

# **Contemporary Orthodontics**

**W. R. Proffit**

**Summary + Notes from Seminars**

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## Proffit Chapter 1:

### Malocclusion and Dentofacial Deformity in Contemporary Society

#### The changing goals of orthodontic treatment

**1000 BC:** First orthodontic appliances

**1850 Kingsley:** Book *Oral Deformities*.  
(1829-1913) First use of extraoral force. 1980: Description of the "Jumping Bite". Emphasis to the alignment of teeth and the correction of facial proportions. Little attention paid to bite relationship. A lot of extractions.

**1890 Angle:** Attention to a concept of occlusion to enable good prosthetic replacement of teeth.  
(1855-1930) 6+6 = key to the occlusion.  
Treatment:  
- No extractions  
- No extraoral force  
- Little attention was spent to facial proportions and esthetics (should be obtained by perfect occlusion).

#### 4 occlusion classes:

- Normal occlusion: Teeth aligned in a smooth catenary curve. The mesiobuccal cusp of 6+6 occlude in the buccal groove of 6-6.
- Class I: Normal relationship of the molars. Line of occlusion incorrect. Not considered as a malocclusion.
- Class II: Lower molar distally positioned in relation to upper molar. Line of occlusion not specified.
- Class III: Lower molars mesially positioned in relation to upper molar. Line of occlusion not specified.

**Martin Dewey:** Proposed subdivisions for Angle cl.I

Line of occlusion mx: Central fossa molars, cingulum canines & incisors  
Line of occlusion mn: Buccal cusps molars, incisal edges canines & incisors  
→ Fit perfect together with normal molar relationship (no aberrations in tooth size...)

**1940-1950:** Reintroduction of extractions to enhance facial esthetics and achieve better stability of the occlusal relationships.  
**Tweed & Begg**

**After WWII:** Cephalometric x-rays.  
(first introduced around 1934 by Hofrath in Germany (Europe) and Broadbeck in the US)  
→ Observation of jaw growth, idea that malocclusions depend on jaw relationship and not only on alignment.

Europe: Introduction of "functional jaw orthopedics."  
America: More fixed treatments.  
Use of extraoral force to obtain correct jaw relationship introduced later (esp. around 1960/1970) when a study was published that sign. skeletal changes can be achieved with functional appliance tx)

Latest studies with x-ray were done mostly before 1980.

**Today:** Concept to correct jaw relationship + soft tissue paradigm.  
Soft tissues state the goals and limitations of modern orthodontics.

#### Soft tissue paradigm:

- 1<sup>st</sup> goal:  
Proportions of the soft tissue integument of the face and the relationship of the dentition to the lips and face are the major determinants of facial appearance.

- Soft tissue adaptions to the position of teeth determine whether the treatment results are stable.
- An ideal occlusion cannot always be the focus of the treatment plan.
- 2<sup>nd</sup> goal:  
Functional occlusion:  
No temporomandibular dysfunctions due to the occlusion which result in soft tissues injuries around the TMJ
  - 3<sup>rd</sup> goal:  
Determine how the soft tissue relationships should be and then plan how the teeth and jaws must be arranged

**TABLE 1.1** Angle Versus Soft Tissue Paradigms: A New Way of Looking at Treatment Goals

Parameter	Angle Paradigm	Soft Tissue Paradigm
Primary treatment goal	Ideal dental occlusion	Normal soft tissue proportions and adaptations
Secondary goal	Ideal jaw relationships	Functional occlusion
Hard and soft tissue relationships	Ideal hard tissue proportions produce ideal soft tissues	Ideal soft tissue proportions define ideal hard tissues
Diagnostic emphasis	Dental casts, cephalometric radiographs	Clinical examination of intraoral and facial soft tissues
Treatment approach	Obtain ideal dental and skeletal relationships, assume the soft tissues will be all right	Plan ideal soft tissue relationships and then place teeth and jaws as needed to achieve this
Function emphasis	TMJ in relation to dental occlusion	Soft tissue movement in relation to display of teeth
Stability of result	Related primarily to dental occlusion	Related primarily to soft tissue pressure and equilibrium effects

- Orthodontics is shaped by biological, psychosocial and bioethical issues.

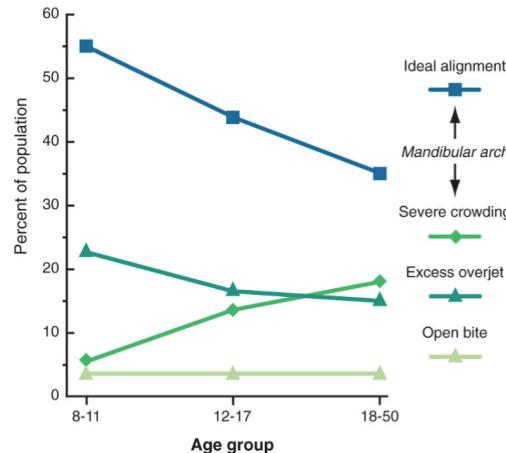
## Epidemiology of Malocclusion

NHANES III  
(Third National Health and Nutrition Examination Survey)

Numbers from US population 1989-1994

### Changes in the prevalence of types of malocclusions from childhood to adult life

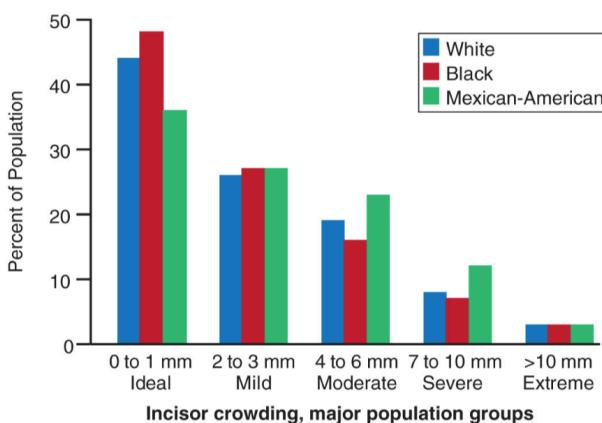
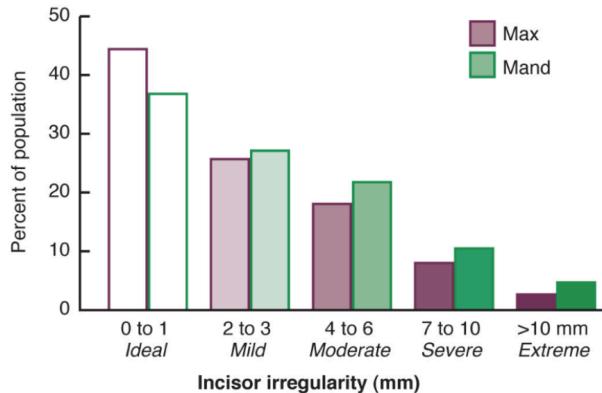
%	Child 8-11	Teens 12-17	Adults
Severe Crowding (7-10mm)	6	15	18
Excessive OJ	23	17	15
Open bite	5	5	5
Severe OB $\geq 5\text{mm}$	20	?	13
Ideal Alignment	55	45	35



- Children 8-11 y:
  - o 55% ideal mn alignment
  - o 25% excessive OJ
- Teenagers 12-17 y:
  - o Mn crowding  $\uparrow \rightarrow$  Ideal mn alignment 45%
  - o Excessive OJ  $\downarrow \rightarrow 15\%$
- Adults:
  - o Mn crowding  $\uparrow \rightarrow 35\%$  ideal mn alignment.
  - o Excessive OJ  $\downarrow \rightarrow 15\%$
- OB prevalence is  $\pm$  constant over all ages groups: 3-4%

## Irregularity index

- = Total of millimeter distances from the contact point on each incisor tooth to the contact point that it should touch.

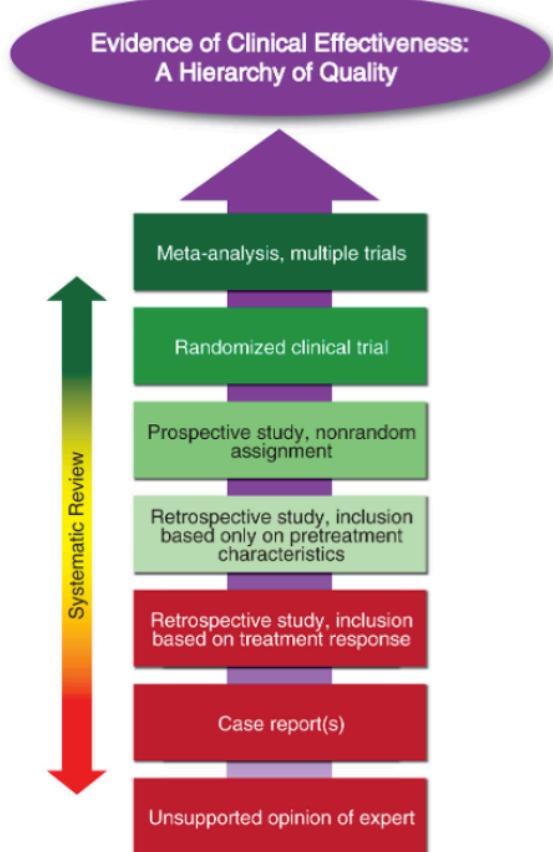


	<ul style="list-style-type: none"> <li>- Irregular incisors: <ul style="list-style-type: none"> <li>• Well aligned incisors (Not clear if 0): <math>\frac{1}{3}</math> population, Mx &gt; Mn.</li> <li>• Mild or moderate irregularity: About <b>45%</b>.</li> <li>• Severe or extreme crowding: <b>15%</b>.</li> </ul> </li> <li>- Mexican-Americans = low income = less orthodontic tx = more irregularity. → The differences are not only due to the ethnicity.</li> </ul>																				
<b>Diastema</b>	<ul style="list-style-type: none"> <li>- Mixed dentition: <b>25%</b> → Disappears or increases with the eruption of 3+3.</li> <li>- Age 12-17y: <b>7%</b>.</li> <li>- If &gt;2 mm → Spontaneous correction is not likely.</li> <li>- Blacks = 2x more likely to have a diastema than Whites or Mexican-Americans.</li> </ul>																				
<b>OJ</b>	<table border="1"> <thead> <tr> <th>Overjet (mm)</th> <th>Percent of population</th> </tr> </thead> <tbody> <tr><td>&gt;10</td><td>~2</td></tr> <tr><td>7-10</td><td>~5</td></tr> <tr><td>5-6</td><td>~10</td></tr> <tr><td>3-4</td><td>~38</td></tr> <tr><td>1-2</td><td>~40</td></tr> <tr><td>0</td><td>~5</td></tr> <tr><td>-1 to -2</td><td>~2</td></tr> <tr><td>-3 to -4</td><td>~2</td></tr> <tr><td>&gt;-4</td><td>~2</td></tr> </tbody> </table> <p>Overjet (mm)</p> <ul style="list-style-type: none"> <li>- Normal OJ = 1-2 mm: about <b>40%</b></li> <li>- Cl. II (tendency): <b>55%</b></li> <li>- Cl. III: <b>5-10%</b></li> <li>- OJ &gt; 5 mm = cl.II: about <b>15%</b> (children 23%, youth 15%, adults 13%)</li> <li>- <b>4%</b> extreme class II/III with need for surgery.</li> <li>- <i>Thilander 2009</i>: Observation period from 5<sup>th</sup> - 31<sup>st</sup> year: <math>\Delta</math> OJ = <b>-0.7 mm</b></li> </ul>	Overjet (mm)	Percent of population	>10	~2	7-10	~5	5-6	~10	3-4	~38	1-2	~40	0	~5	-1 to -2	~2	-3 to -4	~2	>-4	~2
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<b>Posterior crossbite</b>	= <b>9%</b> of the populations (7.6% Mexican-Americans, 9.1% whites, 9.6% blacks)																				
<b>Ethnical differences:</b>	<ul style="list-style-type: none"> <li>- Class II: Whites of Northern European descent ↑</li> <li>- Class III: Asians ↑</li> <li>- Class III or open bite: Africans &gt; Europeans</li> <li>- Deep bite: Africans &lt; Europeans</li> </ul>																				

<b>Why is malocclusion so prevalent?</b> <ul style="list-style-type: none"> <li>- Present prevalence of malocclusions &gt; few hundred years ago.</li> <li>- Crowding and malalignment of teeth were unusual until relatively recently.</li> </ul>	
Changes in the last 1000 years	<ul style="list-style-type: none"> <li>- Size of individual teeth ↓</li> <li>- Number of teeth ↓</li> <li>- Size of the jaws ↓</li> <li>- 3<sup>th</sup> incisor, 3<sup>th</sup> premolars, 4<sup>th</sup> molar disappeared.</li> <li>- Malocclusion increased after transition from rural villages to the city.</li> <li>- Today 852±258 (last tooth of each group) are missing more and more often.</li> </ul>
Formation of malocclusion	<ul style="list-style-type: none"> <li>- Bad match tooth size &amp; number with jaw size → crowding, malalignment.</li> <li>- Reduction of jaw size because of reduced use (softer food) (relation is hard to document).</li> <li>- Dental caries &amp; periodontal disease accompany diet changes and could influence the development of malocclusions.</li> <li>- <u>Types of crowding:</u> <ul style="list-style-type: none"> <li>○ <b>Primary crowding:</b> Mismatch of tooth size and jaw size.</li> <li>○ <b>Secondary crowding:</b> Loss of space due to caries, undermining resorption, early loss of baby teeth, ankylosis, eruption 7-7 before 5-5.</li> <li>○ <b>Tertiary crowding:</b> Due to late changes in growth. Mn growth with incisor contact → incisors tip lingually.</li> </ul> </li> </ul>
<b>Who needs treatment</b>	
<b>Psychosocial problems</b>	<ul style="list-style-type: none"> <li>- Severe malocclusion = Social handicap.</li> <li>- Negative influence: <ul style="list-style-type: none"> <li>• Difference in teachers' expectations</li> <li>• Student's progress in school</li> <li>• Employability</li> <li>• Competition for a mate</li> </ul> </li> <li>- Psychic distress is not directly proportional to the anatomic severity of the problem. The self-esteem defines the impact of a physical defect.</li> <li>- Patients seek orthodontic treatment to improve their psychological well-being rather than to improve the function.</li> </ul>
<b>Oral Function</b>	<ul style="list-style-type: none"> <li>- Malocclusion makes function more difficult, but NOT impossible. → Extra effort is required to compensate for the anatomic deformity.</li> <li>- Pain in the TMJ is mostly caused by muscle fatigue and spasm (grinding the teeth) and not because of the dental occlusion.</li> <li>- <i>Macfarlene, 2009:</i> Strongest correlation coefficients for malocclusion -TMJ disease = 0.3-0.4. NO associations for the majority of patients for a dental malocclusion and TMD → Orthodontics as primary tx for TMJ disease is not indicated!</li> <li>- <i>McNamara:</i> Malocclusion related with TMJ problems: <ul style="list-style-type: none"> <li>• OJ &gt; 6mm</li> <li>• Slide CO / maximal Intercuspidation</li> <li>• Crossbite with shift</li> <li>• Loss of multiple posteriore teeth</li> <li>• Severe anterior open bite.</li> </ul> </li> </ul>
<b>Susceptibility ↑ to trauma, periodontal disease or tooth decay</b>	<ul style="list-style-type: none"> <li>- 1/3 children with untreated class II experience trauma, but they are mostly minor.</li> <li>- Age &lt;9 y, previous trauma &amp; cl.II → 8.4x risk for trauma → tx indicated.</li> <li>- Burden, 1995: Lower lip interposition = most important risk factor for trauma.</li> <li>- Extreme OB can damage the palatinal tissue of the upper incisors → early loss of incisors → tx indicated.</li> <li>- Little / no impact of malocclusions on disease of teeth and supporting structures. → Oral hygiene is much more important.</li> <li>- Orthodontic treatment does not increase the chance of later periodontal problems.</li> <li>- Note: Prevention of root resorption. E.g. ectopic teeth.</li> </ul>

### Type of treatment: Evidence based selection

- Tx must be evidence-driven!
- Differentiate between statistical and clinical significance.



