S, Cunningham SJ, Horrocks EN. The effect of two alternative methods of canine exposure upon subsequent duration of orthodontic treatment. *Int J Paediatr Dent.* 1998;8(2):123–9.
21. Parkin N, Furness S, Shah A, Thind B, Marshman Z, Glenroy G, Dyer F, Benson PE (2012) Extraction of primary (baby) teeth for unerupted palatally displaced permanent canine teeth in children. *Cochrane Database Syst Rev* 12:CD004621. doi:10.1002/14651858.CD004621.pub3
22. Buchner HJ. Root resorption caused by ectopic eruption of maxillary cuspid. *Int J Orthod.* 1936;22:1236–7.

23. Power SM, Short MB. An investigation into the response of palatally displaced canines to the removal of deciduous canines and an assessment of factors contributing to favourable eruption. *Br J Orthod.* 1993;20(3):215–23.
24. Leonardi M, Armi P, Franchi L, Baccetti T. Two interceptive approaches to palatally displaced canines: a prospective longitudinal study. *Angle Orthod.* 2004;74(5):581–6. doi:[10.1043/0003-3219\(2004\)074<0581:TIATPD>2.0.CO;2](https://doi.org/10.1043/0003-3219(2004)074<0581:TIATPD>2.0.CO;2).
25. Naumova J, Kuroi J, Kjellberg H. A systematic review of the interceptive treatment of palatally displaced maxillary canines. *Eur J Orthod.* 2011;33(2):143–9. doi:[10.1093/ejo/cjq045](https://doi.org/10.1093/ejo/cjq045).
26. Baccetti T, Leonardi M, Armi P. A randomized clinical study of two interceptive approaches to palatally displaced canines. *Eur J Orthod.* 2008;30:381–5.
27. Bonetti GA, Parenti SI, Zanarini M, Marini I. Double vs single primary teeth extraction approach as prevention of permanent maxillary canines ectopic eruption. *Pediatr Dent.* 2010;32(5):407–12.
28. Bonetti GA, 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 Dentofac Orthop.* 2011;139(3):316–23. doi:[10.1016/j.ajodo.2009.03.051](https://doi.org/10.1016/j.ajodo.2009.03.051).
29. Koletsi D, Pandis N, Polychronopoulou A, Eliades T. Does published orthodontic research account for clustering effects during statistical data analysis? *Eur J Orthod.* 2012;34(3):287–92. doi:[10.1093/ejo/cjr122](https://doi.org/10.1093/ejo/cjr122).
30. 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](https://doi.org/10.2319/031013-205.1).
31. Naumova J, Kuroi 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.* 2015a;37(2):209–18. doi:[10.1093/ejo/cju040](https://doi.org/10.1093/ejo/cju040).
32. Naumova J, Kuroi J, Kjellberg H. Extraction of the deciduous canine as an interceptive treatment in children with palatally displaced canines-part II: possible predictors of success and cut-off points for a spontaneous eruption. *Eur J Orthod.* 2015b;37(2):219–29. doi:[10.1093/ejo/cju102](https://doi.org/10.1093/ejo/cju102).
33. 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 Dentofac Orthop.* 2009;136(5):657–61. doi:[10.1016/j.ajodo.2008.03.019](https://doi.org/10.1016/j.ajodo.2008.03.019).
34. Baccetti T, Sigler LM, McNamara JA Jr. An RCT on treatment of palatally displaced canines with RME and/or a transpalatal arch. *Eur J Orthod.* 2011;33(6):601–7. doi:[10.1093/ejo/cjq139](https://doi.org/10.1093/ejo/cjq139).
35. Sigler LM, Baccetti T, McNamara JA Jr. Effect of rapid maxillary expansion and transpalatal arch treatment associated with deciduous canine extraction on the eruption of palatally displaced canines: A 2-center prospective study. *Am J Orthod Dentofac Orthop.* 2011;139(3):e235–44. doi:[10.1016/j.ajodo.2009.07.015](https://doi.org/10.1016/j.ajodo.2009.07.015).
36. Silvola AS, Arvonen P, Julku J, Lahdesmaki R, Kantomaa T, Pirttiniemi P. Early headgear effects on the eruption pattern of the maxillary canines. *Angle Orthod.* 2009;79(3):540–5. doi:[10.2319/021108-83.1](https://doi.org/10.2319/021108-83.1).
37. Armi 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):370–4. doi:[10.2319/062210-339.1](https://doi.org/10.2319/062210-339.1).
38. Olive RJ. Orthodontic treatment of palatally impacted maxillary canines. *Aust Orthod J.* 2002;18(2):64–70.
39. Olive RJ. Factors influencing the non-surgical eruption of palatally impacted canines. *Aust Orthod J.* 2005;21(2):95–101.
40. Moher D, Hopewell S, Schulz KF, Montori V, Gotzsche PC, Devereaux PJ, Elbourne D, Egger M, Altman DG. CONSORT 2010 explanation and elaboration: updated guidelines for reporting parallel group randomised trials. *Br Med J.* 2010;340:c869.

Andrew DiBiase and Paul Jonathan Sandler

Abstract

An increased overjet in the primary or mixed dentition is a common reason to seek orthodontic treatment and is usually indicative of an underlying class II malocclusion. This can be due to a variety of factors, including digit sucking, a lip trap or an underlying skeletal II base relationship. Treatment timing has been controversial, with proponents of early treatment claiming it results in greater growth of the mandible and better outcomes for the patient. However, evidence from several large randomised clinical trials investigating early treatment for class II malocclusion have refuted this, essentially showing few clinical differences in outcome for patients who underwent an early course of treatment in the mixed dentition compared to those treated comprehensively in adolescence. However, patients treated early do seem to experience less dentoalveolar trauma than those treated later, although this is generally not severe, and it is debatable whether the slight reduction in risk justifies the cost and burden to the patient of early treatment. Another justification for early treatment is psychological outcome. An increased overjet has been shown to make a child a target for bullying, and there is weak evidence that early treatment can help these patients. If early treatment is embarked upon, there are several modalities that can be used, one of which is a functional appliance. These appliances primarily reduce an increased overjet by dental movement, retroclining the upper incisors and proclining the lowers. There is a small increase in mandibular length, but this disappears with normal growth. Most patients will need a further course of treatment, which will

A. DiBiase (✉)

East Kent Hospitals University NHS Foundation Trust, Canterbury, UK

e-mail: andrewdibiase@nhs.net

P.J. Sandler

Chesterfield Royal Hospital, Chesterfield, UK

University of Sheffield, Sheffield, UK

e-mail: JonSandler57@gmail.com

mean maintaining overjet reduction in the transition from early mixed to permanent dentitions. In most cases, it is more efficient and less demanding on patient compliance to delay treatment until early adolescence in the late mixed dentition, as clinical outcome is likely to be the same.

Introduction

Class II malocclusion is very common in most developed countries, and the features can be seen even in the primary dentition. It usually presents as an increased overjet, which is often a cause of concern to the patient or their parents, and as a consequence, a specialist opinion is frequently sought. While it is possible to treat class II malocclusions in the mixed and sometimes even in the primary dentition, considerable discussion and debate has taken place as to the ‘ideal timing’ of treatment. This chapter will explore this controversial area.

Incidence

Class II malocclusions are common in Western societies and Caucasian patients of Northern European descent, with an incidence of up to 25% having been reported in 12 years old in the UK [1] and 15% of 12–15 year olds in the USA [2]. Class II is less common in Afro-Caribbean and East Asian populations and they tend to be a higher propensity to class III malocclusions.

The prevalence of a class II molar relationships is relatively high in the primary dentition [3]. This reduces in untreated subjects as they enter the mixed and early permanent dentition as the mandibular first molars migrate mesially with exfoliation of the second primary molars. This also reflected in a reduction in the overjet to a lesser extent, although this is less likely to occur if the overjet is over 6 mm [3]. Once a class II buccal segment relation has become established in the permanent dentition, it will tend to be maintained, even with good mandibular growth. This is because dentoalveolar compensation occurs thus maintaining the occlusion despite the growth [4]. As a consequence, class II malocclusions do not usually self-correct without active intervention.

Aetiology

Skeletal

The majority of class II malocclusions present with some degree of mandibular retrognathia [5]. Maxillary prognathism is much less common and is often associated with vertical maxillary excess. Vertical face type and growth are variable, ranging from patients with hypodivergent facial growth patterns and increased overbite to those who have a hyperdivergent pattern, an increased anterior low face height and a skeletal anterior open bite.

Soft Tissue

The teeth sit in a 'zone of balance' between the soft tissues of the lips and cheeks buccally and the tongue lingually. It is therefore unsurprising that the soft tissues have a significant influence on the position of the developing dentition. The position and competence of the lips has a bearing on the position of the labial segments. If the lower lip rests behind the upper incisors, these will be proclined and the lowers reclined, resulting in an increased overjet. This is known as a lip trap and the lip pattern described as potentially competent. If the lower lip position is higher, it can result in retroclination of the upper central incisors but still rest behind the lateral incisors allowing them to procline, resulting in the classic presentation of a class II division 2 incisor relationship.

Lip incompetence and hypotonic activity, often associated with hyperdivergent facial growth, can result in the tongue having more influence on the incisors. Clinically this manifests itself as bimaxillary proclination and a reduction in the overbite. If this occurs with a class II skeletal pattern, an increased overjet can result as well as a reduced overbite or possibly an anterior open bite.

It has been argued that tongue position and poor lip posture are the primary aetiology of class II malocclusion in childhood [6]. The theory goes that due to nasal obstruction, oral breathing predominates, resulting in lip incompetence and an open mouth posture. The tongue position then drops and the maxillary arch narrows, resulting in crowding and a downwards and backwards growth rotation of the mandible. This in turn shortens the lower dental arch resulting in secondary crowding appearing in the mandibular dentition. There is limited evidence for this hypothesis from primate experiments and human studies looking at the effect of adenoidectomies on the growth in children and adolescents [7].

Advocates of this argument encourage early treatment for class II malocclusion usually consisting of a combination of myofunctional appliances and oral exercises designed to restrain the tongue and establish lip competence. By doing this, it is believed that greater anteroposterior mandibular growth will result, thereby correcting the class II malocclusion. There is however no scientific or clinical evidence to support this philosophy or to justify this type of early treatment. What is more, the treatment modalities advocated are extremely demanding on compliance, extend over many years and have by the clinicians' own admissions a very low success rate compared with other types of treatment.

Digit or Thumbsucking

Nonnutritive sucking habits are common in many societies but usually stop in the primary dentition [8]. If this persists into the mixed dentition, this can affect the dental arches and occlusion, the severity being dependent on the duration of the habit [9]. Clinically this often results in the development of a posterior crossbite and an increased overjet as the upper arch narrows, the upper incisors are proclined and the lower incisors retroclined [10]. This can also result in a reduced overbite or an anterior open bite [11].

Indications for Early Treatment

There has been much debate regarding the optimum time to start treatment for class II malocclusions, and early treatment has many advocates. The proposed advantages of early treatment are:

- Maximise growth potential
- Psychosocial benefits
- Reduce risk of dentoalveolar trauma
- Good compliance in younger patients
- Reduce need or complexity of second phase of treatment
- Better overall outcomes

However set against this are the following contraindications:

- Extended treatment time
- Retention problematic during transition of dentition
- Physiological cost of prolonged treatment
- Use up patient cooperation
- Cost to patient and parent—both economic and time

Many of the claims in favour of early treatment were based on retrospective research with small sample sizes, which were often compared to historic controls. In the 1990s, several large randomised clinical trials were set up to try and address the fundamental question of timing for the treatment of class II malocclusions: two in the USA and one in the UK [12–15]. The studies in the USA investigated the use of functional appliances, a Bionator, versus headgear or observation. They were based in dental schools with treatment carried out by a limited number of operators and sometimes involved patient incentivisation to comply with the study. They therefore investigated the *efficacy* of treatment, i.e. the provision of care under ideal conditions rather than its effectiveness. The UK-based study compared early treatment with a functional appliance, a Twin Block, to an observation group. Treatment in this study was carried out by numerous operators in hospital-based orthodontic departments in the UK. It therefore investigated the *effectiveness* of treatment, i.e. the provision of care under conditions that are more relevant to the setting where the proposed treatment is usually carried out. The studies initially reported following the first phase of treatment. The patients were then followed through comprehensive orthodontic treatment in adolescence [16–18]. Overall over 600 patients were initially enrolled in these studies with almost 500 completing them, and so to date, they provide the best evidence available on the outcomes and benefits of early treatment for class II malocclusions. So we need to look at the claims outlined above on the supposed benefits of early treatment in specific relationship to these studies.

Growth

There is no doubt early treatment is effective at reducing an increased overjet, and this is achieved by a combination of dental and skeletal effects. Therefore all three studies reported positive results following the initial treatment including a relative increase in mandibular length measured cephalometrically in the patients treated with the functional appliances and maxillary restraint in those treated with headgear. However once the patients were followed through to the end of the study, these differences had disappeared, and there was no difference skeletally between the patients who had undergone early treatment and those who had undergone later treatment. Therefore to date there is no evidence that early treatment for class II malocclusion has any lasting impact on growth, and therefore 'to achieve better growth' is not a reason to undertake early treatment.

Psychosocial Health

There is an increasing body of evidence that the presence of a malocclusion can have a negative impact on an individual's quality of life and psychological health. This is particularly relevant to class II malocclusions which can be particularly aesthetically conspicuous. Both in childhood and adolescence compromised aesthetics can make an individual more susceptible to teasing and bullying. Bullying is endemic within school populations in most countries. In the presence of a malocclusion, bullying has a negative impact on self-esteem and oral-health-based quality of life including lower levels of social competence, athletic competence, self-esteem related to physical appearance and general self-esteem [19]. In addition bullied individuals report higher levels of symptoms, functional limitations and emotional and social impact from their malocclusions. Combined, these factors can have a long-term negative impact on individuals and are associated with both poor psychological and physical health, including low self-esteem, depression, anxiety, poor academic performance, truancy, crime, mental health problems and suicide.

Despite no evidence of long-term impact of early treatment on self-esteem, it appears to result in a short-term increase in self-esteem and a reduction in the self-reported levels of bullying, as well as a positive impact on the oral health-related quality of life [13, 14, 20]. In certain well-motivated individuals, therefore, early treatment may well be very beneficial (Figs. 9.1, 9.2, 9.3 and 9.4). However this needs to be done on the understanding that ultimately it may result in extended treatment times and quite possibly a second course of treatment with no discernable difference in the final outcome, compared to one course of treatment in adolescence.



Fig. 9.1 8-year-old girl who presented with a 15 mm overjet who was being bullied at school about her dental appearance



Fig. 9.2 Patient from Fig. 9.1 in treatment with a Twin Block functional appliance



Fig. 9.3 Patient from Fig. 9.1 following early treatment



Fig. 9.4 Pre- and post-treatment lateral cephalograms for patient from Fig. 9.1

Prevention of Dentoalveolar Trauma

An increased overjet of over 6 mm has been associated with a higher incidence of trauma to the upper labial segment particularly when associated with lip incompetence [21]. The incidence is highest in children in the mixed dentition, i.e. patients aged 8–11 years old. While the trauma is usually mild, usually consisting of fractures within enamel, it can on occasion be more severe such as fractures into dentine and the pulp, root fractures and rarely avulsion resulting in complete tooth loss, all of which have long-term consequences in terms of treatment and cost.

