Growth and Development

Timing

The third and most important factor of growth in terms of clinical applications is the timing of growth. The biological clock of growth is set differently for different individuals. A particular growth event may occur at different times in different individuals. Some children mature early. They are taller in childhood than average growers since they have matured faster than average, but they may not be particularly tall in adulthood. Late-maturing children are shorter than average in childhood since they mature late. They will eventually gain normal stature in adulthood.
One important factor in the timing of growth is the sex of the individual. Girls attain puberty earlier than boys. The timing of menarche (attainment of sexual maturity) in girls also shows a great variation. Some girls mature faster than others. Timing of growth is an important consideration when growth modification procedures are considered in the treatment plan

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GROWTH RHYTHM AND GROWTH SPURTS

Growth charts reveal that human growth does not proceed at a uniform rate throughout life. There appears to be a rhythm in growth process. The growth rhythm can be readily appreciated in gaining of the body height. There are periods of sudden accelerated growth interspersed with periods of relative quiescence. Such rapid increase in growth rate is termed as a “growth spurt”. Three major growth spurts can be seen during postnatal development. The timing of growth spurts differ in boys and girls. Generally, girls precede boys in growth spurts by approximately two years.

1. Infantile/childhood growth spurt: Up to 3 years in both sexes
2. Mixed dentition/juvenile growth spurt: 7–9 years in females; 8–11 years in males
3. Adolescent growth spurt: 10–12 years in females; 12– 14 years in males

Infantile Growth Spurt

Body length increases from a neonatal range of 48–53 cm to about 75 cm during first year after birth and increases by 12–13 cm in the second year. Thereafter, 5–6 cm is added each year.

Childhood Growth Spurt

The growth spurt at this period is less pronounced than the other two growth spurts.

Adolescent Growth Spurt

In longitudinal growth curves, an increase in the velocity of growth occurs between 10–12 years in girls and 12–14 years in boys. This rapid increase in growth is termed as the "adolescent growth spurt" (Fig. 1). Accentuated growth associated with the pubertal period is more dramatic and is thought to be caused by the physiological alteration in hormonal secretion. Adolescent growth spurt occurs earlier in girls than in boys and it lasts for 2–2.5 years in both sexes. Rapid raise in growth during adolescence is most obvious in the increase in height, while weight gain is more variable.

Girls gain around 16 cm in height during the spurt with a peak velocity at 12 years of age. Boys gain about 20 cm in height with a peak velocity at 14 years of age. The growth rate may reach a 10 cm increase in height per year. After peak height velocity (PHV) is attained, the growth rate declines. Noticeable growth is believed to stop at 18 years in females and 20 years in males.

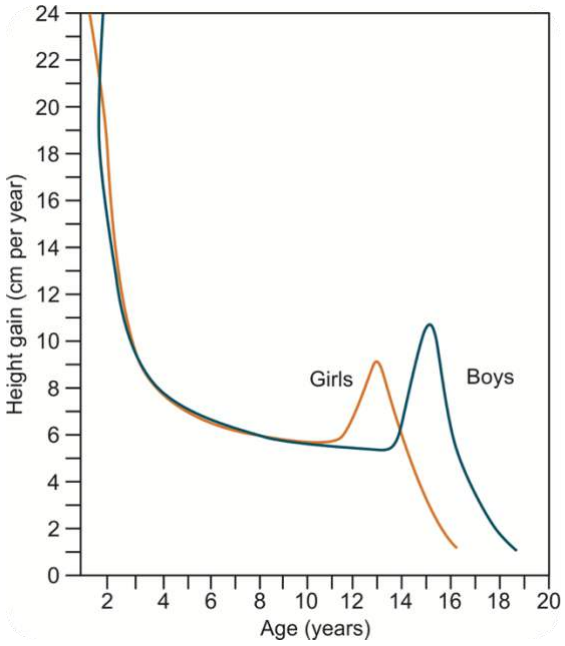


Figure 1 Adolescent growth spurt: Accentuated growth associated with pubertal period occurs earlier in girls than in boys
and it lasts for 2–2.5 years in both the sexes

Clinical Significance of Growth Spurts

- Adolescent growth spurt has significant clinical implications in orthodontics.
- Differences between the timing of growth spurt in males and females has to be kept in mind.
- Treatment of skeletal malocclusions by growth modification using orthopedic and functional appliances is best carried out during adolescent growth spurt, e.g. chin cup appliance to restrict mandibular advancement in treatment of class III malocclusion; Frankel functional regulator to stimulate mandibular growth in the treatment of class II malocclusion. It is advisable to continue appliance wearing until the cessation of adolescent growth spurt so as to obtain stable results.
- Expansion of dental arches (e.g. rapid maxillary expansion) responds well during adolescent growth spurt and the results are more likely to be stable.
- Certain patients with gross skeletal discrepancies may require orthognathic surgical correction and surgery is best carried out after the cessation of active growth.

