Craniofacial growth, development and deformities

Lectures for PhD 1st semester

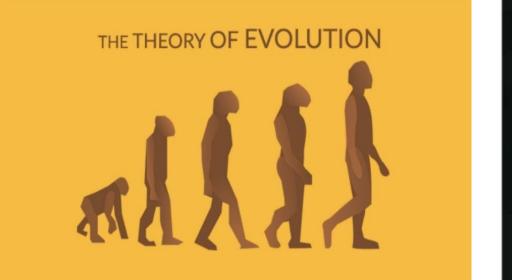
Assist. Prof. Dr. Mehdi Alrubayee BDS 1992

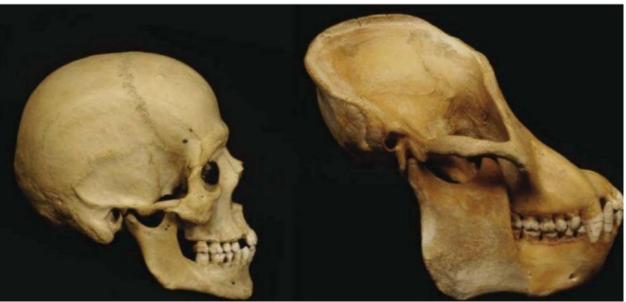
MSc Oral Surgery 2000-2003 University of Baghdad MSc Orthodontics 2005-2007 University of Baghdad

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Prenatal and Postnatal development of craniofacial region The difference of craniofacial region of human and apes

- 1- erect posture
- 2- expanded brain size with higher intelligence rate and languages





GROWTH vs DEVELOPEMENT

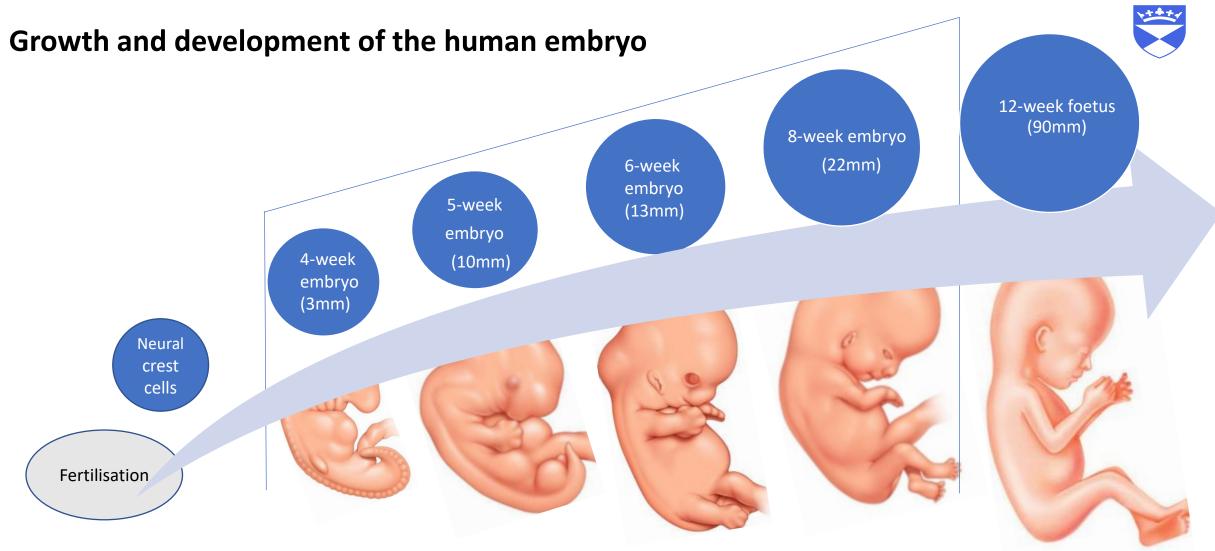
Moss defined the growth as a gradual INCREASE in age, size, weight or height i.e. change in the measurable parameters.

However, there are other definitions:

- The self multiplication of living substance JX Huxely.
- Increase in size, change in proportion & progressive complexity.- Krogman
- Entire series of sequential anatomic & physiological changes taking place from the beginning of prenatal life to senility
 Meredith.
- -Quantitative aspect of biologic development per unit of time-Mayers

Development is the progressive changes in size, shape, complexity and function under genetic potentials (genotype) into functioning mature systems (phenotype)

or it is the progress in maturity



The most critical period of the craniofacial development spans the 4th to 8th week of i.u. life and controlled by NCCs which are sensitive to genetic and environmental teratogenic factors.

Prenatal development of craniofacial region

1- embryonic period, from fertilization through the 8th week

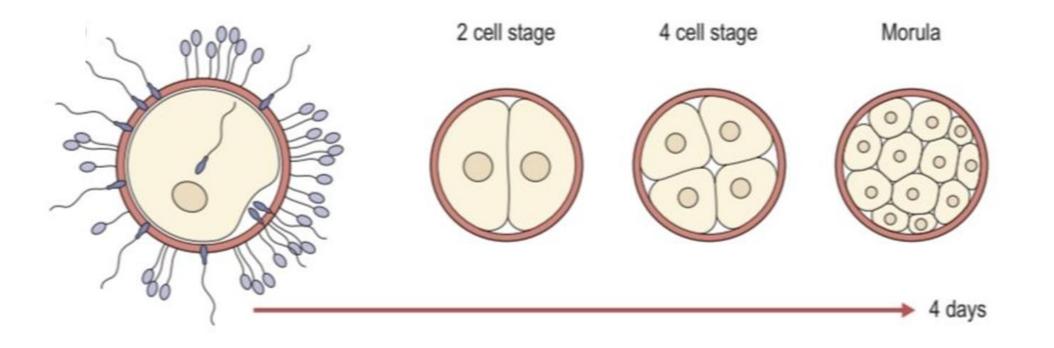
2- fetal period, lasting from week 9 to 39

"Weeks 4 - 8 are the most critical because of organogenesis from the original 3 germ layers"

Bishara, 2001

Fertilization

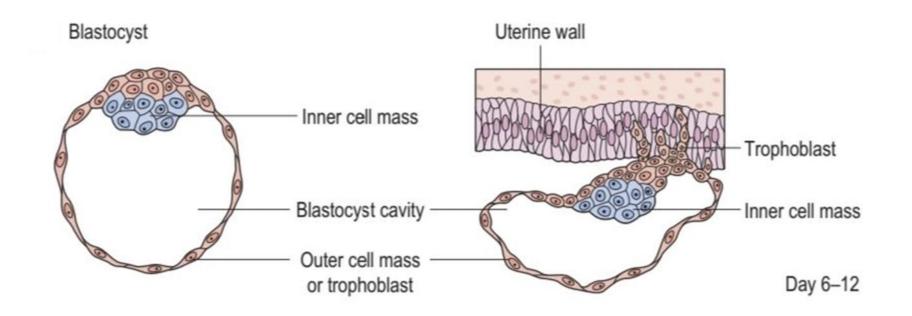
Sperm + ovum = zygot - series of mitotic divisions give rise to 16 cell morula



Implantation in the uterine wall Blastocyst + Trophoblast + embryoblast

Cells of morula organized in outer (trophoplast or future placenta) and inner cell (the embryo itself) masses forming the early embryo known as blastocyst.

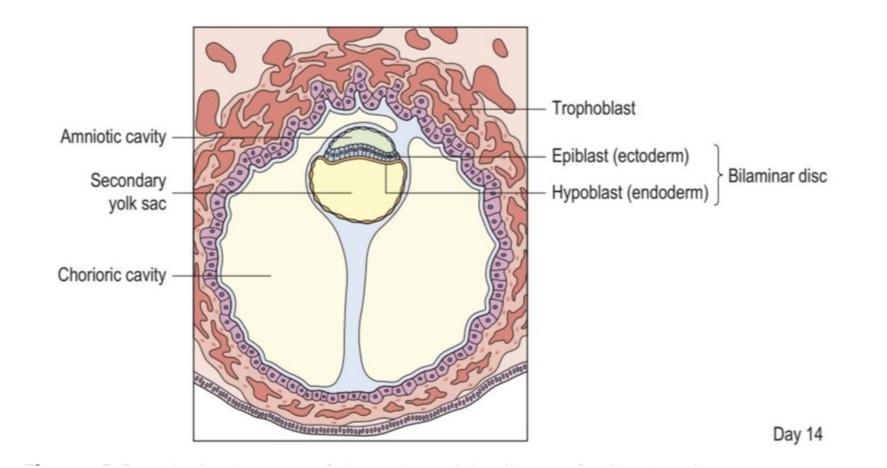
The trophoblast cells adhere and then invade the uterine wall to erode the capillaries to communicate the maternal circulation and provide nutrition for embryo.



Bilaminar embryonic disk

The inner cell mass will differentiate into 2 layers : epiblast (future ectoderm) and hypoblast (future endoderm)

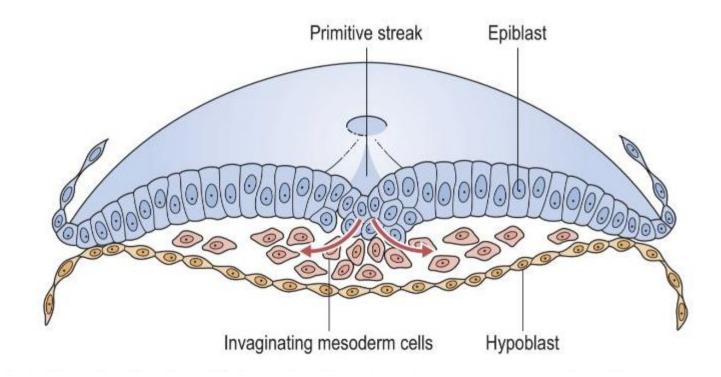
Both together form the bilaminar disc of the embryo

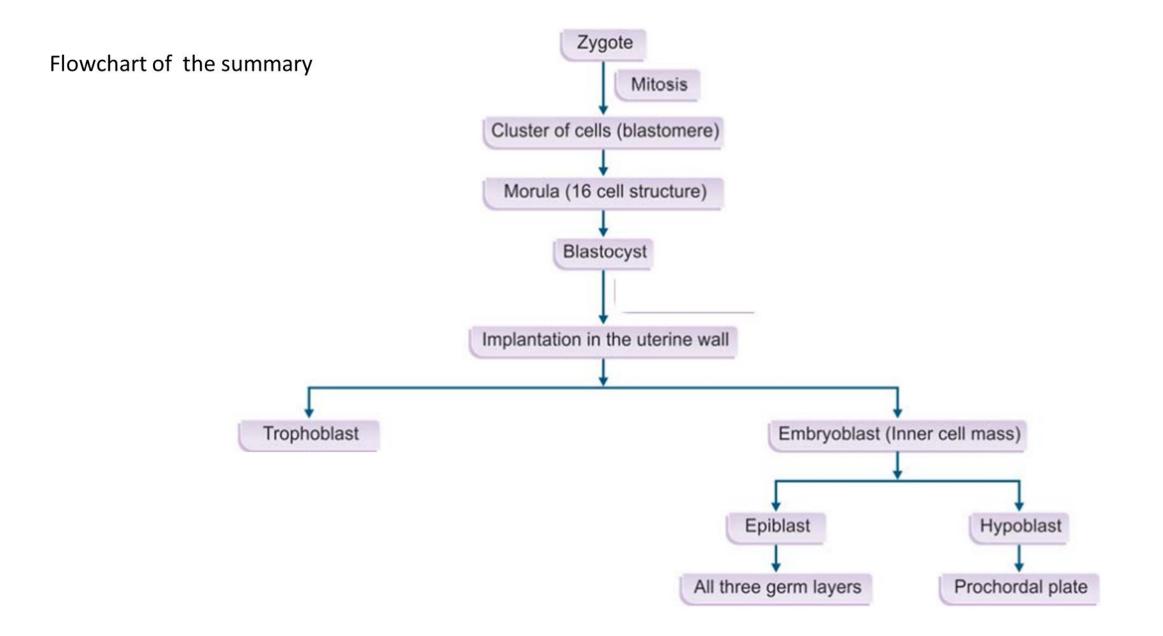


Gastrulation

after 21st day the 3rd germ layer or mesoderm is formed

Cells from the epiblast migrate through the primitive streak which is a raised structure on the surface of epiblast and invaginate themselves as a mesodermal cell layer between the epiblast and hypoblast

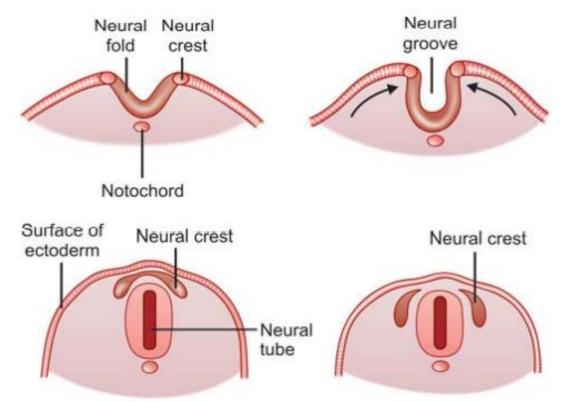




Neurulation

Notocord: a major regulator that will develop into vertebral column, induces the development of the neural tube by folding of ectoderm to form the neural fold.

