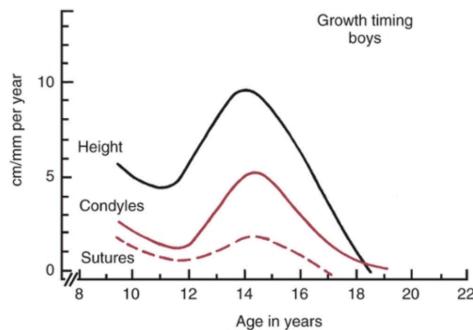


- Factors influencing the timepoint of puberty:

- Genetic / nationality / ethnicity: E.g. Dutch boys at age 10y are taller than American boys.
- Environmental.
- Cultural: Children in cities mature earlier than in rural villages (esp. in less developed countries).
- Season: Growth is faster in spring and summer than in fall and winter.
- Girls: Onset of menstruation requires a certain amount of body fat:
→ slender girls / athletes = later / interruption of the menstruation.

- Growth of jaws correlates with the growth in height at puberty:

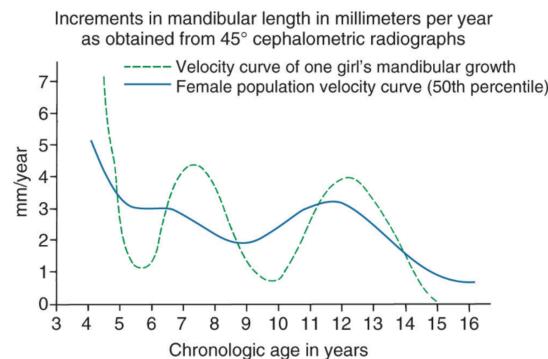
Adolescent growth spurt in length of the mn and to a smaller extent in the mx → Face becomes less convex.



On average, the adolescent spurt in growth of the jaws occurs at about the same time as the spurt in height, but it must be remembered that there is considerable individual variation.

- Girls:

- A *juvenile* acceleration in jaw growth take place 1-2 y before the *adolescent* growth spurt for some girls.
- The adolescent growth spurt precedes the final transition of the dentition in early maturing girls.
- Growth ends when premolars & molars erupt.



Longitudinal data for increase in length of the mandible in one girl, taken from the Burlington growth study in Canada, demonstrates an acceleration of growth at about 8 years of age (juvenile acceleration) that is about equal in intensity to the pubertal acceleration between ages 11 and 14. Changes of this type in the pattern of growth for individuals tend to be smoothed out when cross-sectional or group average data are studied.

- Boys:

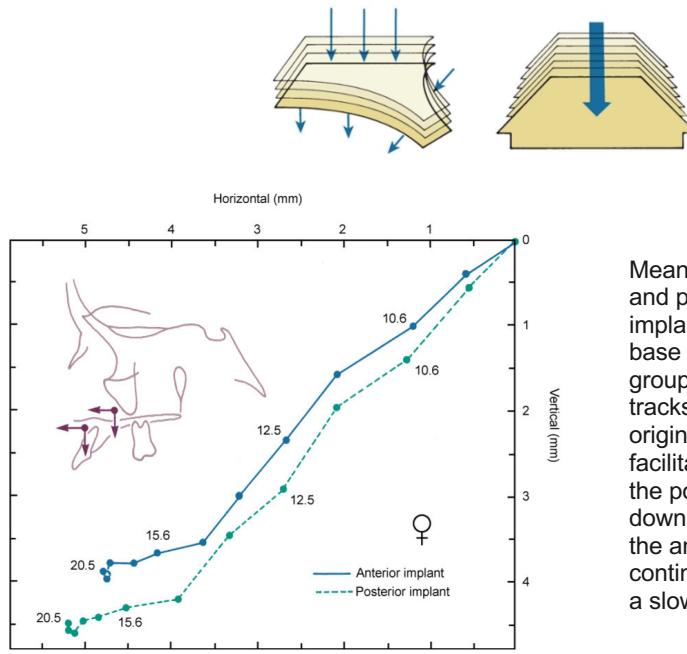
- If a juvenile growth spurt of the jaw occurs, it is mostly less intense than the growth acceleration at puberty.
- A considerable amount of physical growth remains in the permanent dentition in slowly maturing boys.

- **Adrenarche:** = Activation of the adrenal gland to produce sex hormones at about age 6y.

- The intensity of the adrenarche seems to be related to the juvenile acceleration in growth.
- Critical level of sex hormones is reached at age 10y → initiation of sexual attraction.

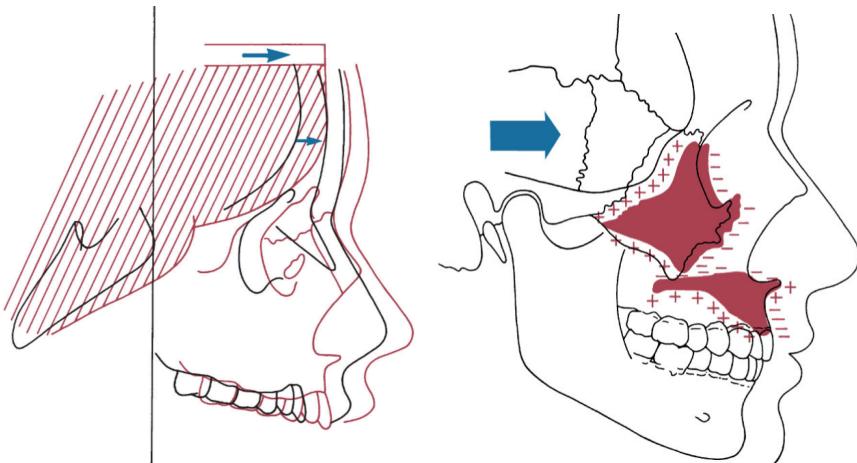
Growth of the nasofacial complex

- Passive displacement by growth of the cranial base which pushes the maxilla forward until age 7y. (synchondrosis closed)
 - Active growth of the maxillary structures and the nose.
 - Maxilla grows downward & forward as bone is added in the tuberosity area posteriorly and at the posterior and superior sutures, but the anterior surfaces are resorbed at the same time.
→ The amount of forward movement of the anterior surfaces is 25% less than the amount of displacement of the maxilla and the mx teeth.
- Exception = palatal vault: Bone is added on the caudal part and resorbed from cranial at the floor of the nose.



Mean growth tracks of anterior and posterior maxillary implants relative to the cranial base and its perpendicular, in a group of Danish girls. The two tracks are shown with their origins superimposed to facilitate comparison. Note that the posterior implant moves down and forward more than the anterior one, with growth continuing into the late teens at a slow rate.

- Nose:
 - Grows ~25% faster than the rest of the face during the adolescent growth spurt.
 - Increase in size of the cartilaginous nasal septum + proliferation of the lateral cartilages alter the shape and overall size (growth of the nasal bone is completed at age 10 y)
 - Continues to grow after the mx and mn have stopped:
→ Apparent prominence of the jaws in relation to the midface is reduced.
- *Björk + Skieller, 1972:*
 - Mx displacement in anterior-caudal direction is normally accompanied by rotation (in general in anterior direction).
 - The inclination of the nasal floor and the orbit to the cranial base remains unchanged, because of compensatory, differential resorption.
 - Mx rotations is mostly in the same direction like the mn rotation (anterior, 1°/y), but only about 1/3 of the extent.
- *Björk + Skieller, 1977:*
 - Mx growth in the sutura mediana posterior > anterior.
 - Age 4y - adulthood: $\Delta +6.9 \text{ mm}$ (5.5.-8.2 mm)
 - Age 6/7y adulthood: $\Delta +4.8 \text{ mm}$
 - Increase of the intermolar width correlates with the sutural growth.
Increase of the intercanine width is smaller.
 - Sutural width age 6/7y adulthood: $\Delta +4.8 \text{ mm}$
 - Intermolarwidth age 6/7y adulthood: $\Delta +3.1 \text{ mm}$
 - Intercanine width age 6/7y adulthood: $\Delta +1.1 \text{ mm}$. (age 4y - adulthood: $\Delta +3.1 \text{ mm}$)
 - Reduction of the arch perimeter: Total **-2.5 mm**
 - Anterior drift of the teeth: $\Delta -1.5 \text{ mm}$
 - Reduction due to the expansion: $\Delta -1 \text{ mm}$.
 - Incisors are 2.5 mm further forward position, molars 5 mm.



Structures of the nasomaxillary complex are displaced forward as the cranial base lengthens and the anterior lobes of the brain grow in size.

As the maxilla is translated downward and forward, bone is added at the sutures and in the tuberosity area posteriorly, but at the same time, surface modeling removes bone from the anterior surfaces (except for a small area at the anterior nasal spine). For this reason, the amount of forward movement of anterior surfaces is less than the amount of displacement. In the roof of the mouth, however, surface modeling adds bone, whereas bone is resorbed from the floor of the nose. The total downward movement of the palatal vault, therefore, is greater than the amount of displacement.

Mandibular growth

- Riolo 1974: Relative steady growth before puberty:

- Body length (Go-Pg): **2-3 mm**
- Ramus height (Cd-Go): **1-2 mm**

TABLE 4.1 Mandibular Length Changes

Data from Riolo ML, et al. *An Atlas of Craniofacial Growth*. Ann Arbor, MI: University of Michigan Center for Human Growth and Development; 1974.