Growth Fields

Bone growth is controlled by so-called "growth fields". Periosteal (outer) and endosteal (inner) surfaces of bones are blanketed by soft tissues and cartilage or osteogenic membrane. With this blanket of soft tissue matrix, the growth fields are disturbed in a characteristic mosaic-like pattern across the surface of a given bone.

Growth fields have either depository or resorptive activity. If the periosteal growth field is resorptive, the opposing endosteal field is depository, and vice versa. Depository and resorptive fields are disturbed characteristically across the entire inner and outer surfaces of the craniofacial skeleton. However, the activity of the growth field is not located in the bone itself. The genetic information resides within soft tissues.

Growth Centers

The term "growth centers" is used to describe very active growth fields, which are significant to the growth processes/mechanisms.

Examples

- Cranial and facial sutures
- Synchondroses of cranial base
- Mandibular condyles
- Maxillary tuberosity

- Alveolar processes

The growth center controls the overall growth of the bone. Genetically controlled growth takes place at growth centers, i.e. they have intrinsic growth potential. Growth centers respond to functional needs; however, they show little response to external influences.

Growth Sites

Growth sites are certain areas of a bone where significant growth of that bone takes place. Growth sites show a marked response to external influences. Unlike centers, growth sites do not control the overall growth of the bone. They do not cause the growth of the whole bone instead, they are simply areas of the bone where exaggerated growth takes place, e.g. certain areas of mandibular condyle and maxillary tuberosity. Growth sites can occur at growth centers, but all growth sites are not growth centers.

MODES OF BONE FORMATION

Bones develop by either of two mechanisms, namely endochondral ossification or intramembranous ossification. Bones developed from endochondral ossification are referred to as cartilaginous bones while bones formed by intra- membranous ossification are called membranous bones.

Endochondral Ossification/Indirect Ossification

In endochondral ossification, a precursor cartilage model (template) is first formed and is then replaced by bone
in an ordered sequence. Endochondral bone formation proceeds as follows:

a. The cartilage model which forms during embryo- genesis (primary cartilage) acts as a miniature template for the development of cartilage bone.
b. The cartilage model becomes surrounded by a highly vascular mesenchyme: the perichondrium. The perichondrium contains osteoprogenitor cells in its deeper layer.
c. Centers of calcification appear within the cartilage primordium matrix. As calcification of cartilage matrix surrounding the cartilage cells proceeds, nutrition to the cartilage cells is cut off resulting in their death. Destruction of cartilage cells lead to formation of empty spaces within the calcifying cartilage matrix.

d. Blood vessels from surrounding perichondrium invade into the calcifying matrix, bringing undifferentiated connective tissue cells.

e. Osteoblasts get differentiated from the undifferentiated connective tissue precursor cells, and deposit (fibrous bone matrix osteoid) on the remnants of calcified cartilage matrix.

f. Osteoid tissue then gets mineralized to form bone.

g. In the craniofacial skeleton, the bones of cranial base and portions of the calvarium are derived from endochondral ossification.

Intramembranous Ossification/Direct Ossification

Intramembranous ossification is the direct formation of bone within highly vascular sheets of membranes of condensed primitive mesenchyme. There is no precursor cartilage model here. At centers of ossification, ectomesenchymal stem cells differentiate into osteoblasts. The newly formed osteoblasts proliferate around the branches and capillary network and produce a fibrous bone matrix (osteoid). As osteoid deposition continues, osteoblast cells get encased in matrix and become osteocytes. Blood vessels are retained within the spaces and become surrounded by bone. A haversian system starts to form which nourishes the bone. Fibrous bone matrix (osteoid) eventually becomes mineralized. Entrapped blood vessels supply nutrients to osteocytes and bone tissue.

