CHAPTER 1

Biological basis for functional appliance therapy

A functional appliance is one that uses the facial muscles and masticatory muscles to produce changes in the position of the individual teeth or arches. Any oral appliance causing a change in the forces of occlusion and alteration in muscular activity is likely to produce displacement of individual teeth or arches. Therefore such appliances can be either removable, inducing a displacement of the mandible by a process of interference or by stimulating an avoidance reflex, or fixed, involving the use of a mechanism causing the mandible to be held in a different position for function.

Facial growth

Maxillary growth occurs primarily by intra-membranous ossification with surface remodelling, resulting in a downward–forward displacement of the maxilla at an angle of approximately 40 degrees to the cranial base.[1] Growth of the maxilla is complex and may be affected by alterations in the sutures of the maxillae. Resorption on the superior surface and the apposition of bone on other surfaces affect the position of the maxillary dento-alveolar complex, with resorption of the anterior surface being typical during the downward–forward growth of the basal bone. However, while apposition of bone occurs on the inferior surface of the palate, resorption occurs on the superior surface, resulting in a net downward displacement (Figure 1.1).

Björk and Skieller's tantalum implant studies have shown that mandibular growth in children and adolescents occurs mainly as a consequence of an increase in condylar length in a posterior and superior direction due to endochondral ossification.[2] Elsewhere mandibular growth is a product of surface apposition and remodelling. Appositional growth does not occur anteriorly at the chin, with chin growth being expressed chiefly at the lateral aspects. Mandibular growth otherwise manifests as remodelling of the alveolus and of the bony areas with muscular attachments. Growth of the ascending ramus primarily occurs posteriorly, with resorption on the anterior aspect (Figure 1.2).

The mandible is not directly attached to the skull, but rather held in position by the muscles, ligaments and tendons, with the condylar head of the mandible being placed in the glenoid fossa within the temporal bone. The synovial articulation between the condyle and the temporal bone is classified as a ginglymoarthroidal joint, as both a ginglymus (hinging) and arthroidal (sliding) element exist, permitting the required mandibular opening and excursive movements during function. Changes in the position of the glenoid fossae will have consequent effects on the position of the mandible.

Orthodontic therapy involving functional appliances therefore might be expected to produce changes in the position of both the maxilla and the mandible, and combinations of growth restraint and growth induction would result in clinical changes in three dimensions. Detailed information on facial growth has been presented by Enlow[1] and Björk and Skieller.[2]

While increases in the absolute mandibular dimensions outstrip those of the maxilla during adolescence, this does not normally result in occlusal improvement in Class II malocclusion without active orthodontic intervention.[3] Based on longitudinal data from growth studies, some straightening of the profile and reduction in facial convexity may occur during the pubertal growth phase,[4] although this has not been a universal finding[5] and little change in the skeletal profile occurs in late adolescence.[6] Foley and Mamandras[7] noted that twice as much mandibular as maxillary growth arose in Class II males and females from 14 to 20 years old based on a North American Caucasian sample. However, a greater increase in absolute mandibular length is to be expected, as its overall dimension is greater than that of the maxilla, with the percentage difference in the increase between mandibular and maxillary less significant; mandibular length also incorporates a profound vertical element, while maxillary growth is usually measured from ANS (anterior nasal spine) to PNS (posterior nasal spine) and is therefore essentially horizontal. Positive occlusal interdigitation may also limit changes in interarch relationships. Moreover, in an analysis of patients with skeletal 2 patterns aged 8 to 18 years and increased overjet who had no orthodontic treatment, 4 mm more forward growth of the mandible than the maxilla was observed, but the occlusion and overjet remained unchanged into adulthood; this lack of change was attributed to the cuspal interdigitation.[8]

The rate of craniofacial growth, particularly of the maxilla and the mandible, is believed to undergo a pre-pubertal peak. The rate of growth is generally limited prior to this period, although a transient juvenile peak in growth rate has been

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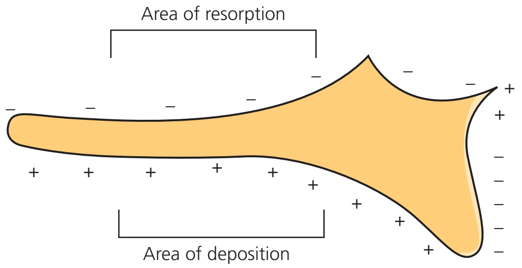

Figure 1.1 Resorption on the superior surface of the maxilla accompanied by deposition on the palate surface leads to an inferior displacement.

