TEXTBOOK

Orthodontic Management of Class II Malocclusion

An Evidence-Based Guide

Martyn T. Cobourne
Editor

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Editor
Martyn T. Cobourne
Faculty of Dentistry, Oral & Craniofacial Sciences
King's College London
London, UK

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Preface

Class II malocclusion represents a significant component of the day-to-day case load for most working orthodontists. It manifests with a wide range of clinical variation and can represent something of a challenge to achieve consistently successful outcomes. This textbook has been written as an evidence-based guide to the clinical management of class II malocclusion and will serve as a useful reference for all clinicians interested in managing this wide-ranging malocclusion.

The chapters have been written by an international group of orthodontists with considerable experience and expertise in the theoretical and practical aspects of class II management. The first section of the book contains a brief overview of the clinical and epidemiological characteristics of class II malocclusion, followed by a discussion of treatment timing and then an extensive overview of the contemporary evidence base relating to outcomes for class II treatment. The second section deals with the practical aspects of managing class II treatment in children and adults and includes chapters on the use of removable and fixed functional appliances, molar distalisation, fixed appliances, and an outline of current aspects relating to aligner-based treatment. These chapters are followed by a separate overview of class II division 2 treatment and a final chapter on orthodontic-surgical management of class II cases.

This text will be of interest to specialist trainees in orthodontics, newly graduated orthodontic practitioners, and those with more experience in managing class II cases. It provides a succinct and definitive overview of strategies aimed at correcting this type of malocclusion with multiple clinical examples and reference to the contemporary evidence base. We hope it will be of relevance to the global orthodontic community and will find its place on their bookshelves.

London, UK — Martyn T. Cobourne

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1 Epidemiological and Clinical Features of Class II Malocclusion

Jadbinder Seehra

1.1 Introduction

A Class II occlusion has been recognised since the 1900s [1]. In the antero-posterior dimension, this malocclusion is subdivided into three categories. A Class II Division 1 incisor relationship is defined as when the lower incisor edges lie palatal to the cingulum plateau of the upper incisors. Typically, the upper incisors are proclined or of an average inclination with an increased overjet (Fig. 1.1). In a Class II intermediate incisor relationship, the upper incisors are upright or slightly retroclined with an increased overjet present. In contrast, in a Class II Division 2 incisor relationship, the upper incisors are retroclined with a minimal overjet (Fig. 1.2). However, in this category, the lower incisors can also be retroclined and the overjet maybe increased [2].

Based on molar relationship, within a Caucasian sample the prevalence of Class II Division 1 and Class II Division 2 malocclusions were reported at 19% and 4% respectively [1]. More contemporary prevalence studies have reported variation in the prevalence of a Class II malocclusion within different genders and ethnicities (Table 1.1).

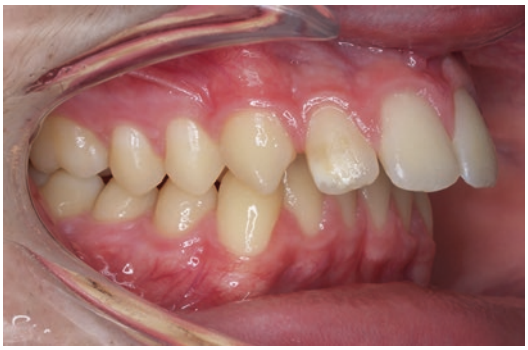

Fig. 1.1 Class II Division 1 incisor relationship

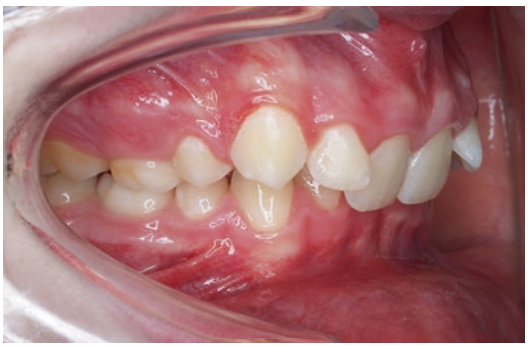

Fig. 1.2 Class II Division 2 incisor relationship

J. Seehra (✉)
Department of Orthodontics, Faculty of Dentistry, Oral & Craniofacial Sciences, Kings College London, London, UK
e-mail: Jadbinderpal.seehra@kcl.ac.uk

