CHAPTER 1
Introduction

An attractive smile with a good display of teeth is important for psychological well-being. There are a number of goals that should be aimed for when considering dental attractiveness, such as the symmetry, alignment, smile line, dental arch shape and gingival contour, as well as the quality and morphology of the dental tissue itself. Orthodontic management of the developing dentition is important to ensure that the established dentition will be in the most aesthetic position. Orthodontics requires a good understanding of facial and dental growth and the effects of occlusal guidance.

In the past it was common practice for patients to be referred to an orthodontist once their secondary dentition was established. This practice allowed many developing problems to significantly worsen and ultimately be more difficult to correct. In many cases, developmental problems can be managed as the dentition develops and early intervention can eliminate some of the complex occlusal problems that can take 24 months or longer to manage with orthodontic appliances.

The premise of early interceptive treatment is to allow the secondary dentition to establish itself in an aesthetic position with the dental units lying within the dental arches. Issues such as skeletal discrepancy are managed within the scope of the genetic potential of each patient and early intervention should be decided on not only with reference to the severity of the discrepancy, but also the aesthetic and emotional needs of the patient and the implications of not undertaking treatment. For these reasons, general dental practitioners (GDPs) must consider early referral to a specialist orthodontist for all patients to ensure that all opportunities for occlusal guidance are taken. Referral at the age of 7 years allows any developing issues to be addressed in a strategic and planned manner.

What do we know about growth?

While a patient is still growing, there exists the possibility of addressing orthodontic problems with orthodontic appliances to alter facial growth. However, the problem in clinical orthodontics is that facial growth continues from birth to early adulthood and the growth pattern cannot be accurately predicted. We do know the average rate and direction of growth, but are aware of different skeletal relationships in three planes of space and of growth rotations that lead to differences in facial form from Class II to Class III, high and low angle and transverse discrepancies. We also have some understanding of the role of the facial

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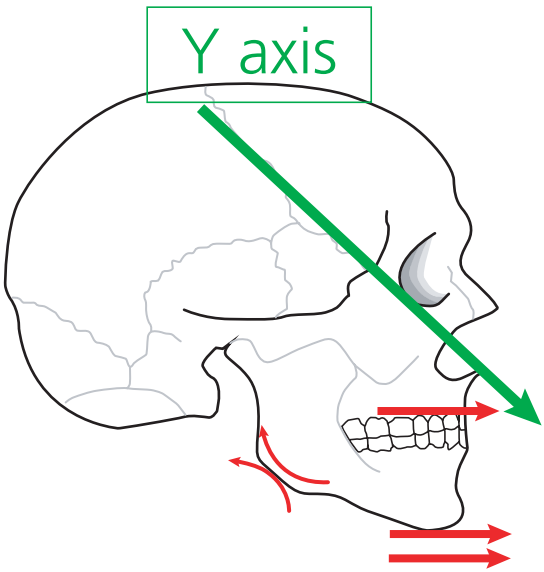

Figure 1.1 Directions of facial growth.

muscles and the influence environmental factors have on the dentition (Figure 1.1). However, we cannot reliably predict the timing of growth or the ultimate amount of growth for any individual until it is almost at an end, even if analytical techniques such as cervical spine and hand/wrist radiograph assessments are employed. In addition, while the soft tissue balance between the tongue, lips and cheeks and how this affects tooth position and dental arch shape can be predicted, we cannot quantify the latter over time or predict accurately the influences that these may have on the dental arch.

Growth and development of the jaws

Growth generally refers to an increase in size, number or complexity by natural development. Development is an increase in the degree of organisation (Proffit, 1993). Craniofacial growth may be divided into four components (Thilander, 1995):

1. growth mechanisms (how new bone is formed);
2. growth pattern (change in size and shape of the bone);
3. growth rate (speed at which the bone is formed);
4. regulating mechanism that initiates and directs the above three factors.

Growth mechanisms

Growth and development of the craniofacial skeleton occurs in two ways:

1. endochondral ossification: growth and ossification of a cartilage model;
2. intramembranous ossification: transformation of mesenchymal connective tissue and deposition of bone on existing bone surfaces.

The bones of the skull base mainly grow and develop by endochondral ossification, and the vault of the cranium and facial skeleton mainly by intramembranous ossification (Enlow, 1990).

Although several ossified areas fuse into large morphological units, remnants of the chondrocranium persist as synchondroses (cartilaginous joints) between the bones in the cranial base. When intramembranously formed bones meet, sutures develop. Bone growth and adaptation can thus proceed due to the separation of bones in the synchondroses and suture areas.