<b>Case reports</b>	<ul style="list-style-type: none"> <li>- Likely to have been selected, because they show the expected outcome.</li> </ul>
<b>Randomized clinical trial</b>	<ul style="list-style-type: none"> <li>- Gold standard for evaluation of clinical procedures.</li> <li>- Patients are randomly assigned in advance to alternative tx procedures.</li> <li>- Methods of randomization: <ul style="list-style-type: none"> <li>o Simple randomization (randomization lists)</li> <li>o Block randomization</li> <li>o Stratification</li> <li>o Minimization</li> <li>o Date of birth / gender ... are not appropriate for randomization</li> </ul> </li> <li>- Blinding and control of bias is essential.</li> <li>- If the sample is large enough → similar distribution of all variables between the groups.</li> </ul>
<b>Meta-analysis, multiple trials</b>	<ul style="list-style-type: none"> <li>- Group the data from several studies of the same phenomenon.</li> </ul>
<b>Cross-sectional studies</b>	<ul style="list-style-type: none"> <li>- Larger sample needed.</li> <li>- Quick.</li> <li>- Individual variation is lost in a smoothed line by taking the average level.</li> </ul>
<b>Longitudinal study</b>	<ul style="list-style-type: none"> <li>- Retrospective or prospective.</li> <li>- Smaller sample needed.</li> <li>- Long observation time period.</li> <li>- Controls must be followed a long period of time equivalent to the tx time.</li> <li>- Cave: Sequential x-rays usually required → ethical not possible.</li> <li>All data for growth studies are based on x-rays from 50 years ago!</li> </ul>
<b>Data transformation</b>	<ul style="list-style-type: none"> <li>- Logarithmic or exponential calculations can shape a curve to a line. → Take in mind while analyzing statistics.</li> </ul>
<b>Evidence based orthodontics</b>	<ul style="list-style-type: none"> <li>- 3 piles: <ul style="list-style-type: none"> <li>o <b>Clinical expertise</b></li> <li>o <b>Evidence from the literature</b></li> <li>o <b>Patient's expectations and desire</b></li> </ul> </li> </ul>

Demand for treatment	
<b>PAR index</b> <i>Richmond et al., 1992</i>	<ul style="list-style-type: none"> <li>- Developed in the UK to evaluate tx results.</li> <li>- Score calculated from 11 measurements and a weighting scale for each characteristic:           <ol style="list-style-type: none"> <li>1. Mx and mn anterior alignment (crowding, spacing)</li> <li>2. Buccal segment occlusion (anteroposterior, transverse, vertical)</li> <li>3. OJ / reverse OJ</li> <li>4. OB</li> <li>5. Midline discrepancies</li> </ol> </li> <li>- The relative importance of each criteria evaluated by a panel of 74 members.</li> <li>- Models are rated before and after tx:           <ul style="list-style-type: none"> <li>o Improvement = min. 30% increase of the PAR score</li> <li>o Great improvement = <math>\geq + 22</math> PAR score points</li> <li>o Good orthodontist:               <ul style="list-style-type: none"> <li>▪ In general, &gt; 70% increase of the PAR score</li> <li>▪ Few / no case without increasement of the PAR score or worsening of the PAR score.</li> </ul> </li> </ul> </li> </ul>
<b>ABO index</b> <i>Developed 1995, latest revision 2012</i>	<ul style="list-style-type: none"> <li>- Developed in the States.</li> <li>- Calculation similar to the PAR index with addition of cephalometric variables.</li> <li>- Points considered: (8 points) Penalty score for deviations from the optimum.           <ul style="list-style-type: none"> <li>o Alignment (line of the dentition)</li> <li>o Sagittal molar relationship</li> <li>o Marginal ridges</li> <li>o Inclination</li> <li>o Occlusal contact of all functional cusps</li> <li>o Interproximal contact of all teeth</li> <li>o OJ</li> <li>o Root parallelism in the OPT</li> </ul> </li> </ul>
<b>IOTN</b> <i>Shaw, 1989</i>	<ul style="list-style-type: none"> <li>- Developed in the UK.</li> <li>- US: Tx is paid if <math>IOTN \geq 3.6</math>. (<math>DHC &lt; 4</math>, <math>AC &lt; 7</math> are not eligible for tx)</li> <li>- <b>Dental health component:</b> Derived from the occlusion and alignment: MOCDO. The most severe occlusal trait is identified and defines the category of the DHC.           <ul style="list-style-type: none"> <li>o Missing teeth</li> <li>o OJ</li> <li>o Crossbite</li> <li>o Displacement</li> <li>o OB</li> </ul> </li> <li>- Five grades for dental health component:           <ol style="list-style-type: none"> <li>1. no need</li> <li>2. mild / little need</li> <li>3. moderate / borderline need</li> <li>4. severe / need treatment</li> <li>5. extreme / need treatment</li> </ol> </li> <li>- <b>Esthetic component:</b> Derived from comparison of the dental appearance to 10 standard photographs. The EC is used only for category 3, to create 10 subgroups.</li> </ul>
<b>Aesthetic component</b> <b>1-4:</b> no / slight need  <b>5-7:</b> moderate / borderline need  <b>8-10</b> definite need for tx	<p>The image shows a 5x2 grid of dental photographs, labeled 1 through 10. Each photograph displays a different dental aesthetic issue, such as crowding, spacing, or misalignment. The first four photographs (1-4) represent 'no / slight need' for treatment. The next three (5-7) represent 'moderate / borderline need'. The last three (8-10) represent 'definite need for tx'.</p>

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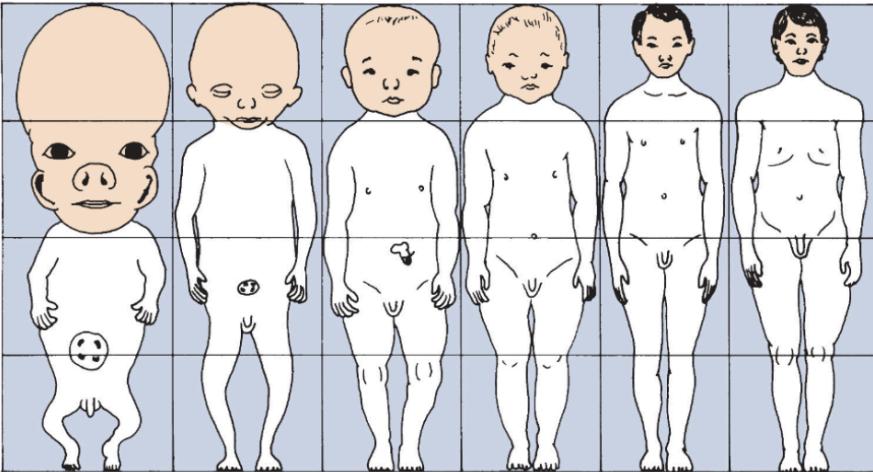
	<p><b>Grade 2 (Mild/Little Need)</b></p> <table border="1"> <tr><td>2.a</td><td>Increased overjet greater than 3.5mm but less than or equal to 6mm with competent lips.</td></tr> <tr><td>2.b</td><td>Reverse overjet greater than 0mm but less than or equal to 1mm.</td></tr> <tr><td>2.c</td><td>Anterior or posterior crossbite with less than or equal to 1mm discrepancy between retruded contact position and intercuspal position.</td></tr> <tr><td>2.d</td><td>Contact point displacements greater than 1mm but less than or equal to 2mm.</td></tr> <tr><td>2.e</td><td>Anterior or posterior open bite greater than 1mm but less than or equal to 2mm.</td></tr> <tr><td>2.f</td><td>Increased overbite greater than or equal to 3.5mm without gingival contact.</td></tr> <tr><td>2.g</td><td>Prenormal or postnormal occlusions with no other anomalies.</td></tr> </table> <p><b>Grade 1 (No Need)</b></p> <table border="1"> <tr><td>1.</td><td>Extremely minor malocclusions, including contact point displacements less than 1mm.</td></tr> </table> <p>- Tx sometimes move individuals only from the severe group to the mild tx needed group and does not eliminates all the characteristics of the malocclusion.</p>	2.a	Increased overjet greater than 3.5mm but less than or equal to 6mm with competent lips.	2.b	Reverse overjet greater than 0mm but less than or equal to 1mm.	2.c	Anterior or posterior crossbite with less than or equal to 1mm discrepancy between retruded contact position and intercuspal position.	2.d	Contact point displacements greater than 1mm but less than or equal to 2mm.	2.e	Anterior or posterior open bite greater than 1mm but less than or equal to 2mm.	2.f	Increased overbite greater than or equal to 3.5mm without gingival contact.	2.g	Prenormal or postnormal occlusions with no other anomalies.	1.	Extremely minor malocclusions, including contact point displacements less than 1mm.
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Who seeks tx	<ul style="list-style-type: none"> <li>- No tx: People do not recognize that they have a problem, cannot afford / obtain tx.</li> <li>- Family income = major determinant for tx.</li> <li>- ~35% demand for orthodontic tx under optimal economic conditions. → Many of these patients with dentofacial conditions within the normal range of variation.</li> <li>- Good facial appearance &amp; no disfiguring dental conditions are associated with more prestigious social positions: → High aspirations for a child → parents seek orthodontic tx for a child.</li> <li>- 25-30% adults in orthodontic cabinet: They did not get tx in childhood, orthodontic appliances are socially more accepted.</li> <li>- Some extend of "enhancements" tx accepted: = interventions to make an individual "better than well" or "beyond normal".</li> <li>- <u>What is considered nice in facial attractiveness:</u> <ul style="list-style-type: none"> <li>• Esthetics which is close to the average which we know.</li> <li>• Symmetry.</li> <li>• Photographs are ranked the same way in different societies → biological influence.</li> <li>• Ask patients before tx what they bother about the most. → Communication = key to treat the patients!</li> </ul> </li> </ul>																

#### Schlundbögen, eng. pharyngeal arches: (unvollständige Auflistung der entstehenden Strukturen)

- 1: Mandibularfortsatz  
Meckelknorpel  
Hammer  
Amboss
- 2: Steigbügel  
Proc. Styloideus  
Zungenbein kleines Horn  
Lig. Stylohyoideum
- 3: Zungenbein grosses Horn
- 4: Schildknorpel
- 5: Ringknorpel
- 6: Verkümmert im Verlauf der Entwicklung

## Proffit Chapter 2:

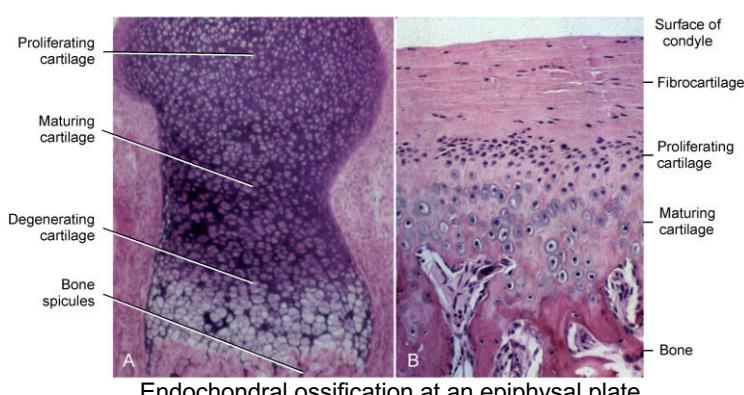
### Concepts of Growth and Development

Growth: Pattern, variability, and timing	
<b>Growth</b>	= Increase in size or number. Anatomic phenomenon.
<b>Development</b>	= Increase in complexity, specialization → loss of potential. Physiologic and behavioral phenomenon.
<b>Pattern</b>	= Represents proportionality or changes of proportional relationships over time.
Development body proportions	<p>- Head to body relationship:</p>  <p>The diagram illustrates the progression of human development through six stages: 2 Month fetus, 4 Month fetus, Birth, 2 Years, 12 Years, and 25 Years. Each stage is represented by a figure standing in front of a vertical grid. The grid shows the relative size of the head and body at each stage. The head is largest in the fetus and gradually decreases in relative size as the body grows. The 25-year-old figure shows a more balanced head-to-body ratio compared to the fetal stages.</p> <ul style="list-style-type: none"> <li>• <b>3<sup>rd</sup> m intrauterine:</b> <ul style="list-style-type: none"> <li>○ Head = <b>50%</b> = <math>\frac{1}{2}</math> of the total body length.</li> <li>○ &gt;50% cranium of total head, VC : NC = <b>1 : &gt;1</b></li> </ul> </li> <li>• <b>Birth:</b> <ul style="list-style-type: none"> <li>○ Head = <b>25%</b> = <math>\frac{1}{4}</math></li> <li>○ Face and jaws relatively underdeveloped compared with the cranium.</li> <li>○ Relationship viscerocranum : neurocranium = <b>1 : 8</b></li> <li>○ Head = <b>45</b> bones.</li> </ul> </li> <li>• <b>Adult:</b> <ul style="list-style-type: none"> <li>○ Head = <b>12%</b> = <math>\frac{1}{8}</math></li> <li>○ Head = <b>22</b> bones → <b>14</b> viscerocranum, <b>8</b> neurocranium.</li> <li>○ Relationship viscerocranum : neurocranium = <b>1 : 2.5</b></li> </ul> </li> </ul> <p><b>- Cephalocaudal</b>    <b>- gradient of growth:</b></p> <ul style="list-style-type: none"> <li>• = Axis of increased growth extending from the head towards the feet.</li> <li>• Explanation: Different tissues systems that grow at different rates are concentrated in various parts of the body.</li> <li>• Postnatal growth lower limbs &gt; upper limbs.</li> <li>• Mandible grows more and later than maxilla.</li> </ul> <p>- Maximal growth takes place in the first 2y of life.</p> <p>- Not all tissues systems of the body grow at the same rate:</p>