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Table 1.1 Prevalence of Class II malocclusion

Study	Ethnicity	Sample size	Age (Years)	Prevalence (%)	Sub-division (%)
Silva and Kang 2001 [3]	Latino	507	12-18	21.5	II Div 1 (91.5%) II Div 2 (8.5%)
El-Mangoury and Mostafa 1990 [4]	Egyptian	501	18-24	21.0	II Div 1 (16.2%) II Div 2 (4.8%)
Horowitz 1970 [5]	White	718	10-12	22.5	
Helm 1968 [6]	Danish	3842	6-18	24.5	
Lew et al. 1993 [7]	Chinese	1050		21.5	
Garner and Butt 1985 [8]	American (Afro-carribean)	445	13-15	16.0	
Garner and Butt 1985 [8]	African	505	13-14	7.9	II Div 1 (7.9%) II Div 2 (0.0%)
Grewe et al. 1968 [9]	Indian	651	9-14	9.6	
Salzmann 1977 [10]	American	7514	12-17	32.0	

1.2 Aetiology of Class II Malocclusion

As with many malocclusions, a multifactorial model consisting of both environmental and genetic factors has been proposed in the aetiology of a Class II malocclusion. Both exposure to alcohol during embryonic development [11] and preterm births [12] have been associated with the development of retrognathic mandibles. Although inter-arch relationships such as overjet, overbite, and molar relationship appear not to be under genetic control [13], environmental factors such as caries experience [14] and non-nutritive sucking behaviours (NNSB) can influence these occlusal traits.

The term non-nutritive sucking behaviour (NNSB) describes habitual sucking of digits, pacifiers, and objects by a child in order to source comfort, a sense of security, and calmness [15]. Although this may be viewed as a normal process of a child's growth and development, depending on the age of the child, the presence of NNSB can result in occlusal disturbance and contribute to the development of a malocclusion which exhibits Class II features [16] [17]. Historically, in the vertical dimension, as a result of reduced alveolar growth [18], an anterior open bite with an associated tongue thrust swallowing pattern can manifest. The horizontal component of forces generated by NNSB can increase the maxillary arch length with concomitant proclination of the maxillary incisors [16]. The size of the resultant increased overjet can be exacerbated by retroinclination of the mandibular incisors [16]. Finally, in the transverse dimension, more commonly in the primary dentition, a posterior crossbite can develop [16]. Systematic evidence has also reported variations in the impact on the occlusion depending on if the NNSB involves the use of a pacifier or sucking of digits. For instance, an increased overjet in the primary dentition is less likely to occur if a pacifier is used. However, the use of a pacifier is associated with an increased risk of developing a Class II canine relationship and posterior crossbite. In contrast, in the mixed dentition, the risk of developing a posterior crossbite and anterior open bite is greater with digit sucking [19]. The vertical and antero-posterior effects on the occlusion tend to diminish once the NNSB has been ceased [16]. However, the more the NNSB persists the greater the risk of developing a malocclusion [19].

Support for the genetic basis of a Class II malocclusion stems from the observation of retrognathic mandibles in patients with congenital

craniofacial abnormalities such as Pierre Robin sequence and Treacher Collins [20]. Additionally, dental anomalies such as ectopic maxillary canines [21] and microdontia [22] which have been proposed to have a genetic aetiology are commonly associated with a Class II Division 2 malocclusion leading to some authors to propose a shared genetic basis for the development of the maxillomandibular discrepancy. More recent studies have provided further insight into the development of a Class II malocclusion [20]. Polygenic inheritance and autosomal dominance models, with incomplete penetrance and variable expressivity, have been proposed for both Class II Division 1 and Class II Division 2 [22, 23, 24]. At a genetic level, a Class II malocclusion characterised by mandibular hypoplasia was detected in four families in which the affected individuals were homozygous for the rare allele of the polymorphism rs1348322 within the NOGGIN gene [25] which has been shown to be involved in mandibular formation in a mice model [26].

