Etiology of Orthodontic Problems

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In the Name of Allah

the Compassionate the Merciful


فاطمة الزهراء

Goals

- Leisure
- Get energy for the week
- Think differently

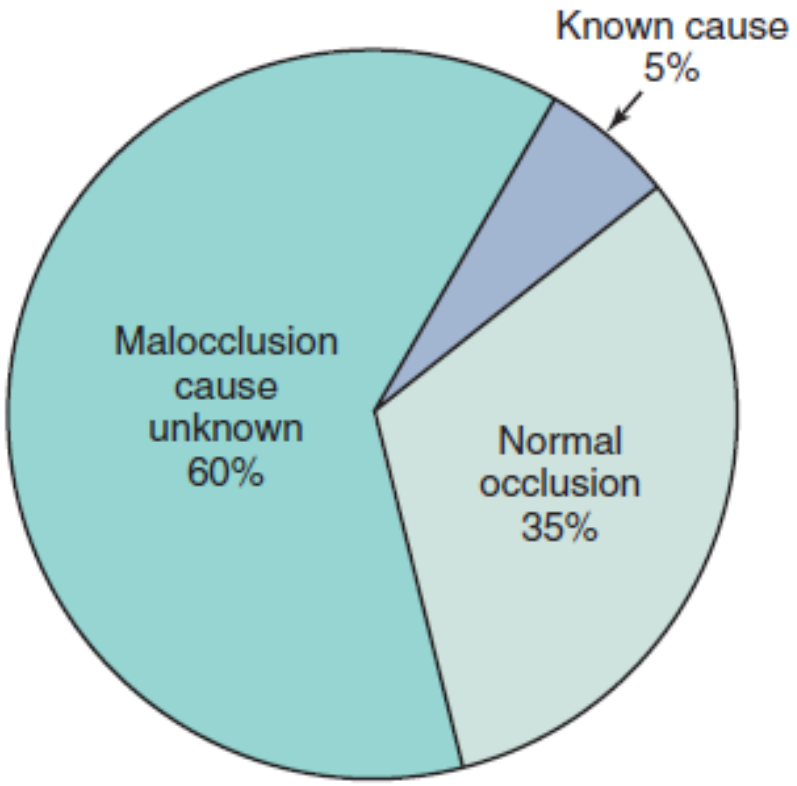
- Bring a new insight for the next session

Malocclusion

- Developmental condition
- Caused by :
  - Not pathologic problem
  - Mod to severe distortion from normal

- Deformity vs Malformation ???
  - Initially formed normally…. Failed to continue normal
  - Congenital

Distribution



Classification

- Specific
  - Embryologic development
  - Fetal and Perinatal
  - Progressive in childhood
  - Adolescence and early Adult
  - Dental Development
- Genetic
- Environmental
  - Equilibrium
  - Mastication
  - Habits (sucking, etc.)
  - Tongue thrusting
  - Respiratory pattern

Disturbances in Embryologic Development

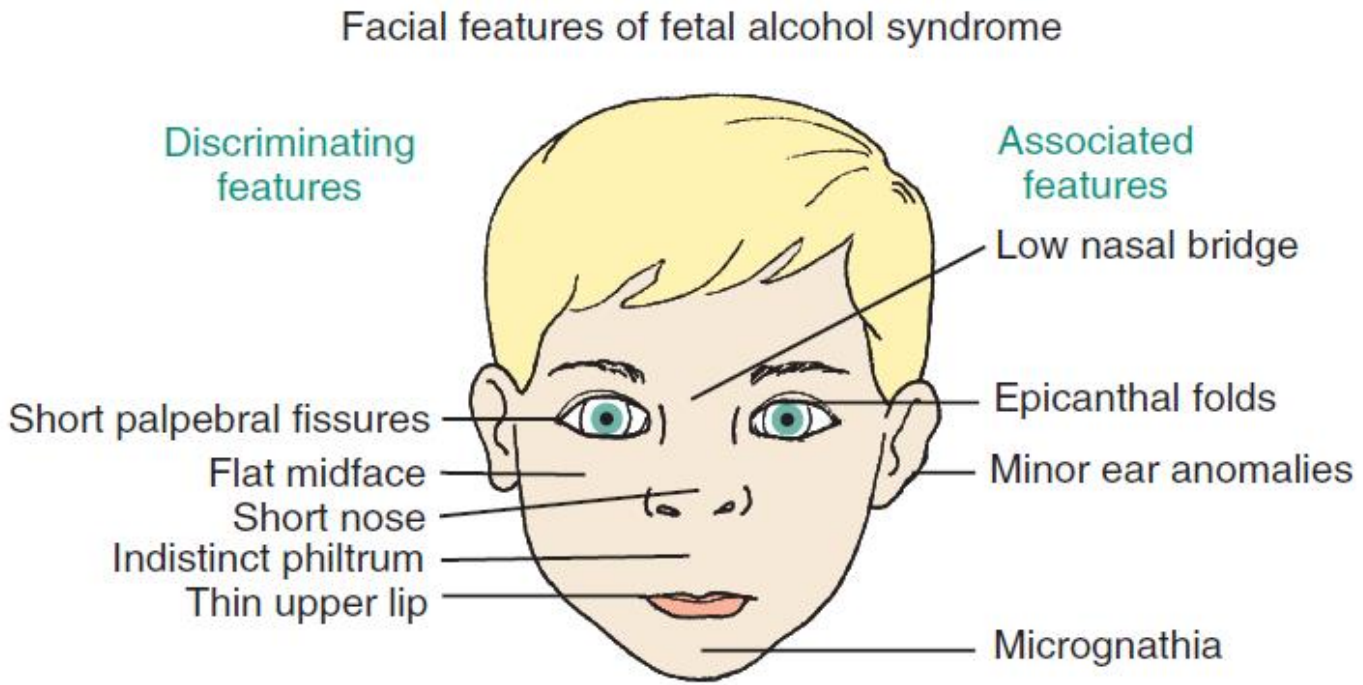
- Usually result in death
- 20%
- Teratogen
  - Low level….specific defect
  - High dose….lethal