The only small positive difference found in the class II RCTs was the slightly reduced incidence in dentoalveolar trauma in the patients who had undergone early treatment. This was not actually found in the individual studies, but when the results were combined in a meta-analysis, a difference between the early treatment group and late treatment group was found with less incidence of new trauma during the study period in the early treatment group [22].

The overall incidence of dentoalveolar trauma in childhood has been reported as 1–3%, and the cost of treatment has been reported to range from US \$2 to \$5 million per one million inhabitants with patients usually requiring 2–9 dental appointments to complete the treatment [23]. Most of the new trauma reported in the RCTs was mild in nature and clinically negligible not requiring treatment, and therefore it is debatable whether the extra cost related to early treatment could be justified. Also much of the trauma occurs in the early mixed dentition

as the permanent incisors erupt due to falls, sports or nonaccidental injury [23]. To have any really meaningful impact, therefore, treatment would need to be started soon after the permanent incisors erupt which may have an impact on compliance and overall duration of treatment, as well as cost. To prevent injury during sport, use of a mouthguard maybe more cost-effective and less demanding than early treatment. Finally even a slight increase in overjet of over 3–4 mm increases the risk of trauma by 21.8% (95% CI 9.7–34.5%) [3, 24]. This relatively would mean many more children would require early treatment, which is again neither cost-effective nor practical, particularly in a state-funded health system. However in certain children with very prominent maxillary incisors and lip incompetence who are particularly physically active and deemed at high risk of dentoalveolar trauma, early treatment can be justified (Figs. 9.4, 9.5, 9.6, 9.7, and 9.8).



Fig. 9.5 9-year-old male in mixed dentition with class II div 1 incisor relationship. Early treatment was carried out as there was gross lip incompetence increasing the risk of dentoalveolar trauma



Fig. 9.6 Patient from Fig. 9.5 in treatment with a Bionator functional appliance



Fig. 9.7 Patient from Fig. 9.5 at the end of the first phase of treatment showing reduction in overjet and improvement in soft tissue profile. The patient went on to have comprehensive treatment with fixed appliances and extraction of the upper left first molar which was hypoplastic

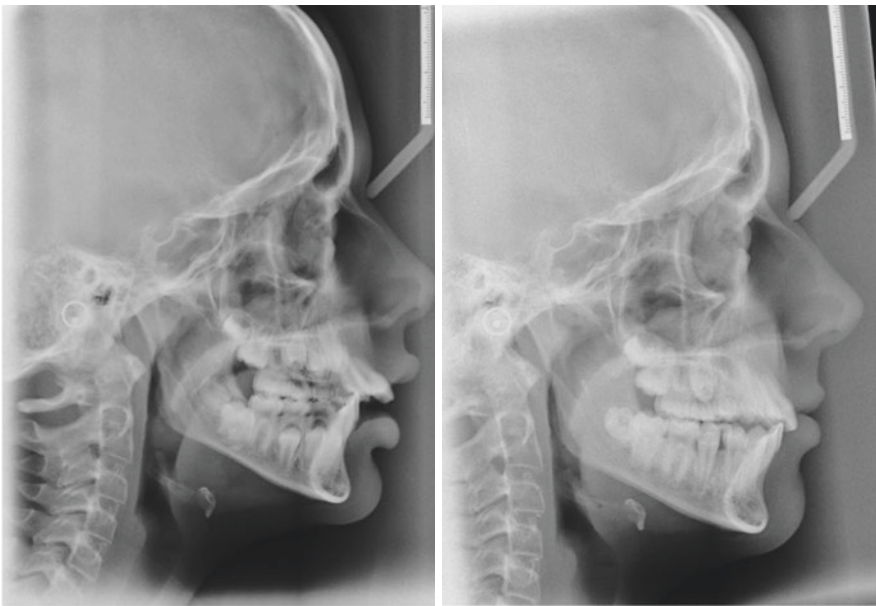


Fig. 9.8 Pre- and post-treatment lateral cephalogram from patient in Fig. 9.5 showing mostly dentoalveolar changes

Compliance

Good patient cooperation is fundamental to successful orthodontic treatment. This is often extremely difficult to gauge, and there appears to be no psychosocial parameters that can predict this. An assessment of potential cooperation can be made by observing the initial patient behaviour and in particular the relationship with the orthodontist or treating clinician [25]. Poor oral hygiene, repeated breakages, failure to wear appliances as instructed and a poor patient/clinician relationship are often indicators of poor overall outcome [26, 27].

Preadolescent children generally make very good patients as their behaviour is more affected by figures of authority such as their parents or the orthodontist. As long as instructions are not abstract or relate to long-term outcomes, compliance levels are generally very good. As a child enters adolescence, their behaviour becomes more influenced by their peer group, and they tend to rebel against authority. Successful treatment therefore involves empowering the patient, so that they feel the treatment is being done for them as opposed to them. This again involves including them in decision-making and developing a good patient-clinician relationship. Fortunately acceptance of treatment in this age group has become easier by the more universal availability of orthodontic treatment in most developed countries and the greater awareness of malocclusion and overall body image, plus the undeniable benefits of orthodontic treatment.

A problem with early treatment can be compliance 'burn out' as early treatment in the majority of cases will extend overall treatment time dramatically, and we know that extended treatment duration has a negative influence on cooperation [28]. Therefore there is a risk that the compliant eight year old will become a disgruntled 12 year old after 4 years in treatment. As no study to date has shown convincingly any major benefits in early treatment, particularly in relation to better outcomes, the argument of better compliance of younger patients cannot really be used to justify it as ultimately the majority of patients will still further treatment in adolescence.

Second Phase of Treatment

The majority of patients undergoing early treatment for class II malocclusion will require a further course of active orthodontic treatment according to the three RCTs previously discussed. This need for treatment can be for a variety of reasons but usually involves relieve of crowding and alignment of the teeth, detailing the occlusion or to fully reduce a residual increased overjet. Furthermore the extraction rates and the duration of any subsequent treatment, usually with upper and lower fixed appliances or the percentage of patients requiring orthognathic surgery, appear to be no different between the patients that underwent phase 1, early treatment and those

patients that waited and had comprehensive treatment at the more usual time in early adolescence. The early treatment of class II malocclusion therefore can also not be justified to reduce the need or duration of second phase of treatment, based on the current scientific evidence available.

Better Outcomes

In the three RCTs critically assessing early treatment for class II, the occlusal outcomes of the treated groups after the initial phase with both functional appliances and headgear were better than those in the control group: both modalities were demonstrated to be effective at reducing an increased overjet. Following comprehensive treatment, however, these differences disappeared. Indeed, overall the patients in the early treatment groups reported significantly longer treatment times overall, had a greater number of visits and, in the UK study, had a poorer occlusal result as measured by the Peer Assessment Rating (PAR) [17]. On the current evidence, therefore, it is not possible to justify early treatment for class II malocclusion on an expectation of a better occlusal result, compared with comprehensive treatment started in the late mixed or early permanent dentition.

Mechanics for Early Treatment of Class II Malocclusion

So overall, while early treatment using the methods outlined later in this chapter can certainly be effective, it is questionable whether it is the most efficient way to treat class II malocclusions. If it is decided, however, that a course of early treatment is justified, and in the patient's best interests, there are a variety of ways that it can be carried out.

Thumb Deterrents

A digit sucking habit should ideally stop before eruption of the permanent incisors; otherwise it can result in long-term dental and skeletal changes, as outlined previously. If persistent into the early permanent dentition, the child should be actively encouraged to stop, and numerous techniques have been described to assist in this. If the child struggles to break the habit on their own, a passive device such as a palatal arch incorporating a thumb or tongue crib can be effective [29] (Fig. 9.9).

Fig. 9.9 An anterior open bite and increased overjet as a result of thumbsucking. A palatal arch with spurs was used to break the habit, and there was an improvement in the incisal relationship



Removable Appliances

A removable appliance with an activated labial bow can be used to reduce an increased overjet in the mixed dentition. This is only appropriate if the upper incisors are proclined and spaced, as the appliance will simply retrocline them by tipping of the teeth. An anterior bite plane can be incorporated to help reduce an

increased overbite, and headgear can also be used as outlined below. It is an inappropriate treatment in patients with a mark skeletal II base relationship, and mandibular retrognathia as simple dentoalveolar tipping is unlikely to produce a satisfactory result.

Functional Appliances

An extremely effective way of reducing an increased overjet in the mixed dentition is with the use of functional appliances. These are a class of orthodontic appliances originally developed in Europe in the early twentieth century that were believed to have an effect on facial growth. While many different designs and systems have been described the basic premise on which they all work is by posturing the mandible forward. This achieves several things: it changes the soft tissue environment and as a result alters forces that influence the position of the dentition. It exerts direct force on the teeth via the appliance, from the forces generated by the stretch of the muscles controlling the mandible trying to return to their resting length. In most cases, this results in a distalising force being transmitted to the upper jaw and the maxillary dentition and a mesialising force being transmitted to the mandible and the lower dentition. It has also been suggested that there is some bony remodelling at the condyle and glenoid fossa. Combining all of these influences is very effective at reducing increased overjets via:

Retroclination of the upper incisors

Proclination of the lower incisors

Distal tipping of the maxillary dentition

Mesial eruption of the mandibular dentition

Some small but worthwhile restriction in maxillary growth

Repositioning of mandible anteriorly with some remodelling of glenoid fossa.

Whether functional appliances have a lasting effect on facial growth has remained one the most hotly debated topics in orthodontics, with passionate supporters of both opposing viewpoints. Advocates claim that the use of a functional appliance results in a significant improvement in appearance as a result of an increase in mandibular growth. Unfortunately many of these claims were based on case reports or retrospective studies, often comparing a small treated group to a historic sample. There was also some evidence from animal studies that mandibular hyperpropulsion with a fixed splint did result in bony change at the condyle and glenoid fossa. Whilst animal models are interesting, these experiments imposed treatment regimes on either rodents or primates that would just not be tolerated clinically. Also while these experiments show histological changes, as class II malocclusions do not exist in the animal models used, it is difficult to imagine how these changes would relate to a meaningful clinical difference in a patient.

With the publication of the large RCTs over the last two decades, it has become apparent that the early use of functional appliances, while very effective at reducing

an increased overjet, appears to have little or no long-term impact on facial growth. This is not to say that by doing nothing, an increased overjet and class II malocclusion will correct spontaneously. Indeed an untreated class II malocclusion will almost certainly persist into adolescence and adulthood due to maintenance of the occlusal relationship irrespective of growth [4]. The clinical effect of these functional appliances therefore appears to be early establishment of a class I occlusion, while then allowing normal condylar growth to consolidate this. And herein lies one of the major problems of early treatment. The most effective time to use these appliances is during the adolescent growth spurt [30]. In females this starts around 10 years of age with the peak at about 11.5 years. In males the growth starts between 11 and 12 years and peaks between 14 and 15 years. If early treatment is undertaken, therefore, it does not coincide with the growth spurt, particularly in males. Treatment will be less efficient than if undertaken in the late mixed or even early permanent dentition. Also unless the achieved results are effectively retained, the beneficial clinical effects will be lost as the patient enters the growth spurt, thus necessitating a second course of functional appliances.

A practical problem with the use of removable functional appliances in the mixed dentition, particularly a largely tooth-borne appliance such as Twin Block, is retention of the appliance in the mouth (see Figs. 9.1, 9.2, 9.3, and 9.4). Primary teeth are generally not ideal teeth to attach a crib to, due to their conical shape and lack of natural undercuts. These teeth can also become mobile as they begin to exfoliate thus further reducing their function for retention. This problem can sometimes be overcome by the addition of composite to create an undercut or by the use of cemented functional appliances such as a Herbst. Finally there is the option of using non-tooth-borne or partially tooth-borne removable appliance such as a functional regulator or Balter's Bionator. The former is not an easy to appliance to wear and is prone to distortion or breakage while the later has the potential advantage of allowing the natural shedding of the primary molars. In a child for whom early treatment is being advocated on psychosocial grounds due to bullying and teasing, the Bionator also has the advantage of them not having to wear the appliance to school, thus avoiding making them more of a target for abuse by their peers (see Figs. 9.5, 9.6, 9.7, and 9.8).

The final problem with early treatment with a functional appliance is knowing what to do once the overjet is reduced to maintain this reduction as the patient enters their adolescent growth spurt and while the permanent dentition establishes itself. Ideally the patient enters a period of retention while the appliance is worn at night, although this may be for several years depending on when treatment was started, which can put a strain on future compliance. Also it may necessitate the use of a further appliance such as a removable retainer with headgear added at night. The second option is to give the child a break from treatment by stopping appliance wear; however this runs the risk of relapse and reappearance of the overjet as the class II malocclusion re-establishes itself. Either way the patient and their carers should be fully informed of these potential outcomes before early treatment is started.

Headgear

Headgear for the treatment of class II malocclusion can be used with a removable or functional appliance, or on its own, and it has been shown to be effective at overjet reduction in the mixed dentition. Classically headgear can be run to maxillary molar bands while the patient wears an ACCO (acrylic cervical occipital) appliance to reduce the overbite and distalise the maxillary molars correcting the buccal segment relationship. In the two RCTs looking at early treatment for class II carried out in the USA, headgear was compared to an observation group and a group treated with a functional appliance [12, 15]. Both in terms of morphological traits and dentoalveolar trauma, there was no difference in the outcomes for the headgear patients compared with the patients treated with a functional appliance after the initial phase of treatment, i.e. both modalities of treatment essentially did the same thing, and both were effective at reducing overjets. As with the patients treated with functional appliances, however, these differences disappeared in the headgear patients compared with the observation group at the end of comprehensive treatment.

Practically, the use of headgear with or without a removable appliance also has the problems of retention and what to do during the transition from the mixed into the permanent dentition.

Fixed Appliances

Similarly to the use of a removable appliance, if space is available with the dental arch, a fixed appliance can be used to reduce an increased overjet. The main problem with the use of fixed appliances in the mixed dentition is bonding brackets and attaching wires to the primary teeth, as this may increase their mobility and thus hasten their loss. This is usually why the use of removable or functional appliances is often preferred.

Conclusions

Class II malocclusion is extremely common and is usually evident in the mixed dentition when it can effectively be treated using a variety of treatment modalities. There is however no evidence that treatment at this stage is superior in terms of morphological outcomes to comprehensive treatment carried out once the permanent dentition has become established. Indeed early treatment will result in an overall greater treatment time, a larger number of appointments and higher cost to the patient or state, depending on who is paying. It can perhaps be justified in terms of risk-benefit analysis in patients with very prominent maxillary incisors with lip incompetence who have an active lifestyle are considered more at risk of dentoalveolar trauma. Similarly early treatment is worth considering in patients with very prominent upper incisors who are experiencing sustained bullying specifically due to their dental appearance. Before treatment is started, however, the patient and their parents or carers need to be fully aware that this course of early

treatment will not result in a better outcome nor will it reduce or eliminate the need for further orthodontic treatment to be carried out at a later stage when the full permanent dentition is established.