1.3 Clinical Features of Class II Malocclusion

1.3.1 Dentoskeletal

Within the literature, longitudinal growth studies have reported great variability in the dentoskeletal components of a Class II malocclusion. These studies tended to employ serial lateral cephalograms to assess the growth and development of the Class II skeletal and dental complex against a sample of "normal occlusions." However, inconsistent use of different cephalometric analyses may explain the observed variability in the reported dentoskeletal components of a Class II malocclusion.

Between the ages of 3 and 7 years, the cranial base and the maxilla are normal. The mandibular corpus and lower facial height are reduced, the gonial angle is large, and the dentoalveolar position of the mandible is in a retruded position. Both the height of the ramus and the skeletal position of the mandible are normal. However, the chin becomes slightly retruded after 5 years of age. In the transverse dimension, the maxilla is deficient. Importantly, the skeletal component of a Class II does not appear to be established during the deciduous dentition [27]. However, this observation is not universal, as mandibular retrusion and a short mandibular length have been reported in a sample of 5–8-year-olds [28]. In contrast, the occlusal features of a Class II are established during this age range. These include distal terminal plane of the second deciduous molars, Class II canine relationship, increased overjet and overbite and both a narrow upper dental arch and maxillary base [27, 28].

Between 8 and 10 years of age, the skeletal components of the Class II malocclusion seem to establish. However, variability exists in both the antero-posterior and vertical dimensions [29]. In the majority of cases, the maxilla is in the normal position. When it deviates from this position it is more likely to be retrusive rather than protrusive. A more protrusive maxilla as a key component of the Class II malocclusion has been reported [30]. However, overall, mandibular retrusion appears to be a common feature of Class II malocclusions [29] which is exacerbated by a shorter total mandibular length [28]. Indeed, in addition to a reduced lower face height proportion, a degree of mandibular retrusion appears to be a key characteristic in both Class II Division I and Class II Division II malocclusions [31]. Dentally, the maxillary incisors can be average, proclined, or of a more retroclined position which is particular to Class II Division II malocclusions [31]. The lower incisors can have an average, retroclined, or proclined inclination [29]. The position of both the upper and lower incisors can be reflective of the degree of dentoalveolar compensation for the underlying skeletal discrepancy. Rather than reporting features from a cohort of radiographs and comparing them to a control group, statistical modelling has been employed to identify dentoskeletal predicators or distinct facial patterns of a Class II malocclusion. Using this method, five vertical and six horizontal (A-F) morphological features of a Class II malocclusion have been described [32] (Table 1.2). As highlighted previously, even with this classification, different features in both the vertical and

Table 1.2 Morphological features of a Class II malocclusion as classified by Moyers et al. [32]

Feature	Type	Dentoskeletal component
Horizontal	A	Normal skeletal profile. Maxillary dentition is protracted resulting in Class II molar relationship, increased overjet and overbite.
	B	Midface prominence with normal mandible size.
	C	Marked Class II profile characterized by retrusion of the maxilla and mandible. Proclined lower incisors. Upper incisors upright or proclined.
	D	Retrognathic profile characterized by a small mandible. Midface is normal or slightly retruded. Mandibular incisors are upright or retroclined. Upper incisors proclined.
	E	Maxillary protrusion with normal or protruded mandible. Maxillary and mandibular incisor proclination.
	F	Milder Class II profile with mandibular retrusion.
Vertical	1	Steep mandibular plane (High angle). Anterior face height greater than posterior face height.
	2	Mandibular, functional occlusal and palatal plane are flatter than normal. Increased overbite present. "Square face" appearance.
	3	Palatal plane tipped upward anteriorly. Steep mandibular plane (High angle) with skeletal open-bite.
	4	Mandibular, functional occlusal and palatal plane are tipped downwards.
	5	Mandibular and functional occlusal plane are normal. Palatal plane is tipped downwards. Skeletal deep-bite.

horizontal dimensions can contribute to the Class II dentoskeletal features.