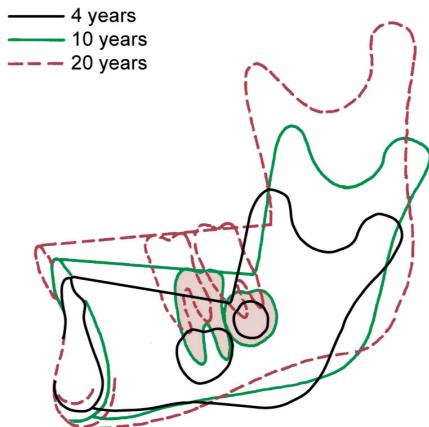
Age	BODY LENGTH INCREASE (mm) (GONION-POGONION)		RAMUS HEIGHT INCREASE (mm) (CONDYLION-GONION)	
	Male	Female	Male	Female
7	2.8	1.7	0.8	1.2
8	1.7	2.5	1.4	1.4
9	1.9	1.1	1.5	0.3
10	2.0	2.5	1.2	0.7
11	2.2	1.7	1.8	0.9
12	1.3	0.8	1.4	2.2
13	2.0	1.8	2.2	0.5
14	2.5	1.1	2.2	1.7
15	1.6	1.1	1.1	2.3
16	2.3	1.0	3.4	1.6

- Gomes et al. 2006: Mandibular growth age 9-18 y:
 - Mn length (Cd-Pg) **4.3 mm/y**
 - Mn ramus (Cd-Go) **3.1 mm/y**
 - Mn body length (Go-Pg) **2.1 mm/y**
- Juvenile and pubertal (adolescent) growth spurt.
- Accentuation of the chin:
 - Area just above between the chin and the dentoalveolar process is resorbed.
 - Forward translation as a part of the overall growth pattern of the mn.
 - Little bone is added.
- Growth changes at the glenoid fossa (Os temporale) influence the translation direction of the mandible:
 - Usually the attachment point moves straight down = no anterior-posterior displacement of the mandible.
 - Occasionally it moves posterior = subtracting the forward projection of the chin.
 - Occasionally the area of temporal bone moves forward and translates the mandible forward.
- Growth in width (1) is completed first, then growth in (2) length and growth in (3) height.
- Growth in length and height of both jaws continue through the period of puberty:
E.g. girls: Mx growth until 14-15 y (2-3 y after 1st menstruation) forward & downward and afterwards tendency for almost straight growth forward.
- Boys & girls: Growth in vertical face height continues longer than growth in length, esp. in the mn.
- Behrents 1984: Growth continues the whole life: V > S > T.
- Increase in face height and concomitant eruption of the teeth continue throughout life, but decrease in adulthood (often not before age 20 y).
- Thilander, 2009: Increase of palatal height
 - 5-16 y: **0.5 mm/y**
 - 16-31 y: **0.1 mm/y**
- Width:
 - Includes the width of dental arches.
 - Completed before the adolescent growth spurt.
 - Exception: Jaws become wider posteriorly when they grow in length.
Mx: Width across the 2nd and 3th molars (if they erupt).
Mn: Molar and bicondylar width.

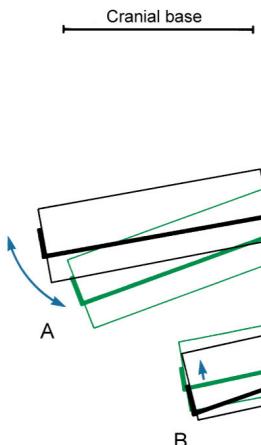
Rotation of the jaws during growth

Condition	Björk	Solow, Houston	Proffit
Posterior growth greater than anterior	Forward rotation		
Anterior growth greater than posterior	Backward rotation		
Rotation of mandibular core relative to cranial base	Total rotation	True rotation	Internal rotation
Rotation of mandibular plane relative to cranial base	Matrix rotation	Apparent rotation	Total rotation
Rotation of mandibular plane relative to core of mandible	Intramatrix rotation	Angular modeling of lower border	External rotation

Proffit: Total rotation = internal rotation – external rotation.
Björk: Matrix rotation = total rotation – intramatrix rotation.
Solow: Apparent rotation = true rotation – angular modeling of lower border.

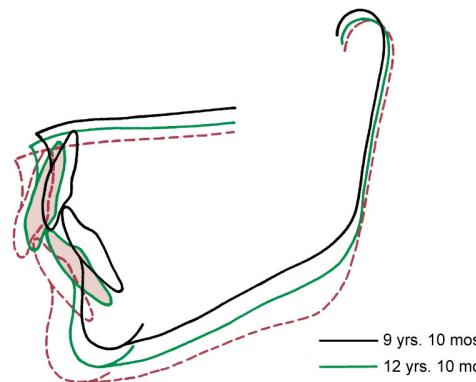
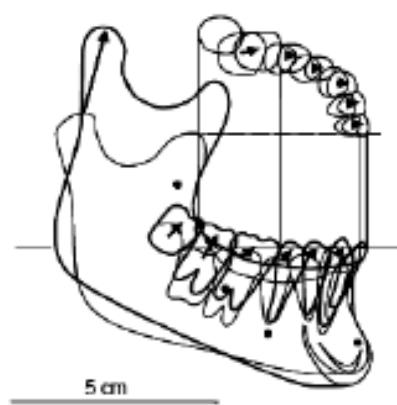
- **Internal rotation:** = Rotation that occurs within the core of each jaw.
Average 15°, 25% matrix rotation, 75% intramatrix rotation.
 - **External rotation:** = Due to surface changes (remodeling)
 - Internal rotation tends to be masked by surface changes and alterations in the rate of tooth eruption.
- | | |
|--|--|
| Mandible | <ul style="list-style-type: none"> - <u>Forward rotation:</u> <ul style="list-style-type: none"> • Core of the mandible rotates in a way that tends to decrease the mandibular plane angle. Happens for most individuals. • Rotation is either around the condyle or centered within the body of the mandible. • A negative sign is given if there is more growth posterior than anterior. - <u>Backward rotation:</u> <ul style="list-style-type: none"> • Anterior dimensions are lengthened more than the posterior ones. • The chin is brought downward and backward. • Given a positive sign.  <p>— 4 years
— 10 years
- - - 20 years</p> <p>Superimposition on implants for an individual with a normal pattern of growth, showing surface changes in the mandible from ages 4 to 20 years. For this patient, there was a 19-degree internal rotation but only a 3-degree change in the mandibular plane angle. Note how the dramatic remodeling (external rotation) compensates for and conceals the extent of the internal rotation</p> |
| <ul style="list-style-type: none"> - Internal rotation has 2 components: <ol style="list-style-type: none"> 1. Matrix rotation: (25%) Rotation around the condyle.
(Björk: rotation of the mn plan relative to the cranial base) 2. Intramatrix rotation: (75%) Rotation centered within the body of the mandible. → Can be measured with implants → Björk.
(Björk: rotation of the mn plane relative to the mn core) - Total rotation = matrix rotation + intramatrix rotation
(Total rotation = Rotation of the mn core in relation to the cranial base (which is judged on ceph)). - Average individual with normal vertical face proportions: <ul style="list-style-type: none"> • 15° internal rotation from age 4y to adult life consisted of 25% matrix rotation, 75% intramatrix rotation. | |

- Not expressed in jaw orientation because it is compensated by surface changes.
- Resorption at the posterior part of the lower border of the mandible.
- The anterior part of the lower border of the mn is unchanged or undergoes slight apposition.

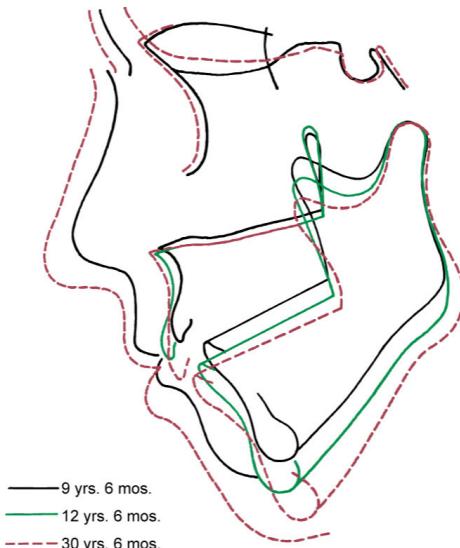


Internal rotation of the mandible (i.e., rotation of the core relative to the cranial base) has two components: **A**, Rotation around the condyle, or matrix rotation. **B**, Rotations centered within the body of the mandible, or intramatrix rotation.

Maxilla	<ul style="list-style-type: none"> - Small and variable degree of internal rotation forward (usual pattern) and backward (also frequent). - Remodeling of the palate. - For most patients, the external rotation is opposite in directions and equal in the magnitude to the internal rotation → net there is no change in jaw orientation. (anterior rotation = resorption at the nasal floor anterior > posterior) <p>Superimposition on implants in the maxilla reveals that this patient experienced a small amount of backward internal rotation of the maxilla (i.e., down anteriorly). A small amount of forward rotation is the more usual pattern, but backward rotation occurs frequently.</p> <ul style="list-style-type: none"> - <i>Björk and Skieller, 1972:</i> <ul style="list-style-type: none"> • Maxilla rotation is normally in the same direction like the mn rotation, but only $\frac{1}{3}$ of the extent. • Apposition and resorption compensate the rotation, so that there is net no change of inclination for the orbit and the nasal floor. - <i>Björk and Skieller, 1977: Changes age 4 y - adulthood:</i> <ul style="list-style-type: none"> • Sutural lowering: 11.2 mm • Apposition at the orbital floor: 6.4 mm • Resorption at the floor of the nose: 4.6 mm • Apposition at the alveolar process: 14.6 mm
Face types: short face type	<ul style="list-style-type: none"> - Excessive forward rotation of the mandible during the growth. (increase in internal rotation + decrease in external compensation) - Low mandibular plane angle, large (? Rather small) gonial angle. - Deep bite. - Crowded incisors through uprighting and lingual positioning. (molars move more mesial than the incisors which are uprighted by the rotation → arch length ↓ = crowding) - Nearly horizontal palatal plane. - 20th century belief: The lingual movement of the incisors is considered more important for the development of a crowding than the mesial drift of the molars. - <i>Björk 1969:</i> <ul style="list-style-type: none"> • Condyle growth in vertical direction → ramus length ↑ or • Middle cranial fossa displaced caudal (bending of the cranial base) → Fossa condylaris displaced caudal. • = Mn lowering > Mn forward displacement But: attachments and ligaments transform the caudal displacement into an anterior rotation.