Growth of the cranial base

Displacement growth of the cranial base is made possible mainly by synchondroses. The spheno-occipital synchondrosis is regarded as the most important growth centre for the cranial base (Sicher, 1952; Björk, 1955). The upper facial skeleton is attached to the anterior cranial base, whereas the mandible is connected to the middle cranial base; thus, the length and growth of the cranial base has an important effect on the jaw relationships (Björk, 1955).

Growth of the mid-face

Scott (1953) suggested that the essential primary elements directing craniofacial skeletal growth are the cartilages, in particular the anterior extension of the chondrocranium, the nasal septal cartilage. The anteroposterior expansive growth of the nasal septal cartilage, which is buttressed against the cranial base posteriorly, is thought to 'push' the mid-face downward and forward and as such, the nasal septum is thought to play an important part in the prenatal and very early postnatal growth of the mid-face. However, opinions differ as to its role in postnatal growth; one view is that growth is secondary to, and compensatory for, passive displacement of the mid-facial bones and the nasal septum plays a significant biomechanical role in maintaining normal mid-facial form (Melsen, 1977; Moss, 1977).

Growth of the mandible

The cartilage of the mandibular condyle is a secondary cartilage and is different in origin and structure from the epiphyseal plate and synchondrosal cartilage (Thilander *et al*., 1976).

The traditional view of the condylar cartilage was that it controlled overall mandibular growth and represented a major growth centre for the entire mandible (Scott, 1962). Koski and Makinen (1963) and Koski and Mason (1964) attempted to grow the condylar cartilages of rats and showed that cartilage only grew when it was explanted with the adjacent bone. Koski and his team interpreted this as confirmation of the views of Moss (1968) that growth of the condylar cartilage is always secondary to forward displacement of the mandible as a result of some outside influence, 'the functional matrix', and only participates in regional adaptive growth. They suggested therefore, that condylar cartilage is not a major growth centre for the mandible, but it does have a great capacity to adapt to mandibular displacement during growth (Koski and Ronning, 1966; Enlow, 1990).

Experimental studies in animals by McNamara *et al*. (1982) have shown that cellular proliferation in the condylar cartilage can be influenced by appliances that displace the jaw forwards. However, the extent to which condylar growth can be modified in amount or direction by external influences is not clear. Displacement growth is made possible by the craniofacial sutures, which have the dual function of permitting growth movement and uniting the bones of the upper facial skeleton (Thilander, 1995). According to the Sutural Theory of Weinmann and Sicher (1947), the intrinsic pattern of expansive proliferative growth in the sutures generates forces that separate the bones and are thus responsible for the displacement of the maxillary complex. When sutures are transplanted to non-functional sites, however, no growth occurs (Ryoppy, 1965). Similarly, it has been demonstrated that growth of the circumaxillary sutures can be reduced or inhibited by the application of force to the maxilla (Elder and Tuenge, 1974). It is now accepted that facial sutures are not centres of active growth, but sites at which adaptive growth can occur in response to environmental demands (Moss and Salentijn, 1969).

All inner and outer bone surfaces of the facial skeleton are associated with a mosaic of functional growth fields. These fields carry out specific, localised growth activities that involve separate areas of resorption and deposition on all periosteal and endosteal surfaces (Enlow, 1990). Growth remodelling by periosteal resorption and

deposition is paced by the growth and functions of the soft tissues in which the bones are embedded (Enlow, 1990). The influence of the periosteum is therefore of greatest significance with respect to the change in size and shape of the bones, i.e. the growth pattern (Enlow and Bang, 1965). The periosteum continues to function as an osteogenic zone throughout life, but its regenerative capacity is extremely high in the young child (Thilander, 1995).

Growth pattern

At birth the face makes up approximately one-eighth of the total volume of the skull, this proportion increasing to one-half in the adult male (Mills, 1983). The change in the size and shape of the bone takes place on the basis of several basic principles: remodelling, cortical drift, relocation, displacement and the 'V' principle (Enlow, 1990). These principles of bone growth will result in the following changes in size and shape of the nasomaxillary complex and the mandible.

Growth of the nasomaxillary complex

The postnatal growth of the maxillary complex occurs by sutural apposition and surface remodelling (Enlow and Bang, 1965).