References

1. Holmes A. The prevalence of orthodontic treatment need. *Br J Orthod.* 1992;19(3):177–82.
2. Proffit WR, Fields HW Jr, Moray LJ. Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the NHANES III survey. *Int J Adult Orthodon Orthognath Surg.* 1998;13:97–106.
3. Dimberg L, Lennartsson B, Arnrup K, Bondemark L. Prevalence and change of malocclusions from primary to early permanent dentition: a longitudinal study. *Angle Orthod.* 2015;85(5):728–34.
4. You ZH, Fishman LS, Rosenblum RE, Subtelny JD. Dentoalveolar changes related to mandibular forward growth in untreated Class II persons. *Am J Orthod Dentofac Orthop.* 2001;120:598–607.
5. McNamara JA Jr. Components of class II malocclusion in children 8–10 years of age. *Angle Orthod.* 1981;51(3):177–202.
6. Mew JR. The postural basis of malocclusion: a philosophical overview. *Am J Orthod Dentofac Orthop.* 2004;126(6):729–38.
7. Woodside DG, Linder-Aronson S, Lundstrom A, et al. Mandibular and maxillary growth after changed mode of breathing. *Am J Orthod Dentofac Orthop.* 1991;100:1–18.
8. Patel A. Digit sucking habits in children resident in Kettering (UK). *J Orthod.* 2008;35:255–61.
9. Singh S, Utreja A, Chawla H. Distribution of malocclusion types among thumb suckers seeking orthodontic treatment. *J Indian Soc Pedod Prev Dent.* 2008;26(26):114–7.
10. Bishara SE, Warren JJ, Broffitt B, Levy SM. Changes in the prevalence of nonnutritive sucking patterns in the first 8 years of life. *Am J Orthod Dentofac Orthop.* 2006;130(1):31–6.
11. 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 Dentofac Orthop.* 2002;121(4):347–56.
12. Keeling SD, Wheeler TT, King GJ, Garvan CW, Cohen DA, Cabassa S, et al. Anteroposterior skeletal and dental changes after early Class II treatment with bionators and headgear. *Am J Orthod Dentofac Orthop.* 1998;113:40–50.
13. O'Brien K, Wright J, Conboy F, Sanjie Y, Mandall N, Chadwick S, 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 Dentofac Orthop.* 2003b;124(3):234–43.
14. O'Brien K, Wright J, Conboy F, Chadwick S, Connolly I, Cook P, et al. Effectiveness of early orthodontic treatment with the Twin-block appliance: a multicenter, randomized, controlled trial. Part 2: psychosocial effects. *J Orthod.* 2003a;124:488–95.
15. Tulloch JF, Phillips C, Koch G, Proffit WR. The effect of early intervention on skeletal pattern in Class II malocclusion: a randomized clinical trial. *Am J Orthod Dentofac Orthop.* 1997;111(4):391–400.
16. Dolce C, McGorray SP, Brazeau L, King GJ, Wheeler TT. Timing of Class II treatment: Skeletal changes comparing 1-phase and 2-phase treatment. *Am J Orthod Dentofac Orthop.* 2007;132(4):481–9.
17. O'Brien K, Wright J, Conboy F, Appelbe P, Davies L, Connolly I, et al. Early treatment for Class II Division 1 malocclusion with the Twin-block appliance: a multi-center, randomized, controlled trial. *Am J Orthod Dentofac Orthop.* 2009;135(5):573–9.
18. Tulloch JFC, Proffit WR, Phillips C. Outcomes in a 2-phase randomized clinical trial of early class II treatment. *Am J Orthod Dentofac Orthop.* 2004;125(6):657–67.

19. Seehra J, Fleming P, Newton T, DiBiase A. Bullying in orthodontic patients and its relationship to malocclusion, self-esteem and oral health-related quality of life. *J Orthod.* 2011;38(4):247–56.
20. Seehra J, Newton JT, DiBiase AT. Interceptive orthodontic treatment in bullied adolescents and its impact on self-esteem and oral-health-related quality of life. *Eur Orthod.* 2013;35:615–21.
21. Schatz JP, Hakeberg M, Ostini E, Kiliaridis S. Prevalence of traumatic injuries to permanent dentition and its association with overjet in a Swiss child population. *Dent Traumatol.* 2013;29(2):110–4.
22. Thiruvengkatachari B, Harrison JE, Worthington HV, O'Brien KD. Orthodontic treatment for prominent upper front teeth (Class II malocclusion) in children. *Cochrane Database Syst Rev.* 2013;11:CD003452.
23. Andersson L. Epidemiology of traumatic dental injuries. *J Endod.* 2013;39(3 Suppl):S2–5.
24. Petti S. Over two hundred million injuries to anterior teeth attributable to large overjet: a meta-analysis. *Dent Traumatol.* 2015;31(1):1–8.
25. Nanda RS, Kierl MJ. Prediction of cooperation in orthodontic treatment. *Am J Orthod Dentofac Orthop.* 1992;102(1):15–21.
26. Fleming PS, Scott P, DiBiase AT. Compliance: getting the most from your orthodontic patients. *Dent Update.* 2007;34(9):565–6, 569–70, 572.
27. Trenouth MJ. Do failed appointments lead to discontinuation of orthodontic treatment? *Angle Orthod.* 2003;73(1):51–5.
28. Bartsch A, Witt E, Sahn G, Schneider S. Correlates of objective patient compliance with removable appliance wear. *Am J Orthod Dentofac Orthop.* 1993;104(4):378–86.
29. Borrie FRP, Bearn DR, NPT I, Iheozor-Ejiofor Z. Interventions for the cessation of non-nutritive sucking habits in children. *Cochrane Database Syst Rev.* 2015;3:CD008694.
30. Baccetti F, Franchi L, Toth L, et al. Treatment timing for Twin-block therapy. *Am J Orthod Dentofac Orthop.* 2000;118:159–70.

Simon J. Littlewood

Abstract

Interceptive treatment of Class III malocclusions is indicated if it reduces damage to the oral tissues, or prevents, or significantly reduces the amount, or severity, of future orthodontic treatment. Patients must be informed that the long-term success of interceptive treatment of Class III malocclusions cannot be guaranteed due to the unpredictability of future growth.

The choice of treatment depends on identifying the aetiology of the Class III malocclusion. The aetiology could be dental, a pseudo-Class III (which is due to a displacement of the mandible caused by a crossbite) or skeletal.

Simple anterior dental crossbites can be successfully treated with removable or fixed appliances in the mixed dentition.

Treatment with chin cup or functional appliances can correct a Class III incisor relationship, but any orthopaedic changes with these appliances are likely to be minimal.

Interceptive treatment with a protraction facemask treatment can reduce the need for future orthognathic surgical correction, when used on patients who are under 10, with a mild to moderate Class III and a retrusive maxilla, and with average or reduced vertical proportions.

Bone anchored appliances may offer the potential for more skeletal changes, but further research is needed in this area.

S.J. Littlewood

Orthodontic Department, St Luke's Hospital, Little Horton Lane, Bradford BD5 0NA, UK
e-mail: simonjlittlewood@aol.com

Introduction

Class III malocclusion was originally defined by Edward Angle in terms of the occlusal relationship of the first permanent molars, with the lower molars mesially positioned relative to the upper molar. A more contemporary definition focuses on the incisors, describing a Class III incisor relationship as the lower incisor tips occluding anterior to the cingulum plateau of the upper incisors.

The prevalence and presentation of Class III malocclusion vary significantly with ethnic background. Prevalence in East Asian populations, such as Japan, Korea and China, can range from 4% to 19%, whereas in European populations the prevalence is much lower: 1–4% [1].

Aetiology of Class III Malocclusions in Mixed Dentition

It is important to identify the aetiology of Class III malocclusions in the mixed dentition as this will determine the most appropriate type of interceptive treatment. The aetiology may be due to skeletal and/or dentoalveolar components.

By definition, the lower incisors lie in front of the cingulum plateau of the upper incisors, often leading to an anterior crossbite of one or more teeth. An orthodontic assessment will help differentiate between a simple dental anterior crossbite, due to locally malpositioned teeth, and a true Class III skeletal discrepancy. Whenever an anterior crossbite is present, it is important to assess whether this is associated with an anterior mandibular displacement of the mandible, which increases the severity of the appearance of Class III. There is also often a skeletal component, with the mandibular dentition held more anteriorly than the maxillary dentition. This could be as a result of the size and position of either jaw: it is important to identify where the discrepancy lies as this may affect the choice of treatment.

In order to decide on the most appropriate interceptive approach, we must diagnose the contributing factors causing the Class III malocclusion. This can be done by: extra-oral assessment, intra-oral assessment (including assessing for any mandibular displacements as a result of anterior crossbites) and cephalometric analysis if required.

Extra-oral Assessment

A profile analysis will look at facial proportions, mid-facial position and chin position, as well as vertical proportions. This will help to determine the presence and location of any skeletal discrepancy. For patients with a retrusive maxilla, there may be increased sclera show below the pupil and flattening of the infraorbital rims in addition to flattening of the area adjacent to the nose.

Intra-oral Assessment

An anterior crossbite of one or more teeth is a common presentation in Class III malocclusions. Whenever there is a crossbite, it is important to look for an anterior mandibular displacement. This premature contact may lead to the mandible being positioned further anteriorly, to allow the patient to close into full intercuspation and obtain a more comfortable bite.

It is also important to look at the inclinations of the upper and lower incisors. In patients with skeletal discrepancies, the soft tissues may tilt the teeth towards each other to allow a lip seal to be achieved. This is known as dentoalveolar compensation, and the degree of existing compensation may dictate what is possible with orthodontic movements of the teeth alone or whether movements of the underlying bones are required.

Cephalometric Assessment

A cephalometric analysis may be required in addition to the clinical analysis to confirm the relative positions of the maxilla and mandible to each other and the base of the skull and to determine the inclinations of both the upper and lower incisors. The combination of clinical and cephalometric information will identify which type of Class III malocclusion can be treated in the mixed dentition and help decide the best interceptive approach.

In the mixed dentition there are effectively three types of Class III malocclusions [1]:

- Dental: Incorrect inclination or position of maxillary or mandibular incisors
- Pseudo: Anterior positioning of the mandible as a result of premature dental contacts deflecting the mandible anteriorly to allow the patient to achieve full intercuspation
- Skeletal: True skeletal discrepancies in the maxilla and/or mandible

Indications for Interceptive Treatment of Class III

Although a Class III malocclusion may be identified in the developing dentition, a decision needs to be made whether it is better to treat at this stage or wait for further dental development and growth. Interceptive treatment of Class III malocclusions should be undertaken if it:

- Prevents damage to the oral tissues
- Prevents or significantly reduces the amount, or severity, of future orthodontic treatment

Damage to oral tissues may occur as a result of an anterior crossbite causing a displacement of the mandible. This may lead to localised attritional wear of the teeth that are in premature contact as the mandible slides forwards into a position where the patient can achieve a maximum intercuspatation and a more comfortable occlusion. It is also possible for irreversible periodontal soft tissue and bony damage to occur. This is due to the lateral forces applied by displacing contacts associated with the anterior crossbites and is more likely to occur if there are problems with oral hygiene.

Additional benefits of interceptive treatment include improving occlusal function and improving the facial appearance. It may also reduce the risk of a developing an abnormal posterior occlusion. This abnormal posterior occlusion can be the result of habitual posturing of the mandible, as the patient finds a more comfortable bite to accommodate abnormal anterior occlusal contacts. It has also been suggested that interceptive treatment has the potential to reduce the need for future orthognathic surgery by causing favourable skeletal changes. This is controversial and will be discussed further in section “[Growth Modification and Orthopaedic Treatment](#)”.

Interceptive treatment of Class III malocclusions is always challenging, due to the unpredictability of future growth. Although it may be possible to correct an anterior crossbite and improve dental arch relationships, the result may relapse as a result of future unfavourable mandibular growth. There have been attempts to develop techniques to predict future growth on an individual basis, but at the present time, it is still difficult to confidently predict the outcome of treatment of Class III malocclusions [2]. Patients should therefore be given a cautious prognosis for their corrected interceptive Class III treatment, due to the unpredictability of future growth.

The following sections will discuss the treatment of simple dentoalveolar anterior crossbites (section “[Treatment of Simple Dento-Alveolar Anterior Crossbites](#)”) and the use of growth modification and orthopaedic movement for malocclusions with a larger skeletal component (section “[Growth Modification and Orthopaedic Treatment](#)”). In both cases, more favourable changes will be seen in patients who:

- Have a definite overbite at the end of treatment, which helps to maintain the correction of any anterior crossbite
- Present with an initial anterior displacement of the mandible due to the crossbite
- Are more compliant and will wear the appliances as directed

Treatment of Simple Dentoalveolar Anterior Crossbites

A simple anterior crossbite can be corrected using either a removable appliance or a fixed appliance. Success is increased if there is a minimal existing proclination of the upper incisors and there is adequate overbite to maintain the correction at the end of treatment.

A removable appliance has an active component anteriorly to procline the upper tooth or teeth to correct the anterior crossbite. This active component can either be a palatal spring, which is activated by the clinician, or a screw, which the

patient activates. The appliance also incorporates retentive components to keep the appliance in place and possibly posterior capping to disclude the occlusion to aid correction of the anterior teeth. A removable appliance can only tip the teeth, so it should only be used if simple tipping movements of the upper anterior teeth are required.

A fixed appliance can also be used (see Fig. 10.1) and is sometimes only bonded on the permanent teeth that are present in the mouth at this age. This appliance is often referred to as a “2 by 4” appliance as it is only bonded on the two upper first permanent molars and the four upper incisors. An active pushcoil, between the molars and the incisors, can be used to procline the incisors. Glass ionomer cement may be placed temporarily as a posterior fixed bite plane on the molars if disclusion is required. Fixed appliances allow bodily movement and correction of rotations. They also have the ability to increase the overbite to improve stability and reduce the compliance required by the patient.