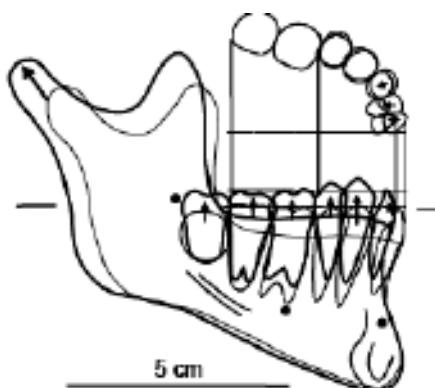
	<ul style="list-style-type: none"> ○ Type 1: center of rotation = condyles (loss of teeth) ○ Type 2: center of rotation = incisors ○ Type 3: center of rotation = premolars (if OJ > 3 mm) ● Incisor are directed forward to compensate the rotation (the incisors inclination within the face stays always \pm the same) <ul style="list-style-type: none"> ○ \rightarrow interincisal angle ↓ ○ \rightarrow anterior crowding ○ Angle incisors - chin = Δ -1 mm/y \rightarrow mentolabial fold more pronounced ● All teeth are anterior directed \rightarrow crowding ● Posterior teeth more upright in relation to the mx plan: <ul style="list-style-type: none"> ○ \rightarrow intermolar angle ↑ ○ \rightarrow interpremolar angle ↑  <p>Cranial base superimposition shows the characteristic pattern of forward mandibular rotation in an individual developing in the “short face” pattern. The forward rotation flattens the mandibular plane and tends to increase overbite.</p>
Face types: long face type	<ul style="list-style-type: none"> - Zusammenfassung der Wachstumsbereiche (Apposition, Resorption) des Unterkiefers - Anteriore Wachstumsrichtung der Kiefergelenke (<i>Berner Skript</i>) <ul style="list-style-type: none"> ● Ausgeprägte Resorption im Angulusgebiet. ● Ausgeprägte Apposition unter dem Kinn. ● Keine Resorption am vorderen Ramusrand. ● Wenig Apposition am hinteren Ramusrand. 

- $= M_n$ displacement in anterior direction > vertical displacement
But: attachments and ligaments transform anterior displacement into a posterior rotation
 - Type 1: center of rotation = condyles (raise of the bite, tooth eruption)
 - Type 2: center of rotation = most distal occluding teeth
- Incisor are more upright positioned to compensate the alveolar prognathisme: \rightarrow interincisal angle \uparrow .
- Lateral teeth are less distal oriented than the anterior teeth \rightarrow crowding.
- Open bite \rightarrow Lip closure difficult.
- Posterior teeth more anterior inclined in relation to the mx plan:
 - \rightarrow intermolar angle \downarrow
 - \rightarrow interpremolar angle \downarrow



The pattern of jaw rotation in an individual with the "long face" pattern of growth (cranial base superimposition). As the mandible rotates backward, anterior face height increases, there is a tendency toward anterior open bite, and the incisors are thrust forward relative to the mandible.

- Zusammenfassung der Wachstumsbereiche (Apposition, Resorption) des Unterkiefers
Posteriore Wachstumsrichtung der Kiefergelenke (*Berner Skript*)
 - Unbedeutende Veränderungen am unteren Corpusrand
 - Ausgeprägte Apposition am hinteren Ramusrand
 - Ausgeprägte Resorption am vorderen Ramusrand



Interaction between jaw rotation and tooth eruption

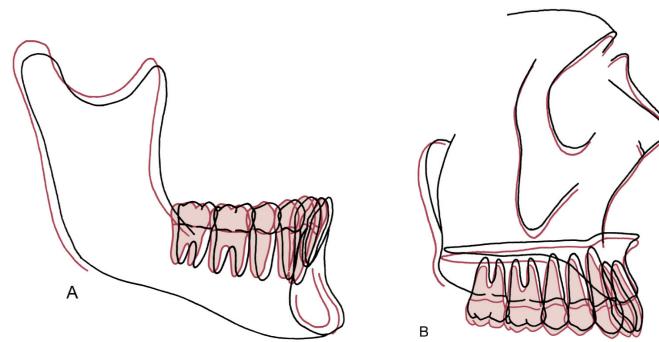
- Rotational pattern of jaw growth influences the magnitude and direction of tooth eruption as well as the ultimate anterior-posterior position of the incisor teeth.
- Maxilla:
Normal tooth eruption is downward and forward.
Movement of the teeth in relation to the cranial base as a combination of translocation: the tooth moves along the jaw in which it is embedded + true eruption (movement of the tooth within its jaw).
 - Forward rotation:
Incisors are tipped forward.
 - Backward rotation:
Incisors are uprighted and their prominence decreased.

	<p>- Mandible: Normal eruption path is upward and forward.</p> <ul style="list-style-type: none"> o Anterior rotation: Incisors are directed more posteriorly and uprighted: → Molars migrate further mesial than the incisors → reduction of arch length = risk for crowding (risk for crowding is more pronounced in the mn, because the internal rotation is more pronounced than in the mx) o Posterior rotation: Incisors are carried forward → dental protrusion. Anterior open bite, if the incisors do no erupt for an extreme distance. <p>- 20th century: Lingual movement of the incisors is considered more important to develop a crowding than arch length reduction due to mesial drift of the molars.</p>
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Maturational and aging changes

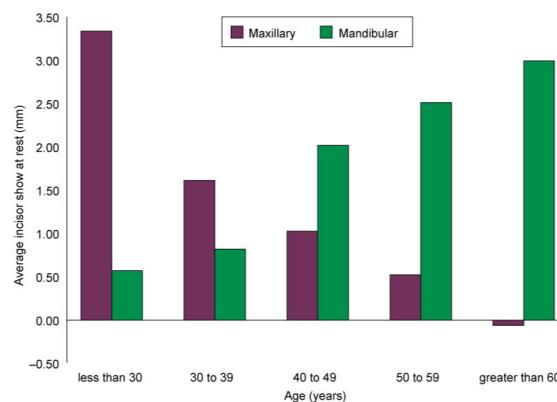
- Facial growth in adults**
- Growth processes decline to basal level after the attainment of sexual maturity and continue to show a cephalocaudal gradient.
 - Size and shape of the craniofacial complex change due to slow continuous growth:
 - Soft tissues > jaw relationship.
 - Vertical > anterior-posterior > width.
 - First pregnancy often produces some jaw growth.
 - Internal rotation and surface changes continue:
 - Males: Net forward rotation, slight decrease in mn plane angle.
 - Females: Tendency towards backward rotation, increase in mandibular plane angle.
 - Occlusal relationships are largely maintained thanks to compensatory changes in the dentition in both genders.
 - Growth pattern associated with the original malocclusion continues.

A, Mean dimensional changes in the mandible for males in adult life. It is apparent that the pattern of juvenile and adolescent growth continues at a slower but ultimately significant rate. **B**, The mean positional changes in the maxilla during adult life, for both sexes combined



- Changes in soft tissue**
- Changes are more pronounced than for the hard tissues.
 - Elongation of the nose: males > females.
 - Augmentation of the soft tissue chin, mentolabial fold more pronounced.
 - Flattening of the lips and coming downward:
 - Adolescence and mid-adulthood:
 - Upper lip lengthening **3.2 mm**
 - Thinner **3.6 mm**
 - Late adulthood:
 - Further **1.4 mm** lengthening and thinning of the upper lip

→ Less exposure of the upper incisors, lower incisors more prominent

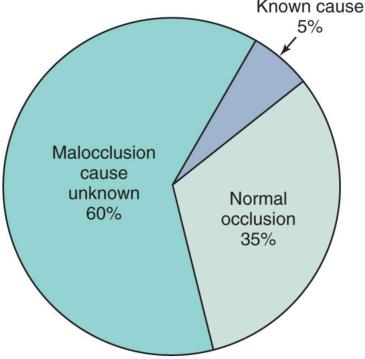


Incisor display at rest as a function of age. With aging, both men and women show less of their upper incisors and more of their lower incisors, so display of upper incisors is a youthful characteristic.

Changes in alignment and occlusion	<p>- Strong tendency for mandibular incisor crowding, 3 theories:</p> <ul style="list-style-type: none"> • Lack of normal interdental attrition in modern diet. <ul style="list-style-type: none"> ○ → Not true. ○ Also crowding when arch length is artificially reduced with premolar extractions. ○ <i>Begg 1954</i>: Interdental attrition is necessary to avoid crowding. • Pressure from the 3rd molars with no room to erupt. <ul style="list-style-type: none"> ○ Difficult to show the force. ○ Also crowding with congenitally missing 3rd molars. ○ Absence of 3rd molars may allow that the whole dentition can shift distally, if late mandibular growth takes place. • Late mandibular growth with rotation. <p>3 ways to react for a patient with a tight anterior occlusion:</p> <ol style="list-style-type: none"> 1. Mandible is displaced distally: <ul style="list-style-type: none"> • Distortion of TMJ function and displacement of the articular disc. • Can happen, but rarely → myofascial pain and dysfunction. 2. Upper incisors flare forward: <ul style="list-style-type: none"> • Maxillary spacing. • Rarely seen. 3. Lower incisors are displaced lingually and become crowded. <ul style="list-style-type: none"> • Usual response. • Occlusal contacts are not even necessary. • Absence of 3rd molars may allow that the whole dentition can shift distal and the crowding is less severe. • If a backward rotation of the mn happens, increasing pressure by the lips put the incisors lingually.
Aging changes in teeth and supporting structures	<ul style="list-style-type: none"> - Pulp chamber becomes smaller: About ½ of original size in the late teens until total obliteration in old age. - Greater exposure of the tooth outside its investing soft tissues as a result of the continuous eruption, not because of downward migration of the gingiva. Accentuated by periodontal diseases. When vertical growth ceases to minimal levels, the gingival attachment should remain at about the same level almost indefinitely. - Occlusal and interproximal wear. Wear facets mostly indicate bruxism nowadays, don't come from the (soft) food anymore.