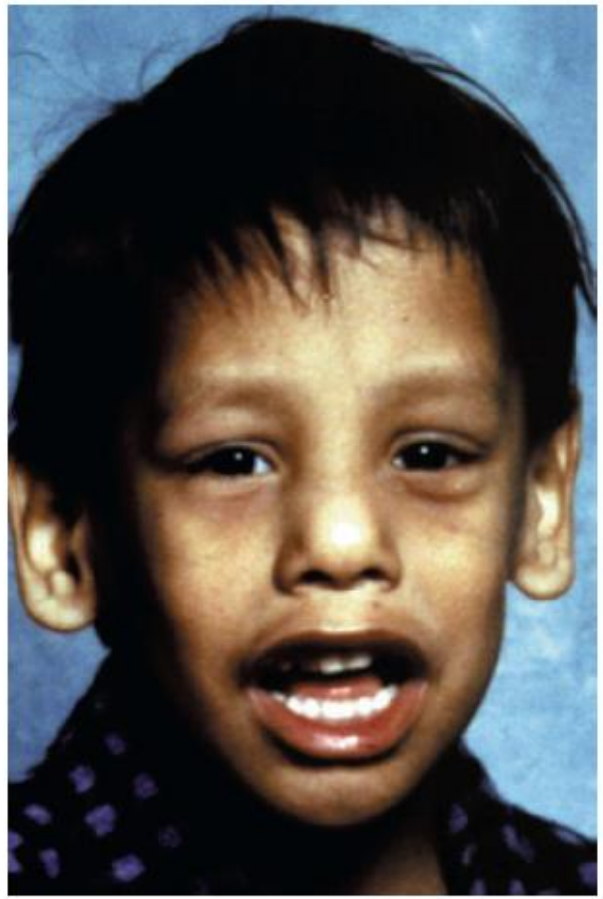
teratogens

TABLE 5.1 Teratogens Affecting Dentofacial Development

Teratogens	Effect
Aminopterin	Anencephaly
Aspirin	Cleft lip and palate
Cigarette smoke (hypoxia)	Cleft lip and palate
Cytomegalovirus	Microcephaly, hydrocephaly, microphthalmia
Dilantin	Cleft lip and palate
Ethyl alcohol	Central midface deficiency
6-Mercaptopurine	Cleft palate
13-*cis* Retinoic acid (Accutane)	Similar to craniofacial microsomia and Treacher Collins syndrome
Rubella virus	Microphthalmia, cataracts, deafness
Thalidomide	Malformations similar to craniofacial microsomia, Treacher Collins syndrome
Toxoplasma	Microcephaly, hydrocephaly, microphthalmia
X-radiation	Microcephaly
Valium	Similar to craniofacial microsomia and Treacher Collins syndrome
Vitamin D excess	Premature suture closure
Zika virus	Microcephaly, brain damage

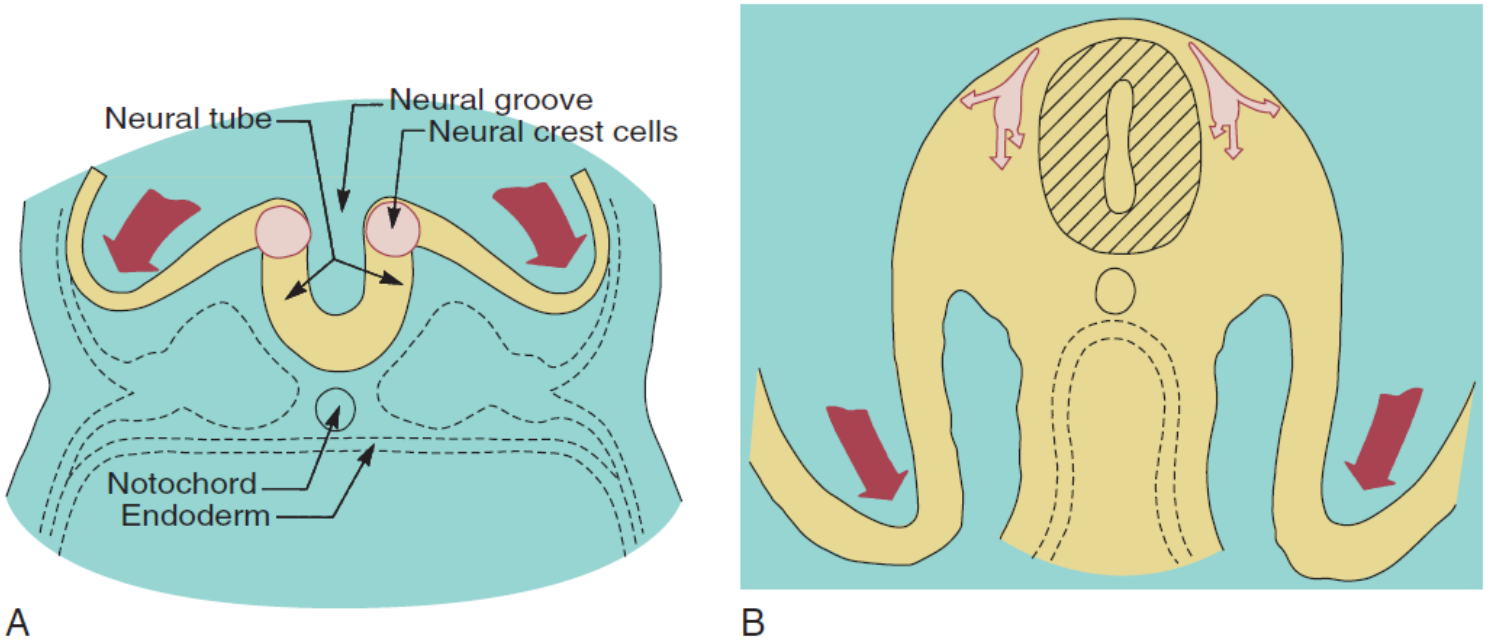
FAS





• **Fig. 5.3** The characteristic facial appearance of fetal alcohol syndrome (FAS), caused by exposure to very high blood alcohol levels during the first trimester of pregnancy.

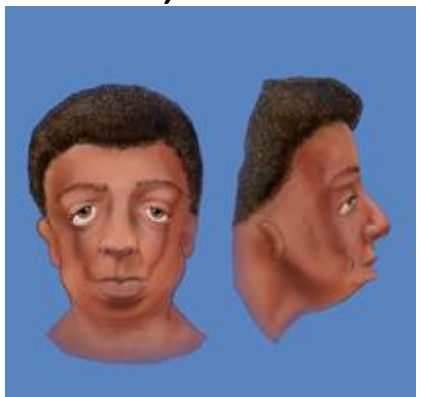
Neural crest

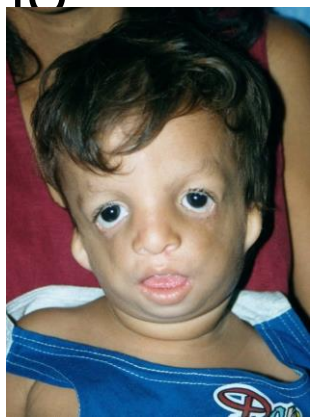


• **Fig. 5.4** Diagrammatic lateral sections of embryos at 20 and 24 days, showing formation of the neural folds, neural groove, and neural crest. (A) At 20 days, neural crest cells *(pink)* can be identified at the lips of the deepening neural groove, forerunner of the central nervous system. (B) At 24 days, the neural crest cells have separated from the neural tube and are beginning their extensive migration beneath the surface ectoderm. The migration is so extensive and the role of these neural crest cells is so important in formation of structures of the head and face that they can almost be considered a fourth primary germ layer. Later stages in the migration can be seen in Fig. 5.2F–H.