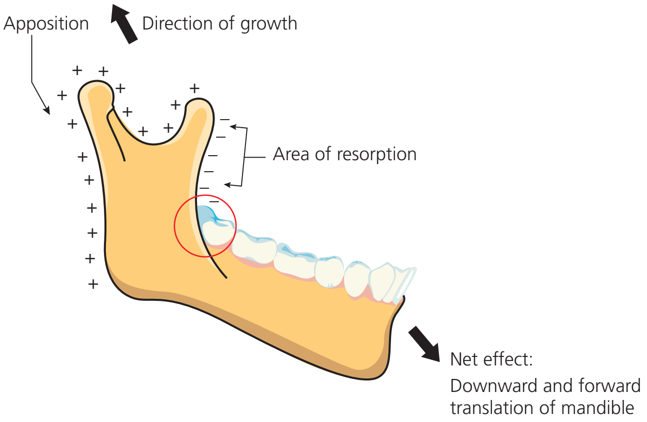

Figure 1.2 Mandibular growth occurs via condylar growth in a posterior and superior direction resulting in downward and forward displacement. Resorption on the anterior surface of the ascending ramus combined with resorption on the posterior surface leads to forward movement of the ramus.

described in females. Riolo et al.[9] described an annual rate of increase in the length of the mandibular body (Gonion–Pogonion) of 1.7 mm and 2.5 mm, respectively, in 8-year-old males and females. The corresponding figures at 13 years were 2 mm and 1.8 mm. Intuitively, therefore, treatment involving growth modification is ideally timed during a period of maximal growth. However, while this sounds relatively simple, a range of techniques directed at timing treatment have been developed and trialled, with limited success (Chapter 4). For example, while the rate of mandibular growth is thought to mirror increases in statural height, there is significant variation.[10]

Arbitrary use of chronological age, typically 10 to 13 years in females and 11 to 14 years in males, continues to be an accepted method of estimating the timing of most efficient and effective growth modification in Class II subjects. However, little difference has been demonstrated in the relative skeletal effectiveness of functional appliances in subjects of mean age 10 years relative to a group treated just after the onset of puberty (mean age 12 years 11 months).[11] Moreover, Pancherz et al., who in earlier research highlighted an increase in condylar growth rate in harmony with increases in statural height,[12] have since reported on the use of the Herbst appliance in skeletally mature patients with demonstrable, albeit limited, skeletal changes based on magnetic resonance imaging of the temporomandibular joints.[13]

Function and craniofacial morphology

Craniofacial growth is believed to be capable of a certain degree of morphological adaptation subject to functional requirements, with function known to be required for normal homeostasis and cellular turnover.[14] This theory is based on the work of Van der Klaauw, subsequently popularized by the American anatomist Melvin Moss.[15] According to the functional matrix theory, facial growth, final shape and dimensions are governed by the role of resident organs and tissues, specifically the senses, and essential functions including eating, cognition and breathing. Moss believed that the properties of important organs were related to underlying skeletal components. In particular, two major functional elements (cerebral and facial) were described with unique tissues and spaces. Moss hypothesized that expansion of each capsular matrix was accompanied and facilitated by bone growth via endochondral and intra-membranous ossification to preserve functional spaces. These hypotheses were supported by experimental evidence demonstrating altered skeletal growth following separation from soft tissue elements, while the presence of enveloping soft tissues led to the observation of normal growth patterns. Applying Moss's concepts to the potential for modification of growth with functional appliance therapy, it could be argued that postural changes with associated soft tissue alteration may be accompanied by a redirection or indeed acceleration of skeletal growth. Moreover, correction of abnormal soft tissue patterns and behaviour was a tenet for the pioneers of functional appliance therapy, many of whom advocated its use to restore normal function and development. Moreover, in animal models altered masticatory function and associated changes in muscular loading have been shown to affect condylar cartilage thickness and chondroblast differentiation.[16, 17]

The malleability of cranial shape following the application of continuous forces during the process of cerebral growth and skull development has been demonstrated in tribal groups. This is apparent in the skulls of indigenous people in South America, where the bandaging of the skull from shortly after birth resulted in significant alteration in the shape of the cranium (Figure 1.3). It would appear that the overall size of the brain has been maintained while the shape of the supporting cranium is significantly altered. Similarly, dramatic changes have been observed in long bones and as a result of other local practices including foot binding, which reduces foot size to an extent by repositioning the bony elements.