In a Class II malocclusion, it is unlikely that the dentoskeletal discrepancy will improve or "self-correct" with further growth [33, 34]. A greater skeletal facial convexity with a retruded mandibular position is maintained into adulthood [35]. However, in adolescence, a reduction in this facial convexity characterised by a decrease in ANB angle has been reported resulting in a marginal improvement in the overjet [36].

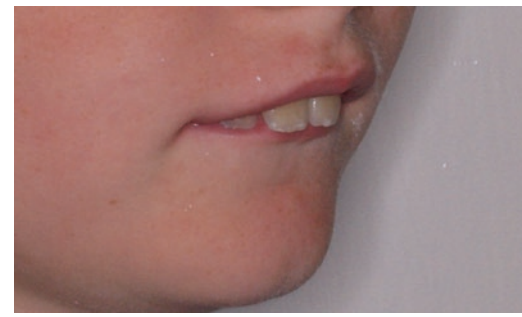

Fig. 1.3 Class II Division 1 incisor relationship complicated by a lower lip trap

1.3.2 Soft Tissues

The position of the lower lip relative to the labial surface of upper and lower incisors plays a role in the aetiology of a Class II Division 1 and Class II Division 2 malocclusion. In a sample of patients with a Class II Division 1 incisor relationship, the presence of a complete lower lip trap has been reported to result in more mandibular incisor retroclination and an increased overjet compared to a matched sample of patients without a lower lip trap [37]. The proclination of the upper incisors in a Class II Division 1 malocclusion can also be exacerbated by a short upper lip and a low lip level with flaccid tone which exerts less lip pressure allowing the upper incisors to "escape control" of the lower lip [38] (Fig. 1.3).

The role of resting pressure exerted from the lower lip and the resultant position of the maxillary incisors is also pertinent in the aetiology of Class II Division 2 incisor relationship. This is clinically described as a high lip level and in this situation the lip has a thicker lip shape but is not hypertonic in nature [39]. However, compared to Class I patients, a higher lip pressure was recorded in patients with a Class II Division 2 malocclusion. Clinically, the resultant effect on the maxillary incisors is a more extruded and

retroclined incisor position [40]. This observation was further confirmed in a cephalometric evaluation which suggests that a high lip-line level is a primary aetiological factor in the development of Class II Division 2 malocclusion [41].

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2 Treatment Timing in the Management of Class II Malocclusion

Martyn T. Cobourne

2.1 Background

Class II malocclusion is characterized by a discrepancy in the sagittal relationship, which leads to a post-normal occlusion. This is often associated with an increased overjet in class II division 1 cases; but if the upper incisors are retroclined, then a class II division 2 occlusion will exist. The extent of the skeletal discrepancy will influence the severity of the post-normal relationship and complexity of potential treatment; and whilst dentoalveolar disproportion often plays a role in the etiology of class II malocclusion, it is the skeletal discrepancy that usually represents the main contributing factor. The maxilla can be too far forward within the facial complex, the mandible can be too far back, or some combination of maxillary prognathia and mandibular retrognathia may co-exist. In many cases, mandibular retrognathia is the defining feature. These discrepancies can also be seen in association with a vertical component, either increased vertical proportions and a reduced overbite or open bite; or reduced vertical proportions and an increased overbite.

M. T. Cobourne (✉)
Centre for Craniofacial Development & Regeneration, Faculty of Dentistry, Oral & Craniofacial Sciences, King's College London, London, UK
e-mail: martyn.cobourne@kcl.ac.uk

In the growing child with a class II skeletal discrepancy, a common treatment approach is to attempt growth modification: most commonly, encouraging upward and forward growth of the mandible and restriction of forward maxillary growth, or some combination of the two. In this chapter we will briefly review the data relating to how the jaws grow within the context of overall body growth, and how successful growth modification strategies can be in the management of class II skeletal discrepancies. Finally, we will discuss the evidence relating to how the timing of class II growth modification strategies can potentially influence treatment outcomes.