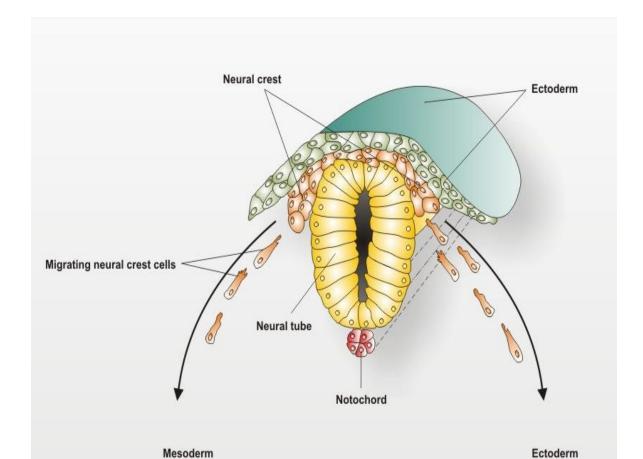
Neural folds deepen to form a groove and then edges of the groove fuse in midline to form the neural tube which represent the future central nervous system.



Neural crest cells

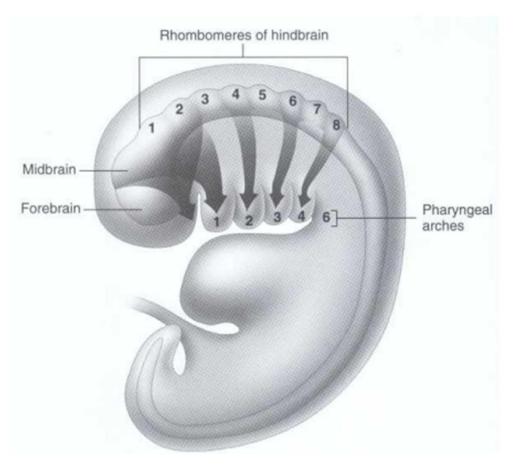
The cells from the crest of the groove form the neural crest cells that proliferate and undergo extensive migration

NCCs are multipotent progenitor cell derived as a strand of cells on the dorsal neural tube, have the capability to differentiate into a vast number of derivatives (<u>Bronner, 2012</u>)



Where Neural crest cells migrate?

- The cephalic region of the neural tube enlarges to form the forebrain, midbrain, and hindbrain.
- Eight bulges called rhombomeres develop in the hindbrain
- NCC from the midbrain and the eight rhombomeres of the hindbrain migrate ventrally to the face and pharyngeal arches where they provide embryonic connective tissue.
- Neural crest from the midbrain and first two rhombomeres contribute specifically to the development of the face and first pharyngeal arch structures



Theories of NCC migration

There are two hypotheses Extrinsic hypothesis suggests the cells interact with the extracellular matrix and adjacent epithelia to determine the final developing tissues (Bronner, 2012)

Or

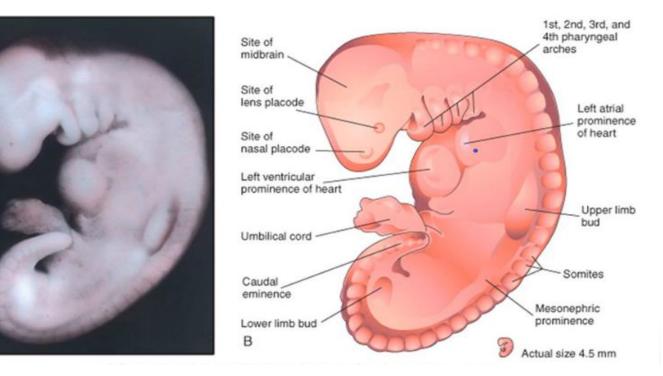
Intrinsic determinants, suggests that NCCs are intrinsically programmed for different developmental cells, (Kuo and Erickson, 2010).

Somite Period/ organogenesis

This is the period of organogenesis from 21st to 31st day post-conception.

There is a complete separation between all three germ layers

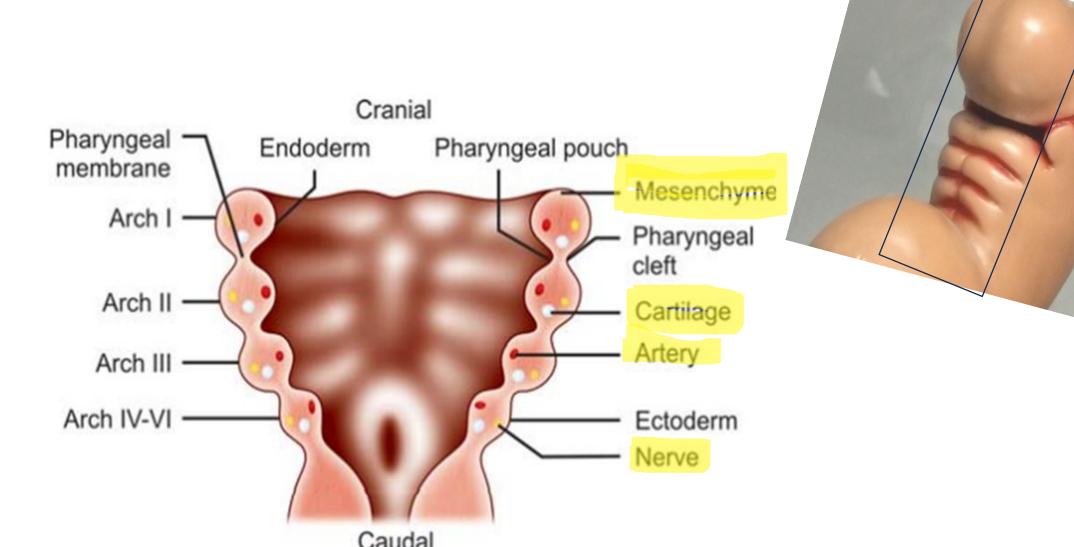
Human embryo 28 days with somites

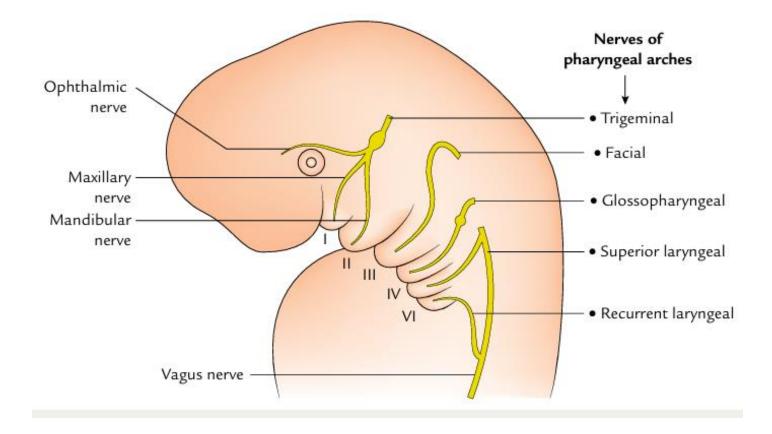


Branchial Arches

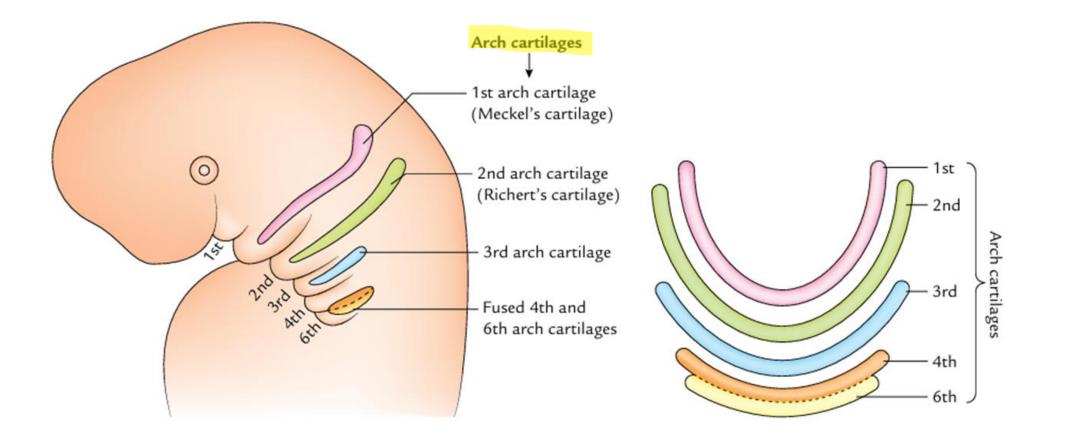
At the 4th week i.u. life, elevations are seen in the ventral foregut resulting in the formation of six pharyngeal arches or branchial arches bilaterally, the fifth arch perishes; finally, only five arches remain

Components of the branchial arch

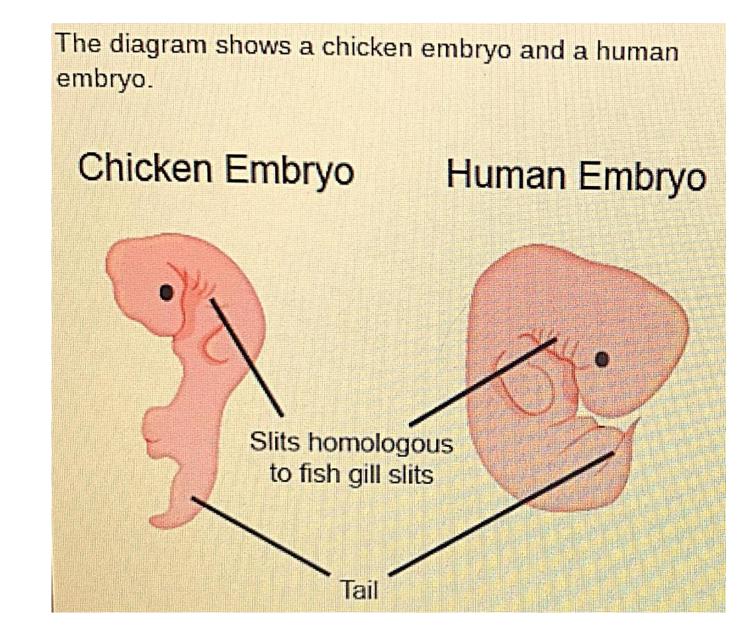


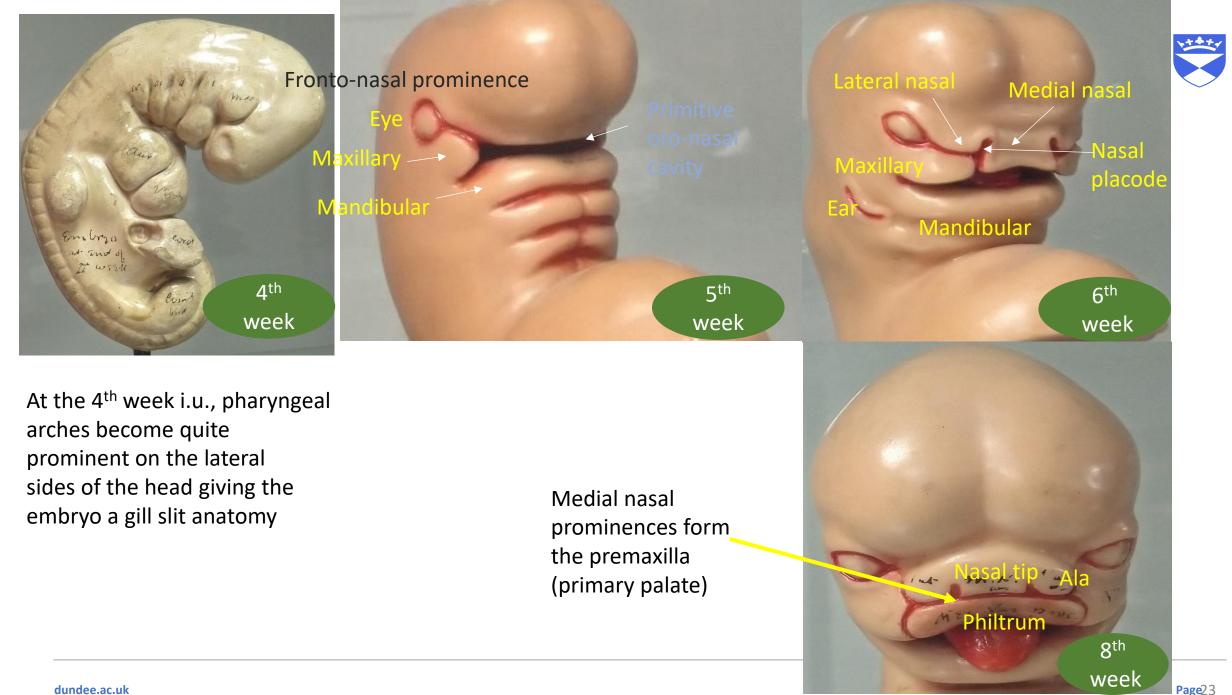


Arch Cartilages



In certain stages human embryo has a gill slit anatomy of a fish/ resembling the chicken embryo





Queen Mary University of London

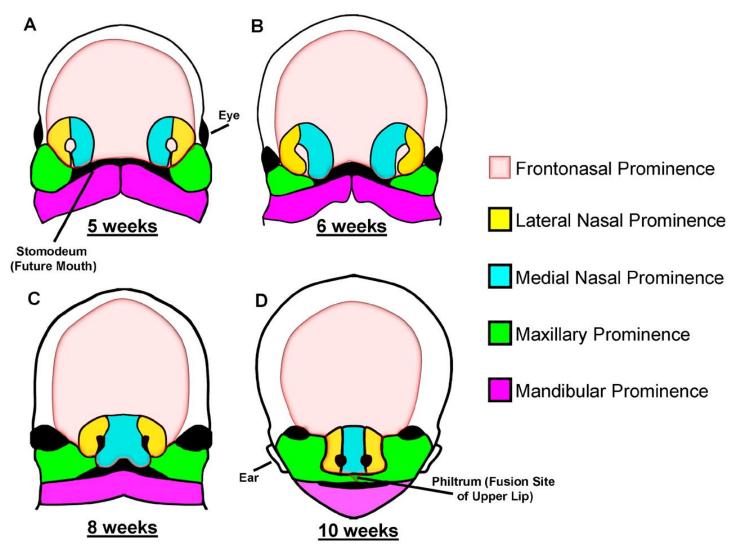
dundee.ac.uk

Development of the face

Key Events in Shaping of the Human Face, Lip and Palate

oropharyngeal membrane:

A junction between primitive oral ectoderm and digestive tube endoderm undergoes a degenerative progressive process of programmed apoptosis by phagocytic and lysosomal activity, uniting the oral cavity and pharyngeal regions of the digestive tube

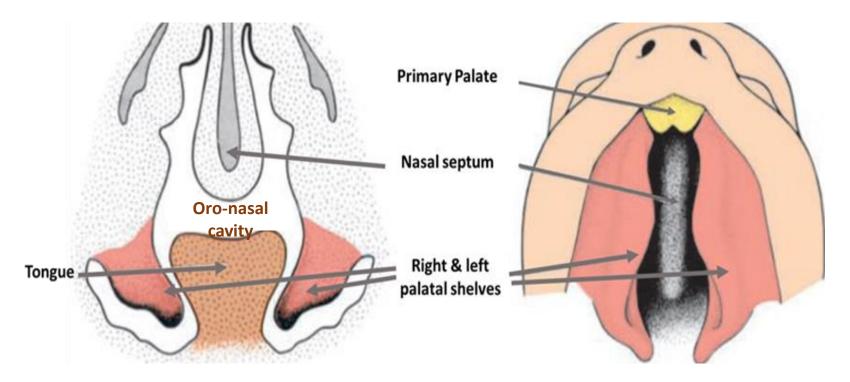


Palatogenesis: Developmental Biology and Morphogenesis of the Palate



The Critical Stages in Development of the Palate

Palatal shelves emerge from the maxillary process inferolaterally on either side of the tongue which fills most of oro-nasal cavity



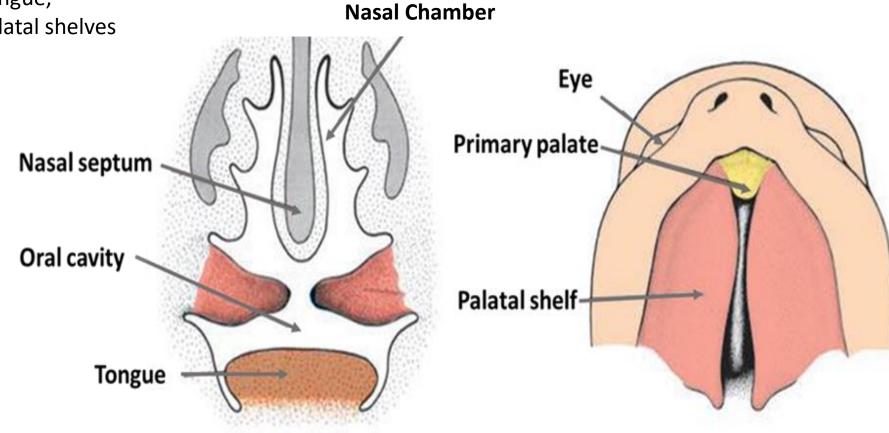
Palatogenesis of a 6.5 –week-old human embryo

Sadler & Langman, 2012

Palatogenesis of 7.5-week-old human embryo.



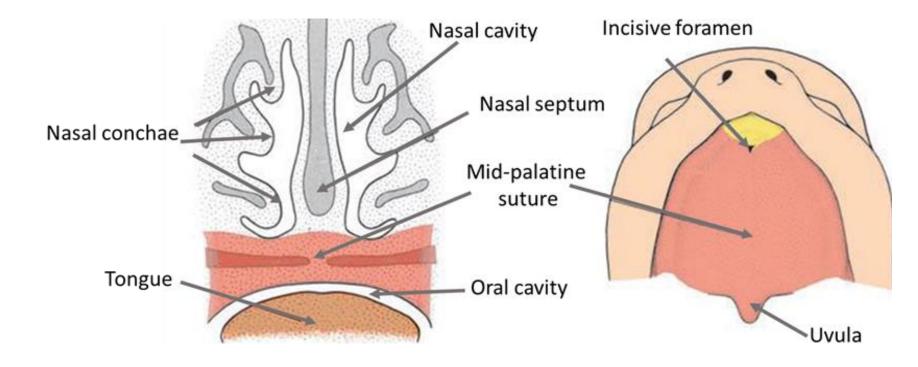
- growth of mandible,
- downward movement of the tongue,
- horizontal orientation of the palatal shelves



Sadler & Langman, 2012

Finely orchestrated developmental program of palate





Palataogenesis of 8-week-old human embryo.

Interference with finely programmed developmental process leads to a consequent variety of isolated and syndromic human defects including cleft palate.

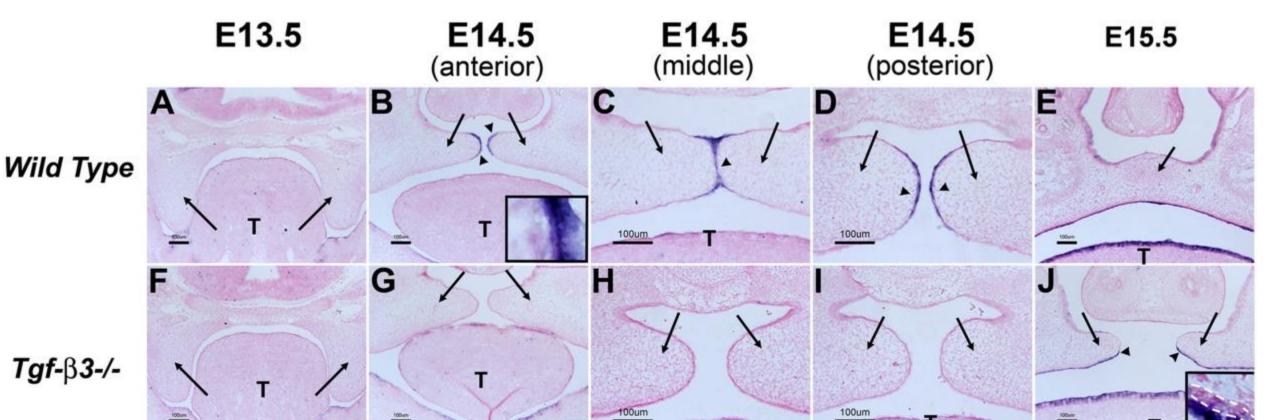
Sadler & Langman, 2012

Fate of Medial Edge Epithelial (MEE) Cells During Palatal Fusion

A transitory mid epithelial seam (MES) derived from the adhered epithelia. medial edge epithelia (MEE) is the he superficial mucous membrane in the contact line

The disintegration of MEE between the two opposing shelves leads to normal midline confluence

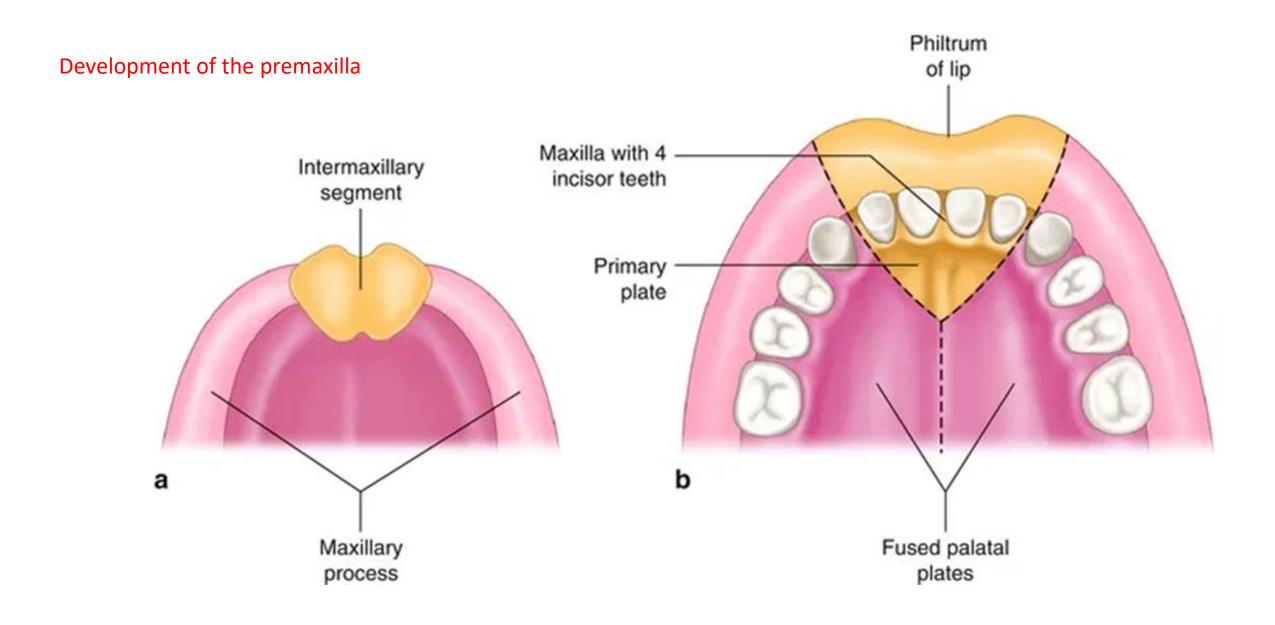
Failure of MEE to disappear will result in aberrant adhesion of the palatal shelves (Alappat et al., 2005) and then a palatal cleft occurs.





There are three concepts explaining how the medial edge epithelial disappears, what are they?

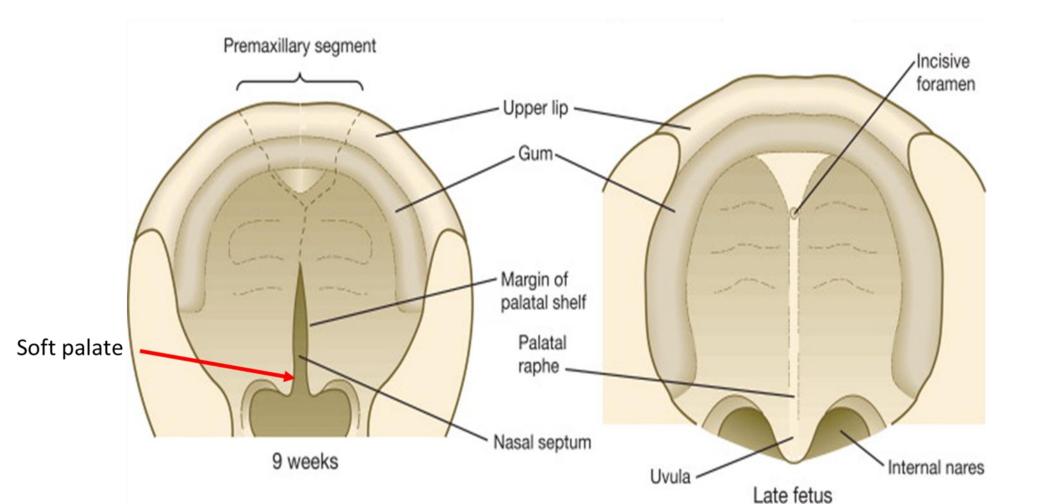
Maximum 300 words in your voice (not copy & paste)



Development of the soft palate

Two separate masses arise at the most posterior portion of the secondary palate.

A selective proliferation of mesenchymal cells located deep in the valley between these two masses results in merging of the masses and obliteration of the valley, contouring of the soft palate and uvula.