Orthodontists involved in changing the facial shape of those with malocclusion would wish to alter similarly the directional growth of the mandible in relation to the maxilla. Positional change in these relationships could be sufficient to correct sagittal, vertical and transverse occlusal discrepancies. It has been recognized that skeletal II discrepancies are primarily related to the position of the mandible relative to the maxilla rather than the overall size of the underlying bones (Figure 1.4).[18–20] McNamara, in an analysis of a North American Caucasian group, has, for example, shown that 49% of skeletal II patterns presented with SNA (Sella–Nasion–A point) values below 81 degrees. Moreover, the SNB (Sella–Nasion–B point) was below 78 degrees in 82% of the sample.[19] Consequently, the majority of experimentation and clinical efforts have been centred on the ability to produce permanent change in mandibular position and dimensions.

Figure 1.3 An example of the effects of cranial binding in a South American female from the Atacama desert. Typically, binding is undertaken for a relatively short period (approximately 6 months) in infancy; the effects are marked and persist into adulthood.

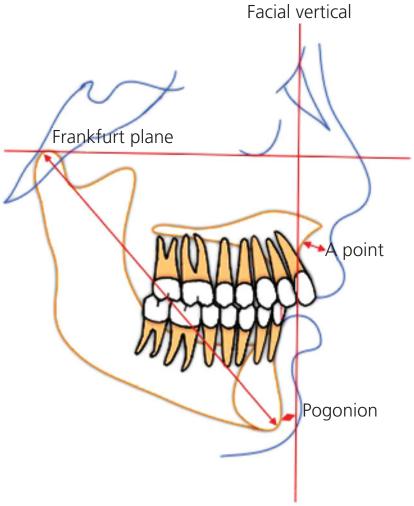

Figure 1.4 Skeletal II discrepancy is typically related to mandibular retrognathia rather than maxillary protrusion. However, analysis of the Burlington, Bolton and Ann Arbor samples demonstrated that 49% of skeletal II patterns were associated with SNA values below 81 degrees. Moreover, SNB was below 78 degrees in 82% of the sample.[19] Therefore, much of the focus of growth modification has been on the propensity to effect lasting change on mandibular position and dimensions. McNamara subsequently developed a cephalometric analysis involving a facial vertical line drawn perpendicular to the Frankfurt plane.[20]

Forward mandibular posture appears to concentrate stresses within the mandibular condyle. Finite element analysis has indicated that stress levels within the condyle are doubled with flexible fixed functional appliances, for example.[21] Moreover, Gupta et al.,[22] in an experimental model, have reported accumulation of tensile stresses in the postero-superior aspect of the condyle with sustained mandibular forward posture. Similarly, tensile forces arise in the glenoid fossa within the posterior connective tissues. It is postulated that these mechanical changes might correlate with enhanced cellular differentiation. While clinical research, most recently in the form of randomized trials, has become the mainstay of this experimentation, laboratory-based experimentation on primates and rodents provides much of our theoretical knowledge on the biological basis for growth modification and functional appliance therapy.