2.2 Mandibular Growth and Growth in Stature

The relationship between height versus chronological age or height-distance curve for a developing child will demonstrate a relatively constant approximate three-fold increase in height from birth through to the age of around 18–19 years (Fig. 2.1, left panel). However, an incremental plot of height change versus chronological age or height-velocity curve will show significant fluctuations in rates of growth. There is rapid growth at birth, progressively decelerating until around 3 years of age; then a more slowly decelerating phase that lasts until puberty, albeit punctuated by a short acceleration of juvenile growth around the

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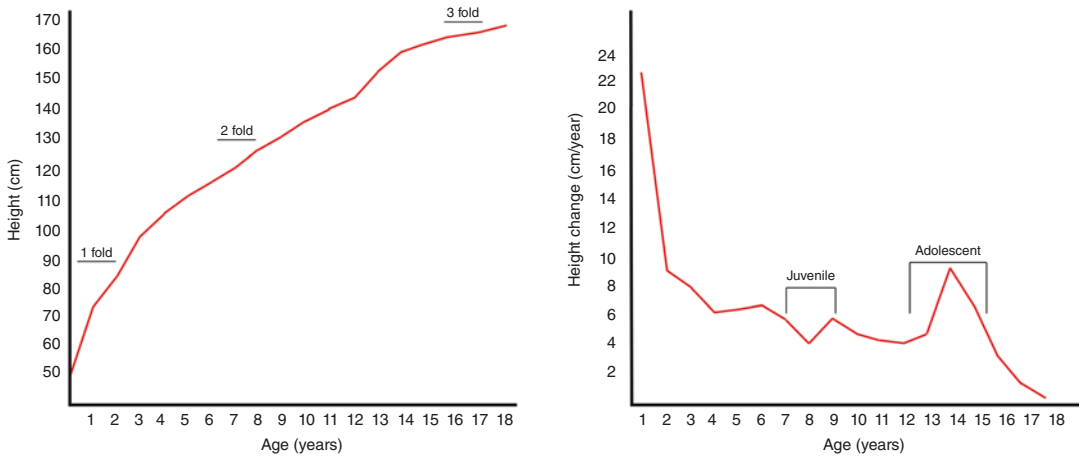

Fig. 2.1 Schematic representation of height-distance (left panel) and height-velocity curves (right panel) for a male from birth to 18 years of age. There is an approximately three-fold increase in height (left panel) and two growth spurts, both juvenile and adolescent—with the adolescent growth spurt being associated with significant changes in height-velocity

age of 6–8 years, followed by the adolescent growth spurt around the ages of 12–14 years, which varies in timing between males and females, and different individuals, and is followed by a progressive deceleration in growth velocity until adulthood (Fig. 2.1, right panel). Thus, height-velocity change does not have a constant relationship with chronological age and will reach a maximum during the pubertal growth spurt.

The mandibular condyle is a key driver of postnatal mandibular growth, and it is known that condylar growth is not constant during development—following a broadly similar pattern to that observed for somatic growth. A correlation between the condylar growth curve and pubertal growth spurt has also been reported [1–3] although this is not a precise relationship and condylar peak velocity does not seem to absolutely coincide with peak height-velocity [4] (Fig. 2.2). In terms of stature, peak height-velocity generally occurs around 12 years of age in females and 14 years in males, with the onset of this growth period generally occurring around 2 years prior to the peak [5]. However, these figures are associated with wide individual variation and there is no universally accepted method of reliably predicting skeletal age or the point of onset associated with an individual's pubertal growth spurt (or more specifically for the orthodontist—mandibular growth spurt). A number of techniques have been described, which can broadly be classified as those associated with clinical evaluation, including chronological age [6], sexual maturity [7], and monitoring of height changes [8]; or more direct