Proffit Chapter 5:

The Etiology of Orthodontic Problems

Specific causes of malocclusion									
Malocclusion	<ul style="list-style-type: none"> - Development condition. Interaction among multiple factors: specific causes, hereditary influences, environmental influences. - Mostly not due to one pathologic process. - Deformity: = issues initially formed normally and then failed to continue normal development. - Malformation: = Tissues did not form normally from the beginning. - Population: <ul style="list-style-type: none"> • 35% normal occlusion • 60 Malocclusion with cause unknown • 5% Malocclusion with known cause  <table border="1"> <thead> <tr> <th>Cause Category</th> <th>Percentage</th> </tr> </thead> <tbody> <tr> <td>Malocclusion cause unknown</td> <td>60%</td> </tr> <tr> <td>Normal occlusion</td> <td>35%</td> </tr> <tr> <td>Known cause</td> <td>5%</td> </tr> </tbody> </table>	Cause Category	Percentage	Malocclusion cause unknown	60%	Normal occlusion	35%	Known cause	5%
Cause Category	Percentage								
Malocclusion cause unknown	60%								
Normal occlusion	35%								
Known cause	5%								
Stages of craniofacial development	<ol style="list-style-type: none"> 1. 1st - 2nd pregnancy week: Germ layer formation. Initial organization of craniofacial structures. 2. 3rd pw: Neural tube formation. Initial formation of the oropharynx. 3. 3rd - 4th pw: Origins, migrations and interactions of cell populations esp. neural crest cells. 4. 5th pw: Formation of organ systems, esp. the pharyngeal arches and the primary (7th pw) and secondary palates (9-10th pw). 5. 8th pw - birth: Final differentiation of the tissues (skeletal, muscular and nervous elements). <ul style="list-style-type: none"> - Embryonic period: Week 1-8 in utero. - Fetal period: Week 9-40 in utero. 								

Disturbances in embryologic development (embryonic period = 1-8 w in utero)

- Defects usually result in death of the embryo.
- Origin of disturbances = genetic and environmental.
→ **teratogens:** Chemicals that cause a specific defect if they are present at low levels, but have a lethal effect if they are present in higher doses.

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

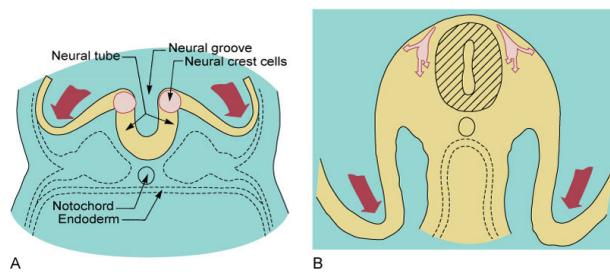
- 5 principal stages in craniofacial development:

Stage	Time in humans	Related syndromes
Germ layer formation and initial organization of structures	Day 17 1-2 w	Fetal alcohol syndrome
Neural tube formation Origin, migration, and interaction of cell populations	Days 18-23 Days 19-28 3 w 3-4 w	Anencephaly Craniofacial microsomia Mandibulofacial dysostosis (Treacher Collins syndrome) Limb abnormalities
Formation of organ systems	Days 28-38 5 w	Cleft lip (6-8 w) and/or Cleft palate (11 w) Other facial clefts
Primary palate 6-8 th week Secondary palate 11 th week	Days 42-55 (Elevation of the palatal shelves at 7.5 w)	Cleft palate
Final differentiation of tissues (skeletal, muscular, nervous elements)	Day 50-birth	Achondroplasia Synostosis syndromes (Crouzons's = Dysostosis craniofazialis, Apert's)

- Tissue origin: (Chiquet)

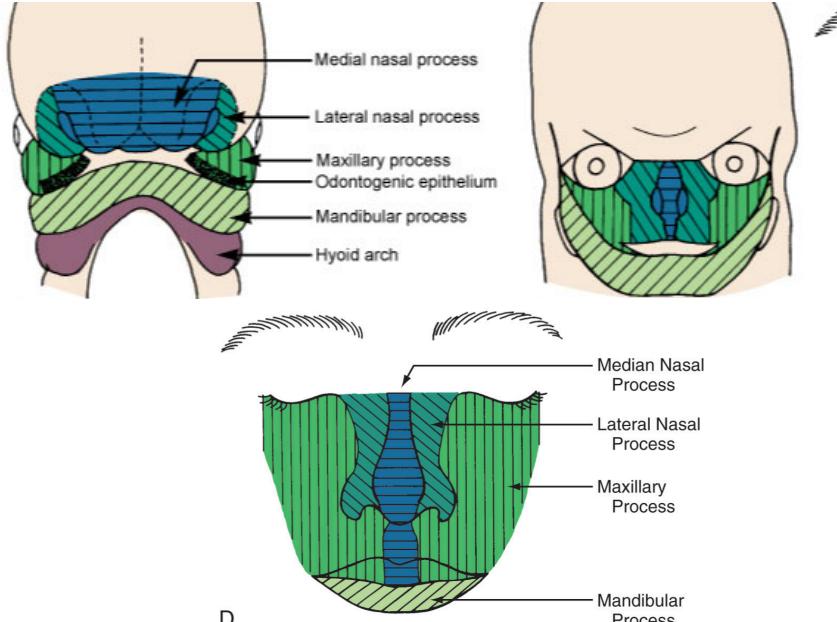
- Neutral crest cells:
 - Bone
 - Cartilage
 - Dermis
 - Paraxial mesoderm:marathon
 - Bloodvessels
 - Muscles
 - Ectoderm:
 - Epidermis
- Neural crest cells:
- Formation at the edge ectoderm / neural tube
 - Are originally ectoderm cells, but they undergo a mesenchymal transformation.
 - Can develop to different cell types in the head are → the whole mesenchymal tissue is of neural crest cell origin.
 - Most structures of the face (skeletal, connective tissues incl. jaws & teeth) are originally derived from migrating neural crest cells.
→ Facial deformities if interferences with the migration of the neural crest cells occur.
 - Many craniofacial anomalies are related to neural crest cell death or migration errors (3-4 w in utero).

- Neural crest cells migrate through the lower (pharyngeal) arches and are important for the formation of the heart and great vessels
→ Explanation for the co-occurrence of craniofacial abnormalities and heart defects e.g. teratology of Fallot.



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 as a fourth primary germ layer.

Syndromes	<ul style="list-style-type: none"> - Definition: <ul style="list-style-type: none"> • $\frac{2}{3}$ of the characteristics must be present. • Sometimes the involved genes are known. • Clear diagnosis is only possible with genetics. - Subphenotypes: = Characteristics are may not fully expressed. (due to the presence of some other genes).
Fetal alcohol syndrome (<i>Germ layer formation and initial organization of structures</i>)	<ul style="list-style-type: none"> - Deficiencies of midline tissue of the neural plate. - Caused by exposure to very high blood alcohol levels during the first trimester of pregnancy. - Delay in dental development and skeletal delay.
	<p style="text-align: center;">Facial features of fetal alcohol syndrome</p>
Zika Virus	<ul style="list-style-type: none"> - Microcephaly and neurologic deficits. - Exposure is harmful at any time of pregnancy.
Stickler syndrome	<ul style="list-style-type: none"> - Defect in cartilage formation → small mandible.
Craniofacial microsomia = hemifacial microsomia (<i>Origin, migration, and interaction of cell populations</i>)	<ul style="list-style-type: none"> - Due to teratogens - Lack of development of the lateral facial areas due to loss of neural crest cells during migration (impeded migration): - External ear is deformed. - Ramus of the mandible and associated soft tissues are deficient or missing. - Defects in the great vessels (teratology of Fallot). - Inner organs are not damaged. - Normal intelligence and life expectation. - Timing for the distraction osteogenesis remains controversial, but social acceptability becomes a factor in the decision. → Intervention to advance the mandible often about 6-8 y of age.
Treacher Collins syndrome = Mandibulofacial dysostosis = Franceschetti (<i>Origin, migration and interaction of cell populations</i>)	<ul style="list-style-type: none"> - Autosomal dominant inherited. - Gene defect: Mutations in a specific gene (TCOF1) leads at least in some cases to the loss of a specific exon → proliferation of neural crest cells is inhibited. - Altered development of cells derived from the neural crest cells. - Characteristics: <ul style="list-style-type: none"> • Lack of mesenchymal tissues. • Underdevelopment of the lateral orbit and zygomatic area. • Ears can be affected. • TMJ developed (= DD to dysostosis otomandibularis).

Cleidocranial dysplasia	<ul style="list-style-type: none"> - Missing clavicles. - Many supernumeraries and unerupted teeth. - Failure of the succedaneous teeth to erupt, because of abnormal resorption of bone and primary teeth. - Nonsuccedaneous tooth eruption is delayed by fibrotic gingiva. - Teeth can sometimes erupt spontaneously when the obstructions are removed. Orthodontic force application is maybe necessary.
Cleft lip and palate <i>(Formation of organ systems)</i> <i>+ only cleft palate: formation of the primary and secondary palate)</i>	<ul style="list-style-type: none"> - Cleft lip: (6-8th w in utero) <ul style="list-style-type: none"> • = Failure of fusion between the median & lateral nasal processes and the maxillary prominence at 6-8th week intrauterine. • Localized lateral to the midline or on both sides. • Likely to have a notch in the alveolar process. • 60% also have a palatal cleft. → May a result of the excessive facial width which follows the CL and places the palatal shelves too far apart to unit in the midline. - Cleft palate (sec.): (11th w in utero) <ul style="list-style-type: none"> • = Incorrect elevation of the palatal shelves related to gene TBX22. • Malformation. - Macrostomia (abnormal wide mouth): <ul style="list-style-type: none"> • = Width of the mouth determined by fusion of the maxillary and mandibular processes at their lateral extent. → Obliquely directed cleft if the fusion failed. - Fusion of the median nasal, lateral nasal and maxillary processes forms the upper lip. - The medial nasal process contributes the central part of the nose and the philtrum of the lip. - The lateral nasal process forms the outer parts of the nose. - The maxillary process forms the bulk of the upper lip and the cheeks. - Possible concurrent tissue abnormality of the lip, primary and 2nd palate. - The time of interference is represented by the various processes which failed to fuse. - Normal lip and primary palate closure are due to the fusion of: <ul style="list-style-type: none"> • Lateral nasal prominence • Maxillary segment • Premaxillary segment (medial nasal prominence) - Premaxillary segment <ul style="list-style-type: none"> • = Portion of the medial nasal prominence that is in closest proximity to the developing oral cavity. • Forms later the philtral part of the upper lip and a portion of the alveolar ridge that contains 1+1 and the medial part of 2+2. (lateral part 2+2 is maxillary tissue origin) 