Signalling pathways control the craniofacial development

Cell Signalling Pathways Implicated in the Palatogenesis:

- The Role of Sonic Hedgehog Signalling SHH
- The Role of Bone Morphogenic Protein Signalling BMP2&7

The Role of Fibroblast Growth Factor Signalling FGF

The Role of Wnt Signalling Pathway

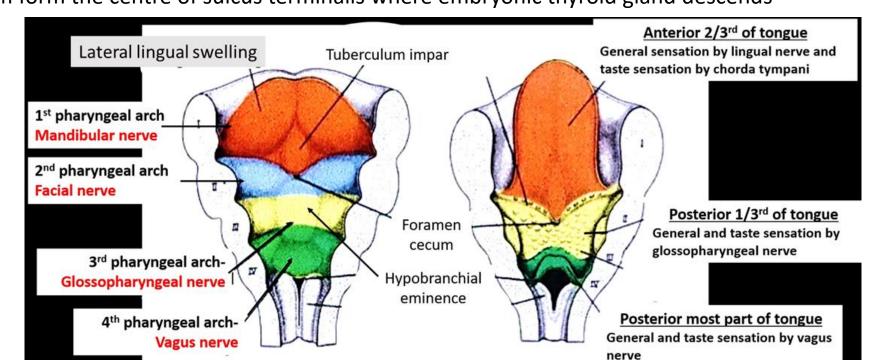
Development of the tongue

Multiple pharyngeal arches shared in development of the tongue (1st, 2nd, 3rd and 4th)

1-Anterior 2/3 are developed near the end of the 4th week as 2 lateral lingual swellings from the 1st arch accompanied with the tuberculum impar

2- hypobranchial eminence is a midline swelling of the 3rd and 4th pharyngeal arches, quickly grows to cover the copula formed earlier from the second pharyngeal arch, and will form the posterior one third of the tongue

3- the junction of ant. 2/3 and posterior 1/3 is called sulcus terminalis 4- foramen caecum form the centre of sulcus terminalis where embryonic thyroid gland descends



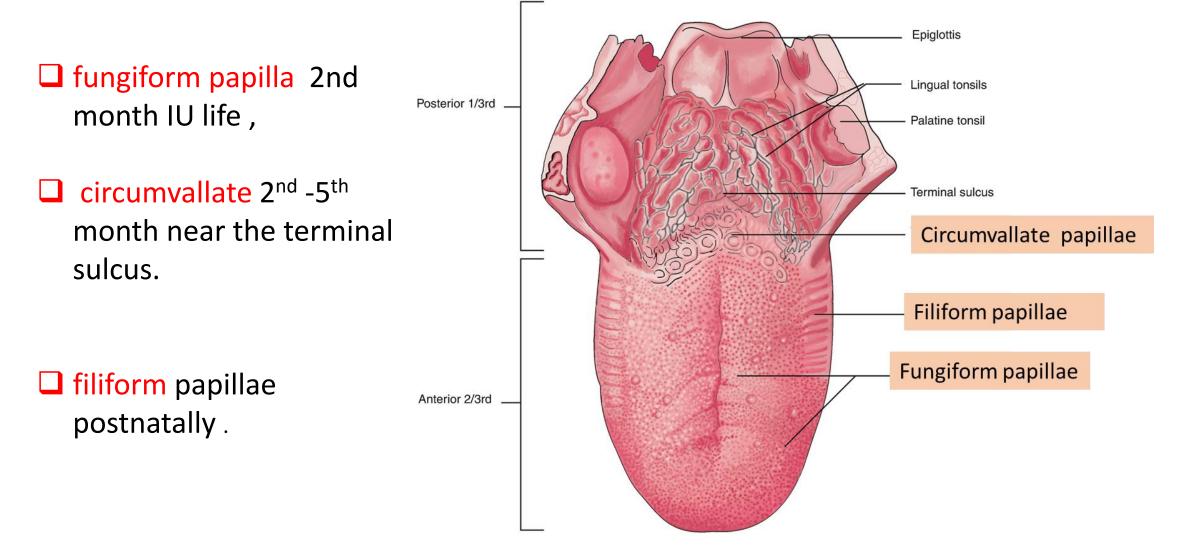
5- because of different origins, the nerve supply of ant. 2/3 is hypoglossal nerve while that of the post 1/3 is glossopharyngeal nerve

Nerve supply and developement arch relationship of tongue

	Anterior 2/3 rd of tongue	Posterior 1/3 rd of tongue	Posterior most portion of tongue
Taste	Chorda Tympani	Glossopharyngeal	Vagus – internal laryngeal
Sensory	Lingual of V3	Glosssopharyngeal	Vagus – internal Iaryngeal
Motor	All extrinsic and intrinsic muscle except palatoglossus – Hypoglossal N, palatoglossus – cranial root of accessory N		
Arch Developement	First	Third	Fourth

www.facebook.com/notesdental

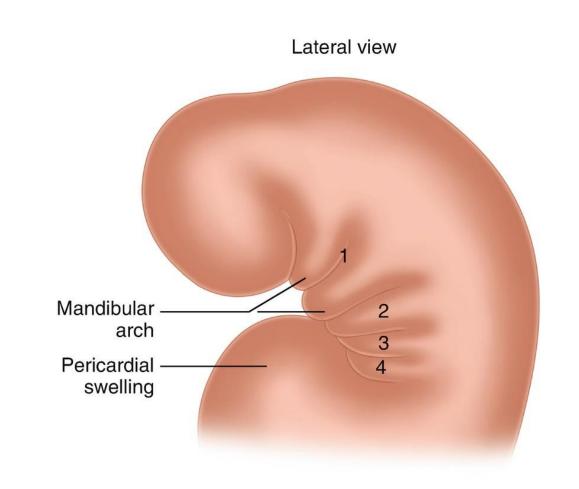
6- taste buds development

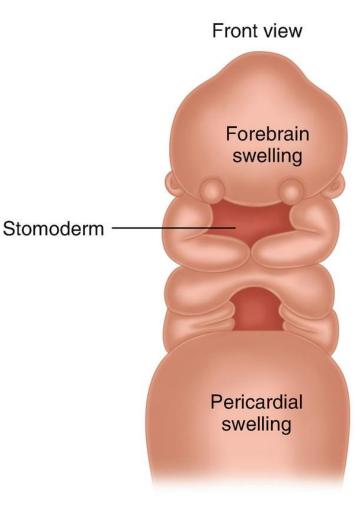


7- at birth tongue occupies
full mouth because of its
large size in relation to the
mouth size and protruding
forward to help in suckling

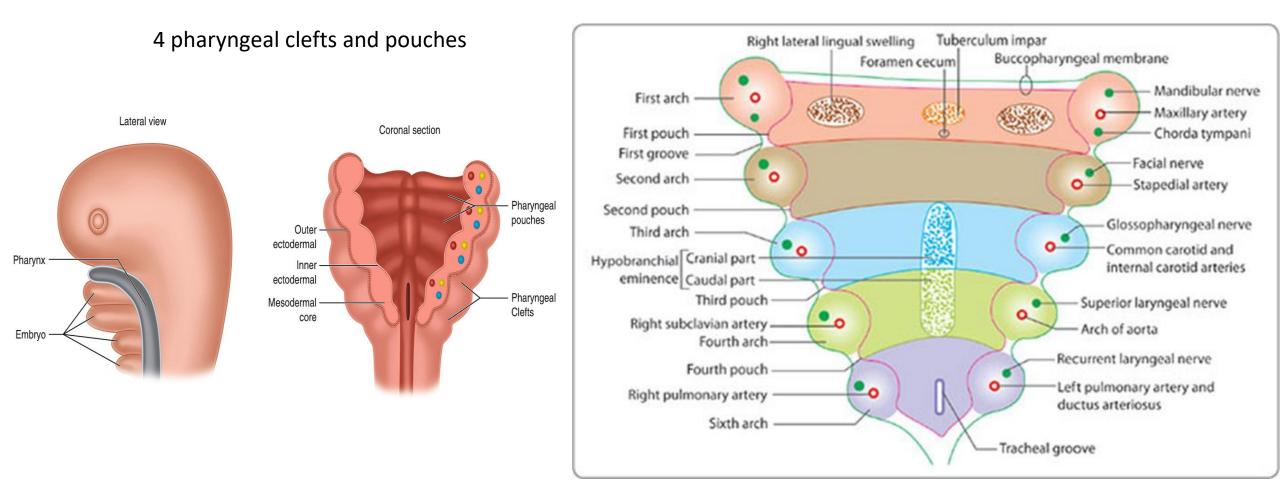


Pharyngeal pouches and clefts/grooves

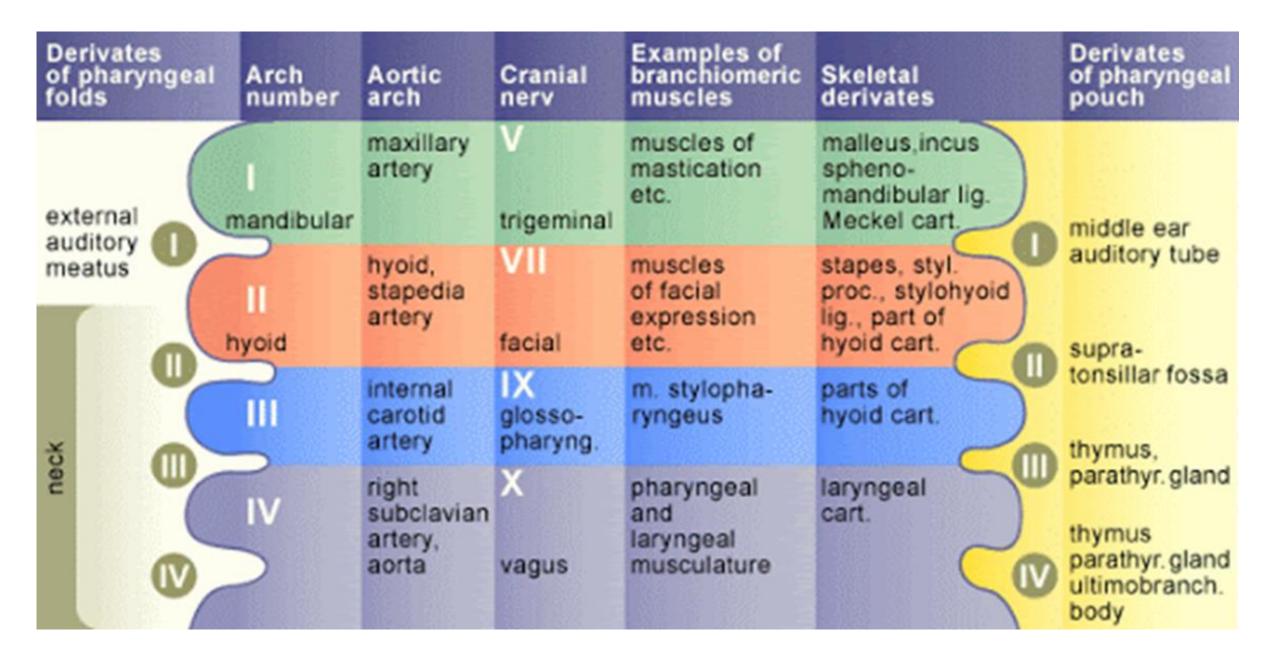




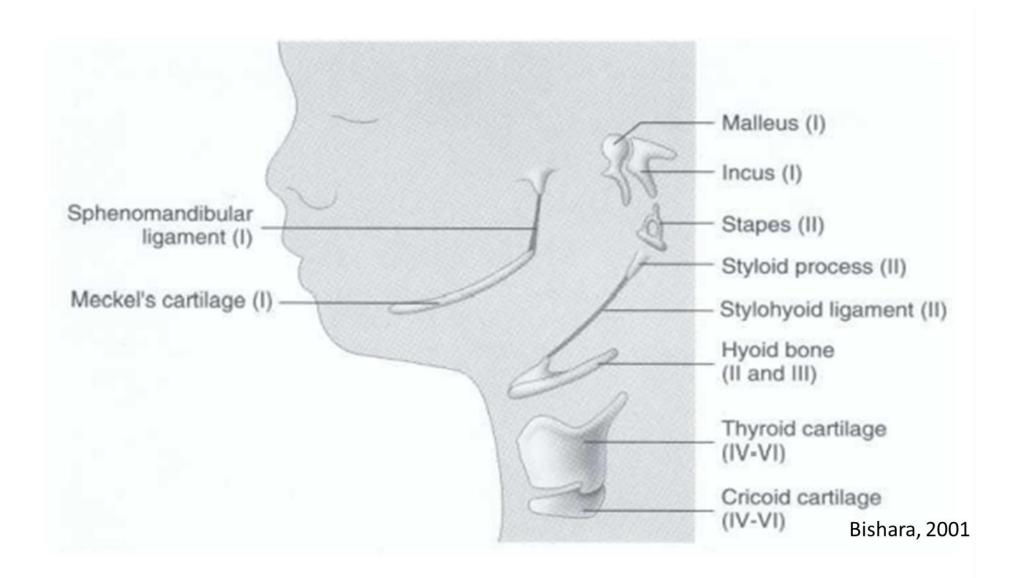
Pharyngeal pouches and clefts/grooves



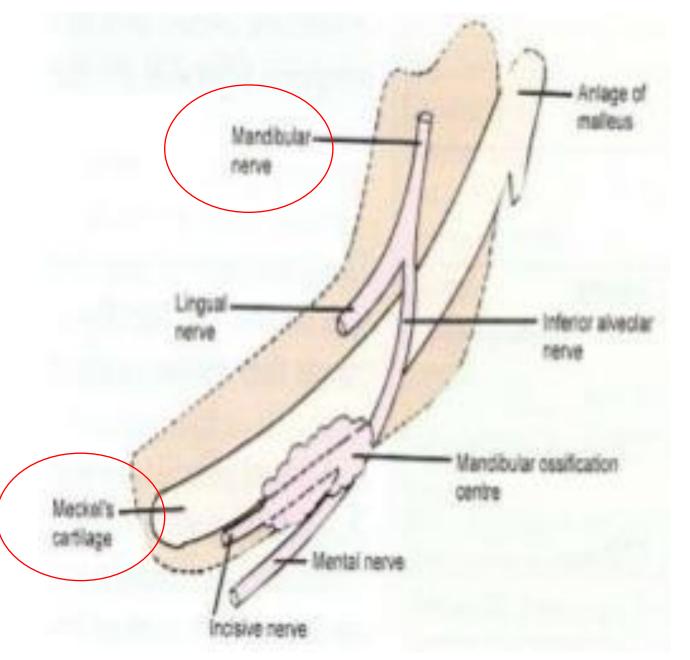
Pharyngeal pouches and clefts/grooves- derivatives



Cartilage components of pharyngeal arches

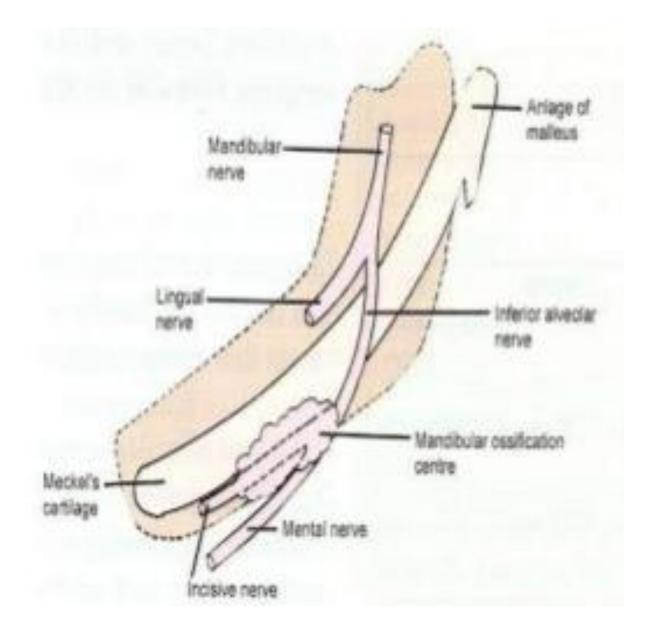


At 4th week IU mandibular branch of the trigeminal n. is the first structure to develop in the primordium of the mandible