There is evidence to suggest that both types of appliances work and the results are equally stable. Fixed appliance treatment is quicker and cheaper and has less

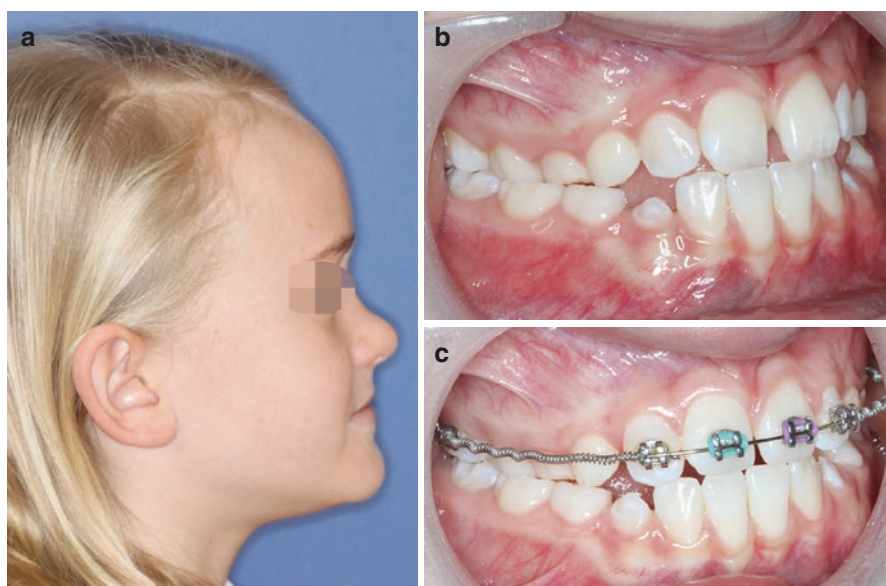


Fig. 10.1 Case demonstrating simple correction of anterior crossbite with 2 × 4 fixed appliance. (a) Start extra-oral lateral view demonstrating Class III skeletal pattern (partly retrognathic maxilla and slightly prognathic mandible). (b) Start intra-oral view demonstrating Class III incisor relationship and anterior crossbite upper right central and lateral incisor. There was a slight anterior displacement of the mandible caused by this crossbite, leading to a “pseudo-Class III”. (c) Fixed “2 × 4” appliance with pushcoil proclining the upper incisors. The patient wore the appliance for 5 months. (d) Final extra-oral lateral view, showing Class I skeletal pattern as a result of the correction of the crossbite, which removed the anterior displacement of the mandible. (e) Final intra-oral view showing correction of the anterior crossbite, with an overbite present to maintain the correction. Future stability will depend on future mandibular growth

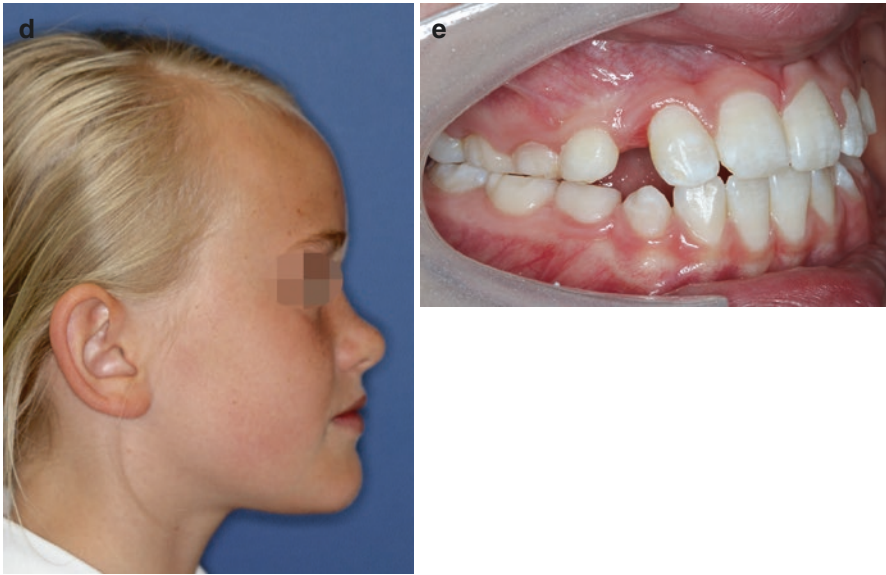


Fig. 10.1 (continued)

effect on the patient's speech than a removable appliance, but patients may complain of slightly more difficulty in chewing and biting initially with the fixed appliance [3–6].

Growth Modification and Orthopaedic Treatment

It has been suggested that it is possible to intercept a developing Class III skeletal malocclusion by using growth modification, leading to orthopaedic treatment. This approach aims to correct the skeletal discrepancy or at least improve it sufficiently to allow treatment with orthodontic camouflage in the future and avoid orthognathic surgery. This orthopaedic approach has been attempted using a variety of approaches, including functional appliances, chin cup therapy, protraction facemask and bone-anchored appliances. The evidence to support each of these approaches will be briefly discussed.

Functional Appliances

Functional appliances have been used to try and modify the skeletal pattern by enhancing the growth of the maxilla and restricting or redirecting the growth of the mandible. Examples include Fränkel functional regulator III appliance (FR III) and reverse twin-block appliance.

The FR III (see Fig. 10.2) has maxillary vestibular shields in the depth of the sulcus. These shields are placed away from the maxilla to stretch the periosteum and

Fig. 10.2 Fränkel functional regulator III appliance (FR III)



encourage anterior development of the maxilla. The lower part of the appliance attempts to restrict mandibular growth or redirect it posteriorly. Research would suggest that it can improve the occlusal relationships, but this is principally due to dentoalveolar changes, proclining upper incisors and retroclining the lower incisors [7]. The FR III can be challenging for patients to wear and subject to breakage, and as the changes are principally dentoalveolar, there may be simpler ways to correct the malocclusion by orthodontic camouflage.

The reverse twin-block (see Fig. 10.3) is a modification of the traditional twin-block, which was originally designed for treatment of Class II. In the reverse twin-block, the blocks are positioned so that there are posterior forces on the mandible and anterior forces on the maxilla. Once again the effects appear to be dentoalveolar, rather than skeletal [8].

It would appear therefore that functional appliances can successfully correct a Class III malocclusion, but this is principally by dentoalveolar changes, with minimal or no effects on the underlying skeletal pattern.

Chin Cup

Chin cup therapy is orthopaedic treatment aimed at modifying the growth of the mandible. The patient is asked to wear the chin cup for over 14 h a day, with forces of 300–500 g directly through the condyle or just behind it. It would appear that it

Fig. 10.3 Reverse twin-block



may redirect the mandible growth vertically, causing a backward rotation of the mandible [9], but often these changes are not maintained in the long term and the normal growth pattern re-establishes itself [10]. This is the principal appliance aimed at the correction of Class III malocclusions that are the result of prognathic mandible. However, as it seems to work by causing a backward rotation of the mandible, with disappointing long-term results, then patients who present in the mixed dentition with marked mandibular prognathism, particularly if associated with increased vertical proportions, are often best treated later with surgery, when their growth is complete.

Protraction Facemask

Protraction facemask, sometimes referred to as reverse headgear, applies a forwards and downwards force to the maxilla and has been shown to be successful in correcting reverse overjets in the developing dentition [11]. The appliance is composed of two components: an external framework that fits on the face and an internal attachment to the maxillary dentition (see Fig. 10.4). The two components are connected by elastics providing forces of 300–500 g per side in a forward and slightly downward vector. The external framework is made up of two pads (one that sits on the forehead and one that sits on the chin), which provide anchorage. There is also a middle bar for the connection of the elastics to the intra-oral attachment to the maxillary dentition.

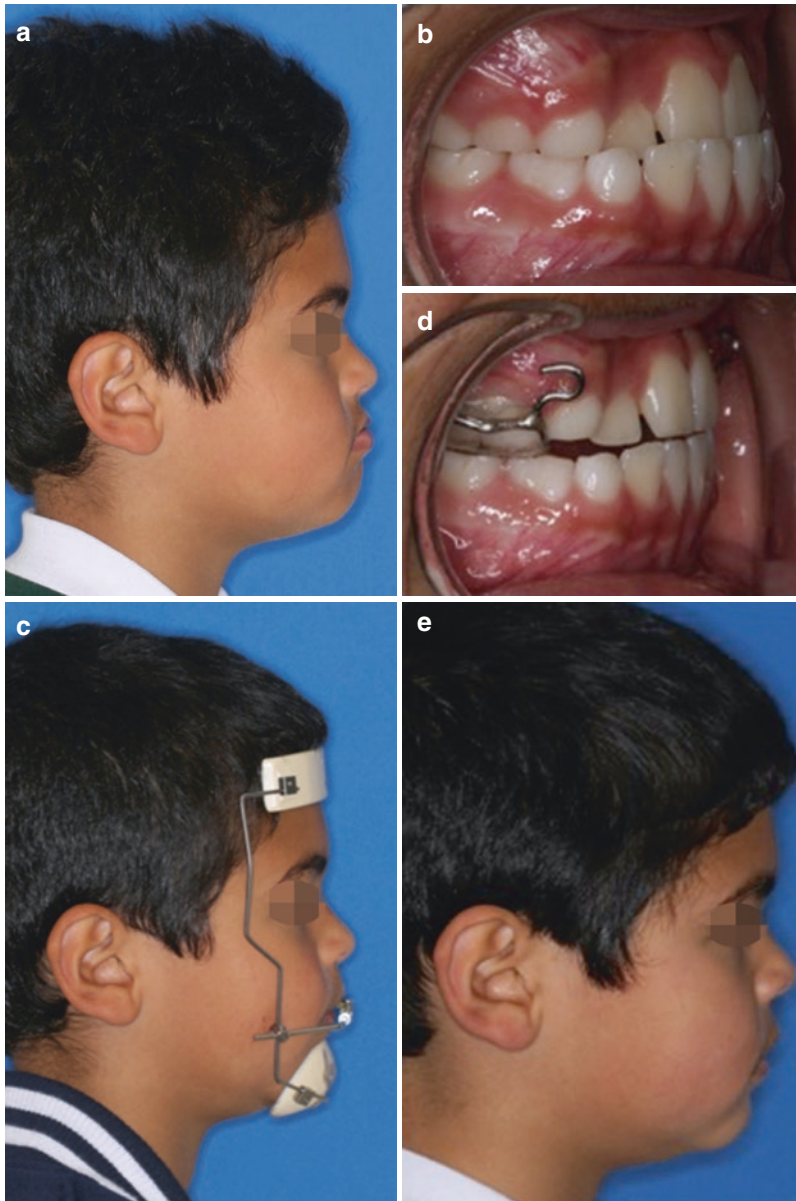


Fig. 10.4 Protraction facemask case. (a) Age 8.5 years pretreatment facial view. (b) Age 8.5 years pretreatment intra-oral view. (c) Facial view of protraction facemask during treatment. (d) Intra-oral view during treatment of bonded RME with hooks for attachment of elastics. (e) Facial view at end of 6 months of treatment. (f) Intra-oral view after 6 months of treatment. (g) Age 11, facial view 2 years after treatment. (h) Age 11, intra-oral view 2 years after treatment

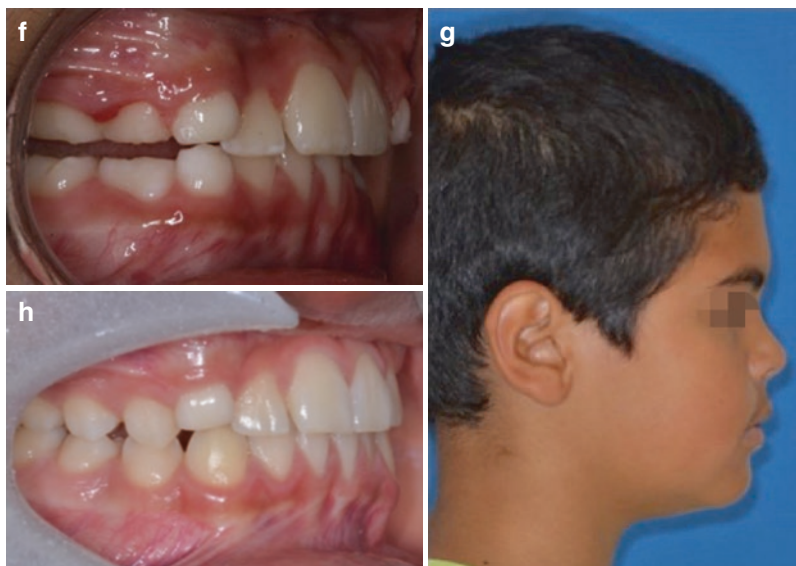


Fig. 10.4 (continued)

There are various designs of attachment to the maxillary dentition, including removable, banded and acrylic-bonded versions. They all incorporate some sort of hooks positioned above the roots of the first deciduous molar (the centre of rotation of the maxilla), for attachments of the elastics. The elastic forces are typically 300–500 g per side and need to be worn 12–14 h per day. The total treatment time is usually 6–9 months.

One controversial area is the use of rapid maxillary expansion (RME) used at the same time as the protraction facemask. Often patients with a Class III skeletal pattern have a small maxilla in the transverse dimension as well as the anteroposterior dimension, so this expansion is a helpful component to the treatment. It has been suggested that this may loosen the circummaxillary sutures and increase the forward movement of the maxilla, although the results of higher quality research seem to suggest that the effects of the RME are minimal [12]. This principle has been taken further by using a technique known as Alt-RAMEC (alternating rapid maxillary expansion and contraction) [13]. The Alt-RAMEC protocol describes alternative weeks of rapid maxillary expansion and constriction, to disarticulate the maxilla without over-expanding. Further high-quality research into RME with protraction facemask is required to determine if this is an appropriate approach.

In a randomised controlled clinical trial comparing protraction facemask with no treatment, it was shown that successful correction of the reverse overjet will happen in 70% of patients, with an average increase in overjet of 4 mm, and a significant skeletal change, principally due to forward movement of the maxilla. The ANB

angle (relating the maxilla to the mandible) improved 2.6° compared to the control at the end of treatment [14]. There were no detrimental effects on the TMJ. Although it was successful skeletally and dentally, there were no detectable psychosocial benefits for the patients who wore the protraction facemask.

These patients were followed up 6 years later to see if the favourable changes were maintained towards the end of growth and in particular to assess whether the interceptive use of a facemask in the developing dentition can help to reduce the need for orthognathic surgery [15]. Of the patients that wore protraction facemask, 36% needed orthognathic surgery at the age of 15, whereas 66% of patients in the control required orthognathic surgery. Encouragingly 68% of patients who wore the protraction facemask had a positive overjet after 6 years. Interestingly, the initial early protraction facemask treatment improvements in the skeletal parameters were not maintained at 6 years follow-up. The reduction in the need for surgery may be as a result of rotational changes in the maxilla and mandible. It may also be due to the accumulation of multiple effects on the occlusion and skeletal pattern, which on their own are insignificant, but collectively reduce the need for orthognathic surgery.

So it would appear that the use of protraction facemask in the developing dentition will correct the Class III malocclusion and reduce the need for orthognathic surgery in the future in the following types of cases:

- Child under the age of 10
- Mild-moderate Class III
- Retrusive maxilla
- Average or reduced vertical proportions

While interceptive treatment of Class III malocclusions can be beneficial in these particular cases, it has been suggested that because the appliances used are tooth-borne, they may lead to less orthopaedic change and unwanted dental changes such as:

- Buccal flaring of molars and extrusion lead to increase in vertical dimensions.
- Arch length decrease due to mesial migration of molars leading to crowding.

In an attempt to overcome the limitations of tooth-borne appliances in the interceptive treatment of Class III malocclusions, bone-anchored appliances have more recently been used.

Bone-Anchored Appliances

As well as trying to overcome some of the unwanted dentoalveolar effects of tooth-borne appliances discussed above, there may also be the potential for bone-anchored appliances to offer greater skeletal changes [16]. These appliances typically involve the use of Class III elastics attached between plates placed in

the mandibular symphyseal region and the infrazygomatic crest (see Fig. 10.5). The success of these mini-plates is related to the surgical technique and the thickness and quality of the bone. Particularly in the maxilla, the bone quality is often not as good until the patient is at least 11 years old, so this interceptive technique tends to be used in slightly older patients than the tooth-borne appliances. The results of initial studies into this bone-anchored approach suggest that it has the potential to offer greater skeletal changes, with less unwanted displacement of the dentition. However, there are unpredictable variations in individual outcomes, and further high-quality research is needed to investigate this technique further.