Peripheral mesenchyme condenses on the outer surface to form fibrocellular periosteum. Further bone deposition occurs from new osteoblasts differentiating from osteoprogenitor cells in the deeper layers of the periosteum. Membrane bones in the craniofacial skeleton are cranial bones, mandible, etc. (most of the bones of the craniofacial skeleton are of intramembranous origin). However, in certain membranous bones, secondary cartilages (not part of the cartilaginous primordium of the embryo) develop and after initiation of intramembranous ossification contribute significantly to their growth, e.g. condylar cartilage in the mandible.

Mechanisms of Bone Growth

After osteogenesis, further growth of bone takes place by several mechanisms. Bones do not grow by simple symmetrical enlargement. Growth of craniofacial bones is complex involving differential growth mechanisms and different types of development for the individual bones. Some of the important growth mechanisms include:

- Bone remodeling
- Cortical drift
- Displacement/translation.

Bone Remodeling

- As said earlier, bone does not grow uniformly in all directions. Selective bone resorption and deposition occurs which is called remodeling.
- Remodeling is the basic growth process providing regional changes in the shape, dimension, and proportions of the bone.
- Along with an increase in size, remodeling facilitates constant reshaping of bone while maintaining the integrity and basic shapes of the bone.
- Remodeling also provides regional adjustments in the bone needed for adapting to the changes in function.

Cortical Drift

Cortical drift and displacement provide means of growth movement in craniofacial development. The cortical plate can be relocated by a simultaneous apposition and resorption process occurring on the opposing periosteal and endosteal surfaces. Such a combination of simultaneous deposition and resorption resulting in a growth movement towards the depositing surface has been described as cortical drift by Enlow (1963).

The bony cortical plate drifts by depositing and resorbing the bone substance on the outer and inner surface respectively in the direction of growth.

If the resorption and deposition take place at the same rate, the thickness of the bone remains constant with only change in the location of the cortical bone. On the other hand, if bone deposition is more than resorption, the thickness of the cortical bone increases along with its movement. For example, the teeth follow the drift of their alveolar bone while the jaw is growing and thus they maintain their position within the surrounding bony structures.

Displacement/Translation

Change in the spatial position of a bone can occur by two types of displacement.

1. Primary displacement occurs where actual enlargement of the bone will change its position in space. In other words, primary displacement of the bone is brought about by its own enlargement. Primary displacement of maxilla in a forward direction occurs due to growth by maxillary tuberosity in a posterior direction (Fig2). The amount of anterior displacement is equal surface of the tuberosity receives continuous deposits of new bone and results in horizontal lengthening of the maxillary arch.

2. Secondary displacement, on the other hand, occurs when the growth of one bone results in a change in the spatial position of an adjacent bone. The nasomaxillary complex grows by secondary translation during the primary dentition period. As the maxilla is attached to the cranial base, growth occurring at the cranial base produces a passive/secondary displacement of the nasomaxillary complex in a downward and forward direction (Fig. 2).