Treacher Collins (Mandibulofacial dysostosis)

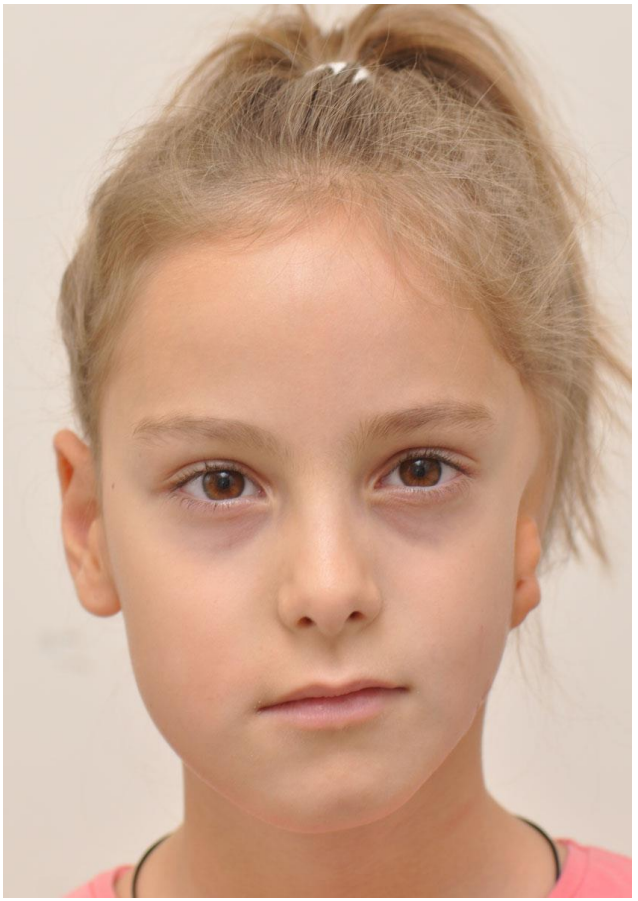
- Autosomal dominant, 40% will have family history, other 60% new mutations
- **TCOF1** gene found on chromosome 5q (**TREACLE gene**)
- Malformation of 1st (& 2nd) branchial arches
- Otologic: Malformed ossicles, auricular deformity, aural atresia, CHL present 30% of time, occasional SNHL
  - 50% will have hearing impairment from EAC and/or middle ear malformations
- Preauricular fistulas, mandibular and malar hypoplasia, antimongoloid palpebral fissures, coloboma of the lower eyelids, may have cleft lip and palate, normal IQ



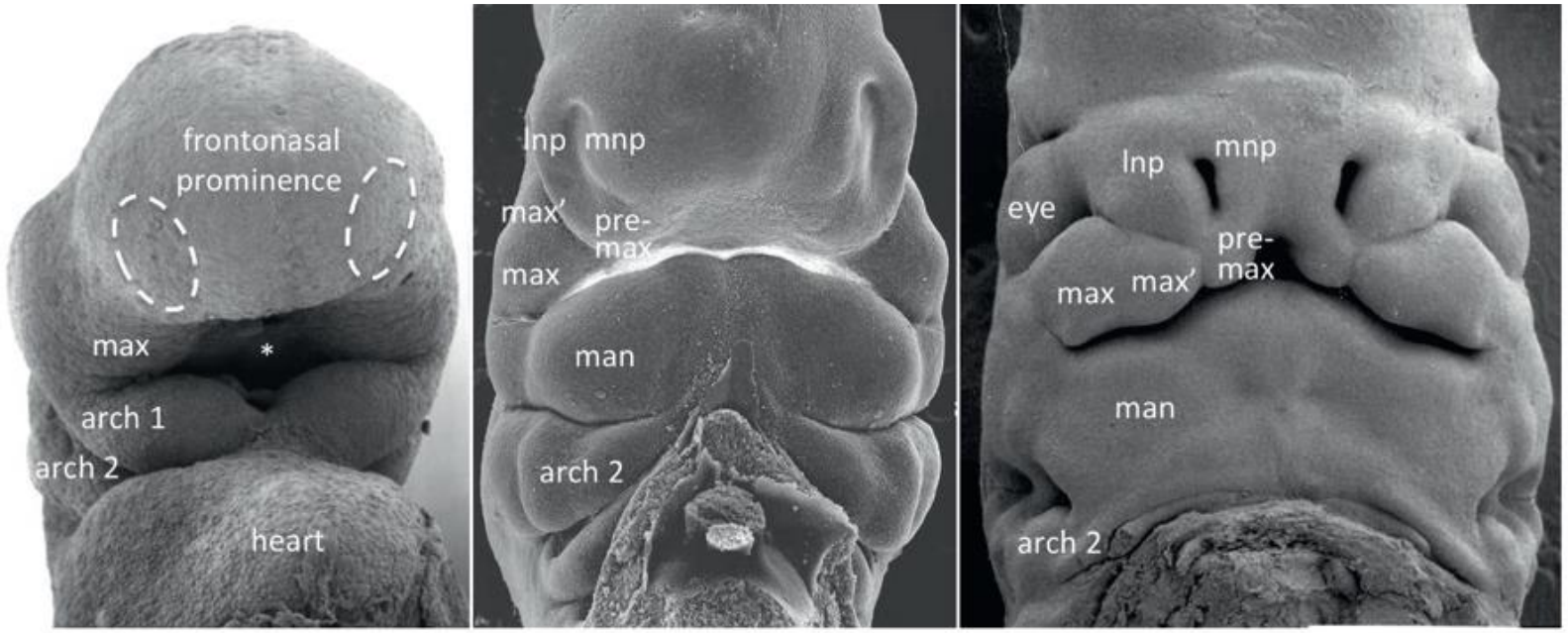


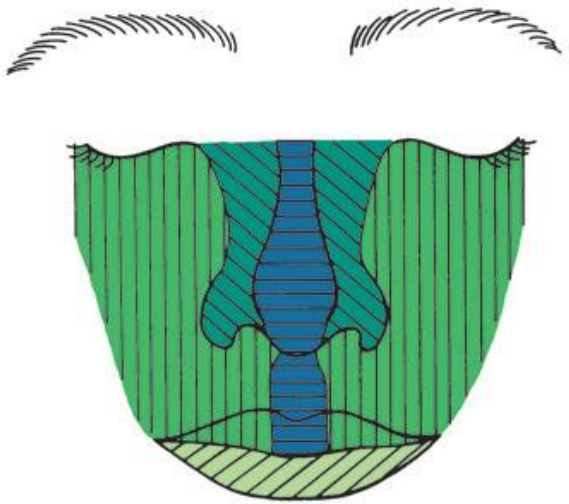
Craniofacial Microsomia (Hemifacial microsomia)