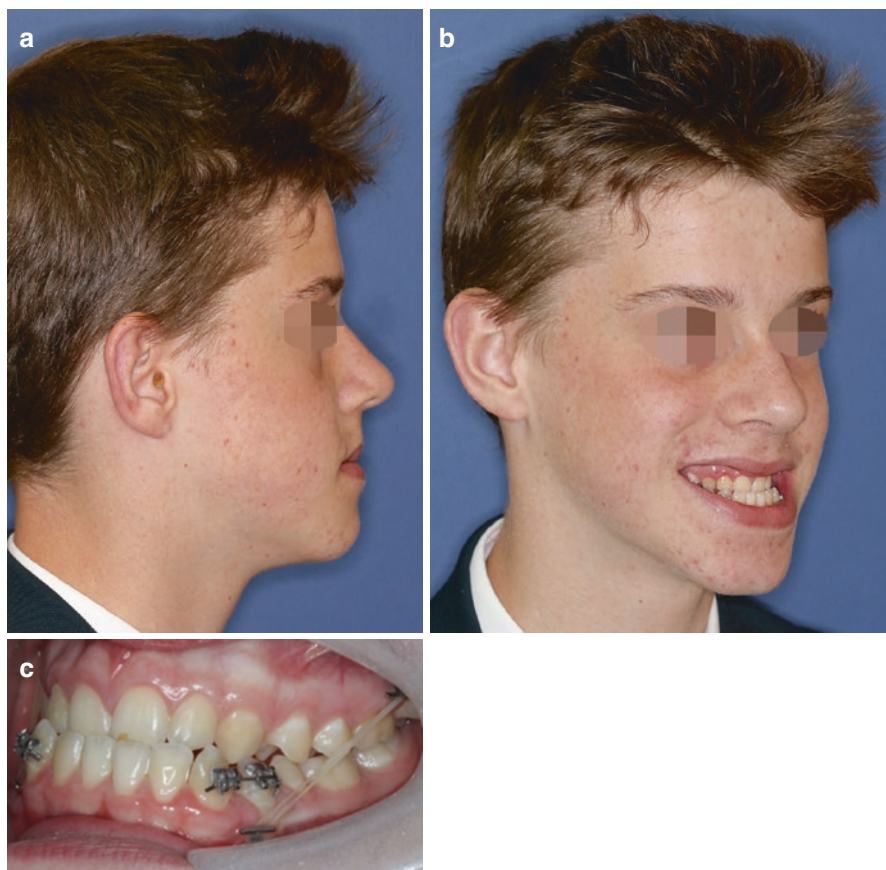


Fig. 10.5 Case demonstrating the use of bone-anchored mini-plates with Class III elastics. (a) Start extra-oral lateral facial view. (b) Start extra-oral three-quarter facial view. (c) Intra-oral view of Class III elastics attached to mini-plates that were placed 2 weeks previously. (d) Four months into treatment extra-oral lateral facial view. (e) Four months into treatment extra-oral three-quarter facial view. (f) Four months into treatment showing intra-oral improvement in occlusion. Use of full-time Class III elastics is ongoing



Fig. 10.5 (continued)

Conclusions

1. Interceptive treatment of Class III malocclusions may be undertaken if it prevents damage to the oral tissues, and/or prevents, or significantly reduces the amount, or severity, of future orthodontic treatment.
2. The long-term success of interceptive treatment of Class III malocclusions cannot be guaranteed due to the unpredictability of future growth.
3. It is important to determine the aetiology of the Class III incisor relationship before deciding on any interceptive treatment. The aetiology could be dental, a pseudo-Class III (which is due to a displacement of the mandible caused by a crossbite) or skeletal.
4. Treatment is more likely to be successful if there is a definite overbite at the end of treatment to maintain the result, in the presence of an initial anterior

- displacement of the mandible due to the crossbite and in patients who are more compliant and will wear the appliances as directed.
5. Simple anterior dental crossbites can be successfully treated with removable or fixed appliances in the mixed dentition.
 6. Treatment with chin cup or functional appliances can correct a Class III incisor relationship, but any orthopaedic changes are likely to be minimal with these appliances.
 7. Interceptive treatment with a protraction facemask treatment can reduce the need for future orthognathic surgical correction, when used on patients who are under 10, with a mild to moderate Class III and a retrusive maxilla and with average or reduced vertical proportions.
 8. Bone-anchored appliances may offer the potential for more skeletal changes, but further research is needed in this area.

References

1. Ngan P, He H, Wilmes B. Chapter 4: Treatment in Class III malocclusions in the growing patients. In: *Orthodontic treatment of Class III malocclusions*. Sharjah: Bentham Science Publishers Ltd; 2014. P. 61–115
2. Fudalej P, Dragan M, Wedrychowska-Szulc B. Prediction of the outcome of orthodontic treatment of Class III malocclusions—a systematic review. *Eur J Orthod*. 2011;33:190–7.
3. Wiedel A, Bondemark L. Fixed versus removable orthodontic appliances to correct anterior crossbite in the mixed dentition - a randomised controlled trial. *Eur J Orthod*. 2015a;32(2):123–7.
4. Wiedel A, Bondemark L. A randomized controlled trial of self-perceived pain, discomfort, and impairment of jaw function in children undergoing orthodontic treatment with fixed or removable appliances. *Angle Orthod*. 2015b;86(2):324–30.
5. Wiedel A, Bondemark L. Stability of anterior crossbite correction: a randomized controlled trial with a 2-year follow-up. *Angle Orthod*. 2016;85(2):189–95.
6. Wiedel A, Norlund A, Petren S, Bondemark L. A cost minimization analysis of early correction of anterior crossbite—a randomized controlled trial. *Eur J Orthod*. 2016;38(2):140–5.
7. Ulgen M, Firatli S. The effects of Frankel's function regulator on the Class III malocclusion. *Am J Orthod*. 1994;105:561–7.
8. Seehra J, Fleming PS, Mandall N, DiBiase AT. A comparison of two different techniques for early correction of Class III malocclusion. *Angle Orthod*. 2012;82(1):96–101.
9. Uner O, YuKsel S, Ucuncu N. Long-term evaluation after chin cup treatment. *Eur J Orthod*. 1995;17:135–41.
10. Sugawara J, Asano T, Endo N, Mitani H. Long-term effects of chin cup therapy on skeletal profile in mandibular prognathism. *Am J Orthod Dentofac Orthop*. 1990;98:127–33.
11. Watkinson S, Harrison JE, Furness S, Worthington HV. Orthodontic treatment for prominent lower front teeth (Class III malocclusion) in children. *Cochrane Database Syst Rev*. 2013; Issue 9. Art. No.: CD003451. doi:10.1002/14651858.CD003451.pub2.
12. Liu W, Zhou Y, Wang X, Liu D, Zhou S. Effect of maxillary protraction with alternating rapid palatal expansion and constriction vs expansion alone in maxillary retrusive patients: a single center, randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2015;148:641–51.
13. Liou EJW. Toothborne orthopedic maxillary protraction in Class III patients. *J Clin Orthod*. 2005;39:68–75.
14. Mandall N, Cousley R, Dibiase A, Dyer F, Littlewood SJ, Mattick R, Nute S, Doherty B, Stivaros N, McDowell R, Shargill I, Worthington H. Is early Class III protraction facemask

- treatment effective? A multicentre, randomized, controlled trial: 15-month follow-up. *J Orthod.* 2010;37:149–61.
15. Mandall N, Cousley R, DiBiase A, Dyer F, Littlewood SJ, Mattick CR, Nute S, Doherty B, Stivaros N, McDowall R, Shargill I, Worthington HV. Early class III protraction facemask treatment reduces the need for orthognathic surgery: a multi-centre, two-arm parallel randomised, controlled trial. *J Orthod.* 2016;43(3):164–75.
 16. De Clerck HJ, Proffit WR. Growth modification of the face: a current perspective with emphasis on Class III treatment. *Am J Orthod Dentofac Orthop.* 2015;148:37–46.

Jayne E. Harrison

Abstract

This chapter describes current evidence regarding early management of posterior crossbites. In order to understand the treatment of posterior crossbite definitions, aetiology, epidemiology and the implications of posterior crossbites will be summarised. Current evidence suggests that there is a need for further long-term epidemiological studies to monitor the effects of a unilateral posterior crossbite (UPXB) and spontaneous correction rate as current evidence is conflicting. In addition, randomised controlled trials comparing competing interventions are justified. These should be methodologically sound, adequately powered and record outcomes that take into account participants' views and experiences of the treatment. They should also report the proportion of cases whose UPXB is corrected and in the follow-up period, remains stable until the permanent dentition is established. Based on current evidence, there is justification for the early orthodontic treatment of UPXB with mandibular shifts. There is some weak evidence that posterior crossbites with mandibular shifts are associated with temporomandibular dysfunction and reduced bite force and that children and adolescents with UPXB show signs of some degree of mandibular asymmetry. Randomised controlled trials suggest that treatment appears to be stable if undertaken in the mixed dentition. The appliance of choice appears to be the quad-helix appliance because the evidence suggests that treatment is quicker; they are more cost-effective and tolerated better than their alternatives, i.e. expansion plate or arch.

J.E. Harrison
University of Liverpool Dental Hospital, Liverpool, UK
e-mail: Jayne.Harrison@rlbuht.nhs.uk

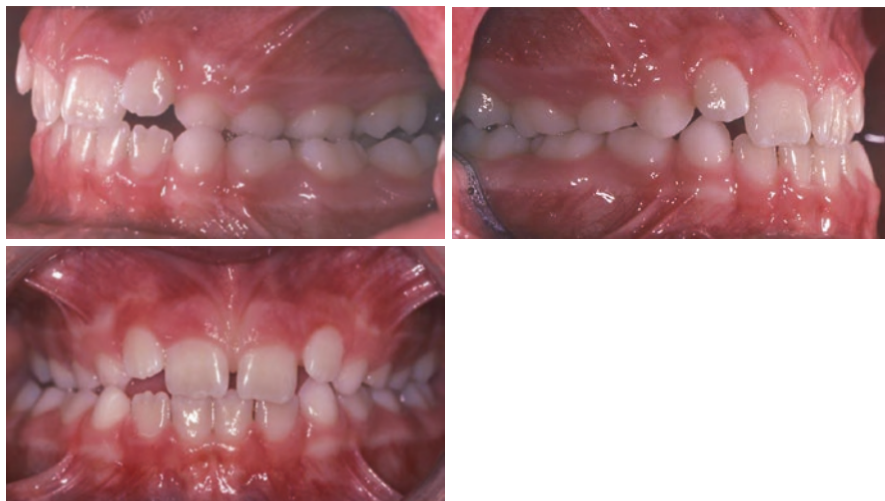


Fig. 11.1 Unilateral crossbite in the mixed dentition

Definitions: Posterior Crossbite

Posterior crossbite is the term used to describe the occlusion when there is a discrepancy between the transverse relationship of the maxillary and mandibular teeth. They can be uni- or bilateral.

Buccal crossbite

- Buccal cusps of the maxillary teeth occlude lingual to the buccal cups of the mandibular teeth (Fig. 11.1).

Lingual crossbite (scissor bite)

- Buccal cusps of the mandibular teeth occlude lingual to the palatal cups of the maxillary teeth.

Aetiology

A posterior buccal crossbite can develop when the maxilla and/or maxillary dental arch width is narrower than the mandible and/or mandibular teeth. A posterior crossbite can occur unilaterally or bilaterally and may develop or improve at any time from when the deciduous teeth erupt to when the permanent dentition becomes established [1–4].

It is unclear what causes posterior crossbites, but they may be due to skeletal, soft tissue, e.g. swallowing [5], dental or respiratory factors [6–10] or develop as the result of a sucking habit, e.g. digit or pacifier sucking [5, 9, 11–14], bottle-/breast-feeding [15, 16] or swallowing pattern [5].

Epidemiology

The prevalence of posterior buccal crossbites varies from 2.4 to 18% of children in the primary dentition [14, 17–21], 8.5–15.1% in the mixed dentition [17, 22, 23] and 5.1–17.8% in the early permanent dentition [17, 22, 24, 25].

The prevalence has been found to be higher in white populations compared with children of African or Asian ethnicity [22, 26, 27]. Whilst early studies suggest that most (50–90%) posterior crossbites in the mixed dentition persist when the permanent teeth erupt and that only in a minority of children the malocclusion self-corrects [4, 28], a more recent, longitudinal study in Sweden found that 24/29 (82%) of posterior crossbites found in children at the age of 3 years had self-corrected by the age of 11.5 years [17].

Implications

Functional Crossbite and Temporomandibular Dysfunction

If the widths of the maxillary and mandibular dentitions are not coordinated, they will not fit into a normal occlusion. If the size of the discrepancy between the widths of the maxillary and mandibular dentition is such that a premature contact in retruded contact position (RCP) occurs, it often (80–97%) causes a displacement of the mandible when closing from RCP into intercuspal position (ICP) resulting in a functional posterior crossbite [4, 29]. Of all the different malocclusions, a posterior crossbite and the displacement on closing, with which it is often associated, have been found by many to increase the likelihood of an individual developing a variety of signs and symptoms related to the temporomandibular joint (TMJ), its associated muscles and the internal disc [30]. However, there is considerable debate about the causal association between a posterior crossbite and its negative impact on the masticatory system with as many authors not finding any association. Some studies have reported a correlation between posterior crossbite with a displacement on closing and TMJ dysfunction in adulthood. However, TMJ dysfunction has many causes; nevertheless, studies of adolescents and adults have shown that some people with a crossbite may have an increased risk of developing TMJ dysfunction (TMD) and show more signs and symptoms of these problems [31–35]. On the other hand, some more recent studies have been less conclusive regarding an association between posterior crossbites and TMD [30, 36–38].

In their recent systematic review, Iodice et al. [30] reviewed the evidence for the association between posterior crossbite, masticatory muscle pain and disc displacement. They undertook a thorough search of the literature from 1966 to mid-2012 that resulted in 2919 citations of which 43 studies met their inclusion criteria and were included in the review. They assessed the quality of the studies on a 10-point scale and then divided the scores into high (9,10), medium (6–8) and low (0–5). It can be seen from Table 11.1 that there were equal numbers of studies in which an association was found between posterior crossbite and the signs and symptoms of

Table 11.1 Association of posterior crossbite with signs and symptoms of TMD

	Association with TMD				No association with TMD			
	All papers			High-quality (≥ 9) number	All papers			High-quality (≥ 9) number
	Number	Mean score	95%CI		Number	Mean score	95%CI	
Disc displacement	15	5.5	6.6, 4.3	2	12	5.8	6.6, 5.0	1
Masticatory muscle pain	9	5.6	6.7, 4.4	0	10	5.8	7.0, 4.6	1
TMD	9	5.6	6.5, 4.6	0	11	5.7	7.3, 5.0	1
Total	33			2	33			3

Data from Iodice et al. [30]

temporomandibular dysfunction (TMD) and those that did not find an association. They noted that those papers in which an association was found had a slightly lower-quality score and were older than those where no association was found; however, the difference in quality was not statistically significantly different for any of the signs or symptoms of TMD, and the rating scale they used was an unvalidated assessment tool.

Growth and Development

Young children with a posterior crossbite only occasionally experience pain or have problems with chewing; however, there is concern that the continued mandibular displacement on closing could potentially have long-term effects on the growth and development of the teeth and jaws leading to a facial asymmetry. Systematic reviews, to evaluate the association between unilateral posterior crossbite (UPXB) and skeletal, dental and soft tissue asymmetry, have been undertaken [40, 41] but they both found conflicting evidence.