Experiments on primates

The condylar cartilage is a secondary cartilage capable of regional adaptive growth, contrasting with primary long-bone epiphyseal articular cartilages. Secondary cartilage appears later in embryonic development, with chondrification of the condyle thought to begin around week 9 *in utero*, and has a distinctive pattern of organization and proliferation with appositional growth, while primary cartilage grows interstitially (see Chapter 3). Primary cartilage is thought to respond to systemic growth stimuli such as hormones, while secondary cartilages only secondarily react to these stimuli. Moreover, while hypertrophic chondrocytes tend to be arranged in columns in long bones, they are organized haphazardly in condylar cartilage; this may favour a multi-directional growth pattern in response to mechanical stimuli. Furthermore, condylar cartilage is not loaded by body weight but by sporadic and intermittent forces applied during mastication, swallowing and parafunctional activity. Mechanical loading and stimuli are prerequisites for normal condylar growth, inducing specific biochemical responses in chondrocytes and pre-osteoblasts. Decreasing the load on the mandibular condyle by reducing occlusal contact has been implicated in a thinner, less dense condylar cartilage layer.[23]

Postural changes have long been considered capable of producing occlusal change, with for example Andreasen highlighting marked occlusal changes with his eponymous appliance, the Andreasen Activator. However, the ability to produce significant skeletal change has been contested primarily since the advent of cephalometry, when evidence began to emerge that orthodontics may be restricted to inducing dento-alveolar change and the concept of the immutability of the skeletal pattern became accepted.[24]

Extrapolation of animal experimentation to the human form is complicated by a range of factors. A specific problem in studying mandibular growth in animals is the nature of the mandible in each species, with unique patterns of attachment of the muscles into the condyle, individual shaped discs and glenoid fossae and specific types of mastication. In an effort to explore growth changes in mammals most comparable to human primates, a number of studies have been undertaken on various species of Macaque monkeys. Nevertheless, there are accepted and influential differences in the pattern and rate of growth between these species and humans, with Macaque, for example, being skeletally mature by the age of 3 years. Moreover, their metabolism is believed to outstrip the rate of human metabolism by a factor of approximately 4 and associated cellular turnover is markedly more rapid than in humans.

In an analysis of Rhesus monkeys, Moyers et al.[25] studied the effects of anterior mandibular displacement produced by occlusal overlay splints at the equivalent of 6 years of age in humans. After 3 months of treatment, a skeletal III pattern was produced with associated overcorrection of the molar relationships to Class III. Treatment-related changes included an increased growth rate at the maxillary tuberosity allied to restraint of vertical maxillary growth in the molar region. In addition, accelerated posterior and superior condylar growth occurred during the treatment period. Dental changes were more limited, with some mesial movement of the lower molars observed. The mandibular growth acceleration was confirmed, as posterior manipulation of the condyles was not possible under general anaesthesia.

An early study by Stöckli and Willert[26] examined the condyle and glenoid fossae of the Macaque *irus* monkeys. Two of the animals had no intervention and six of the experimental animals had 5 mm forward displacement of the mandible with a cemented splint; the animals were sacrificed at different periods to compare the nature of the growth at pre-specified intervals. The conclusion was that the condyle had a characteristic pattern of growth. The condyle was shown to have an outer surface, the articular surface, formed primarily of fibrocartilage. The layer immediately underneath is referred to as the intermediate cellular proliferative layer. This area is cartilaginous in nature, with thickening induced in the layer of cartilage and an increase in the number of cells in response to prolonged mandibular displacement. The third layer of hyaline cartilage is essentially a cartilage being replaced gradually by bone with consolidation. The overall effect of this forward displacement of the mandible was increased length in the bone, which was greater than expected in the non-intervention group. The proliferative area was shown to be increased up to five times more in experimental animals and it was also noted that an increased layer of cellular proliferation occurred in the glenoid fossae.

Further studies by McNamara et al.[27–29] in Macaque monkeys incorporating tantalum implants have identified similar changes and highlighted the relevance of treatment timing of the treatment, based on reported changes in the electromyographic (EMG) activity in the lateral pterygoid muscles. Observation was made of the length of time required to produce additional bone rather than cartilage, with the latter being a less permanent structure. It was concluded that the mandible should be advanced in a step-wise way with gradual advancement rather than single-step activation, with the objective of repeated activation of the lateral pterygoid muscles being postulated to result in additional growth of the condyles. However, Sessle et al.,[30] in a study with a sample of just 4, suggested that the impact of progressive advancement (1.5–2 mm every 10–15 days) on activity within the lateral pterygoid, masseter and anterior digastric was not markedly different to that associated with larger, one-step activation.