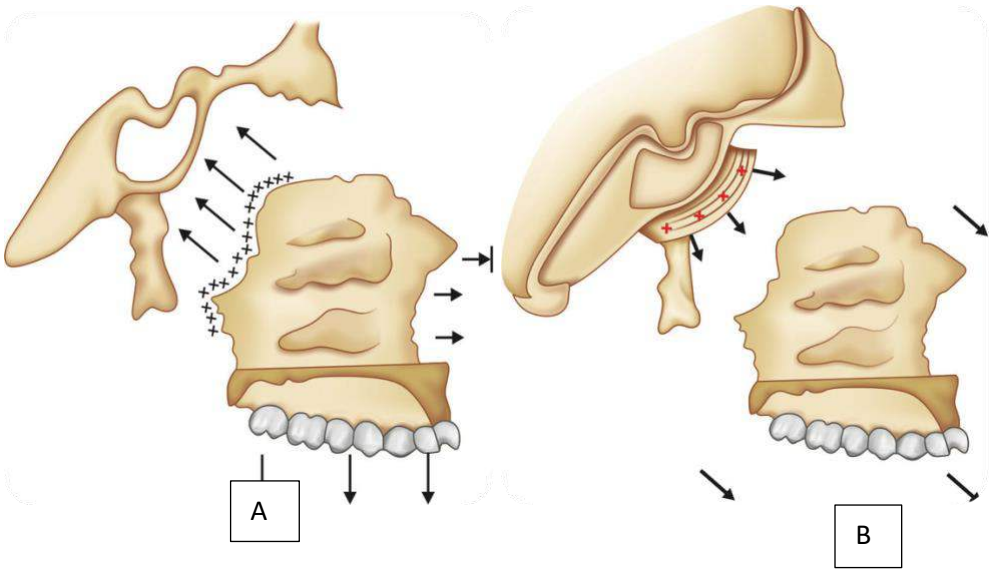


Figure 2 shows A: Primary displacement of the maxilla in a forward direction occurs due to growth by maxillary tuberosity in a posterior direction.

B: Secondary displacement of bone

THEORIES OF CRANIOFACIAL DEVELOPMENT

A number of theories have evolved over the years in an attempt to understand the complex nature of craniofacial development. The major theories of growth are described below.

Genetic theory by Brodie
Sutural dominance theory by Sicher
Cartilaginous theory by Scott
Functional matrix theory by Melvin Moss
Servosystem theory by Petrovic

Other theories related to craniofacial growth are:

Von Limborgh's compromise theory
Enlow's expanding 'V' principle
Enlow's counterpart principle/growth equivalent
concept Neurotrophism

Functional matrix theory by Melvin Moss

Moss noted that the head is a composite structure, operationally consisting of a number of vital functions such as olfaction, respiration, vision, digestion, speech, audition and neural integration. Each function is carried out by a group of soft tissues which are supported and/or protected by related skeletal elements. Taken together, the soft tissues and the skeletal elements related to a single function are termed as "functional cranial component."

A functional cranial component performing a function consists of two components:
1. Skeletal unit: All skeletal elements associated with a single function.
2. Functional matrix: All soft tissues associated with a single function.

Skeletal unit: The totality of all the skeletal elements associated with a single function comprises a skeletal unit. The skeletal unit may be made of bone, cartilage and tendons. The skeletal unit in turn consists of micro and macroskeletal units.

- Microskeletal unit: The term "microskeletal unit" is used when a bone is comprised of several contiguous skeletal units. For example, maxilla and mandible are comprised of a number of microskeletal units. The mandible is made of alveolar, condylar, angular, gonial, coronoid and basal microskeletal units while the maxilla is comprised of orbital, palatal, pneumatic, alveolar and basal skeletal units.

- Macroskeletal unit: The term "macroskeletal unit" is used when the adjoining portions of a number of neighboring bones are united to function as a single cranial component. For example, the entire cranial vault is a macroskeletal unit protecting the brain and the whole mandible is a macroskeletal unit.

Functional matrix: The totality of all the soft tissues associated with a single function is termed as "functional matrix".
The functional matrix has two components:

- Capsular matrix
- Periosteal matrix

Capsular matrix: The capsular matrices bring about spatial changes in the position of bone, i.e. translation by acting indirectly and passively on their skeletal units. These changes in the spatial position of skeletal units are brought about by the expansion of their enveloping capsules. Translative growth is not caused by apposition and resorption of bone.
Examples of capsular matrices are:

- Neurocranial capsule (skin and dura mater) which is controlled by the growing brain thrusting the bony calvarial plates .
- Orofacial capsules (comprising of skin and oral mucosa) within which the facial bone arise, grow and are maintained.

Periosteal matrix: The periosteal matrices act directly and actively on their related skeletal units, causing more local changes in the size and shape of the skeleton, i.e.

"transformation/remodeling". The periosteal matrices bring about transformation by acting directly upon skeletal units via the periosteum and resulting in bone apposition and resorption. Examples of the periosteal matrices include the muscles, tendon, nerves, blood vessels, glands, etc.

Summarizing the functional matrix theory, craniofacial growth is the result of both changes in the "capsular matrices", causing spatial changes in the position of bones (translation) and by "periosteal matrices", causing more local changes in the size and shape of the bones (transformation/remodeling).

Clinical Applications of Functional Matrix Theory

Application of force by orthodontic appliances tends to alter the functional matrix. Alteration of periosteal matrix (teeth) produces changes in microskeletal unit (alveolar bone); alteration of capsular matrix (dentofacial orthopedics) produces changes in macroskeletal unit (jaws).