Talapaneni and Nuvvula [40] found 15 relevant articles, but 4 were excluded due to high risk of bias leaving 11 studies for evaluation of which 6 were considered to be of low methodological quality [42–46], 2 of medium quality [47, 48] and 3 of high quality [49–51]. In Iodice et al. [41], the association of UPXB with skeletal asymmetry, EMG activity, bite force, masticatory muscle thickness and chewing cycle was investigated, and 45 studies were included which were assessed for their methodological quality using a scoring system they had developed previously [30]. They found that 4 studies were of high quality (score 9, 10), 39 medium quality (score 6–8) and 6 of low quality (score 0–5).

The evidence from the studies identified by Talapaneni and Nuvvula [40], for the association of a mandibular asymmetry with a UPXB with displacement, gave conflicting results. Whilst three studies in young children and adolescents demonstrated a significant association between UPXB and mandibular asymmetry [43, 48, 49], a further three could not find any association [45, 46, 50].

Similarly, there were four studies where an association between UPXB and condylar asymmetry in the glenoid fossa was found [42, 43, 49, 51] and two that did not [45, 46]. In addition, they found three studies where an association between PUXB and mandibular positional asymmetry related to the cranial floor was found [48–50]. On balance, they concluded that there was weak evidence to support the view that children and adolescents with UPXB show signs of some degree of mandibular asymmetry; however, they did not think that failure to correct a UPXB in childhood would necessarily result in an asymmetry later in life. It should be noted that five of the studies reported that participants had a positional asymmetry rather than a structural asymmetry.

Similarly, Iodice et al. [41] found 19 studies that investigated the association between UPXB and skeletal asymmetry. Of these, none were found to be of high quality (score 9, 10), 16 were of medium quality (score 6–8) and 3 of low quality (score ≤ 5). Again, the association between UPXB and asymmetry was split: 12 studies of medium quality, with a mean score of 6.7 and mean time since publication of 7.1 years, found a significant association [43, 44, 46, 52–60], whereas 7 studies, with a mean score of 6.2 and mean time since publication of 9 years, did not find an association [34, 45, 61–65].

On balance, there appear to be slightly more studies published that report an association between crossbite and mandibular asymmetry than finding no association; however, this may be a reflection of publication bias. As the overall quality of these studies was medium, no definitive conclusions can be drawn about the association of mandibular asymmetry and posterior crossbite.

Functional Changes in the Masticatory Muscles

It is thought that because spontaneous correction of a posterior crossbite is rare, the long-term effects of a functional displacement lead to changes in the masticatory muscles [39]. This can be seen as differences in the bite force [66] and/or muscular activity at rest, during swallowing and/or during chewing [49, 67] of children with a unilateral crossbite (UPXB). The functional changes in the masticatory system have been evaluated using electromyographic (EMG) activity [67] as well as patients' bite force [68] and masticatory performance [69].

Andrade Ada et al. [39] and Iodice et al. [41] have undertaken systematic reviews assessing the relationship between posterior crossbite and functional changes including EMG activity, bite force and the chewing cycle. Andrade Ada et al. [39] assessed the methodological quality and analysed data from 8 of the 494 articles identified by their search strategy. They assessed the quality of the studies using a point scale derived from tools designed for assessment of randomised controlled trials [70, 71]. Of these studies, one was deemed to be of high quality [67], six of medium quality [49, 66, 68, 72–74] and one of low quality [75]. In Iodice et al. [41], 45 studies from the 2184 identified by the search strategy were included. They assessed the quality of the studies using a tool developed previously [30].

EMG Activity

Andrade Ada et al. [39] reported on two studies that had assessed EMG activity, at rest and during swallowing, chewing or clenching to assess differences in muscular activity between children with a posterior crossbite and control groups [49, 67]. The results of the studies varied with no significant difference being found at rest or swallowing between children with a posterior crossbite and those with a normal occlusion. However, significant differences were seen during chewing and clenching with more activity in the anterior temporalis muscle on the crossbite side but reduced activity in the masseter on the crossbite side. On the other hand, Iodice et al. [41] reported on 11 studies that had investigated the relationship between crossbite and EMG activity, all of which reported a significant association [39, 67, 76–84]. It does appear, therefore, that children with a posterior crossbite have asymmetric EMG activity; however, it must be remembered that this is not necessarily pathological.

Bite Force

Bite force was assessed in six studies [52, 66, 68, 72, 73, 75] in which it was found that the maximum bite force was significant less and the number of occlusal contacts significantly fewer in children with a UPXB compared with children with normal occlusions. However, the implications of this on daily life are unclear.

Treatment

Most treatments for posterior crossbites (PXB) aim to widen the upper arch in order to remove any premature contacts and co-ordinate the archs to prevent functional displacement. Appliances have included the quad-helix appliance [85–89, 104], expansion arch [86], expansion plate [85, 87–90] and rapid maxillary expansion appliances, e.g. Hyrax [91–95, 95] and Haas [92, 94, 96, 96], whilst other treatments have been directed towards treating the cause of the posterior crossbite. For example, for patients with breathing problems, an adenoidectomy may be advocated, and for those with sucking habits, habit breakers can be considered [11]. In addition, removal of any occlusal interferences, causing a premature contact and mandibular shift, has been advocated, e.g. by grinding [4, 97] or addition of onlays [87]. Most treatments have been used at each stage of dental development; however, cooperation in the younger patients may be a problem, so treatment in the late mixed to early permanent dentition may be preferable.

The treatment of posterior crossbites has been the subject of several recent systematic reviews [98–101]. Zuccati et al. [101] and Agostino et al. [98] only included treatments that had been assessed in randomised controlled trials (RCTs), whereas Zhou et al. [100] included non-randomised clinical trials, and Petré et al. [99] included prospective and retrospective studies with concurrent untreated controls or normal sample controls and controlled clinical trials comparing at least two treatment strategies as well as RCTs. Although, between them, they identified several

studies, most of the studies had serious problems including lack of power due to small sample size, bias and methodological weaknesses including lack of blinding and inadequate randomisation. Nevertheless, Zhou et al. [100] and Agostino et al. [98] concluded that the quad-helix appliance might be more successful than removable expansion plates at correcting posterior crossbites and expanding the intermolar width for children in the early mixed dentition (aged 8–10 years), noting that the expansion plate failed in about a third of patients. In their review, Zhou et al. [100] concluded that slow maxillary expansion was more effective at expansion than rapid maxillary expansion, but Petren et al. [99] thought that there was insufficient evidence available at the time to make any firm recommendations. Details of studies included in these systematic reviews are listed in Table 11.2.

Expansion Appliances

Maxillary expansion can be achieved using fixed or removable appliances, and the rate of expansion can be slow or rapid. Slow expansion occurs at a rate of approximately 0.5 mm/week, whereas rapid expansion takes place at 0.5 mm/day. It appears that once the crossbite has been corrected, the transverse relationship will be maintained [88].

Fixed Appliances

Quad-Helix (Fig. 11.2)

This is fixed to the first permanent molars using molar bands that are connected by a framework of 0.9 mm stainless steel wire that includes four helices—two anterior (level with the first deciduous molars/premolars) and two posterior (distal to the first permanent molars) [102]. The helices are positioned at either end of the medial arms of the framework which are continuous across the anterior bridge. The lateral arms extend from the molar band to the first premolar or canine as required. The framework can be soldered to the molar bands (Fig. 11.2a) or inserted into a sleeve on the palatal surface of the bands (Fig. 11.2b). The quad-helix can be activated to expand equally or differentially depending on the amount of activation placed in the anterior and posterior helices. It is usually activated by half a tooth's width bilaterally to give slow expansion.

Expansion Arch: Slow Expansion

An expansion arch is made from 1.135 mm (0.045") round stainless steel wire that is bent and coordinated to the shape of the patient's dental arch and expanded and then inserted into the extra-oral traction tubes on the first molar bands [86]. It is held away from the brackets of the fixed appliance by means of small offset bends mesial to the buccal tubes. Anterior support to the expansion arch can be provided by stainless steel ligatures to the central incisor brackets and/or distal to the canine brackets. It can be activated by 5 mm bilaterally to give slow expansion.

Table 11.2 Studies assessing interventions for treating posterior crossbites

Study	Year	Method	Participants	Intervention(s)	Comparison(s)	Outcome(s)	Results
Ingervall [80]	1995	CCT	35–15 TPA, 20 TPA + BRT 17 male, 18 female Age—6.75–15.9 years	Expanded TPA	Expanded TPA + buccal root torque	Crossbite correction Molar inclination Treatment time	Molar expansion TPA—median 4.8 mm IQR 1.3 mm; TPA + torque—5.7 IQR 1.0
Lindner [97]	1989	CCT	76–38 per group 35 male, 41 female Age—4.3 years	Grinding canine	No treatment	Crossbite correction at 9 years of age	Grinding 19/38 Control 6/38
Sandikcioglu [89]	1997	CCT	30–10 per group Age 6.6–8.9 years	1. Expansion plate 2. Quad helix	1. Hyrax RME	Dental and cephalometric data	Molar expansion Plate—3.6 SD 2.1 QH—5.1 SD 3.1 RME—5.4 SD 2.3
Schneidman [95]	1990	CCT	50–25 per group 23 male, 27 female Age—7–15 years	Four-point expansion RME	Two-point expansion RME	Molar and canine expansion	No statistically significant difference between the groups
Tsarapatsani [105] (16 year follow-up of Lindner 1989 and Lindner 1986)	1999	CCT	29 from original 105 11 male, 18 female Age—20 years	Grinding canine	Quad-helix	Crossbite correction Masticatory performance Asymmetry	2/29 (7%)—still had functional XB XB correction Grinding 57% QH 60%

Tullberg [103] (16–19 year follow-up of Lindner 1989 and Lindner 1986)	2001	CCT	44 from original 105 18 male, 26 female Age 21 years	1. Grinding canine 2. Quad-helix	No treatment	TMD signs and symptoms (S&S)	No statistically significant difference between the groups in any of the TMD S&S
Asanza [91]	1997	RCT	14–7 per group 7 male, 7 female Age—8.5–16 years	Banded Hyrax RME	Banded Hyrax RME	Molar expansion Cephalometric changes	Molar expansion Banded—mean 6 mm Bonded—mean 7 mm
Garib [92]	2005	RCT	8–4 per group All female Age—11.4–13.9 years	Tooth-tissue borne Haas RME	Tooth borne Hyrax RME	Molar expansion Inclination posterior teeth	Molar expansion Haas mean 8.1 SD 0.6 Hyrax mean 8.2 SD 0.9
Godoy [85]	2011	RCT	99–33 per group 41 male, 58 female Age—8 years	1. Quad-helix 2. Expansion plate	No treatment	Crossbite correction Treatment time Molar and canine expansion	Stability of XB—relapse QH—3/33 Expansion plate—3/33 Treatment time QH 4.24 months SD 2.05 Expansion Pl 6.12 months SD 3.25
Kilic [93]	2008	RCT	39–21 banded, 18 bonded 10 male, 29 female Age—13.5 years	Banded Hyrax RME	Banded Hyrax RME	Molar expansion Molar tipping and inclination	Molar expansion Banded—mean 6.7 mm (SD 1.99) Bonded—mean 7.3 mm (SD 1.45)

(continued)

Table 11.2 (continued)

Lagravere [105]	2010	RCT	62–20—TAME, 21 BAME, 21 no treatment	1. Tooth anchored maxillary expander (TAME) 2. Bone anchored maxillary expander (BAME)	No treatment	Molar expansion Inclination Pain	Molar expansion At 6 months: Hyrax—mean 5.83 mm (SD 1.54) Bone anch—mean 5.75 mm (SD 1.98) At 12 months Hyrax—mean 4.24 mm (SD 1.69) Bone anch—mean 4.03 mm (SD 1.49) Molar expansion: 'significant differences were found'
Lamparski [96]	2003	RCT	30–15 per group 15 male, 15 female Age—6.6–14.6 years	Four-point expansion RME	Two-point expansion RME	Molar and canine expansion	Canine expansion: Four-point mean 3.03 mm Two-point mean 1.7 mm
Lippold [106]	2013	RCT	82–40 slow exp, 42 no treatment Male/ Female—unclear Age—7.25 years	Bonded Hyrax slow expansion	No treatment	Maxillary and mandibular dimensions at 12 months	Molar expansion Hyrax—mean 5.1 mm Control—mean 0.8 mm
Martina [107]	2012	RCT	50–23 slow, 27 rapid expansion 13 male, 13 female Age—10 years	Two-band slow expansion	Two-band rapid expansion	Molar expansion	Canine expansion Hyrax—mean 3.6 mm Control—mean 1 mm Molar expansion SME—mean 6.3 mm SD 2.1 RME—mean 5.7 mm SD 1.6

McNally [86]	2005	RCT	60–30 per group 30 male, 30 female Age—11–16 years	Quad-helix	Expansion arch	Molar and canine expansion at 12 weeks	Molar expansion QH—mean 4.54 mm SD 1.27 Expansion arch—mean 5.09 mm SD 1.67 Canine expansion QH mean 1.4 mm SD 1.75 Expansion arch mean 2.12 mm SD 1.11
						Comfort	At start of treatment QH—75% slightly uncomfortable; 3.7% extremely uncomfortable Expansion arch—79% slightly uncomfortable; 3.7%, extremely uncomfortable After 1 week QH—19.7% totally comfortable EA—51.9% comfortable Analgesics QH—21% took painkillers EA—37% took painkillers
						Appearance	QH—25% disliked the appearance. EA—70% disliked the appearance
Mossaz-Joelson [108]	1989	RCT	10–5 per group 6 male, 4 female Age—8.6–12 years	Bonded Minne expander	Banded Minne expander	Molar expansion	Bonded—mean 7.9 mm SD 1.5 Banded—mean 5.3 mm SD 1.9
						Canine expansion	Bonded—mean 6.4 mm SD 1.1 Banded—mean 5.3 mm SD 1.9

(continued)

Table 11.2 (continued)

Oliveira [94]	2004	RCT	19–9—Haas RME, 10 Hyrax RME 6 male, 13 female Age 7.3–14.6 years	Tooth—tissue borne Haas RME	Tooth borne Hyrax RME	Molar expansion Cephalometric variables Treatment time Haas mean 170.4 days Hyrax mean 159 days	Molar expansion Haas 8.49 mm SD 2.33 Hyrax 3.73 mm SD 2.64 Treatment time Haas mean 170.4 days Hyrax mean 159 days
Oshagh [90]	2012	RCT	35–25 conventional, 10 spring screw 11 male, 24 female Age 8–14 years	Conventional expansion screw	Spring loaded expansion screw	Molar and canine expansion Arch size changes	Molar expansion Conventional 1.09 mm SD 1.16 Spring 1.02 mm SD 1.86
Petren [87]	2008	RCT	60–15 per group 26 male, 34 female Age—8.7 years	1. Quad-helix 2. Expansion plate 3. Composite onlays	No treatment	Crossbite correction Molar expansion Canine expansion	No significant difference in discomfort— from paper—no data given QH 15/15 Expansion plate 10/15 Onlays 2/15 QH mean 4.6 mm SD 1.19 Expansion plate mean 3.5 mm SD 1.54 Onlays mean 0.5 mm SD 0.46 Control mean 0.4 mm SD 0.43 QH mean 2 mm SD 1.18 Expansion plate mean 2.7 mm SD 2 Onlays mean 0.63 mm SD 0.7 Control mean 0.3 mm SD 0.25 QH mean 4.8 months SD 3.52 Expansion plate mean 9.6 months SD 3.04