- Teratogen
- 1st pharyngeal arch
  - Lat face, ears, ramus, muscle and facsia



Cleft


frontonasal prominence
max
*
arch 1
arch 2
heart
lnp
mnp
max'
pre-max
max
man
arch 2
lnp
mnp
eye
pre-max
max
max'
man
arch 2

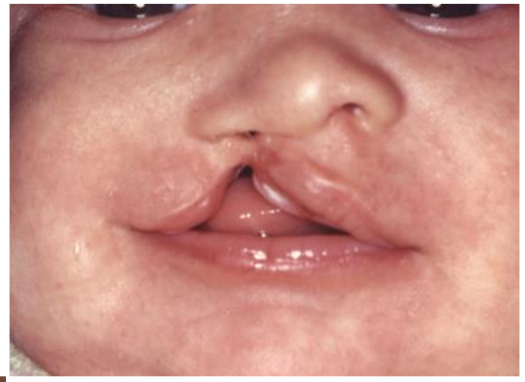


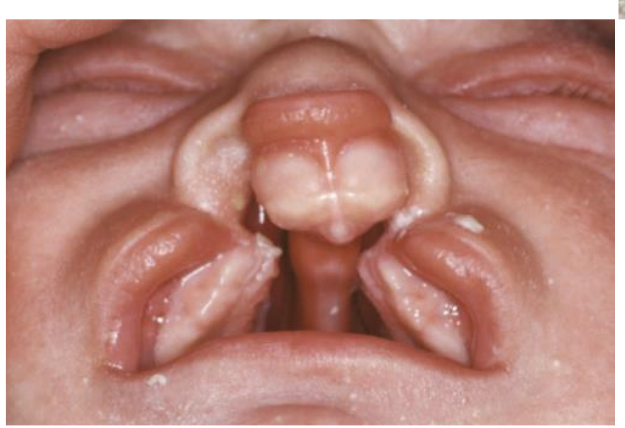
Cleft lip & palate Characteristics

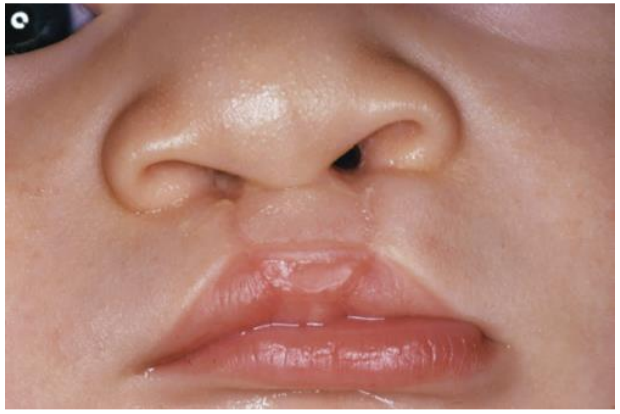
- Stomodeum: MNP+Max+Mand
- Lip &primary palate: LNP+Max+MNP(premax)
- Premax: Ulip Philtrum, alveolar ridge of 1 and 2 (medial)

- 7^{th} week: complete lip closure
- 9^{th} and 10^{th}: sec palate
- 60%: lip+palate
  - Tissue abnormality in pr and sec palate
  - Lip clefting….excessive facial width….pal shelves do not reach

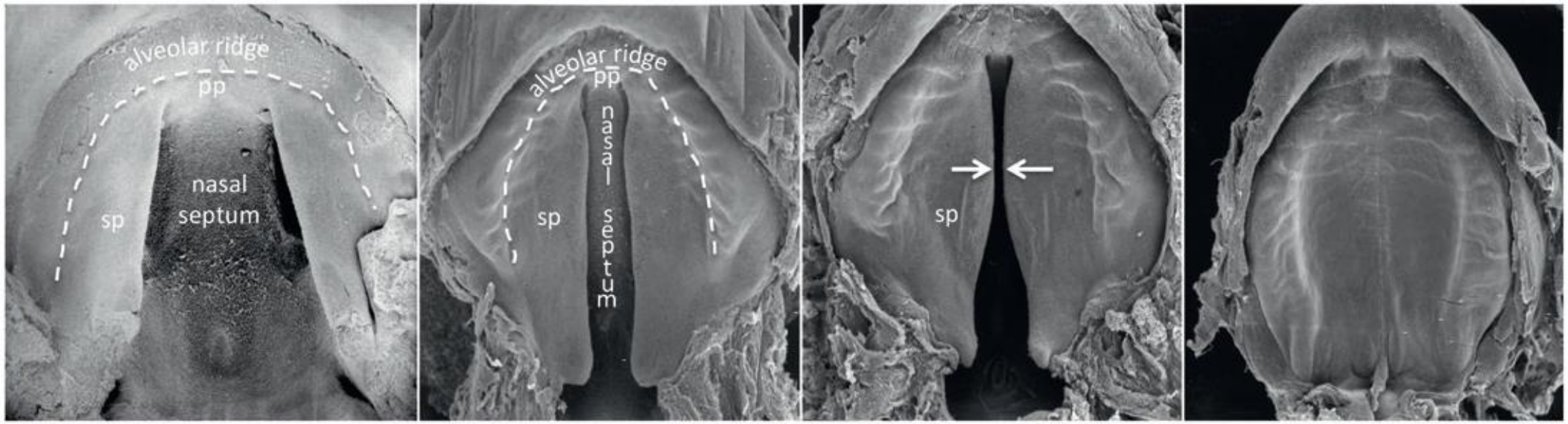
Lip palate Cleft types







Palatal fusion



Facial clefts

- Macrostomia: …..+……
- Oblique facial clefts: ……+…….


پیامبر مهربانی‌ها (ص)
از یکدیگر جدا نشوید و به هم پشت نکنید و به هم
السنن الکبری، ح ۲۱۰۶۰
دشمنی و حسادت نورزید و با هم برادر باشید.
hadis_gram