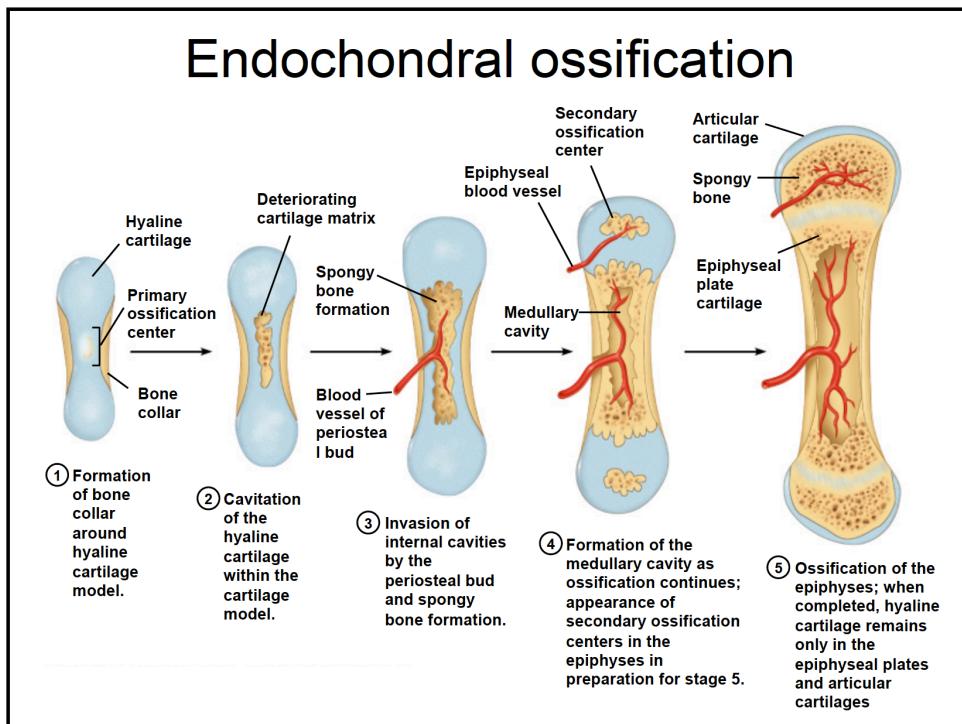
	<p>General = general body involving:</p> <ul style="list-style-type: none"> <li>• Bones</li> <li>• Muscles</li> <li>• Viscera</li> </ul>
	<ul style="list-style-type: none"> <li>- Muscular and skeletal elements grow faster than the brain and the central nervous system → reflected in the relative decrease of the head size after birth.</li> <li>- Maximum disparity in the development of different tissues systems occurs in late childhood.</li> <li>- Reasons for growth variations: <ul style="list-style-type: none"> <li>• Normal variation</li> <li>• Influences outside the normal (e.g. illness)</li> <li>• Timing effects</li> </ul> </li> <li>- Growth concepts <ul style="list-style-type: none"> <li>• <b>Predictability</b></li> <li>• <b>Variability:</b> Decide if an individual is at the extreme of the normal variation or outside the normal range.</li> <li>• <b>Timing:</b> The same event happens for different individuals at different times: = the biologic of different individuals is set differently. → Better us the developmental age than chronological age to reduce timing variability. The same growth patterns are expressed at different times chronologically, but not physiologically.</li> </ul> </li> </ul>
Growth charts	<ul style="list-style-type: none"> <li>- Standard growth charts (m/f) to evaluate a given child in relation to his peers.</li> <li>- Age vs. height / weight.</li> <li>- 30% percentiles from down = 30% of the children are smaller</li> <li>- 50% percentile = 50% of the children are in this range. (but don't have to have exactly this height/weight)</li> <li>- Deviations from the usual pattern are visible and quantitative expressed.</li> <li>- Children outside the range of &lt;3%   &gt;97% → Special examination required before accept them just as an extreme.</li> <li>- In the first 1-2y of life changes of the percentile are normal, the child establishes his growth. (<i>Mullis, 2009</i>)</li> <li>- Later, a child's growth should plot along the same percentile line. → Prominent changes are indices for growth abnormality.</li> </ul> <ul style="list-style-type: none"> <li>- Sexual maturation is accompanied by a growth spurt: <ul style="list-style-type: none"> <li>→ Ask parents if they had to buy new shoes, clothes (trousers)...</li> <li>→ Ask parents to measure the child regularly.</li> </ul> </li> <li>- <i>Hägg, 1980:</i> Onset of menstruation is after the growth peak, the growth rate is descending. Pubertal voice change happens for boys at the beginning of the growth spurt.</li> <li>- <i>Mullis, 2009:</i> <ul style="list-style-type: none"> <li>○ Average for menarche in Switzerland = 13.7 y.</li> <li>○ Puberty onset girls: 10.7 y (8-14 y). Start = breast development.</li> <li>○ Puberty onset boys: 11.7 y (9-15 y). Start = testis size ≥ 4 ml.</li> <li>○ Obese children = early puberty = shorter body length.</li> <li>○ Minimum body fat mass required for menarche.</li> <li>○ Growth &lt;4 mm/y needs special examination.</li> <li>○ <b>Prognostic body height = (Mother + Father ± 13 cm)   2</b> (boy + 13 cm, girl -13 cm)</li> </ul> </li> <li>- The earlier the adolescent growth spurt occurs, the more intense it appears to be. The later the menarche, the less intense (smaller) the growth spurt.</li> <li>- Intensity of growth spurt is very different from one patient to the other.</li> <li>- Velocity curve (age vs height <i>gain</i> in cm) show accelerations and decelerations better than distance curves (age vs height) → clarify growth changes by varying the method of display.</li> <li>- Mathematical transformation (e.g. log-function) can help to understand data easier and to find a correlation.</li> <li>- Skeletal cl.II: Optimum time for tx = peak of growth spurt. If you treat before, most of the effect is only dental.</li> <li>- Skeletal cl.III: Growth until 18/21 y. Little growth takes place also later, but in a much smaller amount.</li> </ul>

Methods for studying physical growth:	
<b>Craniometry</b>	= Measurements on dry skulls. <ul style="list-style-type: none"> <li>- Precise.</li> <li>- Cross-sectional: The same individual can be measured only once.</li> <li>- Variability within the sample can conceal details of the growth pattern.</li> </ul>
<b>Anthropometry</b>	= Landmarks established on dry skulls measured in living individuals by using soft tissue points overlying the bony landmarks. <ul style="list-style-type: none"> <li>- Cave: Soft tissue introduces variation!</li> <li>- Longitudinal: An individual's growth can be followed directly, making the same measurements at different times.            → A lot of information, relatively small numbers of subjects necessary, individual's variations can be highlighted.</li> </ul>
<b>Cephalometric radiology</b>	= Direct measurement of bony skeletal dimensions over time with precise head orientation and control of magnification. <ul style="list-style-type: none"> <li>- Superimposing of x-rays possible to detect changes.</li> <li>- Cave: Only 2D.</li> </ul>
<b>3D imaging</b>	<ul style="list-style-type: none"> <li>- <b>Axial CT</b> (computed axial tomography): 3D reconstruction of the cranium &amp; face.</li> <li>- <b>CBCT</b> (cone beam computed tomography): cheaper, less radiations than CT.</li> <li>- <b>MRI</b>: no radiation, soft tissue better represented than hard tissue.</li> <li>- <b>3D photography</b>: measurements of facial soft tissue dimensions and changes.            Cave: No stable structures for superimposition.</li> </ul>
<b>Vital staining</b>	= Dyes that stain mineralizing tissues / soft tissues are injected into an animal. <ul style="list-style-type: none"> <li>E.g.: <b>alizarin</b> (discovered by Mr. Hunter, marks regions with active growth, tetracycline, radioactive markers).</li> <li>- Not possible to perform in humans for diagnostics. (can occur inadvertently)</li> </ul>
<b>Radioactive tracers</b>	<ul style="list-style-type: none"> <li>- Becomes incorporated in tissues e.g. technetium 99m.</li> <li>- Indication: diagnosis of localized growth problems.</li> <li>- Not suitable for growth studies in general.</li> </ul>
<b>Implant radiography</b>	<ul style="list-style-type: none"> <li>- Insert metal pins in the skeleton → incorporated in the bone → visualized on x-rays. (Arne Björk, Copenhagen)</li> <li>- Large increase in the accuracy of a longitudinal cephalometric analysis.</li> </ul>
Genetic influences on growth	<ul style="list-style-type: none"> <li>- Important <b>homeobox genes</b>:            = Genes with a DNA binding domain → activation of gene expression.            The activation of the homeobox genes is regulated by a gradient of morphogenes.           <ul style="list-style-type: none"> <li>• <b>Msx-1</b>:                Tooth formation                Basal bone                Formation of palatal shelves</li> <li>• <b>Msx-2</b>:                Alveolar process</li> <li>• <b>Dix-1, Dix-2</b>:                Dental mesenchyme                Epithelium of mx and mn arch mesenchyme</li> </ul> </li> <li>- Expression of <b>BMP4</b> and <b>FGF8</b> at the proximal resp. distal processus of the mandible, allows the formation of different tooth types at different locations. The growth factors repress reciprocal their expression.</li> <li>- If the <b>Hedgehog pathway</b> activity is decreased, it causes holoprosencephalic and hypotelorism</li> <li>- <b>Hedgehog signaling</b> acts at two distinct steps in <b>disk morphogenesis: condyle initiation and disc-condyles separation</b> during the formation of the TMJ.</li> <li>- Gene expression can be upregulated and downregulated by mechanical stresses            → Direct: via the integrin network.            → Indirect: Opening / closure of ion channels → paracrine signal.            → Interaction between different tissues can regulate growth and development.            (e.g. muscles that attaches to the mandible)</li> <li>- Growth factors &amp; cognate receptors regulate embryonic processes for cell growth and organ development + postnatal growth, wound healing, bone remodeling, homeostasis.</li> <li>- Some gene locations for class III and PFE have already been identified.</li> <li>- 25'000 genes are important in craniofacial development.            → Unlikely that genetic analysis will become important for ordinary tx planning, but some important information can be obtained for difficult skeletal malocclusions.</li> </ul>

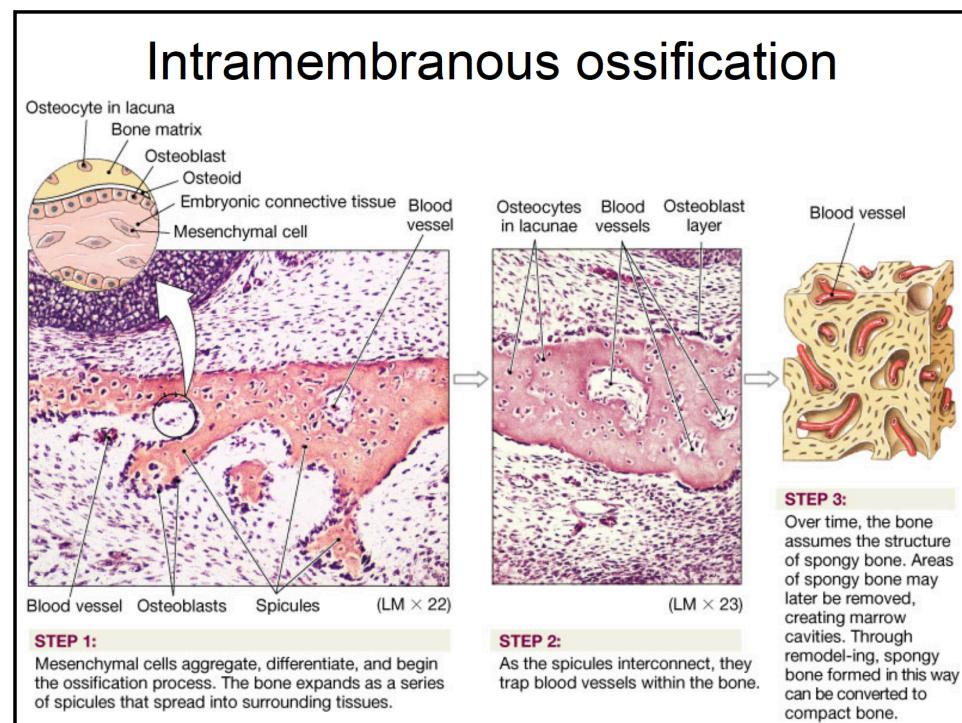
The nature of skeletal growth	
<b>Hypertrophy</b> <b>Hyperplasia</b> <b>Secretion of EZ-material</b>	= Increase in the size of an individual cell. = Increase in the number of cells. = Increase in size independent of the number or size of the cell themselves. → All 3 processes are involved in skeletal growth.
<b>Interstitial growth</b>	- Growth of the soft tissue (cartilage mostly behaves like soft tissue). - Occurs by a combination of hyperplasia and hypertrophy always within the tissues. - Impossible when mineralization takes place or only at the surface, not within the mineralized mass. E.g. on the periosteum of the bone. → Direct / surface apposition = formation of new cells in the periosteum, secretion of EZM, mineralization and formation of new bone. - Prominent aspect of overall skeletal growth because a major portion of the skeletal system is originally modeled in cartilage.
<b>Enchondral ossification</b>	= Transformation from cartilage into bone.  - Process of enchondral ossification: 1. Formation of a bone collar around a hyaline cartilage model. 2. Cavitation of the hyaline cartilage within the cartilage model. 3. Invasion of internal cavities by the periosteal bud and spongy bone formation. 4. Formation of the medullary cavity as ossification continues: appearance of secondary ossification centers in the epiphyses in the preparation for stage 5. 5. Ossification of the epiphyses: when completed, hyaline cartilage remains only in the epiphyseal plate and articular cartilage.  - Examples: o Bones that participate in joints and bear weight o Condylus o Cranial base  - <u>3<sup>th</sup> month intrauterine:</u> Development of a cartilage skeleton of the chondrocranium. Cartilage = nearly avascular tissue, internal cells are supplied by diffusion through the outer layer → thickness is limited.  - <u>4<sup>th</sup> month intrauterine:</u> Ingrowth of vascular elements → formation of centers of ossification. Cartilage continues to grow, but is replaced by bone. Areas of cartilages from the old chondrocranium may persist between large sections of bones.  - <u>Bones of extremities:</u> Areas of ossification are in the center of the bones ( <b>diaphysis</b> dt. Schaft) and at both ends ( <b>epiphysis</b> ). Uncalcified cartilage between epiphysis and diaphysis = <b>epiphyseal plate</b> = major center for growth <ul style="list-style-type: none"><li>• Zone of dividing cartilage cells at the end of each epiphyseal plate.</li><li>• Cells are pushed towards the diaphysis → hypertrophy → secrete EZM → degenerate when the matrix mineralizes → are replaced by bone.</li></ul>