A limitation of these studies is that the overall effect of functional therapy in normal primates is to produce a frank reversed overjet with a true skeletal III relationship, as the selection of skeletal II animals is not possible. These changes arise primarily as a consequence of an elongation of the mandible. A study involving implants and electromyographic sensors using Herbst appliances on primates found that occlusal correction was predominantly (70%) attributable to skeletal change from a combination of maxillary restraint, mandibular condylar growth and glenoid fossa remodelling, with 30% of the change due to dental movement. Despite the apparent limitations of animal-based research,[31] these findings have since repeatedly been corroborated within clinical research.[32, 33]

Other animal studies

A number of studies have been undertaken on the condyle of rodents, particularly rats. Apart from the obvious morphological differences (Figure 1.5), significant key growth-related differences exist that complicate extrapolation into humans. For example, using collagen X expression and capillary endothelium as surrogate measures of maximal mandibular growth, growth rate may peak as early as days 38–56 in the rat.[34] Furthermore, rat alveolar bone tends to be denser than in humans and bone plates are without marrow spaces; there are also marked differences in the arrangement of periodontal fibres.[35] Although rat condyles also have a specific arrangement, with a different discal attachment and very much larger lateral pterygoid muscles, it has been possible to evaluate the treatment-induced changes histologically with various types of functional appliances. Petrovic et al.[36, 37] highlighted the presence of prechondroblasts in the proliferative layer beneath the surface fibrous capsule. These prechondroblasts tend to proliferate and are increased in number when a functional appliance is placed and the animal has the mandible actively postured forwards. Surgical incision of the lateral pterygoid muscle was shown to prevent this change from occurring; the lateral pterygoid muscle was, therefore, identified as a critical active component inducing additional condylar growth. This finding lent further support to the concept that the muscle should be activated incrementally to ensure that additional growth was maintained throughout the functional phase. It has been noted, however, that the lateral pterygoid muscle in rats has a greater bulk and more extensive attachment than that of primates.[38] Nevertheless, further work on

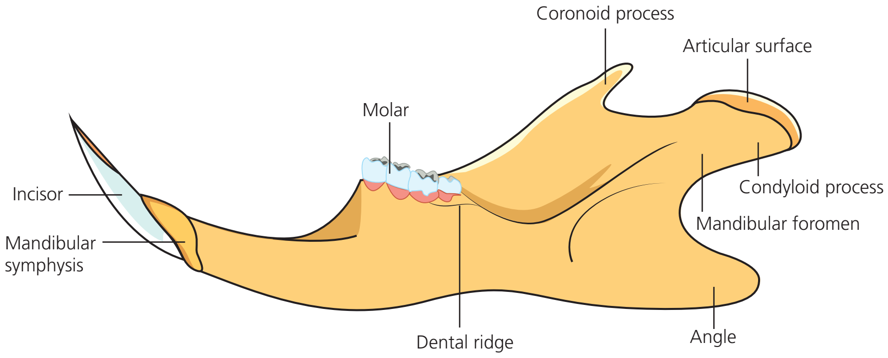

Figure 1.5 Schematic representation of the mature rat mandible. The rabbit and mouse mandibles have a similar morphology with a short ramus and relatively pronounced angle.

the rat model by Petrovic et al. has highlighted that mandibular advancement induced a significant time-related thickening of the prechondroblast–cohondroblast layer, with bone deposition along the posterior border of the ramus inferior to the condylar cartilage over a period of 6 weeks.[39]

Further *in vitro* research by Rabie[40] and co-workers produced detailed information on the cellular changes occurring in the condyles, highlighting the biochemical cascades induced by stimulation of the lateral pterygoid muscle resulting in vascular infiltration into the retrodiscal tissues, and also provided details on the regulation of collagen synthesis. They also observed the requirement for Type II collagen in the cartilage to be regulated by the agent Sox9 resulting in Type X collagen, which is required prior to the ossification of the cartilage. It was observed that this ossification took approximately 5 months to occur from placement of the experimental appliances. This 5-month phase in the rat is likely to be indicative of a much more prolonged period in humans or primates. These authors, therefore, advocate incremental stimulation of the lateral pterygoid muscle to induce meaningful additional bone growth at the condyles, resulting in supplemental growth beyond that which could be anticipated during normal maturation. A pivotal role for Sox9 and the development of collagen II and X has also been highlighted in a mouse model,[41] with their upregulation and secretion shown during condylar regeneration subsequent to experimental condylectomy.