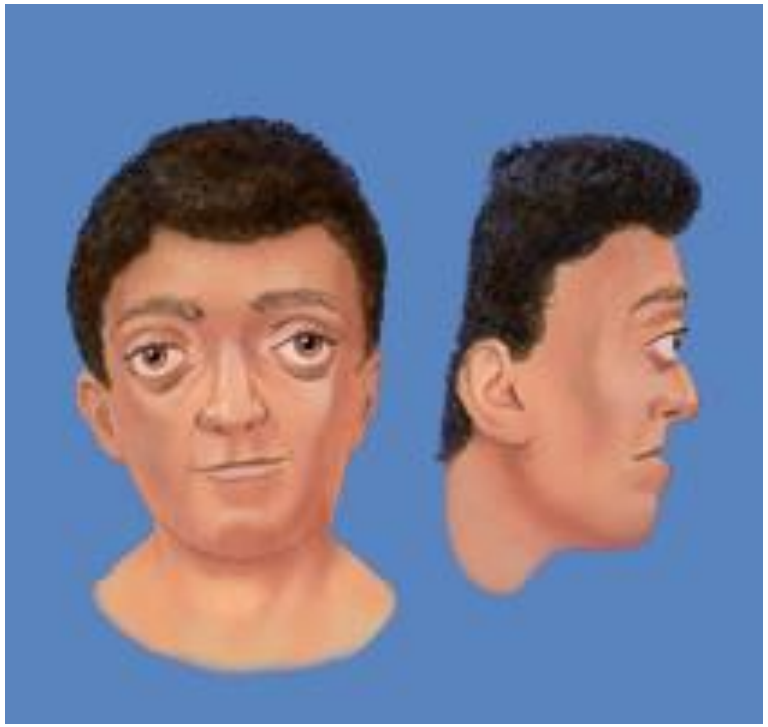
Craniosynostosis

- Fetal period
- Fusion
- Mutation in FGF R genes
- Early surgical release (6-9 ms)

Crouzon's syndrome

- Autosomal dominant, 50% due to spontaneous mutations, complete penetrance, variable expresivity
- Due to mutation of **FGFR-2** (Fibroblast Growth Factor Receptor) gene (**10q26**)
- Common findings:
  - Craniosynostosis (pre-mature fusion of the cranial sutures)
  - Hypertelorism
  - Exophthalmos
  - Midface hypoplasia
  - Mandibular prognathism
  - Parrot-beaked nose
  - **No Syndactyly or cervical fusion**
  - Cognitive function normal to severe mental retardation

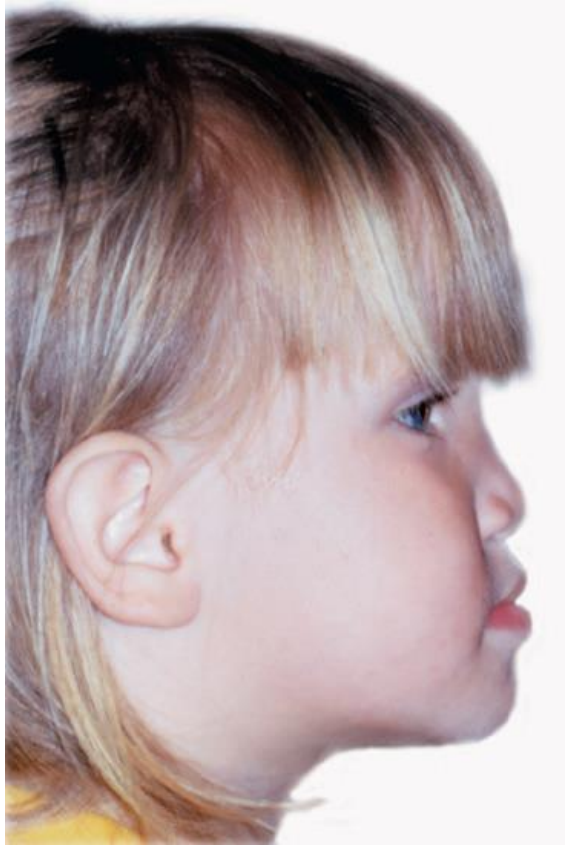
Crouzon Syndrome



- Coronal and sagittal sutures are most commonly involved
- Cloverleaf skull is rare and occurs in the most severely affected individuals.
- Hydrocephalus (progressive in 30%)