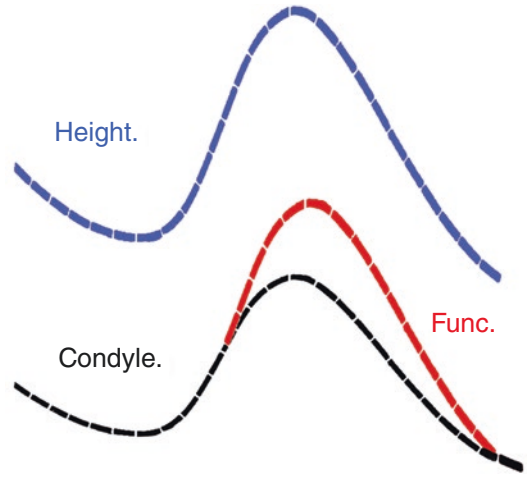

Fig. 2.2 Evidence exists to suggest that there is a correlation between the adolescent growth spurt (height-velocity; blue hatched line) and condylar growth-velocity (black hatched line). There is less evidence that the use of a functional appliance during the pubertal growth spurt can produce accelerated condylar growth (red hatched line). The x-axis represents chronological age spanning the pubertal growth spurt (increasing age from left to right); the y-axis represents height-velocity (blue) and mandibular condylar growth-velocity (black)

assessment of skeletal maturity based upon radiographic investigation, which has included development of the dentition [9], maturation of bones in the hand-wrist [10], or cervical vertebrae [11]. Orthodontists have investigated the relative merits of these techniques for many years and the literature is replete with conflicting data [3, 12–14]. The use of individual hand-wrist radiographs (or indeed, serial radiographs) to predict the pubertal growth spurt is not sufficiently accurate for use in clinical orthodontics [15, 16] and does not seem to afford any meaningful correlation with growth increases in mandibular length [17]. The additional radiation associated with the taking of hand-wrist radiographs as a method of estimating skeletal age in relation to orthodontic treatment cannot be justified. The cervical vertebral maturation (CVM) method has been described as a useful alternative to the hand-wrist radiograph for growth rate estimation [11]. The method is based upon morphological characteristics of the second to fourth cervical vertebrae, which are identifiable on a collimated lateral skull radiograph and therefore do not require any additional exposure to radiation beyond that for a normal pre-treatment examination (assuming of course, that assessment of CVM is not the specific reason for taking the lateral skull radiograph). There is conflicting data within the current literature that CVM represents a more accurate assessment than the hand-wrist method in predicting skeletal maturation—although on balance, the weight of evidence would suggest that it does [18–21]. However, there do not seem to be any significant advantages of CVM in assessing skeletal age or predicting the pubertal growth spurt in comparison to chronological age [12].

2.3 Treatment Changes Induced by Class II Growth Modification

Classic dentofacial orthopedic treatment in class II cases aims to maximize forward growth of the mandible whilst restraining growth of the maxilla. This can be achieved with the use of a functional appliance and/or the application of extra-oral force through headgear. The essential philosophy relating to these approaches is that condylar growth can potentially be accelerated through stimulation of the condylar cartilage, whilst maxillary sutural growth can be restrained through the class II forces established by a postured mandible and certainly through the application of headgear directly to the maxilla. In relation to the condyle, acceleration of growth may then ultimately lead to a larger mandible (and by inference although not necessarily by logic) correction of the class II skeletal pattern (Fig. 2.3).

Animal studies have shown evidence of molecular, cellular, and dimensional changes accompanied by growth and remodeling of the condyle (and glenoid fossa) when the mandible is habitually postured forward with a fixed intra-oral appliance [22, 23]. However, human clinical studies investigating clinically relevant growth changes related to functional appliance treatment are less convincing. The evidence base is generally low-level—being composed predominantly of retrospective case-control studies, some prospective