Rabie et al. further investigated the expression of vascular endothelial growth factor (VEGF) secondary to incremental advancement in an allied study[42] on bony apposition posteriorly in the glenoid fossa. VEGF expression was found to increase and to coincide with new bone formation; increases in both were observed in the experimental group. It appears, therefore, that sustained postural change induces a series of tissue responses producing increased vascularization and bone formation, and that this pattern may be attributable to candidate biochemical markers. Moreover, using similar methodology Tang and Rabie have identified a pivotal role for Runx2, a transcription factor required for chondrocyte maturation and osteoblast differentiation, in the regulation of endochondral ossification following mandibular advancement.[43] A further analysis involving Sprague–Dawley rats has highlighted upregulation of fibroblast growth related factor (FGF8) following mandibular advancement over periods ranging from 3 to 30 days.[44] Cellular enlargement and differentiation were observed in both the condylar cartilage and the glenoid fossa during treatment with bony apposition by endochondral ossification in the condyle and intra-membranous ossification in the glenoid fossa.

Further research on a rabbit model has highlighted the role of matrix-metalloproteases (MMPs), particularly collagenases such as MMP-1 and MMP-13, on removal of extra-cellular matrix, inducing chondrocyte enlargement and differentiation required for bony apposition.[45] MMP expression following forward posture may be amplified by exogenous local administration of transforming growth factor beta and insulin-like growth factor in the inferior joint space. Experimental research of this nature using exogenous hormone delivery in the animal model directed at supplementing mandibular growth has yet to be translated into humans.

The visco-elastic theory

A particular type of activator appliance was developed by Harvold[46] (Figure 1.6) involving activation with an increased vertical dimension very much beyond the rest position, with the objective of stretching the facial musculature and soft tissues. This appliance was underpinned by a different philosophy, with Harvold postulating that changes in mandibular growth could be induced by this passive stretch; the family of appliances that this spawned became known as myotonic appliances.

Woodside et al.[47] evaluated the effects of a fixed functional appliance again in an animal study on Macaque monkeys, finding that increased activation of the appliance by 7–10 mm resulted in forward movement of the mandible without significant growth in its length. These changes arose due to marked cartilage proliferation in the glenoid fossa, which was most apparent in the growing juvenile. Subsequently, the changes achieved with Twin Block and Herbst appliances were