- Rapid palatal expansion by widening of palatal sutures is a form of orofacial orthopedics.
- Repositioning of maxillary segments in cleft patients alter the macroskeletal unit.
- Anterior bite plane used in treatment of deep bite stimulates supraeruption of posterior teeth.
- Activator stimulates the growth of condyle.
- Frankle's functional regulator stimulates both the periostealnmatrix through lip pads and buccal shields; capsular matrix by altering oropharyngeal spaces.
- Inter-arch elastics, head gears, facemask, chincup have direct effect on functional matrices by alteration of muscular behavior and spaces.

Expanding 'V' Principle by Enlow

The concept of expanding 'V' principle was put forward by Enlow. The 'V' principle is an important facial skeletal growth mechanism since many facial and cranial bones have a 'V' configuration or 'V' shaped regions.

In "V' shaped bones/areas, bone resorption occurs on the outer surface of the 'V' and deposition on the inner surface. As the remodeling continues, the 'V' moves away from its tip and enlarges simultaneously. In this way growth as well as movement of the bone occurs simultaneously.

Such an increase in size and the simultaneous movement of the bone in the shape of 'V' is called the expanding 'V' principle (Fig. 3).

Such a growth process results in:

1. Enlargement in the overall size of the 'V' shaped area.
2. Movement of the entire 'V' structure towards its own wider end.
3. Continuous relocation.

Most of the craniofacial bones including the mandible, maxilla, and palate grow on an expanding 'V'. Growth of the palate is one of the best examples of expanding the 'V' principle. Deposition occurs on the palatal periosteal surface and resorption occurs on the side of the nasal floor. In this way, the palate expands in a lateral direction and also moves downwards (Fig. 3 B).

It is easier to visualize the mandible as a 'V' shaped bone than the maxilla because of its horseshoe shape. The Ramus of the mandible grows on an expanding 'V' and the interramal width of the mandible also increases by expanding the 'V' principle (Fig. 3 C). The condyle remodels according to the expanding 'V' principle and the neck of the condyle gets lengthened.

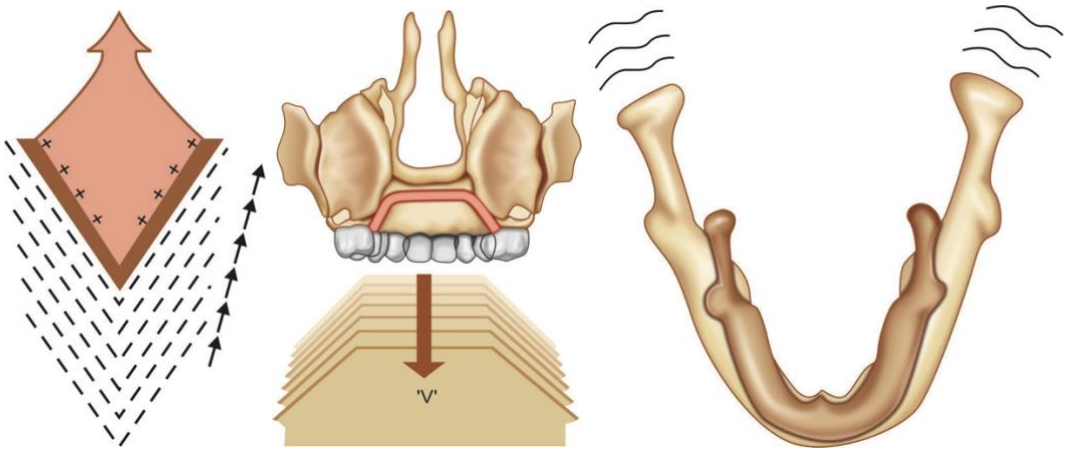


Figure 3 **A** Expanding 'V' principle: In "V'-shaped bones/areas, bone resorption occurs on the outer surface of the 'V', and deposition on the inner surface. As the remodeling continues the 'V' moves away from its tip and enlarges simultaneously—giving rise to simultaneous growth as well as movement of the bone; **B:** Growth of the palate is a good example of expanding 'V' principle. Deposition occurs on the palatal periosteal surface and resorption occurs on the side of the nasal floor causing downward and lateral expansion of the palate; **C:** Mandibular growth follows the expanding 'V' principle