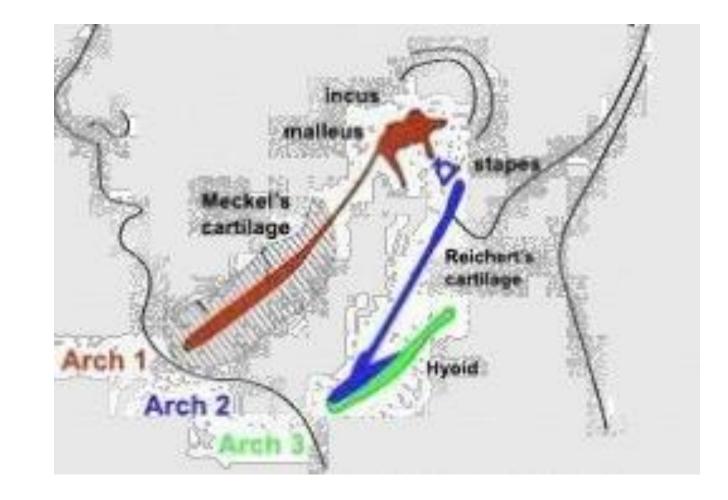


A rod-like cartilaginous core, Meckel's cartilage, develops close to junction of the distal and middle thirds of the mandibular nerve

Mandibular nerve passes forward to divide into lingual nerve (medial to cartilage) and inf. dent. nerve (lateral to cartilage)



Distally, Meckel's cartilage articulates with the cartilaginous cranial base in the petrous region of the temporal bone, where it gives rise to the malleus and incus bones of the inner ear.

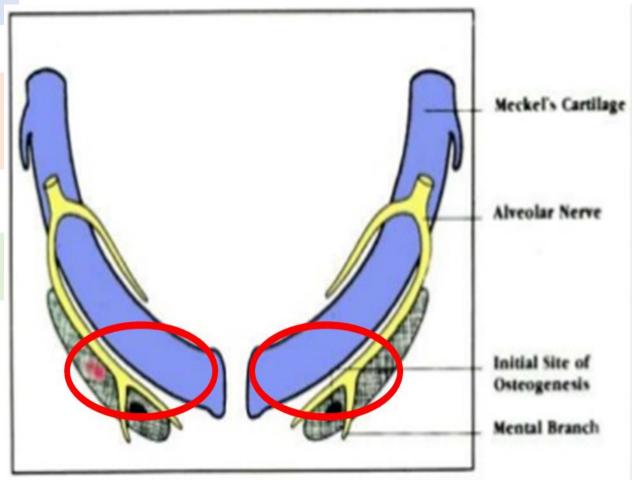


intramembranous or endochondral??

Meckel's cartilages extend proximally to join each other at the symphysis by mesodermal tissue

A perichondral membrane lateral to Meckel's cartilage shows condensation of mesenchyme at the division of the inf. dent. nerve

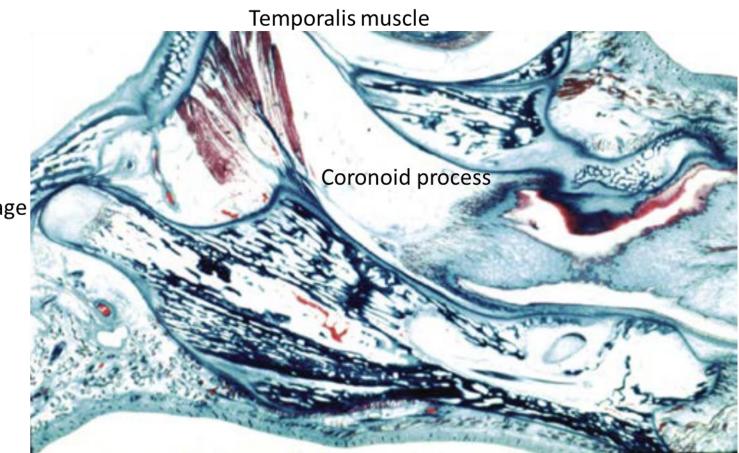
At 6th week, a centre of ossification appears in the lateral condensation



Inferior mandibular canal

medial and lateral bony plates extend anteriorly to the symphysis, posteriorly to the area where mandibular nerve divides and these plates join beneath the nerve forming inferior mandibular canal

Mandibular condyle cartilage



secondary cartilages

between 10th and 14th week of IU life, 3 secondary cartilages appear in the growing mandible.

Condylar cartilage :

respectively.

This is the most important large secondary cartilage. The condylar process appears as a separate carrot-/cone- shaped blastema of cartilage (secondary cartilage from membranous ossification), articulate with the squamous (membranous) portion of the developing temporal bone. This cartilage is replaced by bone except its

upper portion which still essential for growth and articulation through adulthood.

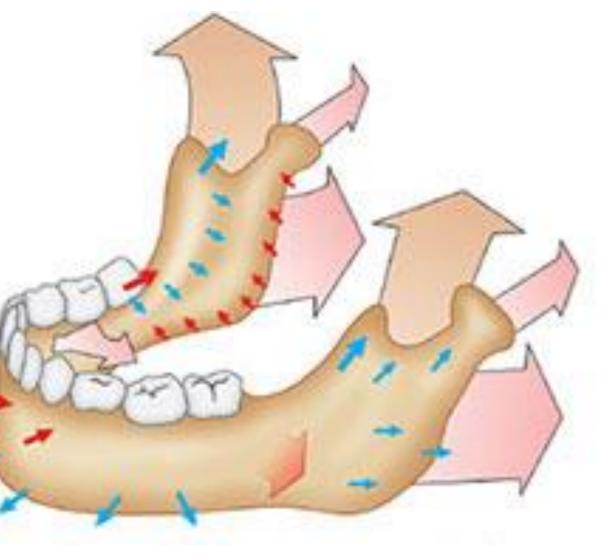
coronoid and symphyseal cartilages are transitory and incorporated later on with adjacent coronoid and body of mandible, Mandibular condyle cartilage

Postnatal Growth of the Mandible

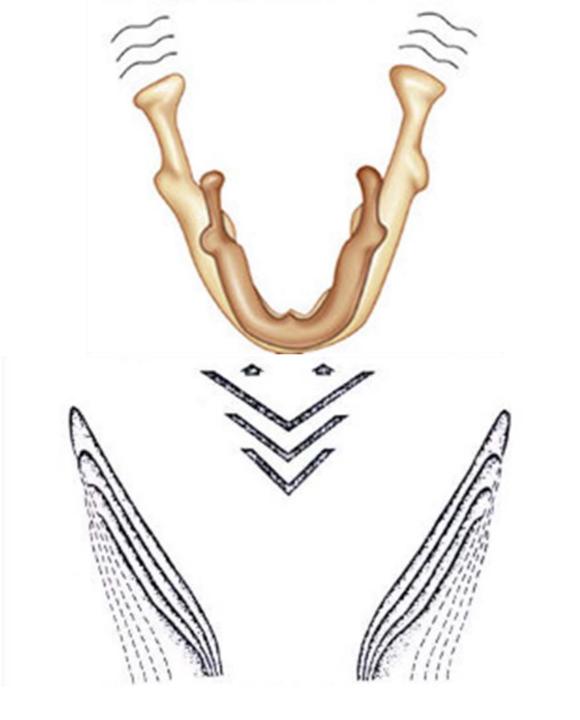
1- The mandible has the greatest postnatal growth potential than any component of the craniofacial complex

2- at birth the body is made of two halves which united at the end of first year

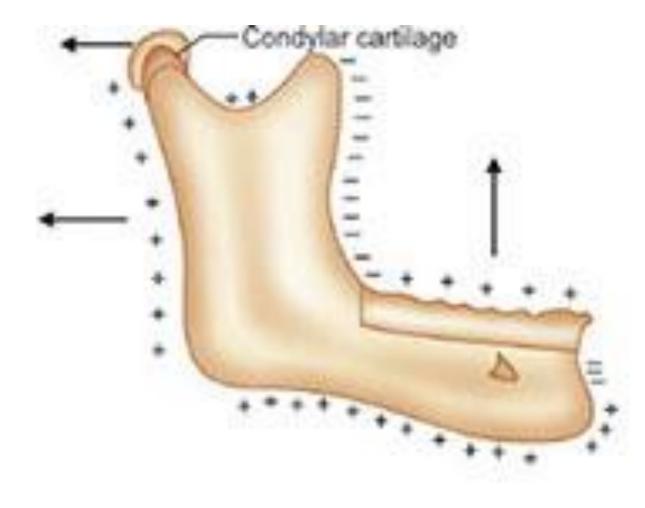
3- At birth, the ramus of the mandible is quite short



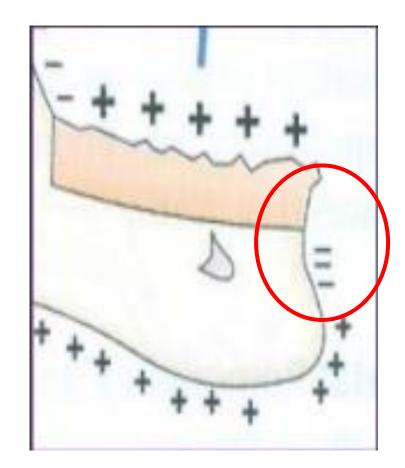
4- mandibular growth follows the expanding "V" principle



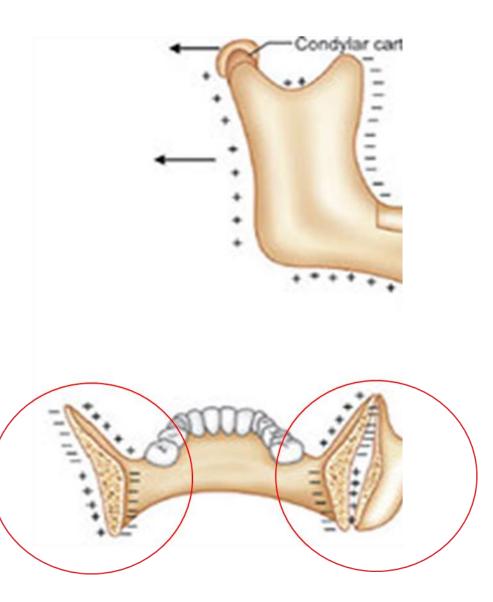
5- functional processes are associated with growth changes of the mandible comprise the gonial process, coronoid process, alveolar process, and bony attachments of the suprahyoid muscles (lingual tuberosity), which allare major sites of postnatal modelling.



6- The cortical region at or just above the chin is the only place on the entire surface of the mandible that remains stable (i.e., does not model) during postnatal growth, which is why it serves as an important site for superimposing successive radiographs.

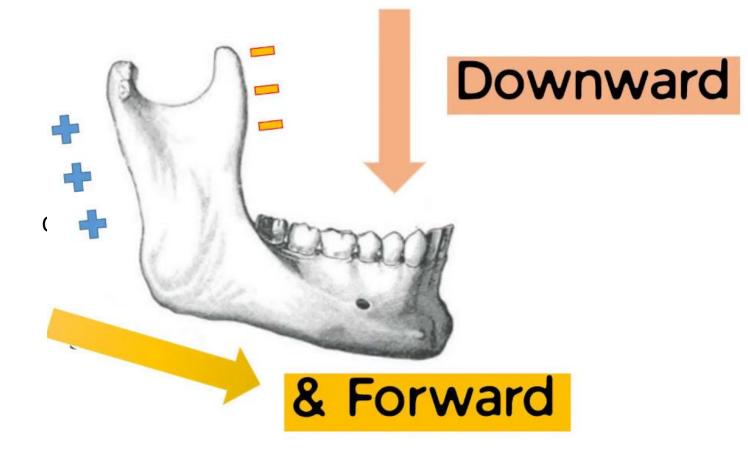


7- The mandible also widens due to bony deposition along its posterior surface



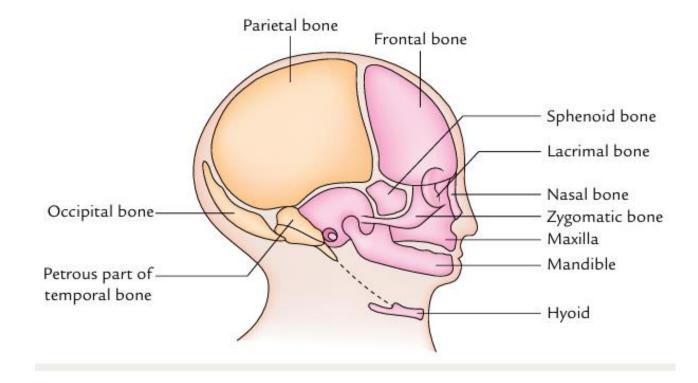
8- The mandible undergoes substantial amounts of true vertical rotation

9- As in the rest of the craniofacial complex, sex differences in mandibular growth are evident with males have larger mandible than female.