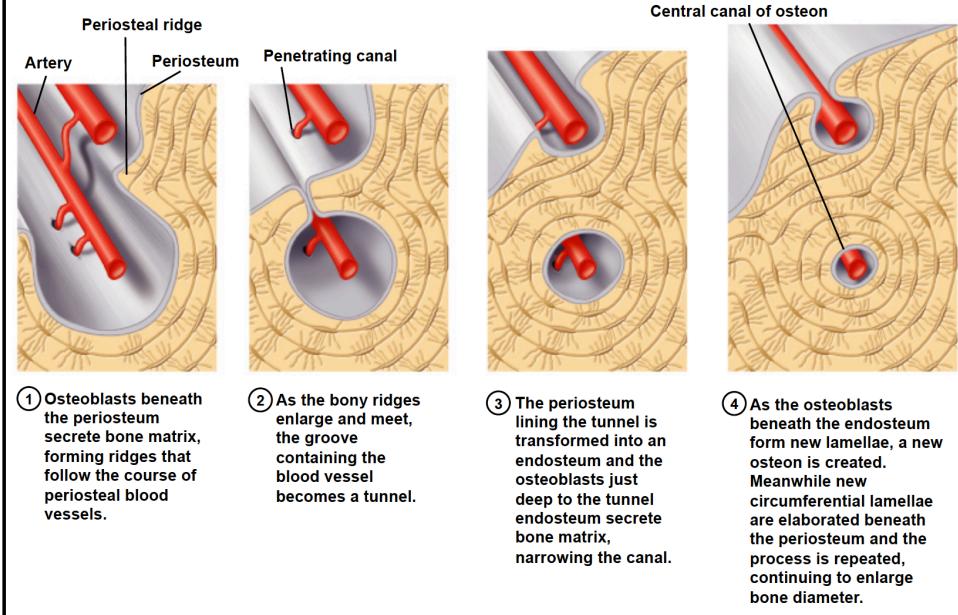
	<ul style="list-style-type: none"> <li>- Growth is possible as long as the cartilage cells proliferation rate is equal or greater than the rate of maturation.</li> <li>- The epiphyseal plates disappear when all the cartilage is replaced by bone. → Growth of the bones is completed except the thickness, which can be altered by the periosteum at the surface.</li> <li>- Fibrocartilage (in the condyle) = 2<sup>nd</sup> cartilage = no primary growth potential, only reactive growth is possible.</li> <li>- Cartilage specific collagen: <b>Type 2,9,11.</b></li> </ul>
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<b>Intramembranous ossification =mesenchymal</b>	<ul style="list-style-type: none"> <li>- = Bone formation (for example by fibroblasts) by secretion of bone matrix directly within connective tissues without any intermediate formation of cartilage.</li> <li>- E.g. cranial vault or both jaws.</li> </ul>
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# Appositional bone growth

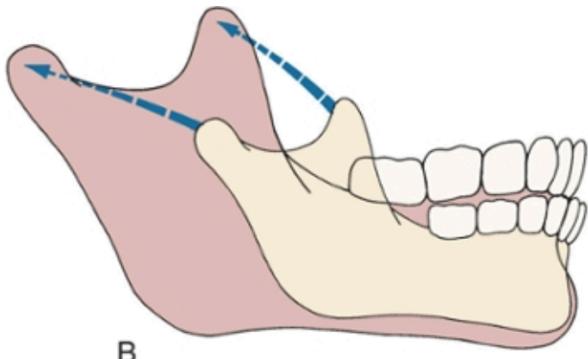


<b>Development of the mandible</b>	- Begins with mesenchyme condensation lateral to the <b>Meckel's cartilage</b> (cartilage of the 1 <sup>st</sup> pharyngeal arch) and proceeds by intramembranous bone formation. - Meckel's cartilage largely disappears as the bony mandible develops. Some remnants (Hammer & Amboss = incus & malleus) in the middle ear. - Condylar cartilage initially develops as secondary cartilage independent and separated → fusion in early fetal life with the mandible.
<b>Development of the maxilla</b>	- Center of mesenchymal condensation in the maxillary process on the lateral surface of the nasal capsule. - Change of shape by resorption and apposition: modeling & remodeling. - Interstitial growth within the mineralized mass is impossible.
Intramembranous	
<b>Modeling</b>	- = Formation of new bone from a cartilaginous predecessor or direct bone formation within the mesenchyme. Adapts structure to function by changing bone size and shape to maintain bone strength when the loading of the bone changes.
<b>Remodeling</b>	- = Changes in the shape of a bone due to resorption and replacement.

Sites and types of growth in the craniofacial complex	
Factors to differentiate growth	<ul style="list-style-type: none"> <li>• Site or location of growth</li> <li>• Type of growth</li> <li>• Mechanism of growth</li> <li>• Determinant or controlling factors</li> </ul>
<b>Wolff's law</b>	<ul style="list-style-type: none"> <li>- = Form follows function.</li> <li>- Correlation between the trabecular orientation in the bone and the main direction of the stress + secondary adaptions of the cortical portion of the bone.</li> </ul>
<b>Cranial vault:</b>  Intramembranous	<ol style="list-style-type: none"> <li>1. Intramembranous bone formation</li> <li>2. <b>Fontanelles: anterior, posterior, sphenoid &amp; mastoid fontanelle</b> = Loose connective tissues that widely separate the bones of the skull at birth (for the deformation in the birth canal) Apposition of bone along their edges eliminates the open space between the fontanelles quickly after birth, but the bones remain separated by thin sutures and fuse eventually only in adult life.</li> <li>3. Remodeling and growth occur primarily at the periosteum lined contact areas between adjacent skull bones = the <b>cranial sutures</b>.</li> </ol> <ul style="list-style-type: none"> <li>- Only responsive growth: Sutures themselves without pressure have no growth potential.</li> <li>- Change in contour: Little bone is removed from the inner surface of the cranial vault, while at the same time new bone is added on the exterior surface.</li> </ul>
<b>Cranial base</b> (occipital, sphenoid and ethmoid bones = bony floor under the brain)  endochondral  intramembranous (only lateral)	<ul style="list-style-type: none"> <li>- Enchondral ossification: bones are initially formed in cartilage and transformed from this model into bone.</li> <li>- <b>Synchondrosis</b>: = band of cartilage that remains between the centers of ossification. Important growth sites: <ul style="list-style-type: none"> <li>• <b>Spheno-occipital synchondrosis</b></li> <li>• <b>Intersphenoid synchondrosis</b></li> <li>• <b>Spheno-ethmoidal synchondrosis</b></li> </ul> </li> <li>- Synchondrosis are built like a two-sided epiphyseal plate: Band of immature proliferation cartilage cells at the center. Bands of maturing cartilage cells and enchondral ossification in both directions.</li> <li>- An immovable joint develops between the bones of the cranial base</li> </ul>
<b>Maxilla (Nasomaxillary Complex)</b>  Intramembranous  <b>Primary translation:</b> = Translation due to the growth of the bone itself.  <b>Secondary translation:</b> = Translation of the bone due to growth of the surrounding structures.	<ul style="list-style-type: none"> <li>- Intramembranous ossification: <ul style="list-style-type: none"> <li>• Apposition of bone at the sutures that connect the maxilla to the cranium and cranial base.</li> <li>• Surface modeling by apposition and resorption</li> <li>• → Growth: Upward &amp; backward. → Movement: Downward &amp; forward.</li> </ul> </li> <li>- Sutures: (nach Sicher, ausg. S. frontonasalis) <ul style="list-style-type: none"> <li>○ <b>S. zygomaticotemporalis</b></li> <li>○ <b>S. zygomaticomaxillaris</b></li> <li>○ <b>S. frontonasalis</b></li> <li>○ <b>S. frontomaxillaris</b></li> <li>○ <b>S. pterygopalatine</b></li> </ul> </li> </ul>

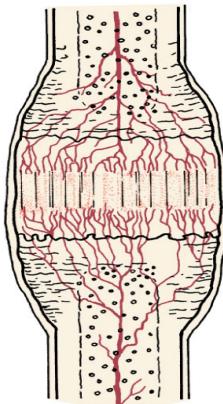
	<ul style="list-style-type: none"> <li>- <u>Until age 6:</u> <ul style="list-style-type: none"> <li>• Growth of the cranial base and growth of the structures behind the maxilla, move the maxilla forward &amp; downward (secondary translation).</li> <li>• New bone is added on both sides of the sutures (primary translation)</li> <li>• The anterior surface (except anterior nasal spine) tends to resorb while the maxilla is carried forward &amp; downward.</li> <li>• A midface deficiency develops in case of failure of the cranial base to lengthen normally (e.g. achondroplasia).</li> </ul> </li> <li>- <u>Age 7y:</u> <ul style="list-style-type: none"> <li>• Only growth at the sutures to push the maxilla further forward.</li> <li>• Sutures remain the same width, the space is filled by bone proliferation.</li> <li>• Various processes of the maxilla become longer: P. zygomaticus, P. frontalis, P. alveolaris.</li> </ul> </li> <li>- <u>Tuberosity region</u> at the posterior border of the maxilla (= free area): Bone is added at the surface, creating additional space for the primary and permanent molars.</li> <li>- <u>Palatal vault</u> (= roof of the mouth and floor of the nose) Translation and remodeling effects are additive: <ul style="list-style-type: none"> <li>• Forward-downward movement.</li> <li>• Bone removal on the nasal side and addition on the oral side.</li> </ul> </li> <li>- Cavity of the palatal plate develops because there is more apposition at the alveolar process than translation of the palatal floor.</li> <li>- Growth in width and length of the palatal develops because of the <b>growth of the suture palatina mediana</b> (posterior more than anterior) and the <b>sutura palatina transversa</b>.</li> <li>- <i>Björk &amp; Skieller, 1977: "Growth of the Maxilla in Three Dimensions as Revealed Radiographically by the Implant Method"</i> <ul style="list-style-type: none"> <li>• Increase in width 6/7y to adulthood: <ul style="list-style-type: none"> <li>◦ Suture: <b>+4.8 mm</b> (4y to adulthood: <b>+6.8 mm</b>)</li> <li>◦ Intermolar width: <b>+3.1 mm</b>.</li> <li>◦ Intercanine width: <b>+1.1 mm</b>. (4y to adulthood: <b>+3.1 mm</b>)</li> </ul> </li> <li>• Mx growth in the median suture: posterior &gt; anterior.</li> <li>• Strong correlation intermolar width &amp; sutural growth. Weaker correlation intercanine width &amp; sutural growth.</li> <li>• Molars compensate for the sutural growth: Increase intermolar width &lt; sutural growth.</li> <li>• Reduction of dental arch length: <ul style="list-style-type: none"> <li>◦ <b>-1.5 mm</b>: Anterior drift of the teeth.</li> <li>◦ <b>-1 mm</b>: Transversal expansion reduces the arch length.</li> </ul> <p>→ Incisors: <b>2.5 mm</b> more anterior positioned at adulthood. → Molars: <b>5 mm</b> more anterior positioned at adulthood.</p> </li> </ul> </li> <li>- <u>Summary: apposition:</u> <ul style="list-style-type: none"> <li>• Tuber (dorsal)</li> <li>• Orbital floor (cranial)</li> <li>• Palatal roof (caudal)</li> <li>• Molar and frontal alveolar process (caudal) and molar vestibular</li> <li>• Proc. zygomaticus (dorsal)</li> <li>• Nasal floor (palatal)</li> </ul> </li> <li>- <u>Summary: resorption:</u> <ul style="list-style-type: none"> <li>• Palatal roof (cranial)</li> <li>• Frontal vestibular alveolar process</li> <li>• Proc. zygomaticus (anterior)</li> </ul> <p>(Note: Ø sure if correct, anterior surface of the zygomatic arch = stable structure according to Björk)</p> <ul style="list-style-type: none"> <li>• Nasal floor (nasal)</li> </ul> </li> <li>- <u>Summary: translation nasomaxillary complex:</u> <ul style="list-style-type: none"> <li>• Maxilla: downward forward. (frontal resorption (exception spina nasalis ant.), apposition dorsal)</li> </ul> </li> </ul>
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	<ul style="list-style-type: none"> <li>Nasal floor: caudal. (resorption nasal, apposition palatal)</li> <li>Orbital floor: cranial. (apposition cranial, resorption caudal)</li> <li>Palate: caudal. (apposition palatal, resorption nasal)</li> <li>Processus zygomaticus: dorsal. (apposition dorsal, resorption anterior)</li> </ul>
<b>Mandible</b>	<p>Endochondral: condyles</p> <p>Periosteal: (intramembranous) all other areas</p> <ul style="list-style-type: none"> <li>- Enchondral activity (cartilage covers the surface of the mn condyle) and periosteal activity (all other surfaces). Displacement by the cranial base that moves the TMJ is only negligible.</li> <li>- Growth of the mn from the perspective of the stable cranial base: Chin moves downward and forward.</li> <li>- <u>Principal sites of growth:</u> <ul style="list-style-type: none"> <li>Posterior surface of the ramus (periosteal apposition) + resorption at the anterior surface of the ramus + modeling: → Mandible becomes longer.</li> <li>Coronoid processus</li> <li>Condylar processus (endochondral replacement + surface modeling): → Ramus grows higher.</li> <li>Posterior remodeling creates space for the second primary and the permanent molars. Early cessation of growth → not enough space for 8-8 → impaction.</li> <li>Minimal changes in the body and chin area.</li> <li>The chin is translated downward and forward but almost inactive.</li> </ul> </li> <li>- <u>Transversal growth:</u> <ul style="list-style-type: none"> <li>Up to 6<sup>th</sup> month of life: 2 bones.</li> <li>Age 6<sup>th</sup> y: Ossification of the midsymphysal cartilage.</li> <li>Age 4-10y: Mandibular condyles grow larger.</li> <li>Apposition vestibular at the alveolar process.</li> </ul> </li> <li>- <u>Summary: apposition:</u> <ul style="list-style-type: none"> <li>Posterior surface of the ramus</li> <li>Alveolar process frontal and molar side</li> <li>Inner side of the lateral side of the ramus</li> <li>Anterior side of the corpus vestibular</li> </ul> </li> <li>- <u>Summary: resorption:</u> <ul style="list-style-type: none"> <li>Anterior surface of the ramus</li> <li>Mandibular posterior bottom edge</li> <li>Vestibular frontal alveolar process</li> <li>Outside lateral side of the ramus</li> </ul> </li> </ul>