The predominant direction of growth of the maxillary complex is posteriorly, with displacement occurring in the opposite anterior direction. As the growing maxillary complex is displaced, anteroposterior dimensions increase mainly by bone deposition on the tuberosity (Enlow, 1990). Simultaneously, the zygomatic region is relocated in a posterior direction (Enlow and Bang, 1965).

Vertical growth of the nasomaxillary complex, similar to the horizontal elongation, is brought about by a combination of growth remodelling and displacement associated with growth at the various sutures of the maxilla (Björk and Skieller, 1977). The palate is relocated inferiorly by a combination of periosteal resorption on the nasal side of the palate and periosteal deposition on the oral side. Simultaneously, periosteal resorption occurs on the anterior surface of the maxilla (Enlow and Bang, 1965).

Growth of the mandible

The postnatal growth of the mandible involves periosteal deposition at many sites. Elongation is brought about by deposition at the condyle and the posterior border of the ramus. Growth remodelling serves to maintain the shape and proportions of the bone as it increases in size (Enlow and Harris, 1964).

Displacement of the mandible is associated with a rotation dependent on the direction of condylar growth. Anterior rotation will take place in individuals with an upward and forward direction of condylar growth, whereas in those with a predominantly backward direction of condylar growth, the mandible will rotate in a posterior direction (Björk and Skieller, 1983).

The rate and direction of condylar growth is a secondary adaptive response to intrinsic and extrinsic biomechanical forces (Enlow, 1990). However, it is debateable whether it can be permanently changed by external forces acting only for a short time.

Growth rate

Craniofacial growth and development is a complex process that is not merely an increase in size; rather it is a differential growth process in which the various structures grow at different rates from birth to maturity (Enlow, 1966). Cross-sectional studies have identified differences in the

growth curves of lymphoid, genital, neural and somatic tissues (Scammon, 1930). Growth of the calvarium follows the neural growth curve and reaches a plateau after 6 years of age. Growth of the facial skeleton follows the somatic growth curve and thus still has growth potential after the age of 6 years.

Although somatic tissues increase in size throughout the growth period, their growth rate is characterised by a decrease from birth with a minor mid-growth spurt at approximately 6–8 years of age, a prepubertal minimum, and a pubertal growth spurt (Brown *et al.*, 1970). The sequence of growth events is predictable, but their timing is quite variable among individuals. The developmental status of a child can be assessed by peak growth velocity in standing height, dental development, skeletal ossification and secondary sex characteristics (Moore *et al.*, 1990). Skeletal age, estimated from hand and wrist radiographs, has proved to be useful in helping to predict adult stature, but does not reliably predict the growth spurt (Houston, 1980). Menarche in girls and voice changes in boys occur soon after the peak of the growth spurt and so these features can be used to indicate whether or not the peak has passed (Hagg and Taranger, 1980). However, these assessments often can only report that growth has peaked and cannot be used to predict when it will peak if this has not occurred.

Growth in stature follows the somatic growth curve and has been investigated more thoroughly than has growth in facial dimensions, but it is a poor indicator of jaw growth (Houston *et al.*, 1997). The peak height velocity (pubertal growth spurt) was found by Tanner *et al.* (1976) to occur on average at 12 years of age in girls and at 14 years in boys, although they found considerable individual variation with a standard deviation of nearly 1 year.

Growth regulating mechanism

The control of a complex morphogenesis requires a precise biological regulatory mechanism for initiating and directing the growth mechanisms, growth pattern and growth rate (Thilander, 1995).

Sicher (1952) postulated that craniofacial growth as a whole was determined by the innate genetic information in the skeletal tissues; Scott (1962) postulated the heredity and expansive growth of the osteogenic tissues to the chondral structures; and Moss and Salentijn (1969) described the functional matrix theory, which refers to all the soft tissues and spaces that perform a given function such as mastication, swallowing and respiration, as being the stimulus for craniofacial skeletal growth.

Petrovic *et al.* (1990) carried out experimental work that led to the development of a cybernetic model of growth regulation, which states that it is the interaction of a series of causal changes and feedback mechanisms that determines the growth of the various parts of the craniofacial complex.

Craniofacial morphogenesis is now considered to be multifactorial, with facial development being influenced by several genes together with various environmental factors (Thilander, 1995). Van Limborgh (1982) has divided the factors controlling skeletal morphogenesis into five groups; namely, intrinsic genetic factors, local and general epigenetic factors, and local and general environmental influences. Essentially, growth is mainly under the influence of genetic factors rather than environmental ones (Figure 1.2).

Figure 1.2 Growth is genetically controlled and largely not under our influence.

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