Development of the skull

(neurocranium and viscerocranium)



1- calvarium/vault intramembranous

Mesenchyme superior and lateral to developing brain is derived from the neural crest cells

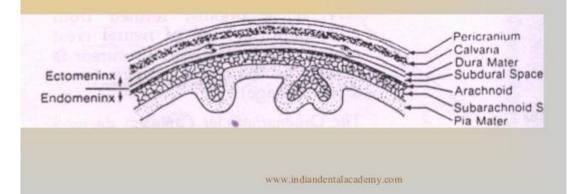
Membranous ossification needle-like bony spicules going to peripheries



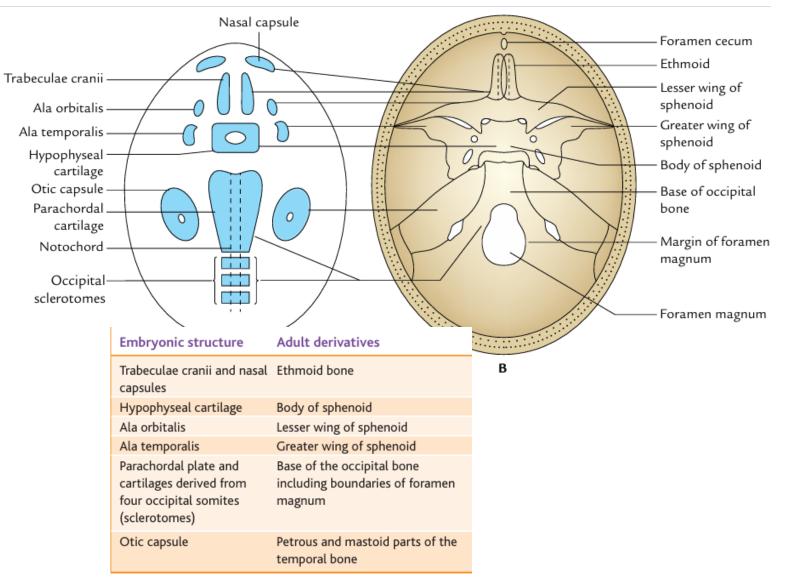
PRENATAL GROWTH

* intramembranous ossification.

MEMBRANOUS NEUROCRANIUM



Base of skull is formed by the fusion of several cartilages



Postnatal growth and development of craniofacial complex

Importance

Proper type and timing of treatment such as

- orthopaedic/functional appliance
- rapid maxillary expansion

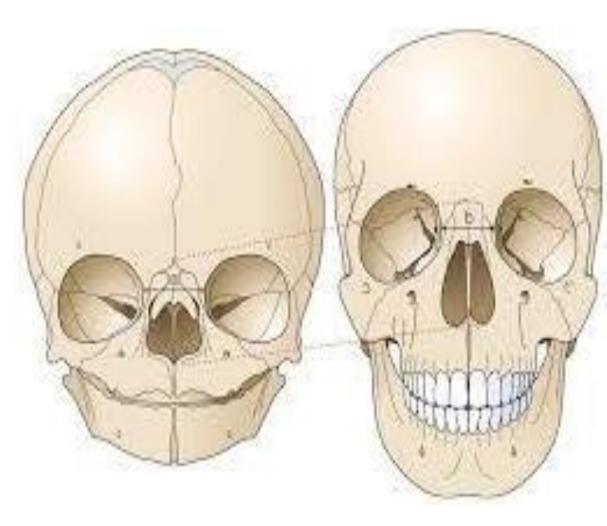
Differences between infant and adult's skulls

1-Infant skull 45 elements separated by cartilage and connective tissues (mandible 2, frontal 2, occipital 2), while adult's 22 bones.

2- bones are widely separated from adjacent bones by loose connective tissues at fontanels

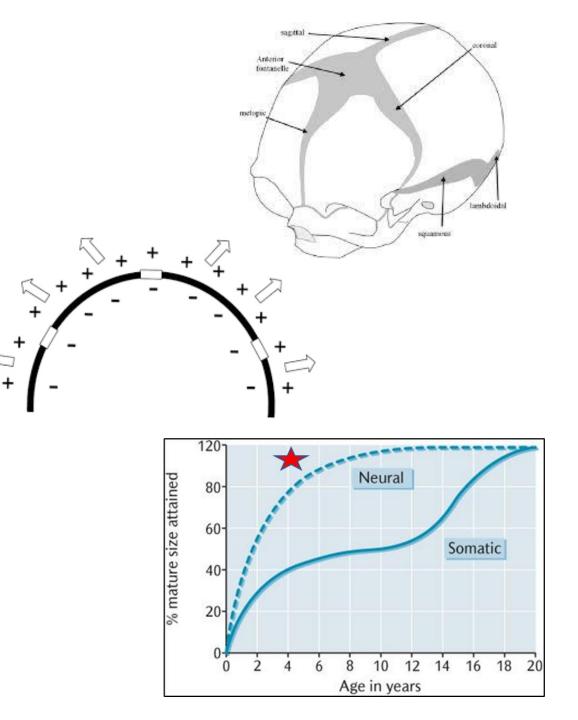
3- Small nasomaxillary complex and mandible give rise to short face

4- Cranium > 50% of the head



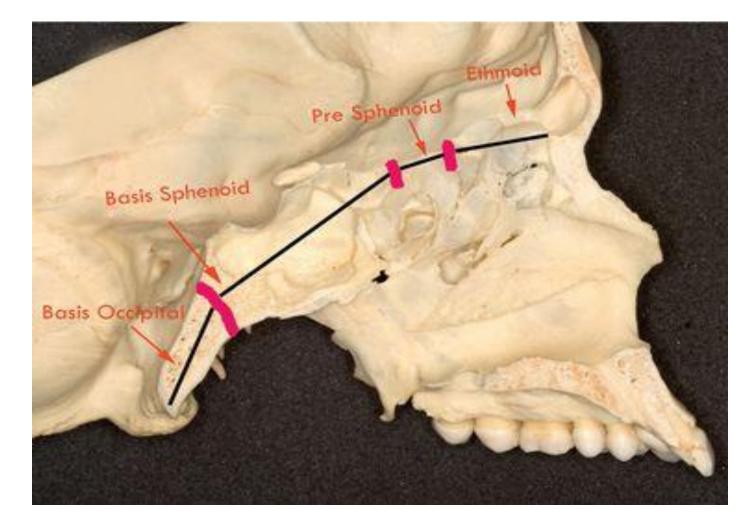
Postnatal growth of the vault

- Rapid growth of coronal, sagittal, temporal and occipital sutures in response to rapidly growing brain
- Endosteal resorption and periosteal apposition lead to increase in overall size of cranial cavity
- Postnatal growth of cranial vault follows the neural growth curve achieving 90% of its growth at first 5 years of life.

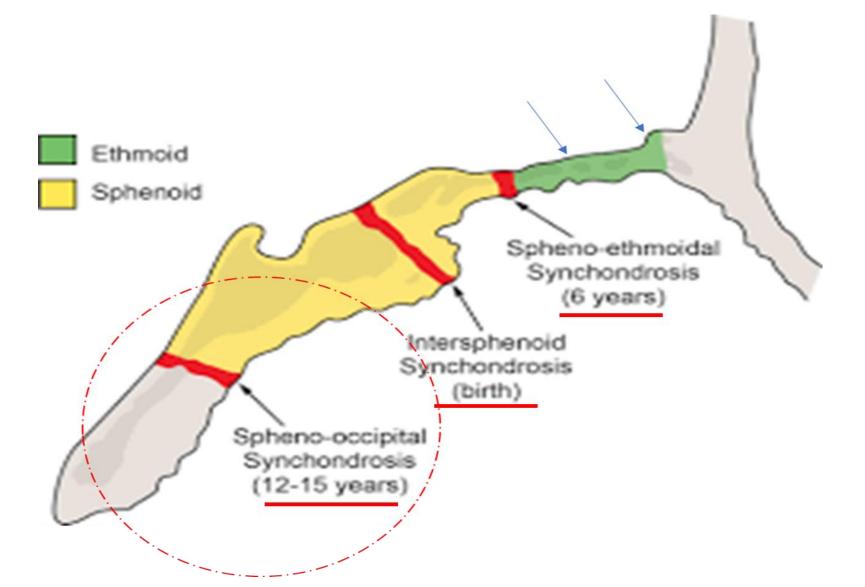


Postnatal growth of the cranial base

- Endochondrally ossified cranial base formed from ethmoid, sphenoid and occipital bone
- Cranial base acts as template for the facial development



A synchondrosis is a hyaline cartilaginous joint subsequently converted into bone Endochondral growth changes take place through a system of synchondroses



Prenatal growth of Maxilla

Maxilla develops from the maxillary process of the first pharyngeal arch by intramembranous ossification

Ossification centre appears around 8th week IU life close to the cartilage of nasal capsule between the division of anterior superior alveolar nerve and infraorbital nerve

Ossification spread in different directions

- ✓ Posteriorly towards the developing zygoma
- ✓ Anteriorly towards premaxilla
- ✓ Superiorly to form frontal process
- ✓ Medially towards palatine process

□ Medial and lateral alveolar plates form a trough surrounding the maxillary teeth germs

□ A secondary cartilage appears close to the zygomatic process of maxilla

The body still small at birth containing a small pea-sized maxillary sinus which enlarges to adult size by gradual resorption of maxilla internal walls

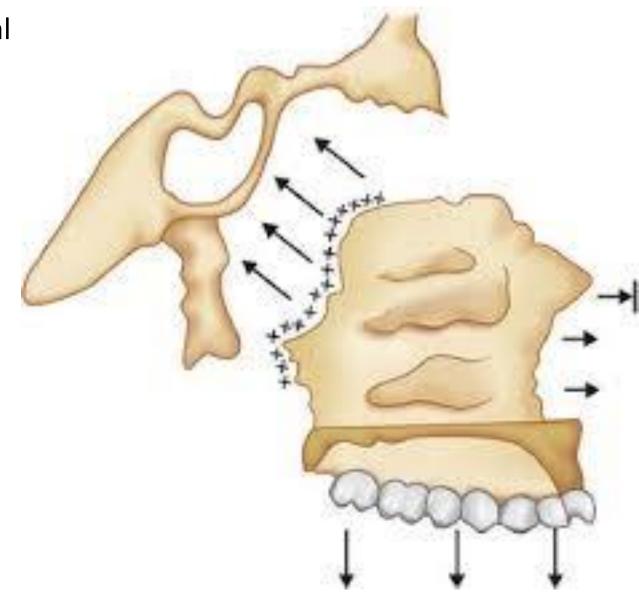


Nasomaxillary complex postnatal growth

Growth of cranial base and cartilaginous nasal septum carry the maxilla forward and downward

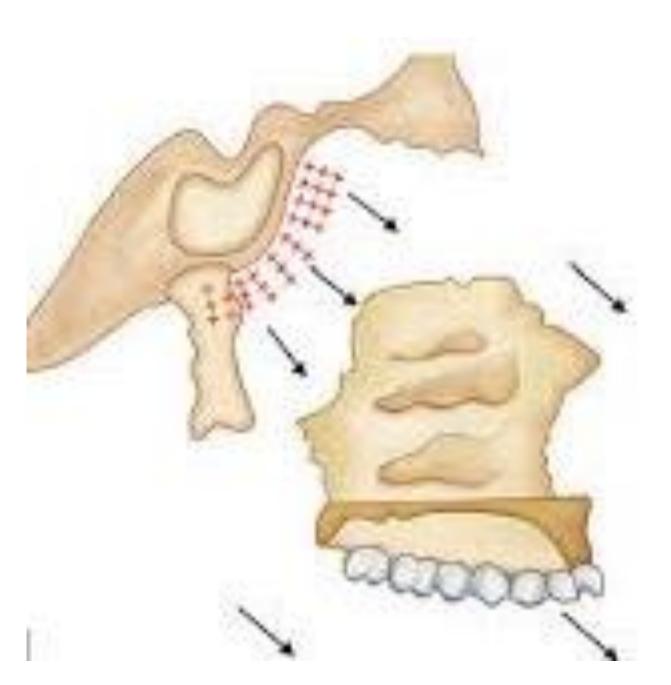
Mechanisms of maxillary growth

- 1- translation/change in spatial position
- Primary translation/displacement: enlargement of the bone size by continuous deposits on tuberosity leading to horizontal anterior displacement.