<b>Facial soft tissues</b>	<ul style="list-style-type: none"> <li>- Do not perfectly parallel the growth of the underlying hard tissues.</li> <li>- <u>Lips:</u> <ul style="list-style-type: none"> <li>• Train behind the growth of the jaws before adolescence, then undergo a growth spurt → lip height relatively short in the mixed dentition.</li> <li>• Lip separation at rest (lip incompetence) is maximal during childhood and decreases in adolescence.</li> <li>• Maximum lip thickness during adolescence, then decrease. → Esthetic problem for women.</li> <li>- <u>Upper lip adolescence and mid-adulthood:</u> (Vig, 1978)           <ul style="list-style-type: none"> <li>◦ Upper lip lengthening <b>3.2 mm</b></li> <li>◦ Thinner <b>3.6 mm</b></li> </ul> </li> <li>- <u>Upper lip mid- to late-adulthood:</u> (Vig, 1978)           <ul style="list-style-type: none"> <li>◦ Further <b>1.4 mm</b> lengthening and thinning of the upper lip</li> </ul> </li> <li>• Lips are framed by the nose and the chin which become both more prominent during adolescence and therefore decrease the relative prominence of the lips.</li> </ul> </li> <li>- <u>Growth of the nose:</u> <ul style="list-style-type: none"> <li>• Nasal bone: Growth completed at about age 10y. Afterwards only growth of the nasal cartilage and soft tissues: → Nose becomes more prominent in adolescence, esp. for boys.</li> </ul> </li> </ul>
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Theories of growth control	
Theories of growth control	<ol style="list-style-type: none"> <li>1. Bone is the primary determinant of its own growth. Genetic control is expressed directly at the level of bone in the periosteum. → Thought not to be true nowadays.</li> <li>2. Cartilage is the primary determinant of growth, bone responds secondarily and passively. This would mean that the genetic control is <b>epigenetic</b>. (= indirect control, whatever source it is)</li> <li>3. The soft tissues matrix is the primary determinant of growth, cartilage and bone responds secondary and passively = also epigenetic control.</li> </ol> <p>→ Theory 2 &amp; 3 are probably true.</p> <ul style="list-style-type: none"> <li>- Growth is influenced by <b>genetic factors</b> and <b>the environment</b>: Nutritional status, physical activity, health or illness.</li> </ul>
<b>Site of growth Center of growth</b>	= Location at which growth occurs. = Location at which independent (genetically controlled) growth occurs.
1. <b>Bone as primary determinant of growth</b>  → not true	<ul style="list-style-type: none"> <li>- Idea: <ul style="list-style-type: none"> <li>o The overall pattern of the craniofacial growth is very constant.</li> <li>o Sutures and endochondral ossification centers were considered growth centers.</li> </ul> </li> <li>- → Not true: <ul style="list-style-type: none"> <li>o Areas of sutures between two facial bones transplanted in another location do not continue to grow → lack of initial growth potential.</li> <li>o Sutures respond to outside influences.</li> </ul> </li> </ul>
2. <b>Cartilage as determinant of craniofacial growth</b>  → true	<ul style="list-style-type: none"> <li>- Idea: <ul style="list-style-type: none"> <li>o For many bones, cartilage grows, while bone merely replaces it.</li> <li>o Confirmation by transplantation experiments.</li> </ul> </li> <li>- Not all cartilage acts the same when transplanted: <ul style="list-style-type: none"> <li>o <u>Spheno-occipital synchondrosis</u>: YES Cartilage also grows when transplanted. → Capable of acting as growth center.</li> <li>o <u>Mandibular condyle</u>: NO Little or no growth observed when transplanted. Study: After fracture of the mandibular condyle with dislocation in children, an excellent chance exists that a new condylar process will regenerate from the periosteum to approximately the same size and that the dislocated old condyle is resorbed. Only 15-20% of children suffer a reduction of growth after a condylus fraction. Whether it occurs, is a function of the severity of the soft tissue injury. → Mn condyle ≠ No growth center.</li> <li>o <u>Nasal septal cartilage</u>: PROBABLY YES Some growth potential when transplanted. Loss of maxilla growth in animal studies when the cartilage is removed. Cave: growth could also be impeded by scar tissue and bad blood supply.</li> <li>o <u>Epiphyseal plate</u>: YES Growth center.</li> </ul> </li> </ul>
3. <b>Functional matrix growth theory</b>  Introduced by Moss, 1960'  → true	<ul style="list-style-type: none"> <li>- Idea: Growth of the face occurs as a response to functional needs and neurotrophic influences. It is mediated by the soft tissue in which the jaws are embedded.</li> <li>- Experiments for support: <ul style="list-style-type: none"> <li>o Small brain = small skull. (microcephaly if pregnant women have a Zika virus infection)</li> <li>o Hydrocephalus (cerebrospinal fluid not resorbed) = big skull.</li> <li>o The size of the eyes determines the size of the orbits.</li> <li>o TMJ ankylosis impairs mandibular growth.</li> <li>o Bonegrowth can be induced at surgical created sites. (= distraction osteogenesis)</li> </ul> </li> </ul>

	<p>- <b>Distraction:</b></p> <ul style="list-style-type: none"> <li>○ = Induction of bone growth at surgically created sites.</li> <li>Start segment separation of 0.5-1 mm per day after a few days of initial healing and callus formation.</li> <li>○ Osteogenesis is possible.</li> </ul>  <ul style="list-style-type: none"> <li>• Fibrous radiolucent interzone with longitudinal oriented collagen bundles at the area of lengthening with proliferating fibroblasts and undifferentiated mesenchymal cells.</li> <li>• Osteoblasts at the edge of the interzone.</li> <li>• Rich blood supply in the mineralization zone at both sides of the interzone.</li> <li>• Zone of remodeling under the mineralization zone.</li> </ul>			
Summary	<ul style="list-style-type: none"> <li>- Growth of the cranium occurs almost entirely in response to growth of the brain.</li> <li>- Growth of the cranial base is primarily the result of endochondral growth and bony replacement at the synchondrosis (with individual growth potential). An influence of the brain is possible.</li> <li>- The maxilla is translated downward &amp; forward as the face is growing and remodeling. Influence of the growth of the cartilage is unknown. Soft tissues and cartilage can contribute to the maxilla forward translation.</li> <li>- The mandible is translated by growth of the muscles and other adjacent soft tissues. New bone at the condyle is added in response to soft tissue changes. Remodeling.</li> <li>- Functional need concept (<i>Piotr</i>):</li> <li>- = Structures adapt to the needs. (larger nostrils if more air is needed)</li> </ul>			
Growth of Craniofacial Units				
<b>Growth</b>	<b>Cranial Vault</b>	<b>Cranial base</b>	<b>Maxilla</b>	<b>Mandible</b>
<b>Sites</b>	<ul style="list-style-type: none"> <li>-Sutures (major)</li> <li>-Surfaces (minor)</li> </ul>	<ul style="list-style-type: none"> <li>-Synchondrosis</li> <li>-Sutures (laterally)</li> </ul>	<ul style="list-style-type: none"> <li>-Sutures</li> <li>-Surfaces</li> <li>-Apposition</li> <li>-Remodeling</li> </ul>	<ul style="list-style-type: none"> <li>-Condyle</li> <li>-Ramus</li> <li>-Other surfaces</li> </ul>
<b>Centers</b>	None	Synchondrosis	None	None
<b>Type (mode)</b>	Mesenchymal (=intramembranous)	<ul style="list-style-type: none"> <li>-Endochondral</li> <li>-Mesenchymal (later only)</li> </ul>	Mesenchymal	<ul style="list-style-type: none"> <li>-Endochondral (condyle only)</li> <li>-Mesenchymal</li> </ul>
<b>Mechanism</b>	Pressure to separate the sutures	Interstitial growth at synchondrosis	<ul style="list-style-type: none"> <li>-Cartilage push (cranial base)</li> <li>-Soft tissue pull</li> <li>-Cartilage pull? (nasal septum)</li> </ul>	Soft tissue pull (neurotrophic?)
<b>Determinant</b>	Intracranial pressure (brain growth)	<ul style="list-style-type: none"> <li>-Genetic (at synchondrosis)</li> <li>-Cartilage pull (at lateral sutures)</li> </ul>	Soft tissue pull (neurotrophic?)	Soft tissue pull (neurotrophic?)

Notes: Zellphysiologie	
Rezeptoren	<ul style="list-style-type: none"> <li>- Zelloberflächen Rezeptoren: für hydrophile Signalmoleküle, z.B. Wachstumsfaktoren.           <ul style="list-style-type: none"> <li>• Ionenkanäle</li> <li>• Enzym gekoppelt, z.B. RTK (receptor tyrosin kinase)</li> <li>• G-Protein gekoppelt → 2<sup>nd</sup> messenger z.B. cAMP, Ca<sup>2+</sup></li> </ul> </li> <li>- Kernrezeptoren: für hydrophobe Signalmoleküle, z.B. Steriodhormone, Transkriptionsfaktoren</li> </ul>
Zellkommunikation:	<ul style="list-style-type: none"> <li>- Endokrin: Hormone</li> <li>- Parakrine: Parakrine Faktoren, z.B. PDGF, TGF-B, Histamine</li> <li>- Neuronal: Neurotransmitter, z.B. Acetylcholin</li> <li>- Kontaktabhängig: Rezeptor-Ligand, z.B. RANK/RANKL</li> </ul>
Zellgedächtnis	<ul style="list-style-type: none"> <li>- Methylierung</li> <li>- Feedback Loops: Anhäufung von Transkriptionsfaktoren und den nachfolgen synthetisierten Proteinen.</li> </ul>
DNA Dogma	<ul style="list-style-type: none"> <li>- DNA → (Transcription) → RNA → (Translation) → Proteine.</li> <li>- DNA = Doppelstrang aus           <ul style="list-style-type: none"> <li>◦ Pyrimidin: Cytosin, Thymin → Uracil in RNA</li> <li>◦ Purin: Guanin, Adenin.</li> </ul> </li> <li>- Basenpaare:           <ul style="list-style-type: none"> <li>◦ G &amp; C</li> <li>◦ A &amp; T resp. U in RNA</li> </ul> </li> <li>- Introns &amp; Extrons.</li> <li>- Enhancer &amp; Silencer Sequenzen zur Regulierung der Transkription.</li> <li>- Differenzierte Zellen haben den gleichen Genom → alle Informationen für die Bildung eines kompletten Organismus.</li> </ul>
Translation	<ul style="list-style-type: none"> <li>- Freie Ribosome: Proteine für IZM.</li> <li>- Gebundene Ribosome: Proteine für Sekretion.</li> <li>- Findet im Zytoplasma statt.</li> <li>- 20 Aminosäuren.</li> </ul>
Transduktion	<ul style="list-style-type: none"> <li>= Umwandlung eines Signals in ein anderes Signal.</li> <li>- Ziel:           <ul style="list-style-type: none"> <li>• Verstärkung</li> <li>• Synergie: Mehrere Inputs für eine Reaktion nötig.</li> </ul> </li> </ul>
Main 2 <sup>nd</sup> messengers	<ul style="list-style-type: none"> <li>- cAMP</li> <li>- Ca<sup>2+</sup></li> </ul>
Gewebe	<ul style="list-style-type: none"> <li>- Verschiedene Zelltypen +</li> <li>- EZM:           <ul style="list-style-type: none"> <li>◦ Kollagen: Zug.</li> <li>◦ Proteoglykane: Kompression.</li> <li>◦ Glykoproteine: Linker / Zelladhäsion.</li> </ul> </li> </ul>
Zellverbindungen	<ul style="list-style-type: none"> <li>- Tight junctions: Dicht.</li> <li>- Adherence junctions: Verbindet Aktinbündel verschiedener Zellen oder Zelle/Matrix.</li> <li>- Desmosome: Intermediäre Filamente zwischen Zellen.</li> <li>- Gap junctions: Ionenpassag. Elektrische Koppelung von Zellen.</li> <li>- Hemidesmosome: Verankerung intermediärer Filamente einer Zelle zur Basalmembran.</li> </ul>
Zellentwicklung	<ul style="list-style-type: none"> <li>- Totipotent → pluripotent → multipotent → oligopotent → unipotent.</li> <li>- Stammzellen vs. differenzierte Zellen:           <ul style="list-style-type: none"> <li>◦ Potential ↑</li> <li>◦ Spezialisierung ↓</li> </ul> </li> </ul>

## Social and behavioral development

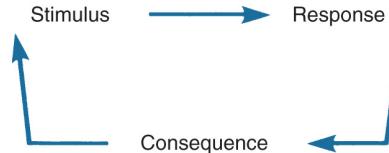
- Humans: Great majority of behaviors are learned and not instinctive → environment influences our behavior.
- Instinct behaviors (sex drive) can be modified by learning.
- The older the individual, the more complex the behavioral pattern and the more important the learned overlay of behavioral will be.
- More complex skills and behaviors appear as learning proceeds. Continues flow model, no distinct stages.
- Mechanisms to learn behavioral responses:
  - o Classical conditioning
  - o Operant conditioning
  - o Observational learning

### 1. Classical conditioning by Ivan Pavlov

- = **Learning by association:**  
Association of a neutral stimulus with one that leads to a specific reaction.
- **Generalization / Reinforcement:**  
Associations tend to become generalized and strengthened every time when the conditioned and unconditioned stimulus occur together.
- **Extinction of the conditioned behavior:**  
Association between the conditioned and the unconditioned stimulus is weakened or disappears when it is not reinforced.
- **Discrimination:**  
Contrary to generalization.
- Pain = unconditioned stimulus.  
Number of aspects of the setting in which pain occurs can become associated with this unconditioned stimulus.
- **Dentist:**
  - *Dental office should look different than a clinic.*
  - *Avoid pain at the first visit.*
  - *Let children look first at other's tx.*

### 2. Operant conditioning theory by B.F. Skinner

- Basic principle = the consequence of a behavior is in itself a stimulus that can affect future behavior. The consequence that follows a response will alter the probability of this response occurring again in a similar situation.



- Individual's determination is considered less important than the unconscious determinant behavior.
- Can be used to modify behavior at any age.
- Reinforcements vs. extinction and generalization vs. discrimination are possible like in classical conditioning.