	<ul style="list-style-type: none"> - Possible teratogens: <ul style="list-style-type: none"> • Aspirin • Cigarette smoke • Dilantin • Folic acid deficit - Timing: <ul style="list-style-type: none"> • Lip closure: 7th week • Closure secondary palate: 9-10th week - Changes on the speech: (<i>Notes</i>) <ul style="list-style-type: none"> • Explosive abgeschwächt: b/p → m, g/k → verschwinden. • Vokale (a,e,i,o,u): Hypernasalität. • Nasenblasen. • ö/ü beeinträchtigt durch die Lippennarben. • Sprache ändert sich meistens zw. 4-5. LJ: Rückverlagerung der Artikulation.
CLP in Switzerland by Joël Beyeler	<ul style="list-style-type: none"> - Formation of the primary palate: <ul style="list-style-type: none"> • Formed by the medial nasal process at 28 d. • Innervation by N. incisivus. (Endbranch of N. nasopalatinus) - Formation of the secondary palate: <ul style="list-style-type: none"> • Formed by elevation of the palatal shelves at 7.5 w. • Apoptosis of cells at the fusion lines and transformation from epithelial into mesenchymal cells. • Fused by a zip mechanism from anterior to posterior. • Involved genes: <ul style="list-style-type: none"> ◦ Msx1: Palatal shelves elongation ◦ TBX22: Lowering of the tongue, palatal shelves elevation ◦ IRF6: Palatal shelves fusion - <u>Etiology of CLP:</u> <ul style="list-style-type: none"> • Environmental factors: <ul style="list-style-type: none"> • Smoking (active or passive) • Hyperthermia • Diabetes Mellitus • Medication (Antiepileptica e.g. Dilantin, Aspirin, cytostatica) • Obesity • ... • Existence of genes or environmental factors do not always in all people lead to CLP, complex interaction. • → Co-Inheritance of a protective genetic background (favorable intrauterine environment). • Genetic factors: <ul style="list-style-type: none"> • Mother with CLP → child: 18-23% • Sibling with CLP → child: 3-8% - <u>Prevalence Switzerland</u> <ul style="list-style-type: none"> • 76'000 newborn per year CH total • 2-2.7% congenital malformations → 6-8% clefts (about 1:500-1000) <ul style="list-style-type: none"> • 41.0% CP (most frequent form in girls) • 14.8% CL • 44.2% CLP (most frequent form in boys) • More frequent in small cantons in Switzerland: → Genetics?! Less mixing? • German part < French part - CL and CP are independent from each other: <ul style="list-style-type: none"> • CL = Fusion defect. • CP = Malformation. - Hypothesis for CP development from the mouse model with BMP7 knockout: <ul style="list-style-type: none"> • No symphysis. • → Lowering of the tongue is inhibited by incorrect attachment of the M. genioglossus. (attaches at the Meckel's cartilage, instead of the symphysis) • → Palatal shelves cannot correctly upright and fuse, because the tongue lies in between.

	<ul style="list-style-type: none"> Possible relation between micrognathia and CLP: Small mandible → tongue cannot be placed anterior. Cave: Early operations can iatrogenic impede further mx growth by scar tissue formation. → Operation protocol and timing must be adapted. → Manipulation of wound-contraction and scar formation (inhibition of myofibroblast contraction). → Tissue engineering: Development of mucosa replacement materials.
Craniosynostosis syndromes <i>(Final differentiation of tissues)</i>	<ul style="list-style-type: none"> Results from the early closure of the sutures (synostosis) between the cranial and facial bones → leads to distortions of the skull. Fusion of sutures which attach the face to the cranial structures behind it can occur with or without fusion of the cranial sutures. Etiology: <ul style="list-style-type: none"> Possible variety of gene mutations, including fibroblast growth factor receptor genes. May caused by an excess of vitamin D? Best timepoint for surgery to release fused cranial sutures = 6-9 m of age. Crouzon's syndrome = Dysostosis craniofacialis: <ul style="list-style-type: none"> Etiology: Prenatal fusion of the superior and posterior sutures of the maxilla along the wall of the orbit which can extend posteriorly into the cranium. → Maxilla can't translate downward and forward. Linked to a mutation in FGFR2 on chromosome 10. Underdevelopment of the midface and eyes. Hypertelorism (wide separation of the eyes). Eyes that seem to bulge from their sockets. (due to under-development of the orbits)
Dental digital syndromes	<ul style="list-style-type: none"> Check the patient's hand for syndactyly in you suspect a syndrome. (several dental-digital syndromes exist)
Achondroplasia <i>(Final differentiation of tissues)</i>	<ul style="list-style-type: none"> = Deficient growth at the synchondrosis of the cranial base → maxilla is not translated forward to the normal extend → midface deficiency.
Dysostosis oculaauricularis (Goldenhaar Syndrom)	<ul style="list-style-type: none"> Etiology: Intrauterine bleeding → not genetic. Symptoms: <ul style="list-style-type: none"> Atypic mandible. Inner organ involved. Normal intelligence and life expectancy
Ectodermal dysplasia <i>(hydrotic or anhidrotic form)</i>	<ul style="list-style-type: none"> Genetic background Symptoms: (Ectodermal tissues = teeth, hair, skin, nails, sweat glands) <ul style="list-style-type: none"> Anodontia / oligodontia. Thin, sparse hair. Malformations of nails / skin. Absence of sweat glands (anhidrotic form). Overclosed appearance (like extreme deep bite) because the alveolar processes fail to develop.
Trisomie 21 <i>(Downs Syndrom)</i> <i>Doriquetto 2019 and other articles.</i>	<ul style="list-style-type: none"> Mikrodontia. Agenesis. Central third of the face underdeveloped → cl.III. Periodontal problems ↑. Facial and oral muscle weakness → open bite. Tongue appears bigger because of the anterior and rather low position in the mouth. Real macroglossia is rare. Interaction of the underdeveloped mx and the tongue volume closes the respiration tracts: <ul style="list-style-type: none"> → Mouth breathing → Crossbite No evidence for caries risk ↑ Roots: Short, taurodontismus

Growth disturbances in the fetal (9-40 w intrauterine) and perinatal period	
Intrauterine Molding	<ul style="list-style-type: none"> - Pressure against the developing face prenatally can lead to distortion of rapidly growing areas: <ul style="list-style-type: none"> • Arm pressed across the face in utero → maxillary deficit. • Head flexed tightly against the chest in utero → mandible inhibited to grow. - Possibility of normal growth after birth and eventually a complete recovery.
Birth trauma to the mandible	<ul style="list-style-type: none"> - Use of forceps on the head in difficult births can damage the TMJ: → subsequent underdevelopment of the mandible.
Pierre Robin anomaly / sequence	<ul style="list-style-type: none"> = Extreme mandibular deficiency at birth. - Multiple causes can lead to the deformity, not a syndrome with a defined cause. - Possible cause: Head tightly flexed against the chest in utero (related to a decreased volume of amniotic fluid) → mn cannot grow forward. - Often related with CLP: The restriction forces the tongue upwards and prevents normal closure of the palatal shelves. - Birth problems possible at birth due to the reduced volume of the oral cavity. - Early mandibular advancement via distraction osteogenesis has been used recently to provide more space for the airways. - Some children have favorable mandibular growth in childhood, but a smaller mandible usually persists and it does not catch up completely → no catch up growth. - Catch up growth is most likely when the cause was mechanical growth restriction. - $\frac{1}{3}$ of the affected subjects have a defect in cartilage formation = Stickler syndrome

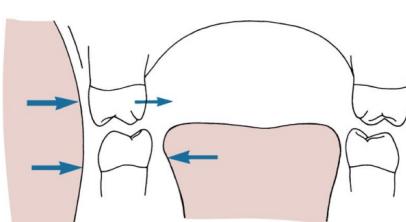
Progressive deformities in childhood	
= Deformities which steadily become worse.	
Childhood fractures of the jaw	<ul style="list-style-type: none"> - 75% of the children with early fractures of the mandibular condylar process have normal mandibular growth afterwards. - Diagnosis is often never made. - Excessive scarring around the TMJ can restrict translation of the condyle. → Asymmetric growth deficiency at the injured side. - Little if any advantage from open surgical reduction of a condylar fracture in children (scarring). Better early mobilization to minimize any restriction of movements → monobloc / functional tx. - Early tx is indicated in case of growth restriction (hybridmonobloc s. later) - Unilateral fracture is more often than bilateral fractures. - Rheumatoid arthritis or a congenital absence of tissue like in craniofacial microsomia can also cause unilateral mandibular deficiency.
Muscle dysfunction	<ul style="list-style-type: none"> - Crucial factors for bone formation: <ul style="list-style-type: none"> ○ Muscular attachments to the bone. ○ Muscles are related to the total of soft tissue matrix which carries the jaws downward and forward. - Dysfunction is mostly caused by damage to the motoric nerves: → underdevelopment of the corresponding part of the face of the affected muscle, deficiency in soft and hard tissues. - Excessive muscle contraction can restrict growth: E.g. torticollis: contraction of M. sternocleidomastoideus. - A decrease in tonic muscle activity allow the mandible to drop downward away from the facial skeleton (e.g. muscular dystrophy) - High risk for recurrence of a malocclusion if it was caused by muscle imbalance. (e.g. open bite caused by weak muscle tonus).
Disturbances arising in adolescence or early adult life	
Hemimandibular hypertrophy (Condylar hyperplasia = old term)	<ul style="list-style-type: none"> - Mostly girls 15-20(30) y old. 85% females, 15% males. - Origin not known. Patients are metabolically normal. - Excessive growth, ø tumor: Normal hard and soft tissue in histologic diagnosis. → Just too much growth = potential to stop growing on its own. - Proliferation of the condylar cartilage (condyle & condylar neck) and mandibular body. The excessive growth occurs in 2 patterns at the condyles: <ul style="list-style-type: none"> ○ Enlargement of the condyle. ○ Lengthening of the condylar neck. (seems to be more likely to stop, but no evidence) - and 2 patterns in terms of: <ul style="list-style-type: none"> ○ Lengthening of the ramus versus ○ Lengthening the body of the mandible - Patients show all possible combinations of excessive horizontal and vertical growth. - Lengthening of the mn body ↑ → Lateral displacement of the center of the chin ↑ - Lengthening of the ramus ↑ → vertical asymmetry ↑ <ul style="list-style-type: none"> ○ Lower position of one side of the chin than the other. ○ Asymmetry at the gonial angles. ○ Canting of the occlusal plane → 2-jaw surgery necessary. - Spontaneous stop of the excessive growth is possible. - Removing the condyle on the affected side stops the excessive growth even though the deviant growth pattern affects the rest of the mn, not just the condyle. - Condylectomy is indicated, if the condyle no longer fits into the condylar fossa. - Condylar shave = Removal of the superior surface of the condylar neck where cellular proliferation occurs. → Can be successful if the excessive growth is an elongation of the condylar neck.
Acromegaly	<ul style="list-style-type: none"> - Caused by an anterior pituitary tumor: → Excessive secretion of growth hormones → excessive growth of the mandible (stops after tumor resection).
Bennett, 2014: Increase of crowding	<ul style="list-style-type: none"> - Maximal increase of crowding with 14-18 y in untreated individuals: <ul style="list-style-type: none"> ○ Parodontal factors ○ Intercanine width ↓ ○ Mn sagittal growth ○ Soft tissue