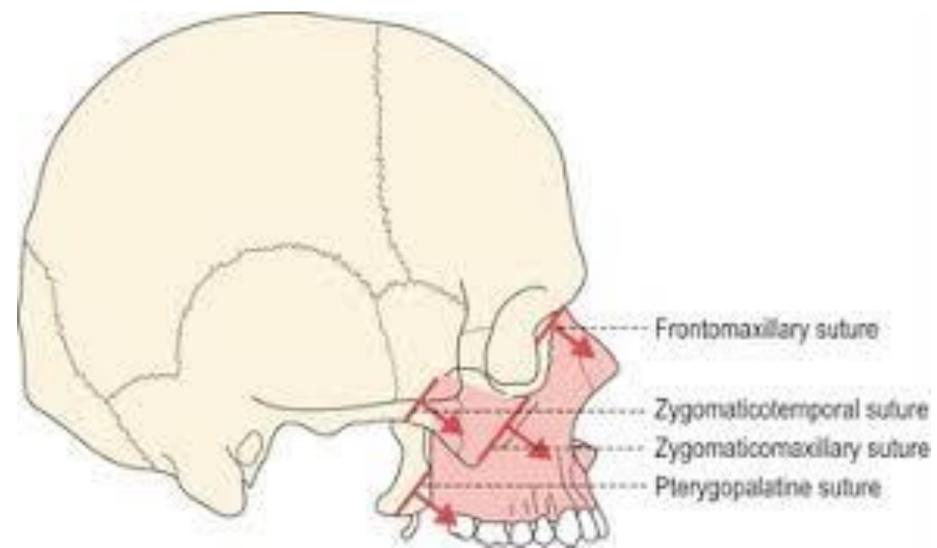


- Secondary translation/displacement occur due to:-
- ✓ growth of the cranial base
- \checkmark and eruption of primary teeth as well

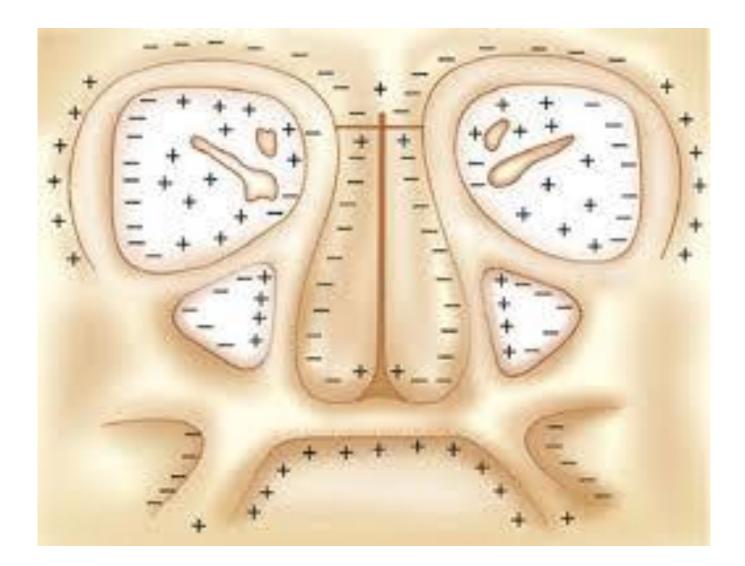
which lead to carrying the nasomaxillary complex downward and forward



2- Sutural growth: maxilla joined with surrounding bones by parallel sutures which are stimulated to form osteogenic sutural membrane



3- bone remodeling by deposition and resorption along endosteal and periosteal surfaces of bone



The Four Stages of Human Growth

From conception to death

- Prenatal : fast rate and very rapid rise in cell numbers
- Postnatal: birth through first 20 years. Decline rate and increase tissue maturation.
- Maturity: body gaining its stable function with maintenance of equilibrium between cell death and replacement
- Aging: decline in functional activities and slow growth process



Regulatory Factors of Growth

- **Genes** control the rate and timing of the growth
- Circulating growth hormones which decline after puberty leading to slow down the growth. Decrease GH leads to dwarfism, while increase leads to gigantism. Thyroxin, thyronine involved in metabolism, while androgen and testosterone-adolescent changes
- **Growth factors** TGF-beta, IGF-I, PDGF, VEGF
- Nutrition : vitamins like vitamin C for connective tissues and vitamins D and A for osteoblast/ osteoclast activities, proteins and minerals
- Secular trends which are a reflect of improved life style and increased health status of the current generation in comparison to the previous ones eg: 15 y old children are now taller by 5 inches than previous generations according to some studies.
- **Illness** may have adverse effect on growth process
- Season and circadian rhythm: height in spring>than in autumn/ weight in autumn> in spring/ growth in height and teeth eruption is more at night than daytime due to fluctuation in hormone release
- **Psychology** stress was found in some research studies to have adverse effect on growth.

How the physical growth can be measured?

1- measurement approach

measuring some criteria on living animals or animal remains , it is less invasive e.g.

- **c**ephalometric analysis
- □ craniometry (measurement of the human skull in different age groups)
- □ anthropometry (measurement of the body).

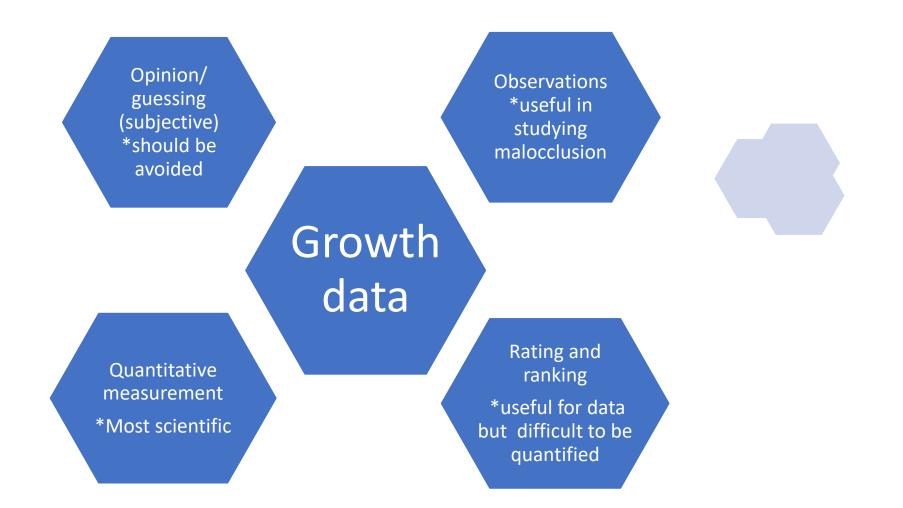
How the physical growth can be measured?

2- experimental approach:

more invasive as it needs to harm or even sacrifice experimental animals by e.g. injecting some stains required for experiment

- □ Vital staining (Hunter & co-workers 18th century) injected dye which stain the mineralised tissue to be studied
- Radioisotopes: injected calcium 45 and technetium 33 and emitted radioactivity was tracked
- Implant radiography (Bjork, 1955): implanting metals in the growing bone as radiographic references

Growth data variations



The 3 types of growth research studies

Cross-sectional: different age groups are studied in one occasion (large no. in short period)

Longitudinal: same sample is studied repeatedly in regular occasions (small sample size/ long period/ results inference should be at the end)

Mixed or semi longitudinal: different age groups are seen longitudinally in regular short periods

How to obtain growth measurement data?

Direct by calliper or tapes

Indirect by radiograph or photos, or dental casts

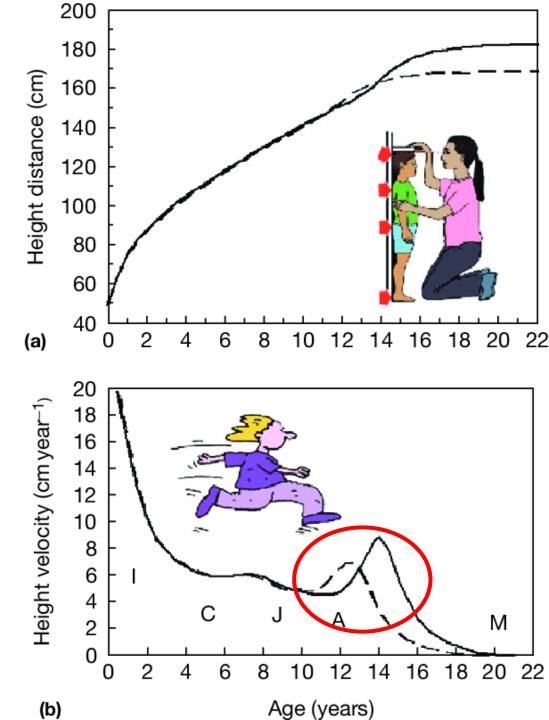
Mixed comparison of direct and indirect such as implant and radiograph

How are the growth data plotted?

Distance curve: Data of height or weight collected from cross-sectional or longitudinal studies are displayed in a cumulative curve. Height of girls (dashed line) steeps earlier than boys (solid line).

Velocity /increment curve: Rate of growth (height or weight) over a period of time (usually longitudinal studies). Height velocity decrease from birth onwards except in adolescent growth spurt where growth accelerated

I, infancy C, childhood J, juvenile A, adolescence M, mature adult (modified from Bogin 1999).





Compare between chronological age and developmental age, with examples?

Maximum 200 words in your voice (not copy & paste)

Normal features of human growth

1- Variability : Different children show different increment of growth, and no two individuals show the same growth. This is because:

- ✓ Heredity
- ✓ Sex
- ✓ Nutrition
- ✓ Racial difference
- ✓ Excessive
- ✓ Climate
- ✓ Socioeconomic and psychological factors
- Timing: biologic clock of growth is different. e.g. girls mature earlier than boys.

Normal features of human growth

2- Pattern/differential growth

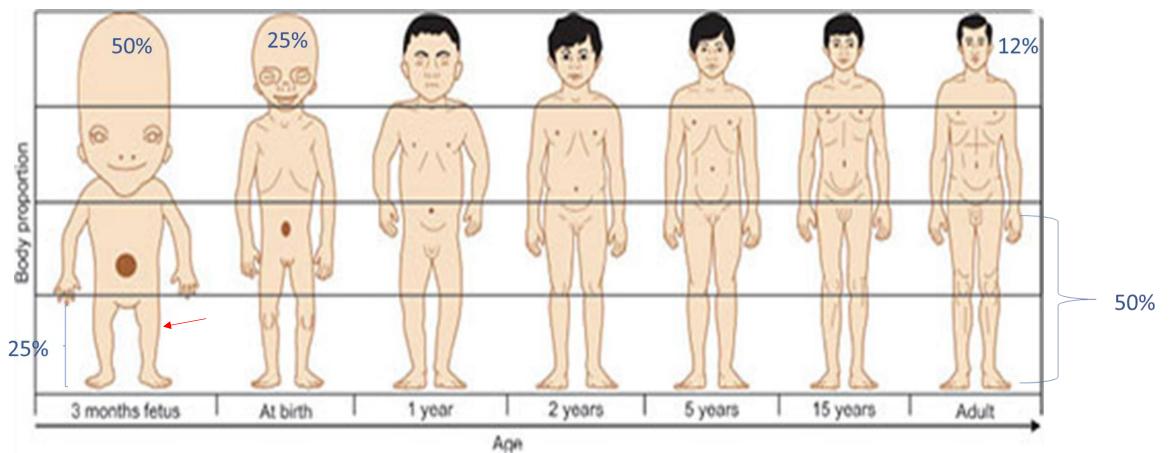
Differential growth means NOT all different organs of body grow at same rate, times and extents (DIFFERENT ORGANS GROW AT DIFFERENT RATES)

3- Allometric growth: proportional change in body size

4- Cephalocaudal **Gradient** (gradual change) of Growth: represent changes in the normal body proportions during normal growth and development.

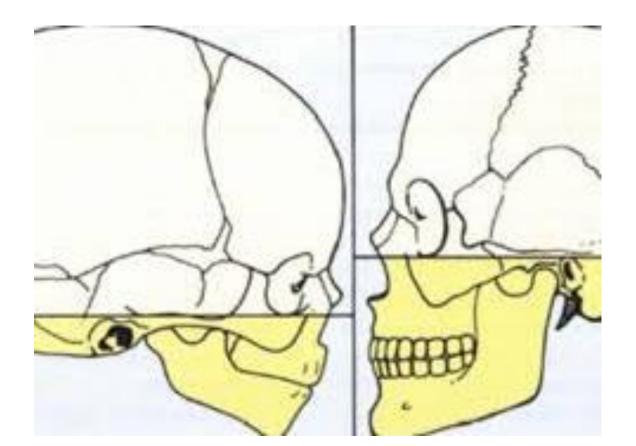
The skull in foetus is much larger while the limbs are rudimentary The face much smaller

Axis of increased growth from head to trunk to feet



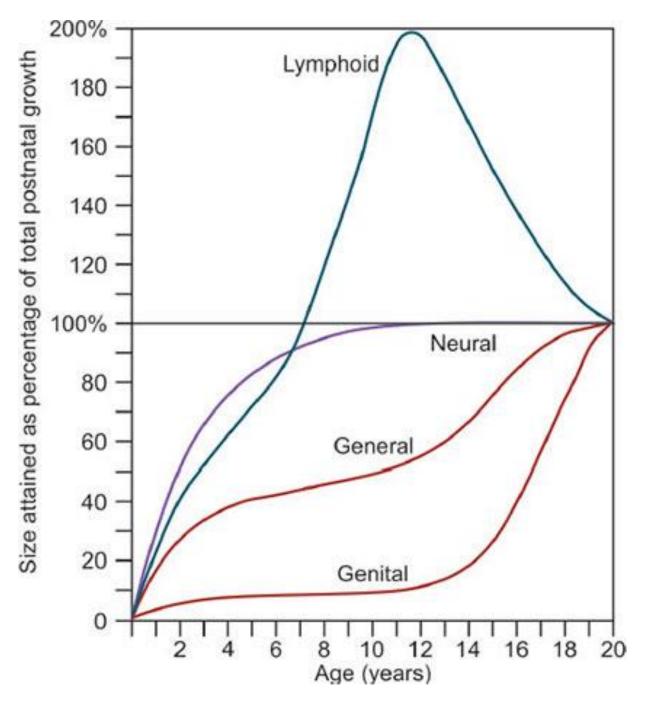
Cephalocaudal gradient in the face

- □ Cranium > 50% of the head
- □ Nasomaxillary complex underdeveloped
- □ Maxilla closer to the brain and growing faster than mandible
- □ Mandible far from the brain growing slower than the maxilla