	Probability of Response Increases	Probability of Response Decreases
Pleasant stimulus (S <sub>1</sub> )	I S <sub>1</sub> Presented Positive reinforcement or reward	III S <sub>1</sub> Withdrawn Omission or time-out
Unpleasant stimulus (S <sub>2</sub> )	II S <sub>2</sub> Withdrawn Negative reinforcement or escape	IV S <sub>2</sub> Presented Punishment

	<ul style="list-style-type: none"> <li>- 4 ways to react:</li> </ul> <ol style="list-style-type: none"> <li><b>1. Positive reinforcement:</b> If a pleasant consequence follows a response, the behavior that led to this pleasant consequence becomes more likely in the future. <i>E.g.: Gift after the dentist's visit.</i></li> <li><b>2. Negative reinforcement:</b> Withdrawal of an unpleasant stimulus after a response. Increases the likelihood of a response in the future. <i>E.g.: No tx for a crying child → cries again when it doesn't want a tx.</i></li> <li><b>3. Omission / time-out:</b> Removal of a pleasant stimulus after a particular response. Children regard attention by others as a pleasant stimulus. <i>E.g.: Withholding attention.</i> Cave: child may react with anger and frustration.</li> <li><b>4. Punishment:</b> An unpleasant stimulus is presented after a response. Effective at all ages. <i>E.g.: Voice control.</i> Cave: fear and anger from the patient.</li> </ol> <ul style="list-style-type: none"> <li>- Both reinforcements increase the likelihood of a particular behavior to reoccur, whereas punishment and omission suppress it.</li> <li>- Voice control: = Speaking to a patient in a firm and controlled voice to gain his attention. Form of punishment. Use with care. Immediate reward after an improvement is necessary.</li> </ul>
<b>3. Observational learning (modeling)</b>	<ul style="list-style-type: none"> <li>= Imitation of a behavior observed in a social context.</li> <li>- 2 stages: <ul style="list-style-type: none"> <li>• Acquisition: of the behavior by observing.</li> <li>• Performance: of that behavior.</li> </ul> </li> <li>- Not all observations are performed: <ul style="list-style-type: none"> <li>• Children can acquire almost any behavior which they observe if it's not too complex to perform for them at their physical level.</li> <li>• Role model: People who are liked or respected are more likely to be imitated by a child (siblings/mother).</li> <li>• Expected consequences of the behavior increase or decrease the likelihood to perform it. → No performance of a behavior if a punishment follows).</li> </ul> </li> <li>- Dentist: <ul style="list-style-type: none"> <li>• <i>Mother = Best predictor how anxious a child will be during tx.</i></li> <li>• <i>Open area with several tx stations → other patients are idols.</i></li> </ul> </li> </ul>

Stages of emotional development	
By Erik Erikson, based on Sigmund Freud	<ul style="list-style-type: none"> <li>- <b>"Eight ages of man"</b>: Psychosocial development proceeds by critical steps.</li> <li>- Turning points, moments of decision in between.</li> <li>- Each stage represents a psychosocial crisis in which individuals are influenced by their social environment to develop more or less towards one extreme of the conflicting personality qualities dominant at that stage.</li> <li>- Chronological age varies, but the sequence of the stages is constant.</li> <li>- Qualities of earlier stages will be evident in later stages due to uncompleted resolution of earlier stages.</li> </ul> <p style="text-align: center;"><b>ERIKSON'S "EIGHT AGES OF MAN"</b></p>
1: <b>Birth - 18 m</b>  <b>Development of basic trust vs. basic mistrust</b>	<ul style="list-style-type: none"> <li>- Depends on a caring and consistent mother or mother substitute, who meets the physiologic and emotional needs of an infant.</li> <li>- Physical growth can be retarded by severe "maternal deprivation".</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• <i>Child has separation anxiety → let parents hold the child during tx.</i></li> </ul> </li> </ul>
2: <b>18m - 3 y</b>  <b>Development of autonomy vs. shame / doubts</b>	<ul style="list-style-type: none"> <li>- Child is moving away from his mother, developing individual identity or autonomy.</li> <li>- If failure: development of shame / doubts in the child's mind about his ability to stand alone and doubts in others.</li> <li>- Parents must provide opportunities to develop independent behavior while protecting the child against consequences of dangerous and unacceptable behavior.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• <i>Offer choices if possible (colors).</i></li> <li>• <i>Make the child think whatever the dentist wants is the child's choice.</i></li> </ul> </li> </ul>
3: <b>3 – 6 y</b>  <b>Development of initiative vs. guilt</b>	<ul style="list-style-type: none"> <li>- Vigorous pursuit of activity of the child. Physical activity and motion, extreme curiosity and questioning, aggressive talking.</li> <li>- Parents must channel the activity in manageable tasks.</li> <li>- Child should express new thoughts and do new things without being made feel bad if it was a failure.</li> <li>- If failure: Guilt from goals that were contemplated but not attained.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• <i>Children are curious in the office.</i></li> <li>• <i>Success in the first appointment helps the child to become more independent.</i></li> </ul> </li> </ul>
4: <b>7 – 11 y</b>  <b>Mastery of skills: industry vs. inferiority</b>	<ul style="list-style-type: none"> <li>- Child works to acquire the academic and social skills that allow him to compete in an environment where recognition is given to those who produce. Child learns the rules by which our world is organized.</li> <li>- Influence of peer group increases.</li> <li>- Parents should present an environment that provides challenges with a chance of being met, rather than guarantee failure.</li> <li>- If failure: Inferiority.</li> <li>- Failure to measure up to a peer group: → personality characteristics of inadequacy, inferiority and uselessness.</li> </ul>

	<ul style="list-style-type: none"> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• <i>Setting attainable intermediate goals, clearly outlining how to reach them.</i></li> <li>• <i>Positively reinforce good behavior.</i></li> </ul> </li> </ul>
<b>5: 12 – 17 y</b>  <b>Development of personal identity vs. role confusion</b>	<ul style="list-style-type: none"> <li>- Includes a feeling of belonging to a larger group and a realization that one can exist outside the family.</li> <li>- Emerging sexuality.</li> <li>- Peer group members become important role models (although some distance necessary to establish one's own uniqueness). Values of the parents / authority figures are rejected.</li> <li>- Health problems considered as problems considered of somebody else.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• <i>Orthodontic tx only if the patient wants it, not only to please the parents.</i></li> <li>• <i>Outline the tx as being done FOR and no TO the patient.</i></li> </ul> </li> </ul>
<b>6: Young adult</b>  <b>Development of intimacy vs. isolation</b>	<ul style="list-style-type: none"> <li>- Success: Person is willing to compromise and even to sacrifice to maintain a relationship, establish affiliations and partnerships.</li> <li>- If failure: Isolation often accompanied by strong prejudices that serve to keep others away rather than bringing them into closer contact.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• <i>Patients think that a change in their appearance will facilitate attainment of intimate relationships.</i></li> <li>• <i>Inform patients that tx. includes all aspects of a person: appearance, personality, emotional qualities, intellect, psychological impact.</i></li> </ul> </li> </ul>
<b>7: Adult</b>  <b>Guidance of the next generation: generativity vs. stagnation</b>	<ul style="list-style-type: none"> <li>- Becoming a successful and supportive parent.</li> <li>- Do a service to the group community and nation.</li> <li>- If failure: stagnation characterized by self-indulgence and self-centered behavior.</li> </ul>
<b>8: Late adult</b>  <b>Attainment of integrity vs. despair</b>	<ul style="list-style-type: none"> <li>- Individual has adapted to the combination of gratification and disappointment.</li> <li>- Feeling that he has made the best of this life's situation and made peace with it.</li> <li>- If failure: Despair, disgust, unhappiness, fear that death will occur before a change of the life occurs.</li> </ul>

Cognitive development	
Theory by Jean Piaget:  4 stages with variable time frame	<ul style="list-style-type: none"> <li>= Development of intellectual capabilities.</li> <li>- Intelligence is a result of biologic adaptation. Every individual is born with the capacity to adjust or adapt to the physical and the sociocultural environment in which he lives.</li> <li>- Intelligence happens as an interplay between two processes: <ul style="list-style-type: none"> <li>• <b>Assimilation:</b> Child assimilates events into mental categories called cognitive structures = classifications for sensations and perception.</li> <li>• <b>Accommodation:</b> Occurs when the child changes the cognitive structures or mental categories to better represent the environment.</li> </ul> </li> <li>- A child's ability to adapt is age related.</li> </ul>
<b>1:</b>  <b>Birth – 2 y</b>  <b>Sensorimotor period</b>	<ul style="list-style-type: none"> <li>- Rudimentary concept of objects.</li> <li>- Ideal that objects in the environment are permanent. (disappear if you don't look at them)</li> <li>- Lack of language capability, simple modes of thoughts are the foundation for the development at this time.</li> <li>- Little ability to interpret sensory data.</li> <li>- Limited ability to project forward or backward in time.</li> </ul>
<b>2:</b>  <b>2 – 7 y</b>  <b>Preoperational period</b>	<ul style="list-style-type: none"> <li>- Children learn to use words to symbolize absent objects.</li> <li>- Use of language in a literal sense, understand words like they learn them.</li> <li>- Children fail to consider aspects such as function. → Understand some words differently than adults.</li> <li>- Children understand the world how they sense it through their 5 primary senses. → Everything else is difficult to understand, capability for logical reasoning is limited.</li> <li>- Child is ego-centered: incapable of assuming another person's point of view.</li> <li>- Animism: Everything is seen as being alive.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• Give dental instruments lifelike names and qualities.</li> <li>• Dental staff should use immediate sensation.</li> </ul> </li> </ul>
<b>3:</b>  <b>7 – 11 y</b>  <b>Period of concrete operations</b>	<ul style="list-style-type: none"> <li>- Improved ability to reason in concrete situations. Abstract level is limited.</li> <li>- Child can use a limited number of logical processes, especially if they involve objects which can be handled and manipulated.</li> <li>- Ability to see another point of view develops.</li> <li>- Animism declines.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• Instructions must be illustrated with concrete objects.</li> </ul> </li> </ul>
<b>4:</b>  <b>11 – adult</b>  <b>Period of formal operations</b>	<ul style="list-style-type: none"> <li>- Thought process has become similar to the one of adults.</li> <li>- Child understand concepts like health, disease...</li> <li>- They can think about thinking, but think that all the others think the same like they.</li> <li>- <b>Imaginary audience:</b> Teenagers feel that they are constantly observed and criticized by those around them → makes them self-conscious and particularly susceptible to peer influence.</li> <li>- <b>Personal fable:</b> Concept hold "because I'm unique, I'm not subject to the consequences other will experience → risky behavior.</li> <li>- <i>Dentist:</i> <ul style="list-style-type: none"> <li>• Child must be intellectually treated like an adult.</li> <li>• Do not try to impose a change of the reality as perceived by the adolescents, help them to see the actual reality that surrounds them more clearly.</li> <li>• Motivate patient to have a try and judge his peers' response.</li> <li>• Help teenager to solve their own problems.</li> </ul> </li> </ul>

## Proffit Chapter 3:

### Early Stages of Development

Late fetal development and birth	
Prenatal	<ul style="list-style-type: none"> <li>- Dental development starts in the 3<sup>rd</sup> month.           <ul style="list-style-type: none"> <li>• 6-8<sup>th</sup> pw: <b>Dental lamina</b> (Zahnleiste) = Initiation</li> <li>• <b>Bud</b> (Knospe) = Morphogenesis</li> <li>• 8<sup>th</sup> pw: <b>Cap</b> (Kappe) = Morphogenesis, start enamel secretion</li> <li>• 12<sup>th</sup> pw: <b>Bell</b> = Cytodifferentiation</li> <li>• &gt;12<sup>th</sup> pw: Matrixsecretion</li> </ul> </li> <li>- Development of all primary teeth and 6+6 starts before birth.</li> </ul>
Birth	<ul style="list-style-type: none"> <li>- Distortion of the head due to the fontanelles (anterior, posterior, sphenoid, mastoid) and missing mandible makes passage through the birth canal easier.</li> <li>- <b>Neonatal line:</b> Line seen in the enamel which is formed at the moment of birth up to a prominent area of stained, distorted or poorly calcified enamel. → Birth = traumatic process followed by a growth cessation and weight decrease in the first 7-10 days postnatal.</li> </ul> <p>The diagram illustrates the timeline of tooth development. It shows the sequence of tooth formation from birth to 3 1/2 years, with specific milestones for each tooth type: Central incisor, Lateral incisor, Cuspid, 1st molar, and 2nd molar. The timeline is divided into several periods: Prenatal period (in utero), Birth, Infancy period (birth to 10 months), Childhood period (10 months to 3 1/2 years), and Infancy ring (10 months). The graph on the left tracks the growth of boys from birth to 36 months, showing length and weight curves. The graph shows a significant increase in both length and weight during the first year, followed by a more gradual increase until 36 months.</p> <ul style="list-style-type: none"> <li>- Growth disturbances lasting 1-2 weeks or more will leave a visible record in the enamel of teeth forming at this time.</li> <li>- Extremely rapid growth in early infancy, with a progressive slowing down after the first 6 m postnatal. Growth curves for boy and girls are nearly identical at this age.</li> </ul>

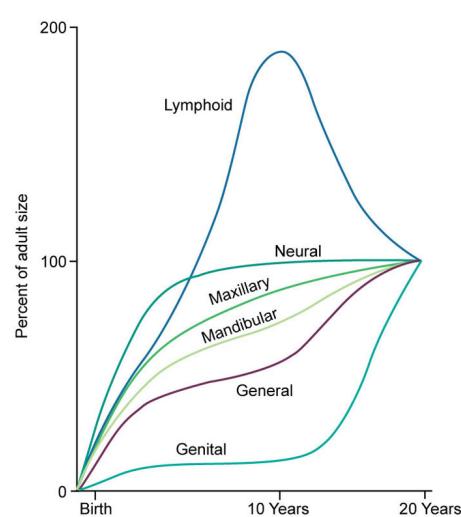
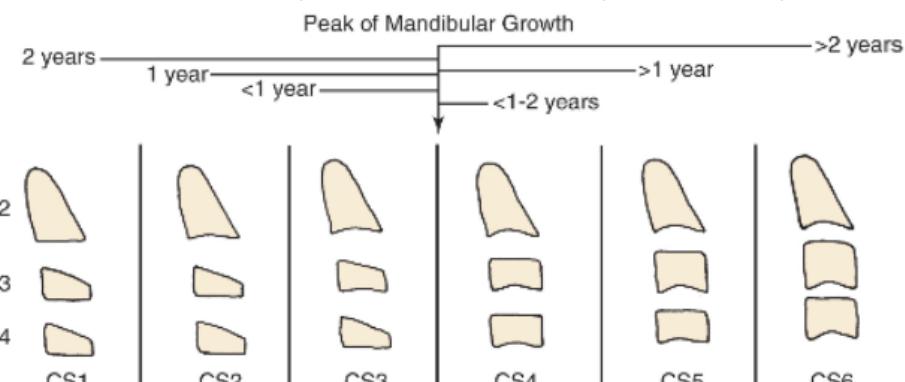
TABLE 3.1 Chronology of Tooth Development, Primary Dentition

Tooth	CALCIFICATION BEGINS		CROWN COMPLETED		ERUPTION		ROOT COMPLETED	
	Maxillary	Mandibular	Maxillary	Mandibular	Maxillary	Mandibular	Maxillary	Mandibular
Central	14wk in utero	14wk in utero	1½ mo	2½ mo	10mo	8mo	1½ yr	1½ yr
Lateral	16wk in utero	16wk in utero	2½ mo	3mo	11mo	13mo	2yr	1½ yr
Canine	17wk in utero	17wk in utero	9mo	9mo	19mo	20mo	3¼ yr	3¼ yr
First molar	15wk in utero	15wk in utero	6mo	5½ mo	16mo	16mo	2½ yr	2½ yr
Second molar	19wk in utero	18wk in utero	11mo	10mo	29mo	27mo	3yr	3yr