Disturbances of dental development	
Congenitally missing teeth	<ul style="list-style-type: none"> - Anodontia: Total absence of teeth. Rare. - Oligodontia: Congenital absence of many but not all teeth (minimum 6). Rare. - Hypodontia: Absence of a few teeth, relatively common. - Genetic basis for missing teeth in most instances: <ul style="list-style-type: none"> o Polygenic multifactorial model of etiology o Linked to WNT10A (56% of the cases) or Pax9 in case of severe or familial non syndromic hypodontia - Environmental factors: <ul style="list-style-type: none"> o Trauma o Jaw surgery o Chemotherapy o Radiotherapy o Early loss of primary tooth - Syndromique and non-syndromique variantes : <p>Tooth agenesis appears as phenotype in >150 syndromes.</p> - Congenital absences of teeth result from disturbances during the initial stages of formation of a tooth: initiation and proliferation. - No permanent tooth if the predecessor is missing. - If only one or a few teeth are missing, the absent tooth will be the most distal tooth of any given type. - Note: Inheritance of familiar, ø syndromic hypodontia <ul style="list-style-type: none"> o Autosomal dominate with incomplete penetrance and variable expression. o Autosomal recessive. o Sex-linked - Ectodermal dysplasia (hidrotic and anhidrotic type): <ul style="list-style-type: none"> o Thin, sparse hair o Absence of sweat glands in the anhidrotic type (present in the hidrotic type) o Missing teeth o Lack of development of the alveolar process o Malformations of the nails / skin (ectodermal tissue)
Malformed teeth	<ul style="list-style-type: none"> - Abnormalities in tooth size and shape result from disturbances during the morphodifferentiation stage with maybe some carryover from the histodifferentiation stage. - 5% population have a significant tooth size discrepancy. Most variable teeth = 2+2. - Influencing factors on tooth variation during development: <ul style="list-style-type: none"> o Genotype: <ul style="list-style-type: none"> ▪ Signal molecules ▪ Receptors ▪ Transcriptionfactors o Epigenetic <ul style="list-style-type: none"> ▪ Specific: e.g. Methylation ▪ General: e.g. Hormons o Interactions <ul style="list-style-type: none"> ▪ Cell/cell ▪ Cell/matrix ▪ Tooth buds o Vascular o Neural o Eruption
Supernumerary teeth	<ul style="list-style-type: none"> - Result from disturbances during the initiation and proliferation stages. - Idiopathic finding or part of a larger disease process or syndrome. → Often seen in patients with cleidocranial dysplasia. - Early removal is often required to obtain reasonable alignment and occlusal relationships.

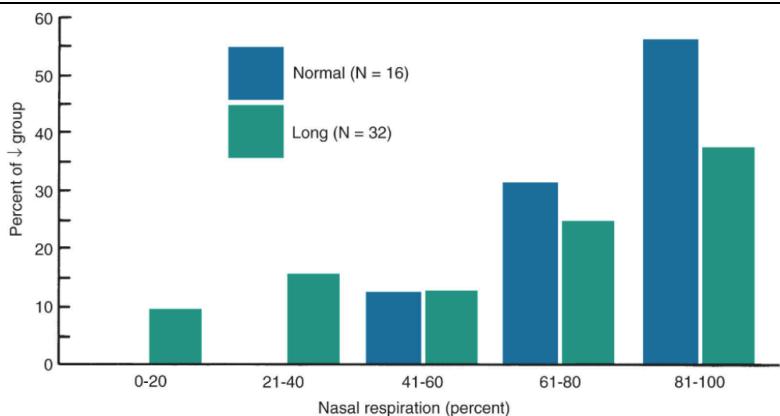
	<ul style="list-style-type: none"> - Distribution: <ul style="list-style-type: none"> ○ Mostly mesiodens in the maxillary midline. ○ Sometimes extra premolars, lateral incisors, 4th molars.
Traumatic displacement of teeth	<ul style="list-style-type: none"> - Trauma to the primary tooth displaces the permanent tooth: <ol style="list-style-type: none"> 1. Disturbance of enamel formation and defects in the crown if the crown is not completely formed at the moment of trauma. 2. If crown completed: <ul style="list-style-type: none"> • Crown displaced in relation to the root • Stop of root formation (shortened roots) • Dilacerations (distortion of the root) - <i>Prof. Becker:</i> Tx of teeth with root dilacerations <ul style="list-style-type: none"> - Early tx is indicated: <ul style="list-style-type: none"> Root formation is possible as long as the apex is open, even with dilacerations. - Difficult to install proper tooth position with severe root distortion. <ul style="list-style-type: none"> • Extraction is maybe necessary. • Apex ectomy + root treatment to avoid perforation of the gingiva if the tooth is going to be aligned. - Lateral displaced teeth by trauma usually should be repositioned as early as possible. Later surgical reposition is may be used if the tooth ankyloses and reposition is not possible.
Invasive cervical root resorption <i>(Prof. Becker)</i>	<ul style="list-style-type: none"> - Sequence: <ul style="list-style-type: none"> ○ Defect in the cement. ○ Dentin around the pulp is resorbed by osteoclasts. Pre-dentin is not affected. No inflammation. No loss of vitality. ○ Resorbed areas are filled by soft tissue. ○ Teeth mimic ankylosis and cannot be moved. - Treatment: <ol style="list-style-type: none"> 1. Exposure of the resorbed area, removal of soft tissue. 2. Provisional glasionomer filling. 3. Apply traction. 4. Root treatment and permanent restoration as soon as the tooth is in position.

Genetic influences	
2 Theories	<ol style="list-style-type: none"> 1. Inherited disproportion between the size of the teeth and the size of the jaws: → Crowding / spacing. 2. Inherited disproportion between the size or shape of the upper and lower jaws: → Improper occlusal relationships.
<p>→ Bone is more related to genes</p> <p>→ Teeth are more related to environmental factors</p> <p>→ Facial type and growth pattern are genetically influenced and can lead to similar responses to environmental influences.</p>	<ul style="list-style-type: none"> - Explanation for the increase in malocclusion in recent centuries: Great increase in outbreeding that occurred as the human population grew and became more mobile → not true. - If malocclusion or a tendency to malocclusion is inherited, the mechanism is not the independent inheritance of discrete morphologic characteristics like tooth or jaw size. - <u>Studies with family members:</u> <ul style="list-style-type: none"> ○ Heritability of craniofacial (skeletal) characteristic is relatively high and increases with age. ○ Heritability of dental (occlusal) characteristics is low and decreasing with age. → Shows an increasing environmental contribution to the dental variation. - <u>Twin studies: Hereditary component for:</u> <ul style="list-style-type: none"> ○ Variations in spacing and tooth position within the dental arch = 69-89% ○ Overbite 53% ○ Overjet 28% - Effect of interracial crosses appears to be only additive, but not multiplicative. - Knowing the type of growth associated with different genetic patterns can help to chose the type and timing of orthodontic and surgical treatment. - Similar malocclusions are likely to be seen in siblings: → Facial types and growth patterns are genetically influenced and lead to similar responses to environmental factors. <p>→ Bone more related to genes → Teeth more related to environmental factors</p> <ul style="list-style-type: none"> - <u>Mn prognathisme:</u> <ul style="list-style-type: none"> ○ 0.316 heritability. ○ Autosomal dominant with incomplete penetration. ○ One major gene was determined as cause, but not only one single gene responsible for the malocclusion. - <u>Puri, 2007:</u> <ul style="list-style-type: none"> ○ Crowding: Patients have in general larger teeth ○ Spacing: Patients have in general smaller teeth
Environmental influences	
<ul style="list-style-type: none"> - Consist largely of pressures and forces related to physiologic activity. <p>1. Equilibrium considerations</p> <p>→ Changes of the equilibrium = changes of tooth and jaw position</p>	<ul style="list-style-type: none"> - Forces are in balance or in equilibrium if an object is subjected to a set of forces, but remains in the same position. (Newton's 1st law) - <u>Tooth:</u> <ul style="list-style-type: none"> ○ Experiences normally forces from masticatory effort, swallowing and speaking, but does not move. ○ Continuous force from an orthodontic appliance applied: → Equilibrium altered → tooth moves. - <u>PDL:</u> <ul style="list-style-type: none"> ○ Constructed to withstand heavy forces of short duration (mastication). Fluid in the PDL space acts as a shock absorber for pressure so that the soft tissues in the PDL are not compressed although bending of the alveolar bone occurs. ○ An impact on the soft tissues occurs only, if the pressure is maintained long enough to squeeze out the fluid. <p>→ Only light forces of long duration (≥ 6h per day) determinate, if there is enough imbalance of forces to lead to tooth movement.</p>