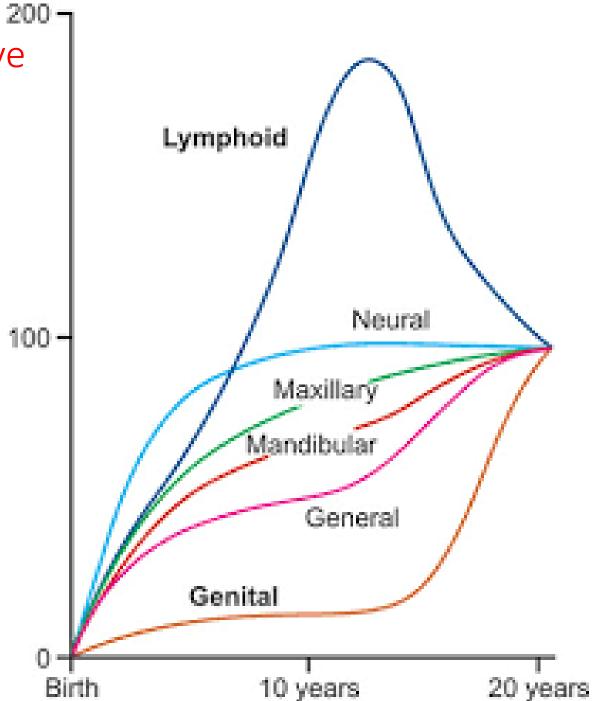
Scammon's Growth Curve

- Lymphoid tissues, during late childhood (10-15y), proliferate 2 folds beyond the adult amount, then undergo involution at same time where genital tissue growth accelerate.
- Neural tissue: at 8y of age, brain is 90% its adult size enabling child to function mentally nearly like adult.
- General body tissues (skeleton, muscles, and viscera) show S-shape with slow growth during childhood and accelerate during puberty



Growth of maxilla on Scammon`s curve

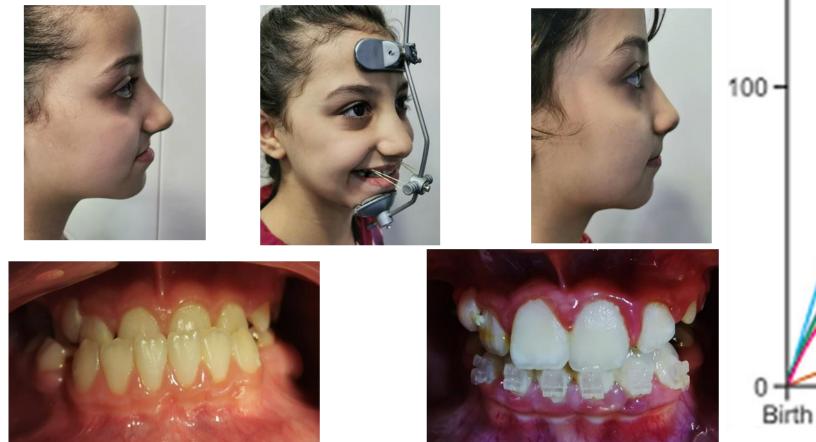
Maxilla growth follows neural growth and ceases earlier than mandible (8 years)

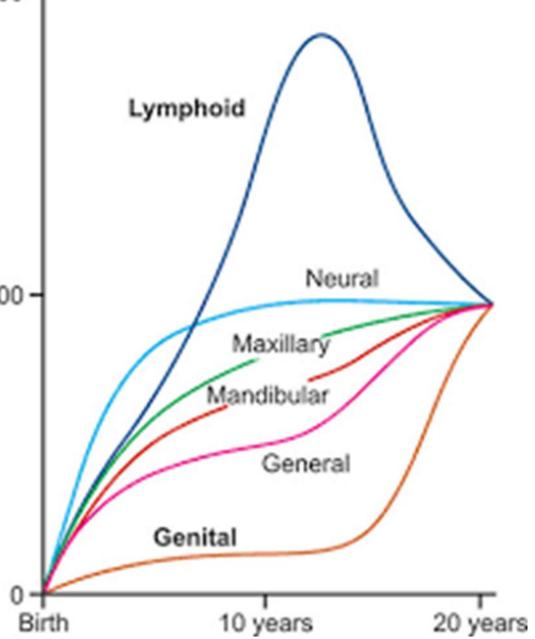


Growth of maxilla on Scammon's curve 200 -

Clinical consideration of Maxillary growth on Scammon's curve

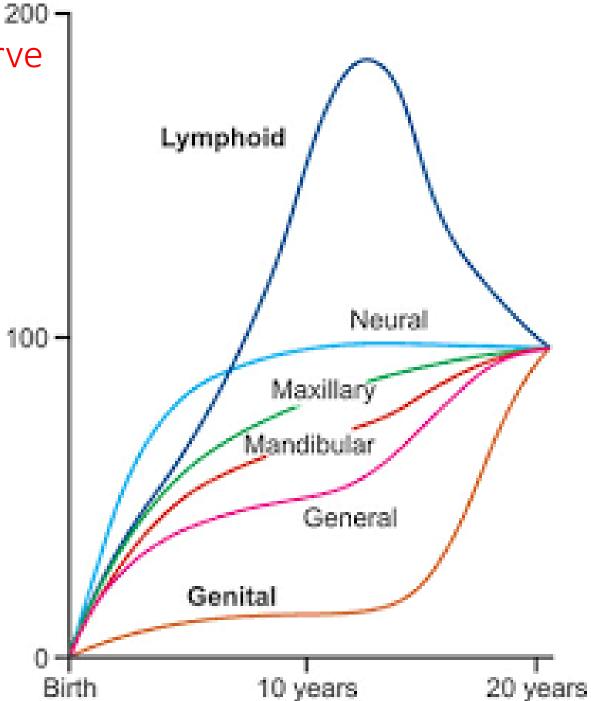
growth modification/promotion of maxilla by face mask should be given around 8y of age





Growth of mandible on Scammon's curve

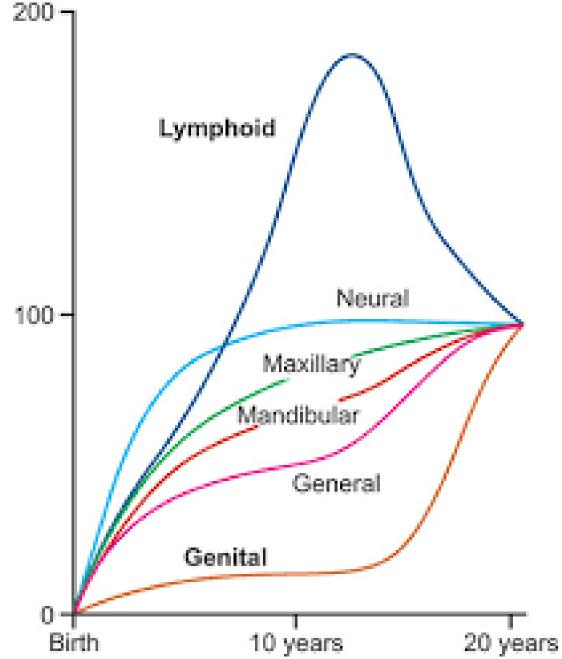
Mandible growth follows general growth and occurring until 18-20 years



Growth of mandible on Scammon's curve

Clinical consideration of mandible on Scammon's curve

growth modification/treatment of mandible should extend until cessation of growth to prevent relapse of class III malocclusion.



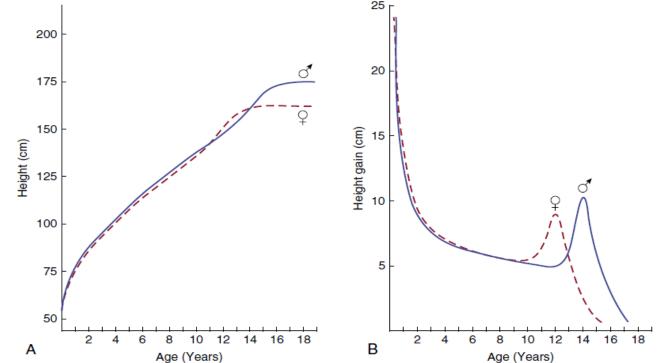
Growth spurts

Mean sudden accelerated growth rate periods scattered within periods of relative rest.

	Girls (Years of age)	Boys (Years of age)	
Infantile/Childhood	3	3	
	Extends over 2 years. body length increases from 50 cm to 75 cm first year and 12 to 13 cm second year. Then, 5-6 cm per year		
Mixed dentition/Juvenile	6-7	7-9	
	Least noticeable		
Pubertal/Adolescent	10-12 (16 cm height gain)	12-14 (20 cm height gain)	

Pubertal growth spurt is more dramatic and thought to be caused by hormonal secretions How long the pubertal growth spurt may last?

Onset of the pubertal growth spurt typically begins about age 10 in girls and lasts approximately 2 years. Boys have later onset (12 years); the entire pubertal period can last 4 to 6 years.



Clinical importance of studying the growth spurt

- Difference in growth spurt between males and females
- orthopaedic and functional appliances better during growth spurt
- Rapid maxillary expanders better during growth spurt
- Retention and stable results after cessation of growth spurt
- Orthognathic surgery after cessation of active growth

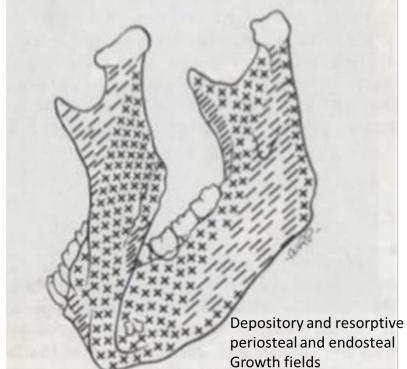
Estimation of growth spurt

1- measurement of body height
 2- x-ray of hand wrist and phalanges ossification
 3- shapes of cervical vertebrae in cephalometric radiograph

Growth field, centre and site

Growth fields: the outside and inside of bone are blanketed by a mosaic-like pattern called growth fields. Half of periosteal surface of whole bone has arrangement of resorptive field while the other have is covered by depository fields.

If a given area of bone has periosteal resorptive fields the opposite endosteal surface of the same area has depository fields and vice versa, this combination will produce drift of whole bone



Growth fields

bone growth is controlled by genetic pacemaker signalling originating from surrounding periosteal soft tissue as well as lining endosteal soft tissue. The periosteal and endosteal growth fields are arranged in mosaic pattern with either depository or resorptive activity. Growth centres: the very active growth fields with an intrinsic hereditary growth potential.

An independent growth takes place genetically controlled. When transplanted, they continue producing a tissue separating force that stimulate bone deposition.

e.g.

- synchondrosis of the cranial base
- mandibular condyle was thought to have a growth centre, however it includes a growth site



Growth sites: are growth fields where a significant exaggerated growth takes place as secondary compensatory activity under an extrinsic control. Growth site may occur in growth centres but they not growth centres.

e.g.

- maxillary tuberosity
- Facial and cranial sutures
- Alveolar processes

Theories of craniofacial development

1- Genetic Theory /Brodie 1930s: proposed that the complete control on pre-planned hereditary growth

2- Sutural Dominance Theory /Sicher, 1940: advocated that the suture act as a "growth centre" within each suture is a genetic information that control the growth amount.

However, research studies proved that sutures are growth sites rather than growth centres and the growth in sutures is adaptive and secondary to functional needs.

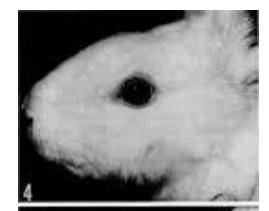
Evidence to objections are firstly, sutures when transplanted to other areas have no innate growth potential and don't continue growth. Secondly, suture responds to unusual pressure by bone resorption rather than deposition.

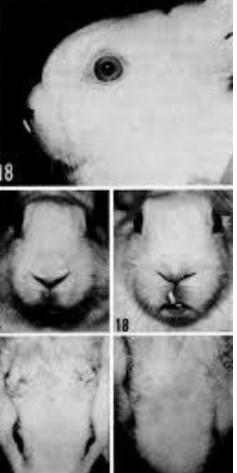
3- Cartilaginous Theory/ Scott 1950s: stated that intrinsic growth controlling factors are present in the cartilage and periosteum, with sutures being only secondary and adaptive.

Hence, nasal septum cartilage and condylar cartilage are major driving force/pacemakers for forward and downward growth of nasomaxillary complex and mandible, respectively.

Evidence:

- Continue growing after transplantation
- Extirpation of nasal septal cartilage cause mid face retardation
- Precursor cartilage give rise to bone like cranial base synchondroses





4- Functional Matrix Theory/ Moss 1969

van der Klaauw's concepts (1946) suggested that skull is formed from units with certain size and shapes determined and affected by their functions.

Moss (1969) stated that "Bones do not grow, bones are grown"

- Moss's model stressed the influence of nonosseous structures on the bony parts
- Soft tissues regulate the growth of skeletal tissues through functional stimuli
- Then the growth of endochondral or intramembranous structures are secondary and compensatory to a primary determinants of functional matrices.

Functional	crania	com	ponents
arretroria			

Functional matrix

Skeletal unit

Periosteal matrix	Capsular matrix e.g. neurocranial (dura matter + skin) /orofacial (mucosa + skin) capsules	macroskeletal unit e.g. Mandible or Maxilla	microskeletal unit Mandible: condyle ,
Act directly on microskeletal unit Leading to changes in size and shape of the skeletal unit by deposition and resorption	Act indirectly on macroskeletal unit Leading to changes in <mark>spacial</mark> position of the skeletal unit		coronoid, gonial, mental , alveolar process, etc. Maxilla: orbital, palatal, alveolar and pneumatic
Causing active growth (transformation)	Causing passive growth (translation)		(maxillary antrum) units

Skeletal unit is defined as the totality of all skeletal elements associated with a single particular function such as speech, swallow respiration

Other craniofacial growth theories

 Van Lomborgh's Compromise/Multifactorial Theory (supported Functional Matrix Theory + accepted Sutural Theory + acknowledged Genetic Theory)

- Servosystem Theory
- Expanding "V" Theory

work

- Servosystem Theory
- Expanding "V" Theory

Maximum 200 words in your voice (not copy & paste)