Infancy and early childhood: The primary dentition years (infancy = birth - 6 months)	
Premature birth (low birth weight)	<ul style="list-style-type: none"> <li>- &lt; 2500 gm at birth → greater risk of problems in the immediate postnatal period.</li> <li>- Normal growth and overcoming of the initial handicap if a premature infant survives. Children are smaller in the first 1-2 y of life, but at age 3y there is normally no difference visible to peer.</li> </ul>
	<p>Catch up growth small for gestational age / &lt; 1750 g</p>
	<p>Growth for boy with growth hormone deficit, tx at 6.2 y</p>
Chronic illness	<ul style="list-style-type: none"> <li>- Normal child: 90% energy is required for survival and activity, 10% for growth. → Less energy available with chronic illnesses.</li> <li>- Chronic illnesses cumulate the growth deficit: <ul style="list-style-type: none"> <li>• The more chronic the illness, the greater the cumulative impact.</li> <li>• The more severe the illness, the greater the impact at any given time.</li> <li>• Relatively brief growth interruptions have no long-term effect.</li> </ul> </li> <li>- Psychologic and emotional stress affects growth in extreme cases the same way as chronic illness (induction of a reversible growth hormone deficiency + disturbance of the appetite center).</li> </ul>
Nutritional status	<ul style="list-style-type: none"> <li>- Chronically inadequate nutrition has an effect similar to chronic illness.</li> <li>- Additional nutritional intake after achieving a certain level of nutritional adequacy is not a stimulus to more rapid growth. → Food is necessary for normal growth, but not a stimulus.</li> </ul>
Secular change in growth and development	<ul style="list-style-type: none"> <li>- Current trend towards more rapid growth and earlier maturation (menarche) is related to better nutrition (more protein, trace minerals &amp; vitamins) and exposure to environmental chemicals with an estrogenic effect (pesticides).</li> <li>- Head and face are becoming taller and narrower in the recent time. This is maybe related to softer food and less functional loading of the facial skeleton.</li> </ul>
Maturation of oral function	<ul style="list-style-type: none"> <li>- <u>Newborn infant:</u> <ul style="list-style-type: none"> <li>• Obligatory nasal breathers. To open the airways, the mandible is positioned downward, the tongue moves downward and forward away from the posterior pharyngeal wall.</li> <li>• Swallowing occurs in the last months of fetal life (amniotic liquid).</li> <li>• Drinking milk: Suckling and swallowing (no sucking!). → Stimulates the mammal's muscles to contract and to squirt milk. The newborn places the tongue in contact with the lower lips (most common position over the day) to receive milk and groove it, to make the milk flow posteriorly in the pharynx.</li> <li>• <b>Infantile swallow pattern:</b> <ul style="list-style-type: none"> <li>○ Active contraction of the musculature of the lips</li> <li>○ Tongue tip in contact with the lower lip</li> <li>○ Little activity of the posterior tongue or pharyngeal musculature</li> </ul> </li> <li>• Suckling reflex and infantile swallow pattern disappear normally during the first year of life.</li> </ul> </li> <li>- <u>Maturing infant:</u> <ul style="list-style-type: none"> <li>• Activity of the elevator muscles of the mandible increases with swallowing.</li> <li>• Use of the tongue in a more complex way.</li> <li>• Maturation of oral function follows a gradient form anterior to posterior: First sounds are formed anterior in the mouth m/p/b.</li> <li>• Mostly some sort of habitual non-nutritive suckling → transition in an adult swallowing pattern starts when the suckling activity stops.</li> <li>• Openbite: <ul style="list-style-type: none"> <li>○ Transition to an adult swallowing pattern is more difficult. Need to seal the anterior space.</li> <li>○ Tongue thrust does not cause an open bite.</li> </ul> </li> </ul> </li> </ul>

	<p>(to short duration of the interposition)</p> <ul style="list-style-type: none"> <li>○ Tongue thrusting is also possible with good anterior occlusion.</li> <li>○ Often tongue trust is necessary to adapt to an open bite.</li> </ul> <p>- <u><b>Chewing movement:</b></u></p> <ul style="list-style-type: none"> <li>○ The transition develops with eruption of the permanent canines.</li> <li>○ Adults with a severe anterior open bite (no canine function) do not achieve an adult chewing pattern.</li> </ul> <p style="text-align: center;">Chewing movements at the central incisor</p> <p style="text-align: center;">Cheese - left side Female, age 24</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="text-align: center; padding: 2px;">Young child</th><th style="text-align: center; padding: 2px;">Adult</th></tr> </thead> <tbody> <tr> <td style="text-align: center; padding: 2px;">1: Moving the mandible lateral with opening 2: Bring mn back to the midline 3: Close</td><td style="text-align: center; padding: 2px;">1: Open straight down 2: Move the jaw laterally 3: Bring the teeth into contact</td></tr> </tbody> </table>	Young child	Adult	1: Moving the mandible lateral with opening 2: Bring mn back to the midline 3: Close	1: Open straight down 2: Move the jaw laterally 3: Bring the teeth into contact
Young child	Adult				
1: Moving the mandible lateral with opening 2: Bring mn back to the midline 3: Close	1: Open straight down 2: Move the jaw laterally 3: Bring the teeth into contact				
<b>Eruption of primary teeth</b>	<ul style="list-style-type: none"> <li>- <b>Natal tooth:</b> Mostly a normal central incisor, not a supernumerary tooth. → Shouldn't be removed. (natal tooth = present at birth 1:2000-6000)</li> <li>- <b>Neonatal tooth</b> = Tooth that erupts in the first 30 days of life.</li> <li>- Eruption sequence is usually preserved.</li> <li>- Dates / timepoints are variable with a normal range up to 6 m: <ul style="list-style-type: none"> <li>○ Delay is usual for preterm infants.</li> <li>○ Ethical differences.</li> </ul> </li> <li>- <b>Primate space:</b> Maxilla between 2/3er, mandible between 3/4er.</li> <li>- Spacing in the anterior part is normal and becomes larger when the child grows and when the alveolar process expands. Spacing is required for proper alignment of the permanent incisors. <i>Stöckli 1994:</i> 70% children have spaces in the primary dentition. <ul style="list-style-type: none"> <li>○ Maxilla: 2.5 mm</li> <li>○ Mandible: 1.0 mm.</li> </ul> </li> <li>- The crowns of the permanent incisors lie lingual to the crowns of the primary incisors.</li> </ul>				

### Late childhood (5/6 y – puberty): The mixed dentition years

Physical development in late childhood (5/6 y)	<ul style="list-style-type: none"> <li>- Maximum disparity in the development of different tissue systems.</li> </ul>  <ul style="list-style-type: none"> <li>- 7y: Neural growth is essentially completed: = brain and brain case are as large as they will ever be.</li> <li>- Lymphoid tissues are beyond adult levels: = large tonsils and adenoids are common.</li> <li>- Growth of sex organs has hardly started.</li> <li>- Rate of general body growth declines from rapid growth at infancy and stabilizes at moderate lower level during late childhood.</li> </ul>
Hand x-rays	<ul style="list-style-type: none"> <li>- Ossification of the bones in the wrist, hand and fingers represent accurate a child's skeletal development status by comparing a patient's x-ray with a standard x-ray.</li> <li>- Today used however only in special circumstances. → The same information is available in lateral cephys regarding the cervical vertebrae.</li> </ul>
Cervical vertebral maturation  (Baccetti, Franchi & McNamara 2005)	<ul style="list-style-type: none"> <li>- Reliability has been questioned, but studies show a good intraobserver &amp; interobserver reliability and applicability. (Baccetti et al, 2015)</li> <li>- CVM is a better predictor for timing of the adolescence growth spurt than chronologic age.</li> <li>- Not valuable to determine if growth has ceased for teenagers with mn prognathisme.</li> </ul> <p style="text-align: center;">Peak of Mandibular Growth</p> 
Behavioral age	Important for dental tx.
Correlations	<ul style="list-style-type: none"> <li>- Development status with chronologic age 0.8 a. → <b>0.64</b> prediction reliability</li> <li>- Dental age with chronologic age 0.7 b. → <b>0.49</b> prediction reliability</li> <li>- <math>\chi^2</math> to predict one characteristic from another (a.: 0.64, b: 0.49).</li> <li>- Good correlation of development ages between themselves: → If advanced, a child is often advanced in different characteristics (skeletal age, weight, reading...).</li> </ul>

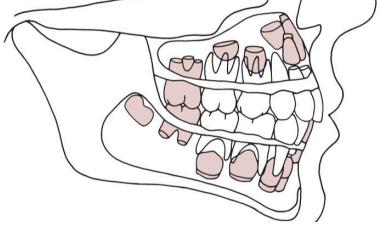
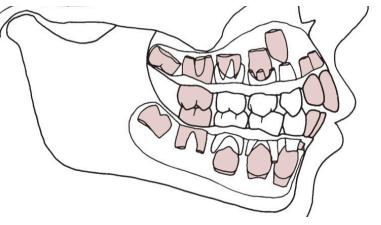
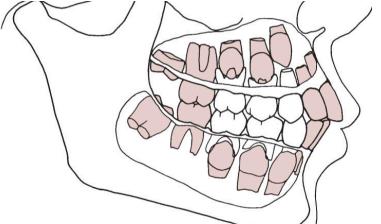
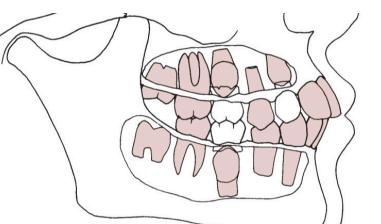
## Eruption of permanent teeth

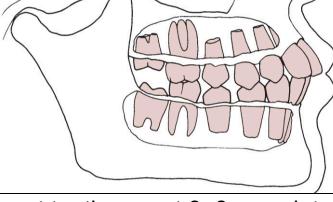
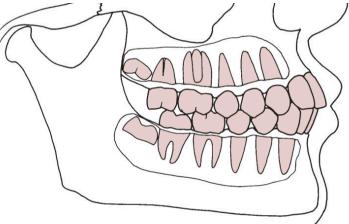
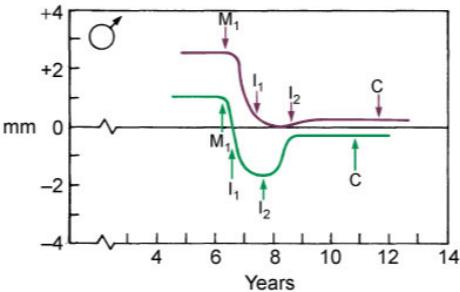
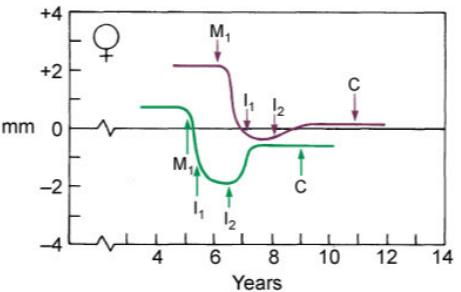
<p><b>1. Pre-emergent eruption</b></p>	<ul style="list-style-type: none"> <li>- Slow labial or buccal drift of a tooth follicle when the crown is being formed. Minimal extent. Does not contribute to eruption.</li> <li>- Eruptive movement starts when crown formation is finished &amp; root formation starts.</li> <li>- Tooth erupts when about ¾ of the root is completed.</li> </ul> <p>Two processes are necessary:</p> <ul style="list-style-type: none"> <li>- <u>I. Resorption of bone and primary tooth roots:</u> <ul style="list-style-type: none"> <li>o Limiting factor for pre-emergent eruption.</li> <li>o Completion of the crown is the start signal = Inhibition removal from: <ul style="list-style-type: none"> <li>▪ Layer of osteoclasts cranial of the tooth germ.</li> <li>▪ Genes necessary for root formation.</li> </ul> </li> <li>o Root formation is not necessary for eruption. <ul style="list-style-type: none"> <li>▪ If the tooth in a follicle is replaced by a metal piece, an occlusal drift will also occur.</li> <li>▪ Tooth with an already completed root can still erupt.</li> <li>▪ Tooth erupts also when the apex area is removed.</li> </ul> </li> </ul> </li> <li>- <u>II. Propulsive mechanism to move the tooth occlusal:</u> <ul style="list-style-type: none"> <li>o Precise mechanism through which the propulsive force is generated remains unknown. Different mechanism for the eruption pre and post-emergence.</li> <li>Theories: <ul style="list-style-type: none"> <li>▪ Cross-linking of maturing collagen in the periodontal ligament. (applies only to post-emergence eruption)</li> <li>▪ Localized variations in blood pressure or blood flow?</li> <li>▪ Forces from contraction of fibroblasts?</li> <li>▪ Alterations in the EZM of the periodontal ligament?!</li> <li>▪ Growth of the pulp and dentin?</li> <li>▪ Apical bone growth?</li> <li>▪ Change in relationship of pressure of the pulp vs. pressure PDL?</li> </ul> </li> <li>o At normal speed, the apex always rests at the same level.</li> <li>o If eruption is mechanically blocked, the proliferating apical area will move in the opposite direction → distortion of the root form = root dilacerations.</li> </ul> <ul style="list-style-type: none"> <li>• Normally the 2 processes operate in concert.</li> <li>• The 2 processes are not controlled physiologically by the same mechanism. → If a tooth is tied to the mandible the overlying bone resorptions proceeds at the usual rate although the tooth cannot erupt → formation of a cystic cavity.</li> </ul> </li> <li>- <u>Cleidocranial dysplasia:</u> <ul style="list-style-type: none"> <li>• Abnormal resorption of bone &amp; primary teeth → Ø eruption of succedaneous teeth.</li> <li>• Nonsuccedaneous tooth eruption is delayed by fibrotic gingiva.</li> <li>• Multiple supernumerary teeth.</li> <li>• Teeth can sometimes erupt spontaneously when the obstructions are removed. Application of orthodontic force is necessary if not.</li> </ul> </li> <li>- <u>Primary failure of eruption:</u> (<i>Frazier-Bowers, 2007</i>) FB says PTH1R, Ø PTHR1. <ul style="list-style-type: none"> <li>• Affected posterior teeth fail to erupt, because of a defect in the propulsive mechanism due to a mutation of the parathyroid hormone receptor gene 1 (<b>PTH1R</b>).</li> <li>• <b>Type 1:</b> Failure at the same time for all teeth → 2<sup>nd</sup> molar more affected than 1<sup>st</sup> molar.</li> <li>• <b>Type 2:</b> Gradient of time → central tooth (normally 1<sup>st</sup> molar) is normally the most severely affected.</li> <li>• <b>Non-syndromic PFE :</b> <i>Heterozygote Mutation des PTHR1ens (dieses Gen kodiert den G-Protein-gekoppelten Rezeptor für das Nebenschilddrüsen-hormon PTH). Bei einer Mutation dieses Gens verändert sich die Rezeptorkonfiguration, wobei PTH nicht mehr binden kann. Der PTHR1 ist auf der Oberfläche von Osteoblasten exprimiert → bei Aktivierung durchs PTH werden Osteoklasten via RANKL stimuliert, OPG ↓, Sclerostin ↓ und Knochenresorption findet statt.</i></li> </ul> </li> </ul>
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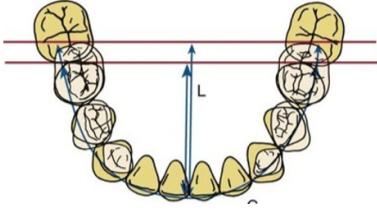
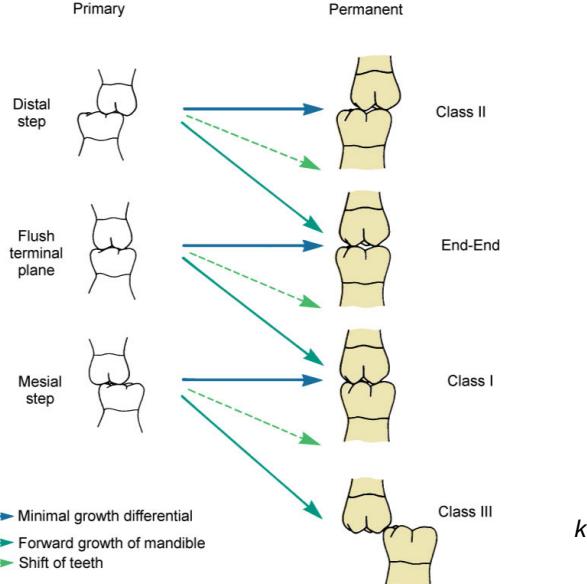
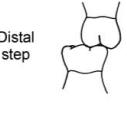
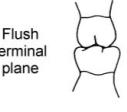
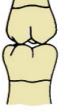
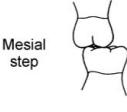
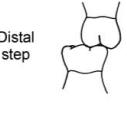
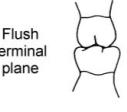
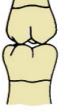
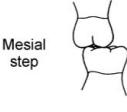
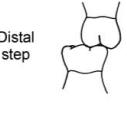
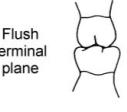
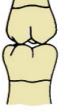
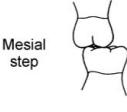
<b>2. Post emergent eruption</b> 	<ul style="list-style-type: none"> <li>- <b>Post-emergent spurt:</b> = Rapid eruption from the time a tooth has penetrated the gingiva until it reaches the occlusal plane.</li> <li>- Root dilaceration usually occurs, if eruption of the tooth is impeded or due to trauma. The tooth can continue to erupt normally, after dilaceration occurred.</li> <li>- <i>Prof. Becker:</i> <ul style="list-style-type: none"> <li>• Early tx start is indicated for teeth with dilacerations. Root formation is possible as long as the apex is open.</li> <li>• May plan apex ectomy + root tx for teeth with severe dilaceration to avoid gingiva perforation.</li> </ul> </li> </ul> <ul style="list-style-type: none"> <li>- <b>Juvenile occlusal equilibrium:</b> = Slow eruption when the tooth reaches the occlusal level of other teeth and is in complete function.           <ul style="list-style-type: none"> <li>• The eruption rate is parallel to the rate of vertical growth of the mandibular ramus.</li> <li>• Eruption only between 8 p.m. - 1 a.m. Stop of the tooth eruption during the day or even a small intrusion occurs. Regulated by growth hormone release.</li> <li>• Applied pressure / force stops eruption only for 1-3 min, if there is any effect at all. → Contact with food, does not explain the rhythm.</li> <li>• Shortening of collagen fibers in the periodontal ligament by cross-linking as eruption mechanism.</li> </ul> </li> </ul> <ul style="list-style-type: none"> <li>- <b>Adult occlusal equilibrium:</b> <ul style="list-style-type: none"> <li>• Extremely slow eruption rate, more rapidly if an antagonist is lost.</li> <li>• Loss of vertical dimension, if the eruption does not compensate the tooth wear.</li> </ul> </li> </ul>
Dental Age	<ul style="list-style-type: none"> <li>- Defined by:           <ul style="list-style-type: none"> <li>○ Which teeth have erupted</li> <li>○ The amount of resorption of the roots of primary teeth</li> <li>○ The amount of development of the permanent teeth</li> </ul> </li> <li>- Change of sequence is a more reliable sign for disturbances than a generalized delay or acceleration.</li> <li>- <u>Variation in sequence with clinical significance:</u> <ul style="list-style-type: none"> <li>• <b>Eruption of 7-7 before mn premolars:</b> Decrease of space can block out 5-5 outside the arch. → Space opening is maybe necessary.</li> <li>• <b>Eruption of 3+3 before maxillary premolars:</b> → Canines are forced labially. (also if there would be enough space if they had erupted after 4+4)</li> <li>• <b>Asymmetries in eruption between the right and left side:</b> X-ray if difference &gt;6m.</li> </ul> </li> <li>- Tooth emerges when root formation is about <math>\frac{3}{4}</math> completed.</li> <li>- Root formation completed 2-3 y after tooth eruption.</li> </ul>