	<p>Equilibrium Influences:</p> <table border="1"> <thead> <tr> <th>Influence</th><th>Force Magnitude</th><th>Force Duration</th><th>Impact</th></tr> </thead> <tbody> <tr> <td colspan="4">Tooth Contacts</td></tr> <tr> <td>Mastication</td><td>Very heavy</td><td>Very short</td><td>No significant impact</td></tr> <tr> <td>Swallowing</td><td>Light</td><td>Very short</td><td>No significant impact</td></tr> <tr> <td colspan="4">Soft tissue pressures of lip, cheek and Tongue</td></tr> <tr> <td>Swallowing</td><td>Moderate</td><td>Short</td><td>No significant impact</td></tr> <tr> <td>Speaking</td><td>Light</td><td>Very short</td><td>No significant impact</td></tr> <tr> <td>Resting</td><td>Very light</td><td>Long</td><td>Important</td></tr> <tr> <td colspan="4">External Pressures</td></tr> <tr> <td>Habits</td><td>Moderate</td><td>Variable</td><td></td></tr> <tr> <td>Orthodontics</td><td>Moderate</td><td>Variable</td><td></td></tr> <tr> <td colspan="4">Intrinsic Pressures</td></tr> <tr> <td>PDL fibers</td><td>Light</td><td>Long</td><td></td></tr> <tr> <td>Gingival fibers</td><td>Variable</td><td>Long</td><td></td></tr> </tbody> </table>	Influence	Force Magnitude	Force Duration	Impact	Tooth Contacts				Mastication	Very heavy	Very short	No significant impact	Swallowing	Light	Very short	No significant impact	Soft tissue pressures of lip, cheek and Tongue				Swallowing	Moderate	Short	No significant impact	Speaking	Light	Very short	No significant impact	Resting	Very light	Long	Important	External Pressures				Habits	Moderate	Variable		Orthodontics	Moderate	Variable		Intrinsic Pressures				PDL fibers	Light	Long		Gingival fibers	Variable	Long	
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Function and dental arch size → Influence yes, but little effect.	<ul style="list-style-type: none"> - Muscle size and activity is reflected in the size and shape of the muscular processes of the jaws: <ul style="list-style-type: none"> ○ → Vertical jaw relationship is affected. ○ → Enlargement of the mandibular gonial angles can be seen in humans with hypertrophy of the mandibular elevator muscle - Not clear, if the masticatory effort influences the size of the dental arches and the amount of space for the teeth, but to some extent it seems reasonable. - If dietary consistency affects dental arch size and the amount of space for the teeth as an individual develops, it must be early as dental arch dimensions are established early. 																																																								
Biting force and eruption → Dental arch size, tooth eruption or vertical dimensions are not influenced in the absence of syndromes. (results no cause)	<ul style="list-style-type: none"> - Maximum biting forces are higher in short face individuals and lower in long-face people than in individuals with normal vertical dimensions. <ul style="list-style-type: none"> ○ → Diagnosis of face type is however possible before the differences in biting forces can be measured: No difference in pubescent children with different face types. ○ The difference is rather an effect than a cause for the malocclusion. (failure of long-face individual to gain strength) - Bite forces do not determine dental arch size, tooth eruption or vertical dimensions in the absence of syndromes. - Muscular dystrophy and related syndromes can affect growth. 																																																								
Sucking and other habits → Can have an impact on the dentition, if long enough applied.	<ul style="list-style-type: none"> - Sucking habits during the primary dentition: little if any longterm effect. - If a mild displacement of the teeth happens at age 3-4 y: → Teeth are restored to their usual positions, if the sucking habit stops and the pressure equilibrium is reestablished. - <u>Persisting sucking habit:</u> Combination of direct pressure on the teeth and alteration in the pattern of resting cheek and lip pressure. <ul style="list-style-type: none"> • Flared and spaced maxillary incisors. • Lingually positioned lower incisors. • Anterior open bite: Interference with normal incisors eruption and excessive eruption of posterior teeth as the jaw is positioned downward. Change of the equilibrium. (1 mm elongation posterior → 2 mm bite opening anterior) • Narrow upper arch (tongue lowered + increased cheek pressure). • Mx arch can become V-shaped because cheek pressure due to contraction of the m. buccinator is greatest at the corners of the mouth. • Changes in symmetry if the thumb is placed on one side instead of the midline. 																																																								



	<ul style="list-style-type: none"> - Different sucking pattern: <ul style="list-style-type: none"> • Short duration, but very intensive: May not a risk for malocclusion. • Long duration, little force (during sleep) → Increased risk for a malocclusion. - <u>Music instruments:</u> Duration too short to make any difference except for highly professional musicians (only minor changes).
3. Tongue thrusting → <i>Tongue thrusting = result of displaced incisors, but NOT the cause.</i>	<ul style="list-style-type: none"> - Populations with tongue thrusting: <ul style="list-style-type: none"> • Young children: <ul style="list-style-type: none"> ▪ Normal transitional stage in swallowing. ▪ Delay, if a sucking habit is present. • Individuals of any age with displaced incisors: Adaption to seal the space between the teeth = result of displaced incisors, but NOT the cause. - Labor study: Not more tongue force against the teeth in individuals who place the tongue tip forward when they swallow than those who keep the tongue tip back. - Adult swallowing pattern is never achieved in 10-15% of the population. The tongue trust swallow is however slightly different than infantile swallow (exception: brain damaged children). - Tongue thrusting is often present in children with good occlusion. - Too short duration to have an impact on tooth position: 1000 x / 24 h swallowing 1 swallowing ~ 1s. - Correction of the tooth position normally causes a change in swallow pattern. → It is neither necessary nor desirable to try to teach the patient to swallow differently before beginning orthodontic treatment. - Only the postural resting position of the tongue is important and can have an effect on the teeth.
1001 Respiratory pattern → <i>Total nasal obstruction is highly likely to alter the pattern of growth and lead to malocclusion in experimental animal and human studies.</i> → <i>Individuals with a high percentage of oral respiration are over-represented in the long-face population.</i> → <i>The majority of individuals with the long-face pattern of deformity have no evidence of nasal obstruction and must therefore have some other etiologic factor as the principal cause.</i>	<ul style="list-style-type: none"> - Respiratory need = primary determinant of the posture of the jaws and the tongue. - Human: <ul style="list-style-type: none"> ○ = Primarily nasal breather. ○ Mouth breather in certain physiologic conditions: exercise, heavy mental concentration, conversation. ○ Greater effort needed to breathe through the nose than through the mouth (warming, cleaning, and humidifying the air). - Chronic respiratory obstruction due to inflammation of the nasal mucosa, allergies, chronic infection, mechanical obstruction or habits lead to mouth breathing and can contribute to a change in the pattern of growth. - BUT: Not all individuals in the long face population have an evidence of nasal obstruction (other etiologic factor!). - Mouthbreathers: <ul style="list-style-type: none"> • Change in posture if nasal obstruction occurs. • Mandible and tongue must be lowered → face height increases. • Posterior teeth overerupt. • Mandible rotates downward and backward → anterior OB ↓ + increased OJ. • Narrow maxilla due the increased pressure from the stretched cheeks. - Long-face and normal-face children are predominantly nasal breathers. - Some long-face children have <40% nasal breathing (no normal-face children with such low values). - More long-face children have a nasal obstruction compared to normal-face children, but the majority are not predominantly mouth breathers. - → Impaired nasal respiration may contribute to the development of the long-face condition, but is not the sole or even the major cause.



- Some lip separation is normal in children, but breathing through the nose is also possible while the lips are apart.
→ Lips separation does not automatically mean that a patient is a mouth breather.
- One partially obstructed nostril should not be interpreted as a problem as the highly vascular nasal mucosa undergoes cycles of engorgement with blood and shrinkage.
(the mucosa of one side is always engorged)
- *Linder-Aronson, 1970:*
Patient with adenoidectomy had stat. sign. greater anterior face height and steeper mn plane angles than the controls, but the differences are quantitatively not large.
- Indication for tonsillectomy (*seminar, zmk*)
 - Apnea / respiration stop due to insufficient O₂ transport to the brain.
 - Asymmetries (suspicion for tumor)
 - Infects with antibiotics intake:
 - >7/y
 - >5/y for 2 years

Discussion with Dr. Gkantidis

- Nearly no malocclusions 35'000 years ago, steadily increase in the years afterwards.
- Little change in genes in that time.
- Nowadays nearly all people have a malocclusion (65%).
- Teeth are more prone for changes than the bones.
- Function has a big influence.
- Mechanical loading is important for bone biology.

Proffit Chapter 6:

Diagnosis and Treatment Planning

1. Diagnosis planning	<ul style="list-style-type: none"> Collection of information and creation of a database. Formulation of a problem list with prioritization. Pathological and developmental problems can be present. → Pathological processes must be under control before tx starts! (they could become worse during treatment) Consider a solution for each single problem and evaluate afterwards the interactions between the solutions. Goal = truth, scientific inquiry. No room for opinion or judgment. Factual arise of the situation.
2. Treatment planning	<ul style="list-style-type: none"> Goal = wisdom. Plan, which a good clinician will follow to maximize the benefit for the patient. Tx is usually comprehensive: → Goal = The best possible occlusion, facial esthetics and stability. <u>Optimal tx-plan:</u> <ol style="list-style-type: none"> Timing of tx: Mostly adolescence: Sufficient growth remaining, self-motivation develops, short course of tx. Timing depends on the specific problem. Complexity of the tx required. Predictability of success with a given tx approach: Prefer an evidence based choice! Patient's and parent's goals and desires. → Must be given a role in the decision-making process. Patient's compliance is a critical issue for success or failure: → Choose a tx that he supports.
	<p>- Orthodontic tx is mostly an elective tx rather than a required one (seldom significant health risks without tx). → Functional and esthetic benefits must be compared to risks and costs.</p> <p>- Interact with the patient to develop the best plan.</p> <p>- Do not concentrate too closely on one aspect of the patient's overall condition, that other significant problems are overlooked. → Not rush to judgement during the initial examination.</p> <p>- Cave: Recognize bias and consciously resist. (habit to characterize problems in terms of own special interests)</p> <p>- Create a database from different sources: <ol style="list-style-type: none"> Interview from questions (written and oral) from the patient and parents. Clinical examination of the patient. Evaluation of diagnostic records: Dental casts, x-ray, photographs. </p>