Petren* [88] *Continuation of Petren 2008	2011	RCT	40–20 per group 14 male, 21 female Age—13.5 years 30/40 from Petren 2008	Quad-helix	Expansion plate	Crossbite correct	QH 19/20
							Expansion plate 15/15 QH mean 3.4 mm SD 1.38 Expansion plate mean 3.5 mm SD 1.19 QH mean 3.2 mm SD 2.28 Expansion plate mean 2.5 mm SD 1.68
Ramoglu [109]	2010	RCT	RME 17–6 males, 11 female Age—8.78 years SD 1.21 SRME 18–7 male, 11 female Age 8.63 years SD 1.09	Acrylic bonded rapid maxillary expansion (RME)	Acrylic bonded semi-rapid maxillary expansion (SRME)	Molar expansion— relapse	Relapse
							QH 1/20 Expansion plate 0/15 QH mean –0.8 mm SD 1.7 Expansion plate mean –0.4 mm SD 1.33
						Canine expansion—relapse	QH mean 0.4 mm SD 1.67 Expansion plate mean 0.2 mm SD 1.09 SRME 5.13 mm SD 1.47 RME 4.77 mm SD 1.53
						Molar expansion— relapse	SRME 5.71 mm SD 1.66 RME 5.11 mm SD 1.81

(continued)

Table 11.2 Continued

Thilander [4]	1984	RCT	61–33 grinding; ± expansion plate; 28 no treatment 24 male, 37 female Age—4–5 years	Grinding ± expansion canine ± expansion plate	No treatment	Crossbite correction at 13 years of age	Grinding only 9/33 Grinding + expansion plate 17/24 Control 6/28
Weissheimer [107]	2011	RCT	33—Haas 18, Hyrax 15 Age—10.7 years; range, 7.2–14.5 years	Haas RME	Hyrax RME	Skeletal and dental measures from CBCT	Molar occlusal expansion Haas 7.70 mm SE 0.20 Hyrax 7.90 mm SE 0.23 Molar apical expansion Haas 2.15 mm SE 0.18 Hyrax 3.14 mm SE 0.21

Also refs

Tullberg M, Tsarapatsani P, Huggare J, Kopp S. Long-term follow-up of early treatment of unilateral forced posterior crossbite with regard to temporomandibular disorders and associated symptoms. *Acta Odontologica Scandinavica*, 2011;59(5):280–284
 Tsarapatsani P, Tullberg M, Lindner A, Huggare J. Long-term follow-up of early treatment of unilateral forced posterior cross-bite. Orofacial status. *Acta Odontologica Scandinavica*, 1999;57(20):97–104

Haas

This is a tooth-tissue-borne expansion appliance that is fixed to the first permanent molars and first premolars using bands and includes a midline expansion screw. The expansion screw is connected to the bands via a framework of 0.9 mm stainless steel wire that is soldered to the bands and embedded in acrylic plates lying on the palate. The expansion screw can be activated at different rates to give slow (0.5 mm/week) or fast (0.5 mm/day) expansion.

HYRAX

This is a tooth-borne expansion appliance that is fixed to the first permanent molars and first premolars using bands and includes a midline expansion screw. The expansion screw is connected to the bands via a framework of 0.9 mm stainless steel wire. The framework is soldered to the bands laterally and medially; it is inserted into mesial and distal tubes in the expansion screw. The expansion screw can be activated at different rates to give slow (0.5 mm/week) or fast (0.5 mm/day) expansion (Fig. 11.3).

Bonded Acrylic Splint

This expansion appliance has acrylic occlusal coverage of the teeth in the buccal segments in the form of 'bite blocks' that are connected to a midline expansion screw via a metal framework of 0.9 mm stainless steel and/or acrylic plates covering the palate. The acrylic 'bite blocks' free up the occlusion by removing cuspal interferences and are cemented or bonded to the teeth. It can be tooth borne or tooth-tissue borne depending on the extent of the palatal coverage by the acrylic. The expansion screw can be activated at different rates to give slow (0.5 mm/week) or fast (0.5 mm/day) expansion.

Minne Expander

This is similar to a tooth acrylic expander but used a spring-loaded screw (Minne expander). The appliance is fixed using bands on the first permanent molars and first premolars that are joined by buccal and palatal connectors to which the screw is soldered. Turning a nut that compresses the spring activates the appliance.

Removable Appliances

Upper Removable Appliance

This appliance is retained using Adam's clasps on the first permanent molars and first premolars or deciduous molars. These are connected by a full palatal coverage, split baseplate of acrylic within which a midline expansion screw is embedded. The expansion screw can be activated to give slow (0.5 mm/week) expansion.

Implications for Research

In view of the current evidence, I think there is a need for further:

- Long-term epidemiological studies to monitor the effects of a unilateral posterior crossbite (UPXB) and the spontaneous correction rate as the evidence is conflicting
- Randomised controlled trials comparing competing interventions that:
 - Are methodologically sound
 - Are adequately powered
 - Record outcomes that take into account the participants' views and experience of the treatment
 - Report the proportion of cases whose UPXB was corrected and, in the follow-up period, remained stable
 - Follow up the children until their permanent dentition is established

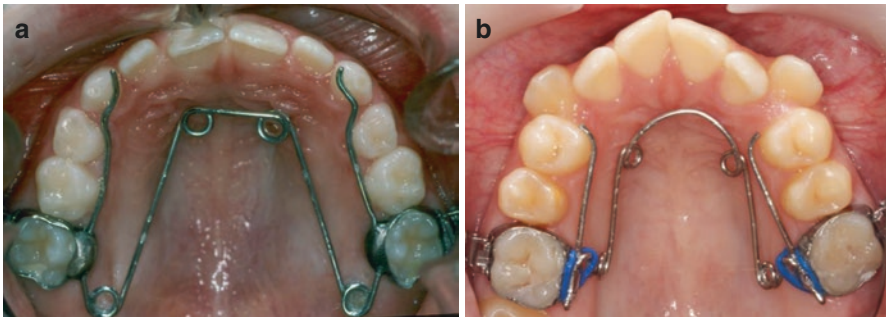


Fig. 11.2 (a) Fixed quad-helix. (b) Removable quad-helix



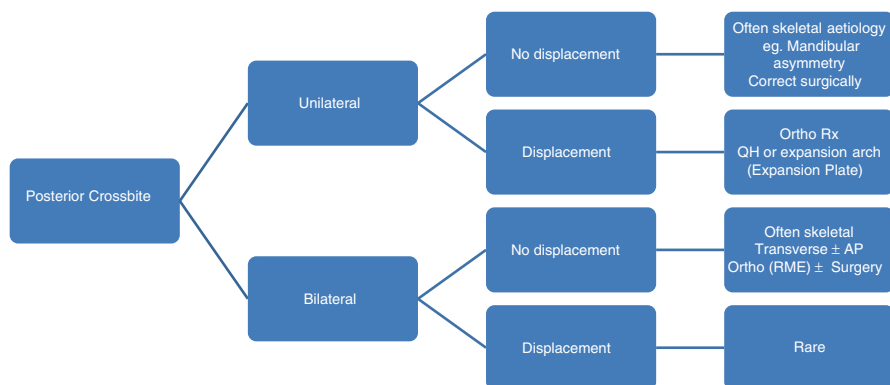
Fig. 11.3 Hyrax-type RME

Implications for Clinical Practice

Based on the current evidence:

- I think that early orthodontic treatment of unilateral posterior crossbites with mandibular shifts is advised because:
 - There is some weak evidence that posterior crossbites, with mandibular shifts, are associated with temporomandibular dysfunction and reduced bite force and that children and adolescents with UPXB show signs of some degree of mandibular asymmetry.
 - Treatment appears to be stable if undertaken in the mixed dentition.
- I would advocate the use of a quad-helix appliance as the evidence suggests that treatment is quicker; they are more cost-effective and tolerated better than their alternatives, i.e. expansion plate or arch.

Posterior Crossbite Treatment Sieve



References

1. Heikinheimo K, Salmi K, Myllarniemi S. Long term evaluation of orthodontic diagnosis made at the ages of 7 and 10 years. *Eur J Orthod.* 1987;9:151–9.
2. Kuroi J, Berglund L. Longitudinal and cost-benefit analysis of the effect of early treatment of posterior cross-bites in the primary dentition. *Eur J Orthod.* 1992;14:173–9.
3. Leighton BC. The early development of cross-bites. *Dent Pract.* 1966;17:145–52.
4. Thilander B, Wahlund S, Lennartsson B. The effect of early interceptive treatment in children with posterior cross-bite. *Eur J Orthod.* 1984;6:25–34.
5. Melsen B, Stensgaard K, Pedersen J. Sucking habits and their influence on swallowing pattern and prevalence of malocclusion. *Eur J Orthod.* 1979;1(4):271–80.
6. Bresolin D, Shapiro PA, Shapiro GG, Chapko MK, Dassel S. Mouth breathing in allergic children: its relationship to dentofacial development. *Am J Orthod.* 1983;83:334–40.
7. Cheng M-C, Enlow DH, Papsidero M, Broardbent BH, Oyen O, Sabat M. Developmental effects of impaired breathing in the face of the growing child. *Angle Orthod.* 1988;58:309–20.

8. Hannuksela A, Väänänen A. Predisposing factors for malocclusion in 7-year-old children with special reference to atopic diseases. *Am J Orthod Dentofacial Orthop.* 1987;92(4):299–303.
9. Linder-Aronson S. Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhinomanometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl.* 1970;265:1–132.
10. Subtelny JD. Oral respiration: facial maldevelopment and corrective dentofacial orthopedics. *Angle Orthod.* 1980;50:147–64.
11. Borrie FR, Bearn DR, Innes NP, Iheozor-Ejiiofor Z. Interventions for the cessation of non-nutritive sucking habits in children. *Cochrane Database Syst Rev.* 2015;(3):CD008694.
12. Dođramacı EJ, Rossi-Fedele G. Establishing the association between nonnutritive sucking behavior and malocclusions: a systematic review and meta-analysis. *J Am Dent Assoc.* 2016;147:926–934.e6.
13. Infante PF. An epidemiologic study of finger habits in preschool children, as related to malocclusion, socioeconomic status, race, sex, and size of community. *ASDC J Dent Child.* 1976;43(1):33–8.
14. Modeer T, Odenrick L, Lindner A. Sucking habits and their relationship to posterior cross-bites in 4-year-old children. *Scand J Dent Res.* 1982;90:323–8.
15. Ogaard B, Larsson E, Lindsten R. The effects of sucking habits, cohort, sex, intercanine arch widths, and breast or bottle feeding on posterior crossbite in Norwegian and Swedish 3-year-old children. *Am J Orthod Dentofacial Orthop.* 1994;106:161–6.
16. Abreu LG, Paiva SM, Pordeus IA, Martins CC. Breastfeeding, bottle feeding and risk of malocclusion in mixed and permanent dentitions: a systematic review. *Braz Oral Res.* 2016;30(1):e22.
17. Dimberg L, Lennartsson B, Amrup K, Bondemark L. Prevalence and change of malocclusions from primary to early permanent dentition: a longitudinal study. *Angle Orthod.* 2015;85(5):728–34.
18. Holm AK. Oral health in 4-year-old Swedish children. *Community Dent Oral Epidemiol.* 1975a;3(1):25–33.
19. Holm AK. Oral health in 5-year-old Swedish children. *Community Dent Oral Epidemiol.* 1975b;3(4):184–9. PubMed PMID: 1056827.
20. Köhler L, Holst K. Malocclusion and sucking habits of four-year-old children. *Acta Paediatr Scand.* 1973 Jul;62(4):373–9.
21. Tschill P, Bacon W, Sonko A. Malocclusion in the deciduous dentition of Caucasian children. *Eur J Orthod.* 1997;19(4):361–7.
22. Brunelle JA, Bhat M, Lipton JA. Prevalence and distribution of selected occlusal characteristics in the US population, 1988-1991. *J Dent Res.* 1996;75:Spec No:706-13.
23. Thilander B, Myrberg N. The prevalence of malocclusion in Swedish schoolchildren. *Scand J Dent Res.* 1973;81(1):12–21.
24. Corruccini RS. An epidemiologic transition in dental occlusion in world populations. *Am J Orthod.* 1984 Nov;86(5):419–26.
25. Foster TD, Day AJ. A survey of malocclusion and the need for orthodontic treatment in a Shropshire school population. *Br J Orthod.* 1974;1(3):73–8.
26. Infante PF. Malocclusion in the deciduous dentition in white, black, and Apache Indian children. *Angle Orthod.* 1975;45(3):213–8.
27. Malandris M, Mahoney EK. Aetiology, diagnosis and treatment of posterior cross-bites in the primary dentition. *Int J Paediatr Dent.* 2004;14(3):155–66.
28. Kutin G, Hawes RR. Posterior cross-bites in the deciduous and mixed dentitions. *Am J Orthod.* 1969;56(5):491–504.
29. Schröder U, Schröder I. Early treatment of unilateral posterior crossbite in children with bilaterally contracted maxillae. *Eur J Orthod.* 1984;6(1):65–9.
30. Iodice G, Danzi G, Cimino R, Paduano S, Michelotti A. Association between posterior cross-bite, masticatory muscle pain, and disc displacement: a systematic review. *Eur J Orthod.* 2013;35(6):737–44.