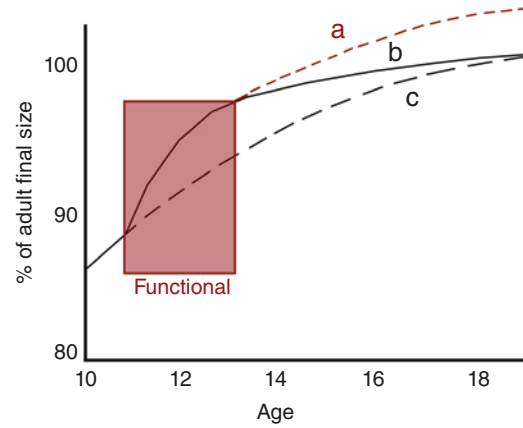

Fig. 2.3 (**a**) Growth stimulation with a functional appliance—growth is accelerated with the functional appliance during treatment (red rectangle) and continues at the expected rate after completion of treatment to produce a larger jaw; (**b**) Growth acceleration with a functional appliance—growth is accelerated with the functional appliance treatment but continues at a reduced rate after completion of treatment to produce a jaw that is ultimately the same size as that achieved with no treatment; (**c**) Normal mandibular growth in the absence of treatment. (Redrawn from Proffit, WR, Fields, HW, Larsson, BE, Sarver, DM. Contemporary Orthodontics, Sixth Edition, Elsevier, ISBN 978-0-323-54387-3)

controlled studies, and only a few randomized clinical trials. Moreover, these investigations rely primarily upon cephalometric analysis to measure growth changes and often focus on mandibular unit length (which often does not correlate with meaningful class II correction in the sagittal plane). These methodological problems notwithstanding, early orthopedic treatment with headgear will provide a posterior translation of the anterior maxilla through an annualized mean reduction in SNA of around 1.6 degrees [24]; whilst analysis of removable functional appliance data has suggested that this treatment can achieve an increase in mandibular unit length of around 2 mm, but this figure is based primarily upon retrospective data and analysis of RCTs alone shows less difference [25]. This is not to say that functional appliances are not effective at correcting a sagittal discrepancy associated with a class II malocclusion, but this seems to be achieved predominantly through dentoalveolar rather than skeletal change [26]. There is some evidence to suggest that they can also have a slight inhibitory effect on sagittal growth of the maxilla over the short term, but this represents less than 1 mm per year [27]. In relation to fixed functional appliances, there is little high-quality evidence that these devices can significantly influence craniofacial growth [28] and dentoalveolar effects also seem to predominate [29] although again, when retrospective data is incorporated into the analysis, maximal changes in mandibular unit length of around 2 mm have also been reported in pubertal patients [30].

Overall, it would seem that the effectiveness of functional appliances is mostly due to early correction of the buccal occlusion and overjet reduction through differential tooth movement, allowing the establishment of a class I relationship that is maintained whilst normal condylar growth catches up [31].

2.4 Do We Get a Better Response with Early Treatment?

Historically, there has been considerable interest amongst orthodontists regarding the relative advantages and disadvantages of early class II treatment. In the broadest sense, some early studies suggested that treating a young child with a class II skeletal discrepancy in the early mixed dentition with a functional appliance and/or headgear could produce significant skeletal changes [32]. This led the advocates of early intervention to claim that starting at this time maximized the success of treatment through enhanced orthopedic change, simplifying any subsequent treatment with fixed appliances in the permanent dentition, and reducing any reliance on dental compensation and extractions. Moreover, it has been argued that early correction of an increased overjet can improve a child's self-esteem and reduce the risk of trauma to the maxillary incisor dentition. However, the data supporting many of these claims was retrospective, and there had been more than a suspicion from some of these studies that the enhanced skeletal growth afforded by early treatment was often lost over the longer term [33].

Recognizing this lack of high-quality evidence, three landmark randomized clinical trials (RCTs) were conducted over a period of around a decade in the late 1990s and early 2000s, two in the United States of America and one in the United Kingdom [34–39]. These trials compared early mixed dentition treatment of class II malocclusion with either a functional appliance (bionator or twin-block) and/or headgear followed by any further treatment required in the permanent dentition, to a single course of comprehensive treatment carried out in early adolescence. The American studies were interested primarily in whether growth could be significantly influenced by early treatment, whilst the UK-based study was more invested in understanding differences in the process of treatment for class II cases, depending upon whether you started early or late. Interestingly, the findings were remarkably similar—early treatment was effective in correcting a class II malocclusion and reducing an increased overjet; however, later treatment achieved this very effectively as well. There were few differences in extraction rates between early and late treatment strategies, but a single course of later treatment did require less appointments and take slightly less time over-

all—although the period of treatment during adolescence was slightly shorter if an early phase of treatment had previously been undertaken. Importantly, at the end of the overall evaluation period, no clinically significant skeletal or dental differences were apparent between children treated early or late [40].