Disturbances in fetal and perinatal period

1. Birth trauma to mandible
2. Intrauterine molding



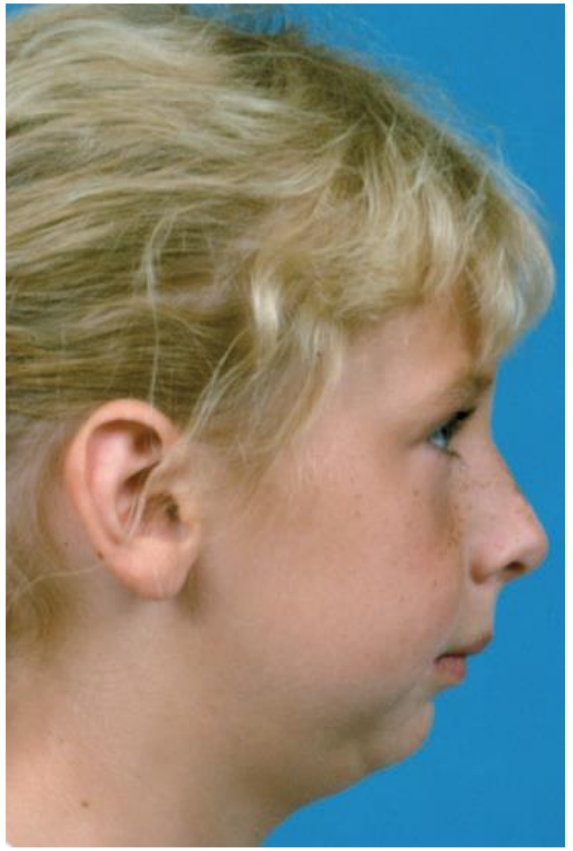
3. Pierre Robin seq

- Decreased amniotic fluid
- Flexed head

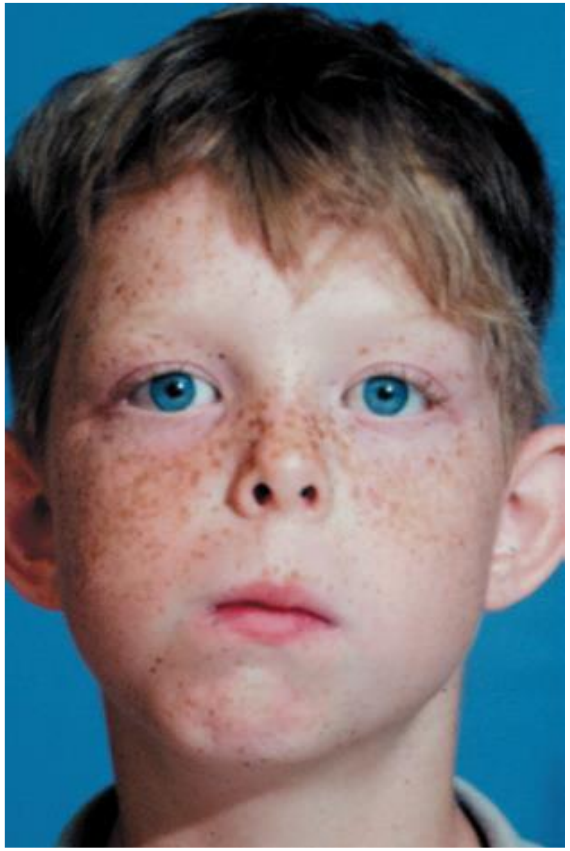
- Cleft
- Tracheostomy
- DO

- Normal growth afterwards
- Not catch-up

- Cartilage
- Stickler syndrome



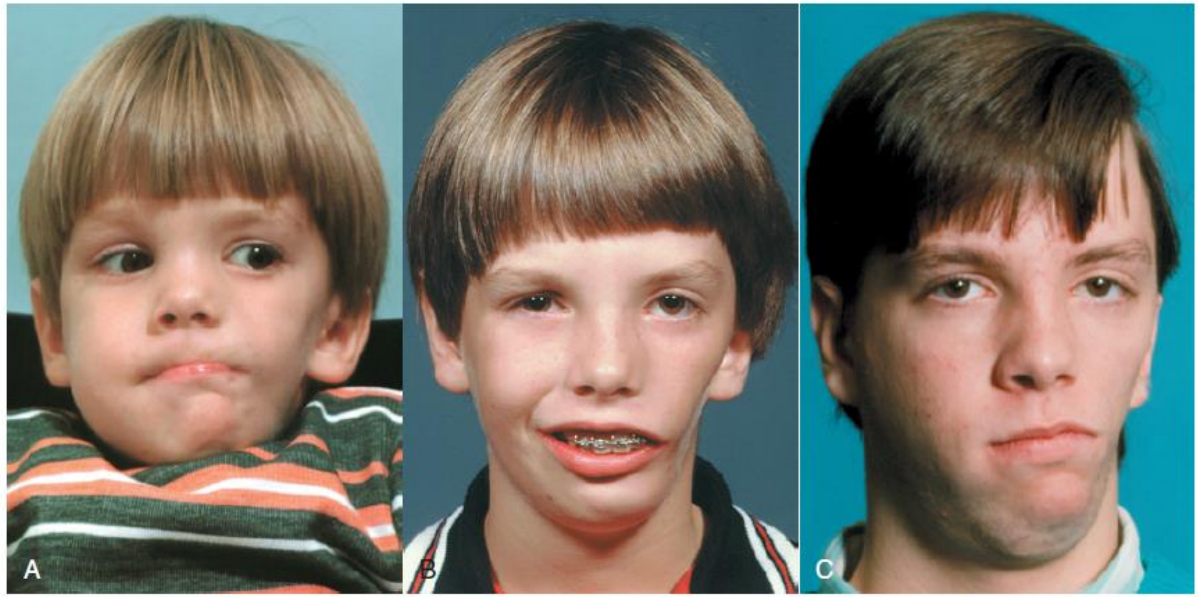
Progressive deformities in childhood



- Fx of jaws
  - Condylar neck
  - 75%
  - Mostly unilat.
  - Mobilization
  - Early Tx
  - DDx:
    - RA
    - Craniofacial microsomia

Progressive deformities in childhood cont'd

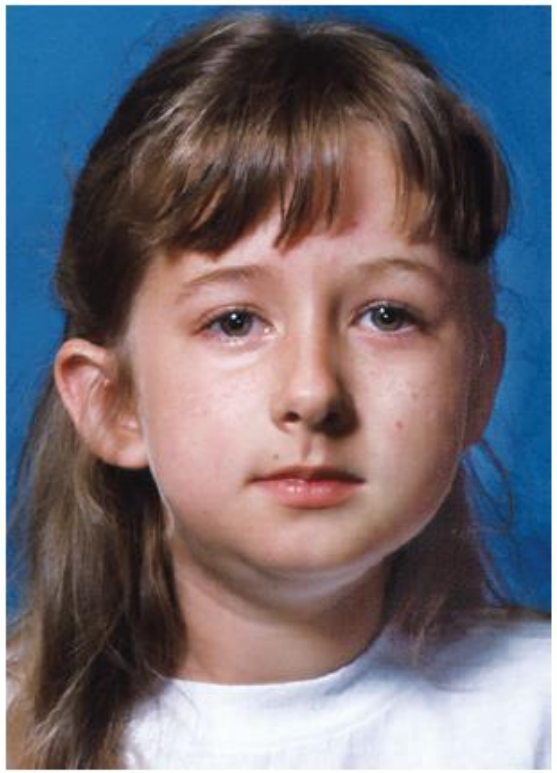
- Muscle dysfunction
- Damage to motor nerve…hard & soft tissue atrophy



• **Fig. 5.16** Facial asymmetry in a boy whose masseter muscle was largely missing on the left side. The muscle is an important part of the total soft tissue matrix; in its absence growth of the mandible in the affected area also is deficient. (A) Age 4. (B) Age 11. (C) Age 17, after surgery to advance the mandible more on the left than the right side. The soft tissue deficiency from the missing musculature on the left side still is evident.

Progressive deformities in childhood cont'd

- Excessive muscle contraction



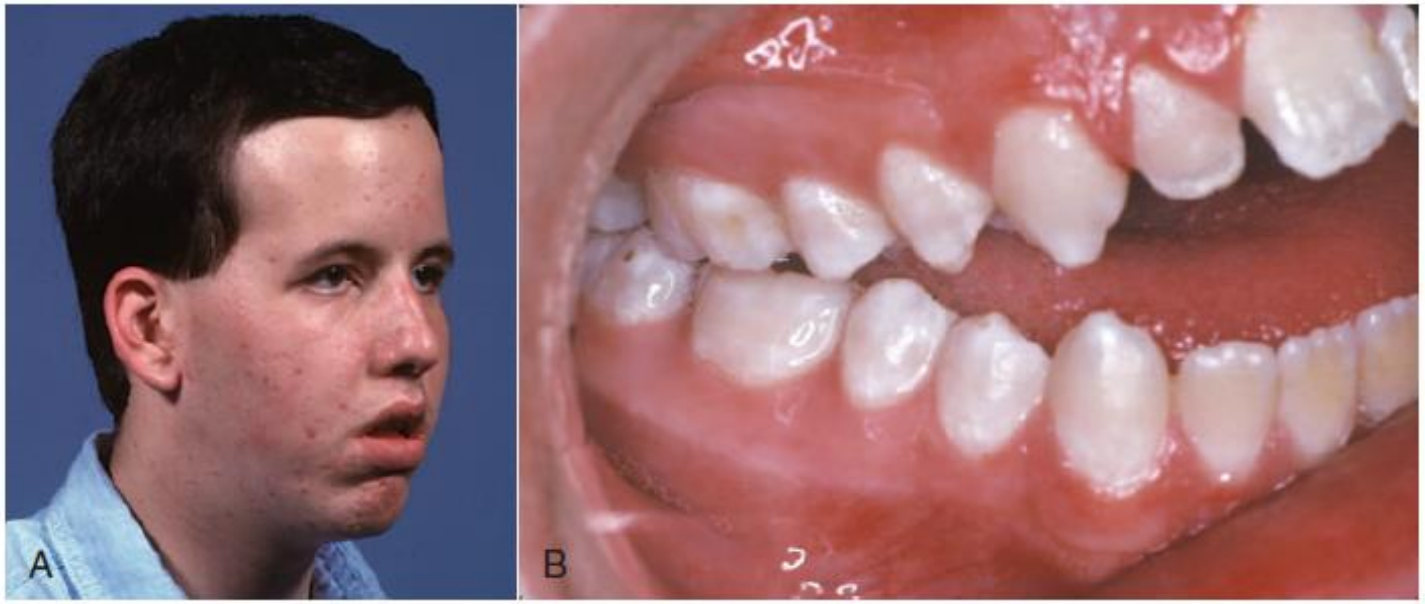
• **Fig. 5.17** Facial asymmetry in a 6-year-old girl with torticollis. Excessive muscle contraction can restrict growth in a way analogous to scarring after an injury. Despite surgical release of the contracted neck muscles at age 1, moderate facial asymmetry developed in this patient, and a second surgical release of the left sternocleidomastoid muscle was performed at age 7. Note that the asymmetry reflects deficient growth of the entire left side of the face, not just the mandible.