31. Egermark-Eriksson I, Carlsson GE, Magnusson T, Thilander B. A longitudinal study on malocclusion in relation to signs and symptoms of cranio-mandibular disorders in children and adolescents. *Eur J Orthod.* 1990;12:399–407.
32. McNamara JA Jr, Turp JC. Orthodontic treatment and temporomandibular disorders: is there a relationship? *J Orofac Orthop.* 1997;58:74–89.
33. Ninou S, Stephens C. The early treatment of posterior crossbites: a review of continuing controversies. *Dent Update.* 1994;21:420–6.
34. O'Bryn BL, Sadowsky C, Schneider B, BeGole EA. An evaluation of mandibular asymmetry in adults with unilateral posterior crossbite. *Am J Orthod Dentofacial Orthop.* 1995;107:394–400.
35. Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal factors. *J Dent Res.* 1993;72:968–79.
36. Gesch D, Bernhardt O, Kirbschus A. Association of malocclusion and functional occlusion with temporomandibular disorders (TMD) in adults: a systematic review of population-based studies. *Quintessence Int.* 2004;35(3):211–21.
37. Thilander B, Bjerklin K. Posterior crossbite and temporomandibular disorders (TMDs): need for orthodontic treatment? *Eur J Orthod.* 2012;34(6):667–73.
38. Thilander B, Rubio G, Pena L, de Mayorga C. Prevalence of temporomandibular dysfunction and its association with malocclusion in children and adolescents: an epidemiologic study related to specified stages of dental development. *Angle Orthod.* 2002;72(2):146–54.
39. Andrade Ada S, Gameiro GH, Derossi M, Gavião MB. Posterior crossbite and functional changes. A systematic review. *Angle Orthod.* 2009;79(2):380–6.
40. Talapaneni AK, Nuvvula S. The association between posterior unilateral crossbite and craniomandibular asymmetry: a systematic review. *J Orthod.* 2012;39:279–91.
41. Iodice G, Danzi G, Cimino R, Paduano S, Michelotti A. Association between posterior crossbite, skeletal, and muscle asymmetry: a systematic review. *Eur J Orthod.* 2016;38:638–51.
42. Hesse KL, Artun J, Joondeph DR, Kennedy DB. Changes in condylar position and occlusion associated with maxillary expansion for correction of functional unilateral posterior crossbite. *Am J Orthod Dentofacial Orthop.* 1997;111:410–8.
43. Kilic N, Kiki A, Oktay H. Condylar asymmetry in unilateral posterior crossbite patients. *Am J Orthod Dentofacial Orthop.* 2008a;133:382–7.
44. Langberg BJ, Arai K, Miner RM. Transverse skeletal and dental asymmetry in adults with unilateral lingual posterior crossbite. *Am J Orthod Dentofacial Orthop.* 2005;127:6–15.
45. Uysal T, Sisman Y, Kurt G, Romoglu S. Condylar and Ramal vertical asymmetry in unilateral and bilateral posterior crossbite patients and normal occlusion sample. *Am J Orthod Dentofacial Orthop.* 2009;136:37–43.
46. Veli I, Uysal T, Ozer T, Ucar F. Mandibular asymmetry in unilateral and bilateral posterior crossbite patients using cone-beam computed tomography. *Angle Orthod.* 2011;81:966–74.
47. Pirttiniemi P, Kantomaa T, Lahtela P. Relationship between craniofacial and condyle path asymmetry in unilateral cross bite patients. *Eur J Orthod.* 1990;12:408–13.
48. Schmid W, Mongini F, Felisio A. A computer based assessment of structural and displacement asymmetries of the mandible. *Am J Orthod Dentofacial Orthop.* 1991;100:19–34.
49. Kecik D, Kocadereli I, Saatci I. Evaluation of the treatment changes of functional posterior crossbite in the mixed dentition. *Am J Orthod Dentofacial Orthop.* 2007;131:202–15.
50. Lam PH, Sadowsky C, Omerza F. Mandibular asymmetry and condylar position in children with unilateral posterior crossbite. *Am J Orthod Dentofacial Orthop.* 1999;115:569–75.
51. Lippold C, Hoppe G, Moiseenko T, Ehmer U. Analysis of condylar differences in functional unilateral posterior crossbite during early treatment – a randomized clinical study. *J Orofac Orthop.* 2008;69:283–96.
52. Castelo PM, Pereira LJ, Andrade AS, Marquezin MC, Gavião MB. Evaluation of facial asymmetry and masticatory muscle thickness in children with normal occlusion and functional posterior crossbite. *Minerva Stomatol.* 2010;59:423–30.
53. Ferro F, Spinella P, Lama N. Transverse maxillary arch form and mandibular asymmetry in patients with posterior unilateral crossbite. *Am J Orthod Dentofacial Orthop.* 2011;140:828–38.

54. Kasimoglu Y, Tuna EB, Rahimi B, Marsan G, Gencay K. Condylar asymmetry in different malocclusion types. *Cranio*. 2015;33:10–4.
55. Kiki A, Kiliç N, Oktay H. Condylar asymmetry in bilateral posterior crossbite patients. *Angle Orthod*. 2007;77:77–81.
56. Kusayama M, Motohashi N, Kuroda T. Relationship between transverse dental anomalies and skeletal asymmetry. *Am J Orthod Dentofacial Orthop*. 2003;123:329–37.
57. Pirttiniemi P, Raustia A, Kantomaa T, Pyhtinen J. Relationships of bicondylar position to occlusal asymmetry. *Eur J Orthod*. 1991;13:441–5.
58. Primožic J, Ovsenik M, Richmond S, Kau CH, Zhurov A. Early crossbite correction: a three-dimensional evaluation. *Eur J Orthod*. 2009;31:352–6.
59. Primožic J, Perinetti G, Richmond S, Ovsenik M. Three-dimensional evaluation of facial asymmetry in association with unilateral functional crossbite in the primary, early, and late mixed dentition phases. *Angle Orthod*. 2013;83:253–8.
60. Takada J, Miyamoto JJ, Yokota T, Ono T, Moriyama K. Comparison of the mandibular hinge axis in adult patients with facial asymmetry with and without posterior unilateral crossbite. *Eur J Orthod*. 2015;37:22–7.
61. Abad-Santamaría L, López-de-Andrés A, Jiménez-Trujillo I, Ruíz C, Romero M. Effect of unilateral posterior crossbite and unilateral cleft lip and palate on vertical mandibular asymmetry. *Ir J Med Sci*. 2014;183:357–62.
62. Cohlma JT, Ghosh J, Sinha PK, Nanda RS, Currier GF. Tomographic assessment of temporomandibular joints in patients with malocclusion. *Angle Orthod*. 1996;66:27–35.
63. Halicioğlu K, Celikoglu M, Yavuz I, Sekerci AE, Buyuk SK. An evaluation of condylar and ramal vertical asymmetry in adolescents with unilateral and bilateral posterior crossbite using cone beam computed tomography (CBCT). *Aust Orthod J*. 2014;30:11–8.
64. Pellizoni SE, Salioni MA, Juliano Y, Guimarães AS, Alonso LG. Temporomandibular joint disc position and configuration in children with functional unilateral posterior crossbite: a magnetic resonance imaging evaluation. *Am J Orthod Dentofacial Orthop*. 2006;129:785–93.
65. van Keulen C, Martens G, Dermaut L. Unilateral posterior crossbite and chin deviation: is there a correlation? *Eur J Orthod*. 2004;26:283–8.
66. Sonnesen L, Bakke M, Solow B. Bite force in pre-orthodontic children with unilateral crossbite. *Eur J Orthod*. 2001;23(6):741–9.
67. Alarcón JA, Martín C, Palma JC. Effect of unilateral posterior crossbite on the electromyographic activity of human masticatory muscles. *Am J Orthod Dentofacial Orthop*. 2000;118(3):328–34.
68. Sonnesen L, Bakke M. Bite force in children with unilateral crossbite before and after orthodontic treatment. A prospective longitudinal study. *Eur J Orthod*. 2007;29(3):310–3.
69. Magalhães IB, Pereira LJ, Marques LS, Gameiro GH. The influence of malocclusion on masticatory performance. A systematic review. *Angle Orthod*. 2010;80(5):981–7.
70. Antczak AA, Tang J, Chalmers TC. Quality assessment of randomized control trials in dental research. *J Periodontol Res*. 1986;21(4):305–14.
71. Jadad AR, Moore RA, Carroll D, Jenkinson C, Reynolds DJ, Gavaghan DJ, McQuay HJ. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials*. 1996 Feb;17(1):1–12.
72. Castelo PM, Gavião MB, Pereira LJ, Bonjardim LR. Masticatory muscle thickness, bite force, and occlusal contacts in young children with unilateral posterior crossbite. *Eur J Orthod*. 2007;29(2):149–56.
73. Rentes AM, Gavião MB, Amaral JR. Bite force determination in children with primary dentition. *J Oral Rehabil*. 2002;29(12):1174–80.
74. Vanderas AP, Papagiannoulis L. Multifactorial analysis of the aetiology of craniomandibular dysfunction in children. *Int J Paediatr Dent*. 2002;12(5):336–46.
75. Sonnesen L, Bakke M, Solow B. Malocclusion traits and symptoms and signs of temporomandibular disorders in children with severe malocclusion. *Eur J Orthod*. 1998;20(5):543–59.

76. Alarcon JA, Martín C, Palma JC, Menéndez-Núñez M. Activity of jaw muscles in unilateral cross-bite without mandibular shift. *Arch Oral Biol.* 2009;54:108–14.
77. Andrade Ada S, Gavião MB, Gameiro GH, De Rossi M. Characteristics of masticatory muscles in children with unilateral posterior crossbite. *Braz Oral Res.* 2010;24:204–10.
78. Ciavarella D, Monsurrò A, Padricelli G, Battista G, Laino L, Perillo L. Unilateral posterior crossbite in adolescents: surface electromyographic evaluation. *Eur J Paediatr Dent.* 2012;13:25–8.
79. Ferrario VF, Sforza C, Serrao G. The influence of crossbite on the coordinated electromyographic activity of human masticatory muscles during mastication. *J Oral Rehabil.* 1999;26:575–81.
80. Ingervall B, Thilander B. Activity of temporal and masseter muscles in children with a lateral forced bite. *Angle Orthod.* 1975;45:249–58.
81. Moreno I, Sánchez T, Ardizzone I, Aneiros F, Celemin A. Electromyographic comparisons between clenching, swallowing and chewing in jaw muscles with varying occlusal parameters. *Med Oral Patol Oral Cir Bucal.* 2008;13:E207–13.
82. Piancino MG, Farina D, Talpone F, Merlo A, Bracco P. Muscular activation during reverse and non-reverse chewing cycles in unilateral posterior crossbite. *Eur J Oral Sci.* 2009;117:122–8.
83. Tecco S, Tetè S, Festa F. Electromyographic evaluation of masticatory, neck, and trunk muscle activity in patients with posterior crossbites. *Eur J Orthod.* 2010;32:747–52.
84. Woźniak K, Szyszka-Sommerfeld L, Lichota D. The electrical activity of the temporal and masseter muscles in patients with TMD and unilateral posterior crossbite. *Biomed Res Int.* 2015;2015:1–7.
85. Godoy F, Godoy-Bezerra J, Rosenblatt A. Treatment of posterior crossbite comparing 2 appliances: a communitybased trial. *Am J Orthod Dentofacial Orthop.* 2011;139:e45–52.
86. McNally MR, Spary DJ, Rock WP. Randomized controlled trial comparing the quad-helix and the expansion arch for the correction of crossbite. *J Orthod.* 2005;32(1):29–35.
87. Petré S, Bondemark L. Correction of unilateral posterior crossbite in the mixed dentition: a randomized controlled trial. *Am J Orthod Dentofacial Orthop.* 2008;133:790.e7–13.
88. Petré S, Bjerklín K, Bondemark L. Stability of unilateral posterior crossbite correction in the mixed dentition: a randomised clinical trial with a 3-year follow-up. *Am J Orthod Dentofacial Orthop.* 2011;139:e73–81.
89. Sandikçiođlu M, Hazar S. Skeletal and dental changes after maxillary expansion in the mixed dentition. *Am J Orthod Dentofacial Orthop.* 1997;111(3):321–7.
90. Oshagh M, Momeni Danaei S, Hematiyan MR, Hajian K, Shokoohi Z. Comparison of dental arch changes and patients' discomforts between newly designed maxillary expansion screw and slow expansion procedures. *J Denti Shiraz Univ Med Sci.* 2012;13(3):110–9.
91. Asanza S. Comparison of Hyrax and bonded expansion appliances. *Angle Orthod.* 1997;67(1):15–22.
92. Garib DG, Henriques JF, Janson G, Freitas MR, Coelho RA. Rapid maxillary expansion – tooth tissue-borne versus tooth-borne expanders: a computed tomography evaluation of dento-skeletal effects. *Angle Orthod.* 2005;75(4):548–57.
93. Kilic N, Kiki A, Oktay H. A comparison of dentoalveolar inclination treated by two palatal expanders. *Eur J Orthod.* 2008b;30(1):67–72.
94. Oliveira NL, Da Silveira AC, Kusnoto B, Viana G. Three dimensional assessment of morphologic changes of the maxilla: a comparison of 2 kinds of palatal expanders. *Am J Orthod Dentofacial Orthop.* 2004;126(3):354–62.
95. Schneidman E, Wilson S, Erkis R. Two-point rapid palatal expansion: an alternate approach to traditional treatment. *Pediatr Dent.* 1990;12(2):92–7.
96. Lamparski DG Jr, Rinchuse DJ, Close JM, Sciote JJ. Comparison of skeletal and dental changes between 2-point and 4-point rapid palatal expanders. *Am J Orthod Dentofacial Orthop.* 2003;123(3):321–8.
97. Lindner A. Longitudinal study on the effect of early interceptive treatment in 4-year-old children with unilateral cross-bite. *Scand J Dent Res.* 1989;97(5):432–8.

98. Agostino P, Ugolini A, Signori A, Silvestrini-Biavati A, Harrison JE, Riley P. Orthodontic treatment for posterior crossbites. *Cochrane Database Syst Rev.* 2014;(8):CD000979.
99. Petré S, Bondemark L, Söderfeldt B. A systematic review concerning early orthodontic treatment of unilateral posterior crossbite. *Angle Orthod.* 2003;73(5):588–96.
100. Zhou Y, Long H, Ye N, Xue J, Yang X, Liao L, Lai W. The effectiveness of non-surgical maxillary expansion: a meta-analysis. *Eur J Orthod.* 2014;36(2):233–42.
101. Zuccati G, Casci S, Doldo T, Clauser C. Expansion of maxillary arches with crossbite: a systematic review of RCTs in the last 12 years. *Eur J Orthod.* 2013;35(1):29–37.
102. Birnie DJ, McNamara TG. The quadhelix appliance. *Br J Orthod.* 1980;7(3):115–20.
103. Tullberg M, Tsarapatsani P, Huggare J, Kopp S. Long-term follow-up of early treatment of unilateral forced posterior crossbite with regard to temporomandibular disorders and associated symptoms. *Acta Odontologica Scandinavica,* 2011;59(5):280–284.
104. Tsarapatsani P, Tullberg M, Lindner A, Huggare J. Long-term follow-up of early treatment of unilateral forced posterior cross-bite. Orofacial status,. *Acta Odontologica Scandinavica,* 1999;57(20):97–104,
105. Lagravere MO, Carey J, Heo G, Toogood RW, Major PW. Transverse, vertical, and antero-posterior changes from boneanchored maxillary expansion vs traditional rapid maxillary expansion: a randomized clinical trial. *Am J Orthod Dentofacial Orthop.* 2010;137(3):304.
106. Lippold C, Stamm T, Meyer U, Végh A, Moiseenko T, Danesh G. Early treatment of posterior crossbite – a randomised clinical trial. *Trials.* 2013;14:20.
107. Martina R, Cioffi I, Farella M, Leone P, Manzo P, Matarese G, et al. Transverse changes determined by rapid and slow maxillary expansion. A low-dose CT-based randomised controlled trial. *Orthod Craniofac Res.* 2012;15:159–68.
108. Mossaz-Joelson K, Mossaz C. Slow maxillary expansion: a comparison between bonded and banded appliances. *Eur J Orthod.* 1989;11:67–76.
109. Ramoglu SI, Sari Z. Maxillary expansion in the mixed dentition: rapid or semi-rapid? *Eur J Orthod.* 2010;32:11–8.
110. Weissheimer A, de Menezes LM, Mezomo M, Dias DM, de Lima EM, Rizzato SM. Immediate effects of rapidmaxillary expansion with Haas-type and hyrax-type expanders: a randomized clinical trial. *Am J Orthod Dentofacial Orthop.* 2011;140(3):366–76.