One further argument for early treatment of class II malocclusion has been to help prevent maxillary incisor trauma [41]. It is well known that an increased overjet is a risk factor for incisor trauma and it is intuitive to conclude that the earlier an overjet is reduced, the less potential risk there is for the child traumatizing their upper front teeth. These RCTs [34–39, 42] and a more recent one based in Sweden [43] did investigate trauma incidence in their samples and collectively found a reduction in the early treatment groups. However, there was much heterogeneity in how trauma was recorded and none of the trials were powered to detect trauma. Interestingly, in all of these trials, a significant number of children had experienced trauma before embarking on early treatment—meaning that as a trauma prevention strategy early overjet reduction needs to be started very early. However, in selected cases with high vulnerability to possible trauma, early overjet reduction might represent a reasonable strategy [44].

2.5 Is Orthopedic Correction of Mandibular Deficiency with a Functional Appliance Enhanced When Treatment Coincides with the Pubertal Growth Spurt?

Those who advocate an orthopedic approach to the management of class II malocclusion argue that the condylar cartilage has a primary role in directing growth of the mandible and will respond positively to forward posture with a functional appliance. Given the known (albeit poorly understood) association between increased mandibular growth and the pubertal growth spurt (see Fig. 2.2), the natural conclusion of this philosophy is that orthopedic functional appliance treatment undertaken during the pubertal growth spurt will be more successful than that carried out either in the pre- or post-pubertal periods. This theory makes a number of assumptions; not least, that the significant gross growth-related changes observed in the condylar cartilages of various juvenile animal models subjected to a variety of mandibular advancement appliances can be extrapolated to humans treated with functional appliances; that the pubertal growth spurt can be predicted with any degree of accuracy in different individuals; that the minimal long-term differences in overall mandibular growth observed between children treated with and without functional appliances is fundamentally wrong (i.e., functional appliances can make the mandible grow larger to a clinically significant degree) and that any accelerated growth beyond what might be achieved without intervention will be essentially linear and contribute to meaningful sagittal correction.

It is difficult to institute high-quality prospective RCTs investigating the influence of treatment timing on orthopedic outcomes in the management of class II malocclusion. Indeed, the accuracy of different methods available to identify whether a child has even entered the growth spurt is questionable—and even if these methods were definitive, the ethics of denying treatment to a child about to undergo their growth spurt as part of an RCT make this area of clinical research challenging. Unfortunately, because of this, the evidence base and therefore appropriate systematic review is problematic [45]. The data relating to removable functional appliances is populated by retrospective studies [46], those using historical growth studies for control groups [47] and indeed, studies that have only investigated either pre-pubertal or post-pubertal subjects in isolation [48]. Current data would suggest an annualized increase in total mandibular length of no more than 2 mm in children treated during the pubertal growth spurt in comparison to those treated before [45]. However, the meta-analysis relating to these data is dominated by one study with a significant risk of bias [47] and if this study is excluded from the analysis there is little difference between groups. Overall, the data to suggest

that timing functional appliance treatment to coincide with the growth spurt will result in any clinically significant difference in mandibular dimensions is weak.

2.6 Conclusions

It would seem sensible when treating class II cases with growth modification to accept that the treatment effects will be essentially dentoalveolar. The evidence that this treatment strategy can elicit clinically significant skeletal change is weak. On this basis, a reasonable strategy would be to start treatment in the late mixed dentition with the expectation that the "growth modification" phase would be complete by the early permanent dentition, facilitating a seamless transition into fixed appliances. Advocating early treatment or treatment during the pubertal growth spurt to encourage clinically significant additional mandibular growth is not supported by the evidence.

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