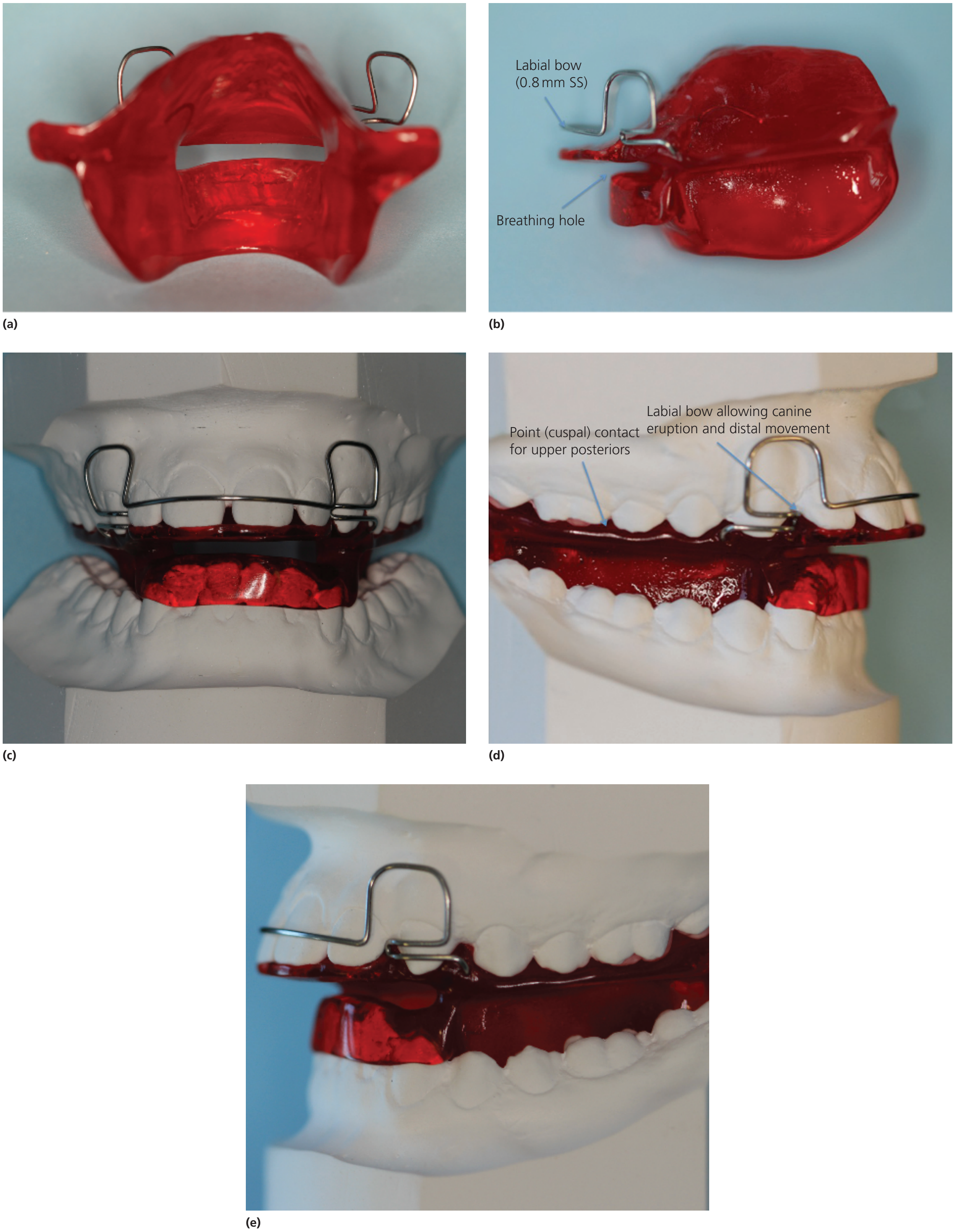

Figure 1.6 Harvold activator (a–e). The inter-maxillary force should theoretically be concentrated on both the maxillary dentition and palate, while the forces are transmitted to the lingual aspect of the mandible rather than the lower teeth. Consequently, well-extended lower impressions with adequate lingual depth, in particular, are required. The postured bite is taken 8–10 mm beyond the freeway space with near maximal protrusion, this degree of vertical opening allows the inclusion of an anterior breathing hole. During fabrication, extensive plaster relief is important in the lower posterior region to promote full eruption and lower arch levelling, while restricting unwanted lower incisor proclination with extension of the lower anterior acrylic onto the labial aspect of the mandibular incisors (c). The molars are afforded space to erupt, particularly in the lower arch to facilitate arch levelling and overbite reduction. An upper labial bow in 0.8 mm spring hard stainless steel may be added to facilitate retention, although more flexible wire may be used where space closure in the upper anterior region is planned. The labial bow should permit eruption and distal movement of the maxillary canines where required. The relief for the upper posteriors is such that it provides cusp tip contact with the upper acrylic plate with no interference, which might inhibit distal movement of the upper posteriors (d, e). These elements are usually introduced during the fabrication stage, with chairside trimming not usually required. The upper anterior aspect of the acrylic plate should extend to the incisal edges of the maxillary incisors to facilitate three-dimensional control, and a relief chamber is provided palatal to the incisors to facilitate intrusion without retraction.