Questionnaire / interview	
Patient's chief concern	<ul style="list-style-type: none"> - Mostly more than one reason to seek orthodontic tx. → Establish their relative importance to the patient: <ul style="list-style-type: none"> • "Do you think you need braces?" • "What bothers you most about your teeth or your appearance?" • "What do you want tx to do for you?"
Medical and dental history	<ul style="list-style-type: none"> - Try to establish the cause of the malocclusion, although mostly this is not possible: Only 5% of the population have a malocclusion from a specific cause. - Provide a background for understanding the patient's overall situation and evaluation of specific concerns. - Growth deficiencies from an old condyle fracture = most common reason for real face asymmetry. - Chronic medical problems are no contraindication for orthodontic tx if they are under control. <ul style="list-style-type: none"> • Diabetes: periodontal breakdown more likely • Arthritis / Osteoporosis: tooth movement maybe difficult because of bisphosphonates
Physical growth evaluation	<ul style="list-style-type: none"> - Growth spurt facilitates tooth movement. - "How rapidly grown recently? Clothes sizes changed? Sexual maturation onset yet? Time of sexual maturation in siblings?" - Observe sexual characteristics. - Consider height-weight records. - Calculating bone age from the vertebrae in the lateral ceph. - Serial cephalometric x-rays = Most accurate way to determine whether facial growth has stop or is continuing.
Social and behavioral evaluation	<ul style="list-style-type: none"> - Motivation for tx: <ul style="list-style-type: none"> • External: Pressure from another individual. • Internal: Comes from the individual and is based on the own assessment of the situation and the desire for tx. Does not develop until adolescence. Better cooperation for tx can be expected. - Cooperation: More likely to be a problem with children than adults. <ul style="list-style-type: none"> • Depends on the extent to which the child sees the tx as a benefit opposed to something he is required to do. • Degree of parental control. - Treating an unwilling child is hardly a good professional judgment, even if the parents force an apparent assent (Zustimmung).

Clinical evaluation																	
Aims	<ol style="list-style-type: none"> 1. To evaluate and document oral health, jaw function, facial proportion and smile characteristics. 2. To decide which diagnostic records are required. 																
Oral health	<ul style="list-style-type: none"> - Any disease or pathology must be under control before orthodontic tx starts: Medical problems, caries, pulpal / periodontal pathologies. - Children with generalized periodontitis need always a proper diagnosis. Often blood diseases are the origin. - Gingiva: <ul style="list-style-type: none"> • Biotype: Defined by genetics <ul style="list-style-type: none"> ○ Thin scalloped 12-81% ○ Thick scalloped ○ Thick flat → Transplant of keratinized gingiva into mobile mucosa remains stable. • Phenotype: Defined by local factors • Detect any areas of bleeding. • Not necessary to establish precise pocket depth: <ul style="list-style-type: none"> → Detect juvenile periodontitis (1er/6er). • Inadequate attached gingiva around crowded incisors may lead to stripping of the gingiva away from the teeth when the teeth are aligned. • Orthodontic tx is possible if periodontal problems are under control. - <i>Prof. Salvi:</i> Risk factors for periodontitis progression and tooth loss due to periodontal reasons: <ul style="list-style-type: none"> • Remaining pockets > 6 mm • BOP > 25% • Furcation involvement > 3 mm (= grade 2 & 3) 																
Jaw and occlusal function	<ul style="list-style-type: none"> - Cerebral palsy / other types of severe neuromuscular disease → Normal adaption to changes in tooth position produced by orthodontics are not possible → post-tx relapse (no stable equilibrium). - Evaluation: <ul style="list-style-type: none"> • <u>Mastication incl. swallowing:</u> <ul style="list-style-type: none"> - Normal coordination and movements. - Severe malocclusion makes mastication more difficult: No socially acceptable manner to masticate, patients avoid certain food, cheek & lip biting occurs during mastication. - Swallowing is almost never affected by malocclusion. • <u>Speech:</u> <ul style="list-style-type: none"> - Can be altered due to malocclusion, but normal speech is also possible with a severe anatomic distortion. - Combination of speech therapy and orthodontics can be helpful if the speech problem is related to the malocclusion. 																
	<table border="1"> <thead> <tr> <th>Speech Sound</th><th>Problem</th><th>Related Malocclusion</th></tr> </thead> <tbody> <tr> <td>/s/, /z/ (sibilants)</td><td>Lisp</td><td>Anterior open bite, large gap between incisors</td></tr> <tr> <td>/t/, /d/ (linguoalveolar stops)</td><td>Difficulty in production</td><td>Irregular incisors, especially lingual position of maxillary incisors</td></tr> <tr> <td>/f/, /v/ (labiodental fricatives)</td><td>Distortion</td><td>Skeletal Class III</td></tr> <tr> <td>th, sh, ch (linguodental fricatives [voiced or voiceless])</td><td>Distortion</td><td>Anterior open bite</td></tr> </tbody> </table> <ul style="list-style-type: none"> • <u>Possibility of sleep apnea related to mandibular deficiency:</u> <ul style="list-style-type: none"> - Oral appliances to advance the mandible can be effective in patients with mild forms of OSAS. • <u>TM-Joint problems:</u> <ul style="list-style-type: none"> - Restricted movements usually indicate a functional problem. 		Speech Sound	Problem	Related Malocclusion	/s/, /z/ (sibilants)	Lisp	Anterior open bite, large gap between incisors	/t/, /d/ (linguoalveolar stops)	Difficulty in production	Irregular incisors, especially lingual position of maxillary incisors	/f/, /v/ (labiodental fricatives)	Distortion	Skeletal Class III	th, sh, ch (linguodental fricatives [voiced or voiceless])	Distortion	Anterior open bite
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	<p>→ Check maximum opening.</p> <ul style="list-style-type: none"> - If the mn moves normally, its function is not severely impaired. - Palpating the muscles of mastication and the TMJ. - Note any sign of joint pain, noise, limitation of mouth opening. - Sirdalud: Myotonolytika for TMJ disorders. - CR is difficult to find in children: (articular eminence is not well developed). <ul style="list-style-type: none"> → A child with an apparent unilateral crossbite often has a bilateral narrowing of the maxillary arch, with a shift to the unilateral crossbite position. → In patients with skeletal class II, the mandible is often positioned more forward and hides the severity of the extend of the mn deficiency (Sunday bite). → Apparent class III relationship resulting from a forward shift to escape incisor interferences from and end-to-end relationship.
Need for antibiotics prophylaxis	<ul style="list-style-type: none"> - <u>Indications: (European Society of Cardiology)</u> <ul style="list-style-type: none"> High risk patients: <ul style="list-style-type: none"> • Prosthetic valves • Prior anamnestic endocarditis • Hereditary cyanotic illnesses (shunt → mix of blood rich and poor of O₂ → blue color of the skin) • Hereditary illness if: <ul style="list-style-type: none"> ◦ OP ≤6 months ago with synthetic material. ◦ Persisting shunt. Moderate risk patients: ø antibiotic prophylaxis, but good oral hygiene. - Amoxicillin 2g or 50mg/kg weight IO/IV 30-60 min before the appointment. Alternative: Clindamycin 600 mg or 20 mg/kg weight 30-60 min before the appointment.
Facial and dental appearance	<ul style="list-style-type: none"> - Overall facial attractiveness influences how people look at teeth and the level of tolerance to different levels of dental attractiveness. - People make judgments regarding intelligence and interpersonal temperament for children and young adults based on the appearance of the teeth. - Different ethnic and national groups view facial esthetics differently. - Gender and overall facial attractiveness influence how people are perceived. - Steps to follow examination: <ol style="list-style-type: none"> 1. Macro-esthetics: Facial proportion in all three planes of space: Asymmetry, excessive or deficient face height, mandibular or maxillary deficiency or excess. 2. Mini-esthetics: Display of the teeth at rest / during speech / on smiling, gingiva / tooth display, buccal corridors. 3. Micro-esthetics: Teeth in relation to each other, tooth proportions in height and width, gingival shape / contour / black triangles.

Macro-esthetics: Facial proportions

TABLE 6.5 Checklist of Facial Dimensions to Evaluate During Clinical Examination

Frontal at Rest	Frontal Smile	Frontal Widths	Profile
To Midsagittal Plane <ul style="list-style-type: none"> Nasal tip Maxillary dental midline Mandibular dental midline Chin (midsymphysis) 	<ul style="list-style-type: none"> Maxillary incisor display Maxillary incisor crown height Gingival display Smile arc Occlusal plane cant? 	<ul style="list-style-type: none"> Alar base Nasal tip Buccal corridor 	Lower Face <ul style="list-style-type: none"> Maxillary projection Mandibular projection Chin projection Lower face height
Vertical <ul style="list-style-type: none"> Lip separation (lips relaxed) Lip vermillion display Maxillary incisor display (lips relaxed) Lower face height Philtrum length Commissure height Chin height 			Nose <ul style="list-style-type: none"> Nasal radix Nasal dorsum contour Nasal tip projection Nasolabial angle Lip <ul style="list-style-type: none"> Lip fullness Labiomental sulcus Throat Form <ul style="list-style-type: none"> Chin-throat angle Throat length Submental contour (fat pad)

Girls: Adolescent Growth total 3.5 y, 3 stages

Stage 1 <i>Beginning of adolescence growth</i>	Appearance of breast buds Initial pubic hair
Step 2 (12 m later) <i>Peak velocity in height</i>	Breast development Axillary hair Darker/more abundant pubic hair
Stage 3 (12-18 m later) <i>Growth spurt ending</i>	Menstruation Broadening of the hips with adult fat distribution Breasts completed
Boys: Adolescent Growth total 5 y, 4 stages	
Stage 1 <i>Beginning of adolescent growth</i>	Fat spurt weight gain Feminine fat distribution
Stage 2 (12 m later) <i>Height spurt is beginning</i>	Redistribution / reductions in fat Pubic hair Growth of penis
Stage 3 (8-12 m later) <i>Peak velocity in height gain</i>	Facial hair appears on the upper lip only Axillary hair Muscular growth with hardener / more angular body form
Stage 4 (15-24 m later) <i>Growth spurt ending</i>	Facial hair on chin and lip Adult distribution / color of pubic and axillary hair Adult body form

- Assessment of development age: Physical development is more important than chronologic age.
- Look for recognizable secondary sexual characteristics.