Progressive deformities in childhood cont'd

- Muscle dystrophy

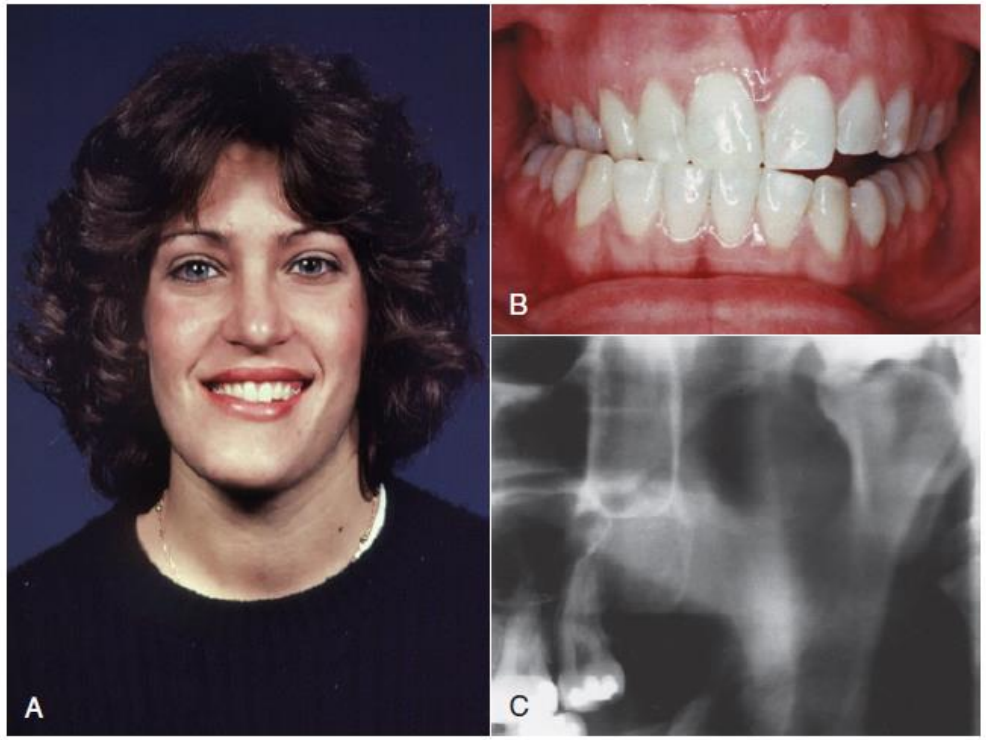
Cerebral pulsy

Muscle weakness



• **Fig. 5.18** (A) Lengthening of the lower face typically occurs in patients with muscle weakness syndromes, as in this 15-year-old boy with muscular dystrophy. (B) Anterior open bite, as in this patient, usually (but not always) accompanies excessive face height in patients with muscular weakness.

Disturbances in adolescence or early adult life



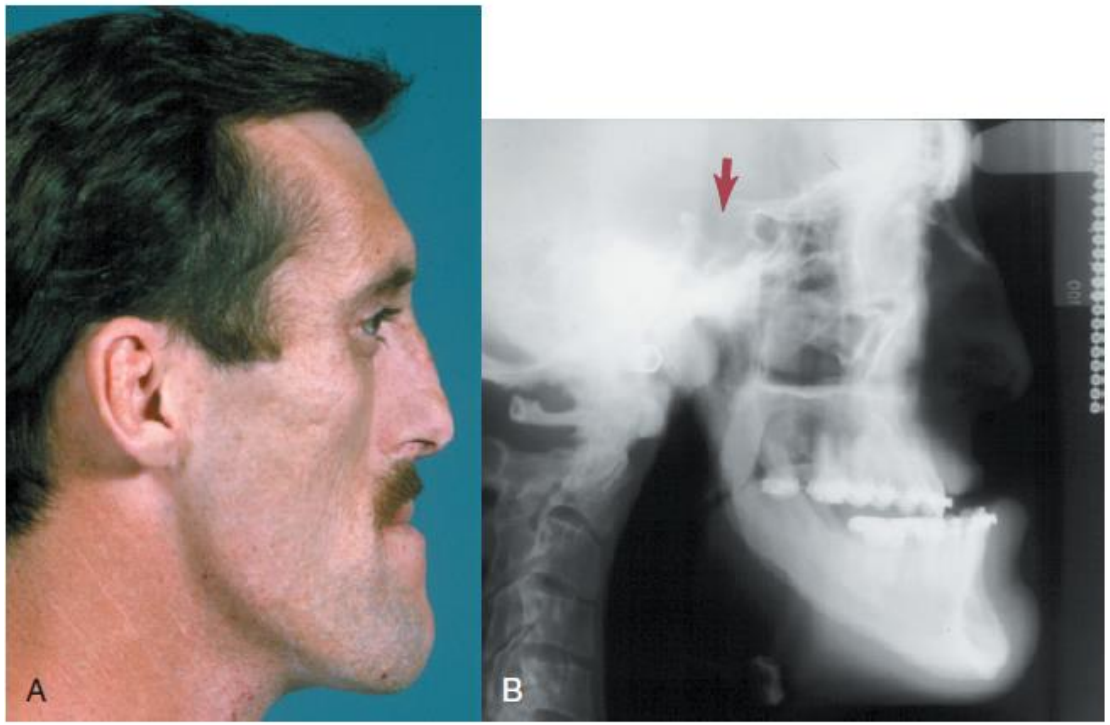
• **Fig. 5.19** (A) Facial asymmetry in this 21-year-old woman developed gradually in her late teens because of excessive growth of the mandible on the left side, after orthodontic treatment for dental crowding during which there was no sign of jaw asymmetry. (B) The dental occlusion shows an open bite on the affected left side, reflecting the vertical component of the excessive growth. (C) Note the grossly enlarged mandibular condyle on the patient's left side. Why this type of excessive but histologically normal growth occurs and why it is seen predominantly in females is unknown.