attributed to visco-elastic stretching forces[48] and these authors described three growth stimuli: displacement, visco-elasticity and referred force from the condyle to the glenoid fossa. They termed this pattern the growth relativity hypothesis. Further investigations by Voudouris et al.[31, 49] involved application of Herbst treatment to Macaque monkeys and identified statistically significant additional growth of the glenoid fossa and condyle in juveniles, with reduced electromyographic postural activity and evidence of comparable levels of growth within the condyle and glenoid fossa. These researchers highlighted the fact that the transition from cartilage to bone was not complete until 18 weeks of therapy. Withdrawal of the postured bite at an earlier stage was subject to antero-posterior relapse. Similar changes in a human subject would require a longer period due to the greater duration of adolescence and slower rate of growth. Voudouris further reported that new bone formation at the condyle and glenoid fossa is related to age and is associated with decreased postural EMG activity of the masticatory muscles, including the lateral pterygoid, masseter and anterior belly of the digastric.[49]

Treatment duration

Fixed functional appliances and removable functional appliances that are worn on a full-time basis will usually correct the overjet and molar relationship within 6 months, but relapse on withdrawal of the appliance is a common finding.[50, 51] A study on rats by Chayanupatkul et al.[52] reported on the histological changes when functional appliances were removed early or following more protracted periods of treatment. The authors found that bone formation is not complete at the condyle following 5 to 7 months of treatment with a Type III collagen remaining. Type III collagen is known to be unstable, leading to emergency-type bone that is less resistant to reversal during function and mastication. The researchers recommended that the treatment time should be doubled to allow replacement bone to be established at the condyle. Extrapolating these laboratory findings to the clinical scenario, it may be reasonable to suggest that at least 1 year of full-time therapy is required to allow establishment of additional bone at both the condyle and glenoid fossa.

Maxillary restraint

All functional appliances used in Class II correction involve either stimulation of the masticatory or facial muscles or stretching of the tissues, which results in transmission of forces to the upper dentition and maxilla. Early animal experiments alluded to restraint of maxillary growth with full-time wear of functional appliances.[29] McNamara et al. also noted occlusal plane changes associated with growth restriction, with the occlusal plane tipping upwards anteriorly secondary to appliance therapy.[29] Consistently in clinical studies with fixed functional appliances, it is noted cephalometrically that reduced forward growth of the maxilla occurs in comparison with untreated subjects. Numerous studies have reported remodelling and an associated change in the position of the glenoid fossa.[48, 50, 53, 54] Some clinicians have designed appliances with the objective of training the mandible to posture forward with an avoidance reflex and the objective of avoiding dento-alveolar movement of the upper and lower dentition,[55–57] but inevitably a degree of dental movement of the upper and lower incisors and reduced maxillary forward movement are evidenced on cephalometric radiographs. Moreover, restraint of vertical maxillary growth has been attempted to encourage a more horizontal vector of forward mandibular growth by restricting downward–backward mandibular rotation. In particular, variants in appliance design in high-angle cases allowing adjunctive use of orthopaedic headgear, such as the Teuscher or van Beek appliance, have been used particularly in Europe. This approach involves high force levels of up to 1 kg directed through the centre of resistance of the maxillary structures, which has been estimated to be apical to the premolars for the maxillary dentition or at the postero-superior region of the zygomaticomaxillary suture for the maxilla.[58, 59] The impact of these appliances in terms of control of vertical growth, however, remains largely unclear, although short-term benefits have been highlighted in non-randomized studies.[60]

Summary

Functional appliances used in the correction of Class II malocclusion have been employed successfully for more than a century. What they have in common is that they all utilize a forward posture of the mandible to transmit forces from the muscles and soft tissues attached to the mandible to produce a more normal occlusion. Developments in appliance design have resulted in a reduced appliance bulk or the ability to fix the appliance to the dentition to allow better patient compliance and more prolonged periods of wear.

Animal experiments point to histological changes that are apparently stable, leading to the development of increased mandibular length. In clinical treatment, however, the same degree of change cannot be expected due to a more gradual rate of biological change and the overall extended duration of human skeletal development. While the emphasis on skeletal effects persists among researchers focusing on both animal models and clinical treatment, it appears increasingly likely that the changes resulting from functional appliance therapy are predominantly dento-alveolar in nature. Nevertheless, important short-term changes in condylar growth manifesting as an increase in mandibular length are likely to result in an improvement in the skeletal II deformity, assuming that the rate of mandibular growth outstrips that of the maxilla. An increase in the lower anterior facial height is also a consistent finding with functional appliance therapy.

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