Hemimandibular Hypertrophy

- Metabolically normal
- 15-20
- Female
- 8-30

- Spontaneous
- Severe …. Condylar removal

Disturbances in adolescence or early adult life cont’d



Acromegaly

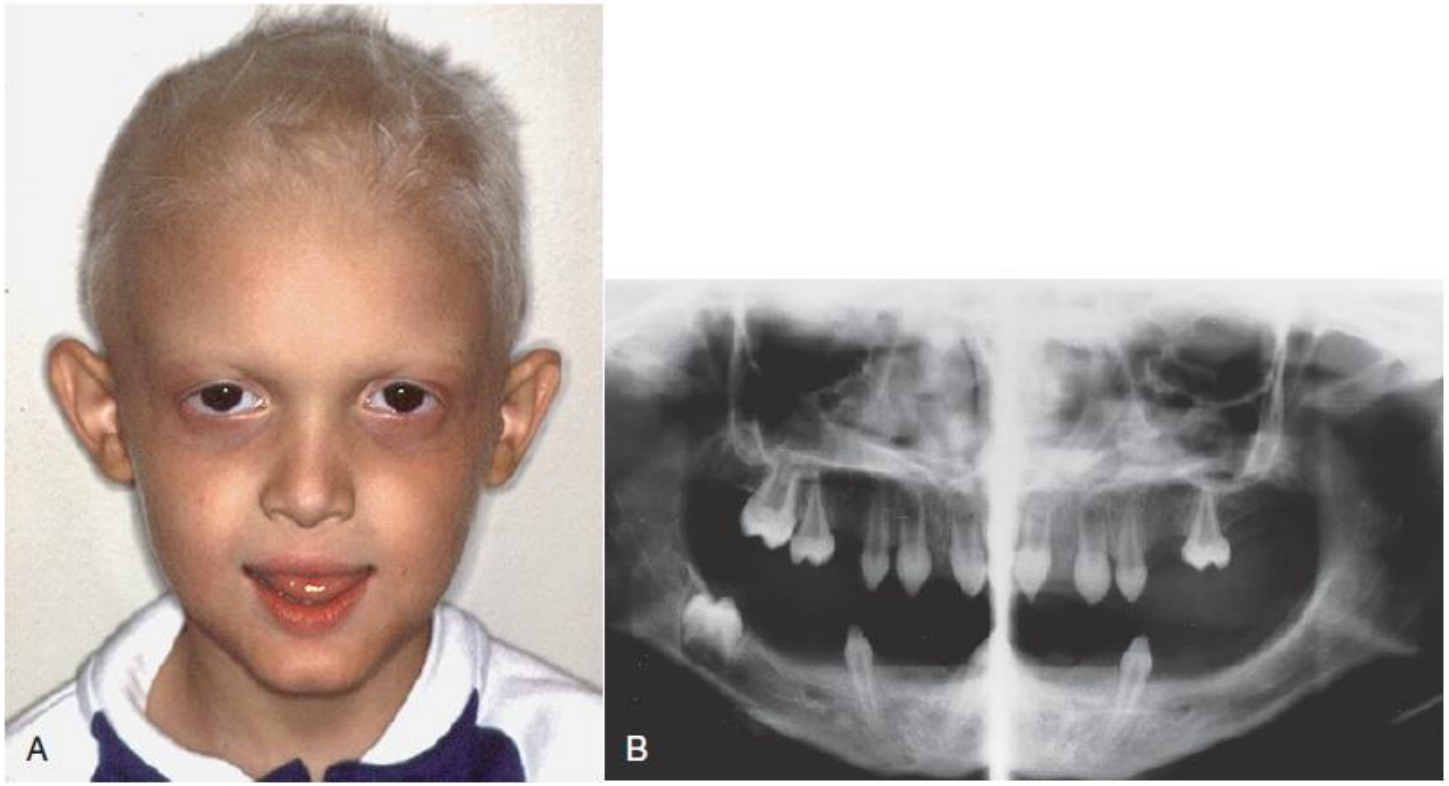
- Ant pituitary gland tumor
- Growth hormone

Disturbances of dental development

Congenitally missing

- Syndromic vs nonsyndromic
  - Anodontia
  - Oligodontia
  - hypodontia

- Ectodermal dysplasia
  - Anodontia/Oligo

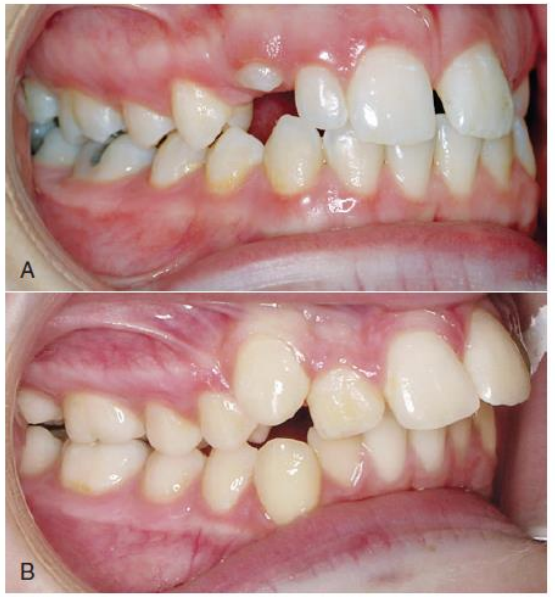


• **Fig. 5.21** (A) A child with ectodermal dysplasia, in addition to the characteristic thin and light-colored hair, is likely to have an overclosed appearance because of lack of development of the alveolar processes. (B) Panoramic radiograph of the same boy, showing the multiple missing teeth. When this many teeth are congenitally missing, ectodermal dysplasia is the most likely cause.

Disturbances of dental development cont'd

Malformed

- Morpho/histodifferentiation
- 5% TSD
- Lateral incisors



• **Fig. 5.22** Disproportionately small (A) or large (B) maxillary lateral incisors are relatively common. This creates a tooth-size discrepancy that makes normal alignment and occlusion almost impossible. It is easier to build up small laterals than reduce the size of large ones, because dentin is likely to be exposed interproximally after more than 1 to 2 mm in width reduction.

Supernumerary

- Initiation/ proliferation

- Cleidocranial dysplasia

Disturbances of dental development cont’d

Traumatic displacement of teeth

- Crown formation…Enamel
- Crown completed….short root/dilaceration

- immediate

Reference

Proffit, 6th edition, Ch.5

Thanks for Your Attention

Any Questions?

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www.drkarandish.ir