TABLE 3.2 Chronology of Tooth Development, Permanent Dentition

Tooth	CALCIFICATION BEGINS		CROWN COMPLETED		ERUPTION		ROOT COMPLETED	
	Maxillary	Mandibular	Maxillary	Mandibular	Maxillary	Mandibular	Maxillary	Mandibular
Central	3mo	3mo	4½ yr	3½ yr	7¼ yr	6½ yr	10½ yr	9½ yr
Lateral	11mo	3mo	5½ yr	4yr	8¾ yr	7½ yr	11yr	10yr
Canine	4mo	4mo	6yr	5¾ yr	11½ yr	10½ yr	13½ yr	12¾ yr
First premolar	20mo	22mo	7yr	6¾ yr	10¼ yr	10½ yr	13½ yr	13½ yr
Second premolar	27mo	28mo	7¾ yr	7½ yr	11yr	11½ yr	14½ yr	15yr
First molar	32wk in utero	32wk in utero	4½ yr	3¾ yr	6½ yr	6yr	10½ yr	10½ yr
Second molar	27mo	27mo	7¾ yr	7½ yr	12½ yr	12yr	15½ yr	16yr
Third molar	8yr	9yr	14yr	14yr	20yr	20yr	22yr	22yr

- Dental age 6y	<ul style="list-style-type: none"> <li>• E 1-1</li> <li>• E 6-6</li> <li>• E 6+6</li> </ul> 
- Dental age 7y	<ul style="list-style-type: none"> <li>• E 1+1, 2-2</li> <li>• Root development 2+2 well advanced</li> <li>• 543+345 stage of crown completion</li> </ul>
- Dental age 8y	<ul style="list-style-type: none"> <li>• E 2+2</li> <li>• 2-3y delay afterwards before further teeth erupt</li> </ul> 
- Dental age 9y	<ul style="list-style-type: none"> <li>• Root formation of 621±126 nearly complete.</li> <li>• 3-3, 4±4 first third of the root complete</li> <li>• Root formation beginning 3+3, 5±5</li> </ul> 
- Dental age 10y	<ul style="list-style-type: none"> <li>• Resorption of the roots of the primary canines and molars</li> <li>• Root formation completed: 21-12, 2+2 nearly</li> <li>• Root formation: 43-34 1/3 4+4 1/2 5-5, 53+35 sign. development</li> </ul>
- Dental age 11y	<ul style="list-style-type: none"> <li>• E 3-3, 4-4, 4+4</li> <li>• Root formation 6±6 completed</li> <li>• Remaining primary teeth: V±V</li> </ul> 
- Dental age 12y	<ul style="list-style-type: none"> <li>• E 3+3, 5±5</li> <li>• E 7±7 nearly</li> </ul> <p>Succedaneous teeth normally complete their eruption before the 2<sup>nd</sup> molars erupt, but not always</p>

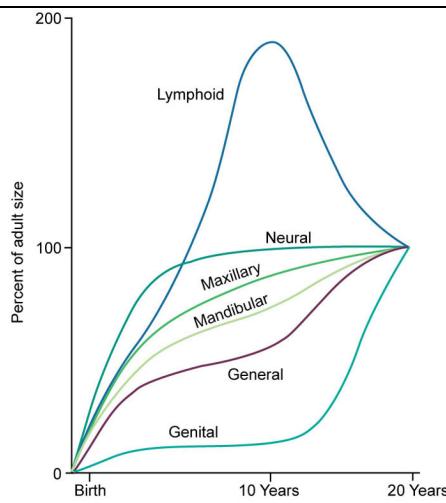
	
Dental age 15y	<ul style="list-style-type: none"> <li>Roots of all permanent teeth except 8±8 complete</li> <li>8±8 apparent on the x-ray</li> </ul> 
Space relationship in replacement of the incisors	<ul style="list-style-type: none"> <li>Permanent incisors in both jaws lay lingual and apical of the primary teeth. → Tendency for mn incisors to erupt lingually in a slightly irregular position.</li> <li>If there is not enough room for the teeth, they are usually displaced labial.</li> <li>Permanent incisors are larger than the primary → Spacing in the primary dentition is crucial. Normally there is no additional spacing in the posterior region. <ul style="list-style-type: none"> <li><i>Thilander 2009:</i> Difference in tooth width with primary - permanent teeth: <ul style="list-style-type: none"> <li>Maxilla: <ul style="list-style-type: none"> <li>21+12: <math>\Delta +7 \text{ mm}   \text{ incl. } 3+3 \Delta +9 \text{ mm}</math></li> <li>IV + IV = 4 + 4</li> <li>5 + 5: <math>\Delta -1 \text{ mm per side}</math></li> </ul> </li> <li>Mandible: <ul style="list-style-type: none"> <li>21-12: <math>\Delta +5 \text{ mm}   \text{ incl. } 3-3 \Delta +7 \text{ mm}</math></li> <li>IV - IV = 4 - 4</li> <li>5 - 5: <math>\Delta -2.7 \text{ mm per side}</math></li> </ul> </li> </ul> </li> </ul> </li> <li>Mx: Normally enough space for 2+2.</li> <li>Mn: On average <math>\Delta -1.6 \text{ mm}</math> space missing for the permanent incisors. → <b>Incisor liability</b> = difference between the space available and the space necessary.</li> <li>Transitory crowding between 8-9 y of age is normal. Normalization when the canines erupt.</li> </ul> <p style="text-align: center;">Available space - incisor segment</p> <div style="display: flex; justify-content: space-around;"> <span>Maxilla —————</span> <span>Mandible —————</span> </div> <div style="display: flex; justify-content: space-around;">   </div> <p style="text-align: center;">Years</p> <p><i>Moorrees, 1965: Space available within the arches: Boys left, girls right. Mn: space for incisors negative for about 2y after eruptions → small crowding.</i></p> <ul style="list-style-type: none"> <li><b>Sources of extra space:</b> <ul style="list-style-type: none"> <li>Slight increase in arch width across the canines: <ul style="list-style-type: none"> <li>Teeth erupt slightly outward as growth continues.</li> <li><b>2 mm</b> gain of space: mx &gt; mn; girls &lt; boys → girls are more likely to have anterior incisor crowding.</li> </ul> </li> <li>Slight labial positioning (proclination) of the central and lateral incisors. Primary incisors are straighter. → <b>1-2 mm</b> gain of space due to a larger circle of the arch.</li> <li>Distal shift of the primary canines when the permanent incisors erupt in the primate space. → <b>Mn: 1 mm</b> gain of space</li> </ul> </li> </ul>

	<ul style="list-style-type: none"> <li>→ <b>Mx: 0</b> (primate space lays mesial III+III)       <ul style="list-style-type: none"> <li>- All changes happen without significant skeletal growth.</li> </ul> </li> <li>- <b>Ugly duckling stage:</b> <ul style="list-style-type: none"> <li>= Maxillary diastema &amp; 2+2 flared labially and distally.</li> <li>Increased risk for impacted 3+3.</li> </ul> </li> <li>- <u>Central diastema:</u> <ul style="list-style-type: none"> <li>o &lt;2 mm normally closes spontaneous when 3+3 erupt.</li> <li>o &gt;2 mm: spontaneous closure unlikely.</li> </ul> </li> </ul>								
Space relationship in replacement of canine and primary molars	<ul style="list-style-type: none"> <li>- <b>Leeway space:</b> <ul style="list-style-type: none"> <li>- Mn: <b>2.5 mm per side</b> (<math>V-V = +2 \text{ mm } 5-5, IV-IV = +0.5 \text{ mm } 4-4</math>)</li> <li>- Mx: <b>1.5 mm per side</b> (<math>V+V = +1.5 \text{ mm } 5+5, IV+IV = 4+4</math>)</li> </ul> </li> <li>- 6er move rapidly forward after loss of the premolars: → Arch length and arch circumference decrease.</li> </ul>  <ul style="list-style-type: none"> <li>- Stöckli 1994: Molar migration is normally smaller than the LWS.       <ul style="list-style-type: none"> <li>o Mx: 1 mm</li> <li>o Mn: 2 mm</li> </ul> </li> <li>- Relationship of primary molars:        <table border="0"> <tr> <td style="vertical-align: top;">           Primary           <ul style="list-style-type: none"> <li>Distal step</li> <li>Flush terminal plane</li> <li>Mesial step</li> </ul> </td> <td style="vertical-align: top;">           Permanent           <ul style="list-style-type: none"> <li>Class II</li> <li>End-End</li> <li>Class I</li> <li>Class III</li> </ul> </td> </tr> <tr> <td style="vertical-align: top;">  </td> <td style="vertical-align: top;">  </td> </tr> <tr> <td style="vertical-align: top;">  </td> <td style="vertical-align: top;">  </td> </tr> <tr> <td style="vertical-align: top;">  </td> <td style="vertical-align: top;">  </td> </tr> </table> <p>         ● Distal step (10%)          &gt; Cl.II          &gt; End to end       </p> <p>         ● Flush terminal plane/normal relationship (30%)          &gt; Cl.1          &gt; end to end       </p> <p>         ● Mesial step (60%)          &gt; Cl.1          &gt; Cl.III       </p> <p>Legend:   Minimal growth differential   Forward growth of mandible   Shift of teeth       </p> </li> <li>- The amount of differential mn growth and molar shift into the leeway space determines the molar relationship.</li> <li>- Flush terminal plane = normal relationship of the primary molar teeth. (or mesial terminal plane with no mn primate spaces)</li> <li>- Class III is almost never seen in the primary dentition, as the mn lies behind the maxilla due to the growth pattern.</li> <li>- Forward movement mn molars &gt; mx molars.</li> <li>- The transition is usually done by one-half cusp (3-4 mm) relative forward movement of the lower molar accomplished by a combination of differential growth and tooth movements.</li> <li>Key variable = amount and direction of mn growth.</li> </ul>	Primary <ul style="list-style-type: none"> <li>Distal step</li> <li>Flush terminal plane</li> <li>Mesial step</li> </ul>	Permanent <ul style="list-style-type: none"> <li>Class II</li> <li>End-End</li> <li>Class I</li> <li>Class III</li> </ul>						
Primary <ul style="list-style-type: none"> <li>Distal step</li> <li>Flush terminal plane</li> <li>Mesial step</li> </ul>	Permanent <ul style="list-style-type: none"> <li>Class II</li> <li>End-End</li> <li>Class I</li> <li>Class III</li> </ul>								
									
									
									

## Proffit Chapter 4:

### Later Stages of Development

Adolescence: The early permanent dentition years	
Adolescence	= Transitional period between the juvenile stage and adulthood when sexual maturation is attained. Adolescent growth spurt, secondary sexual characteristics, physiological changes, fertility, change from the mixed in the permanent dentition.
Initiation of adolescence	<p><b>Diagram illustrating the hormonal feedback loop during adolescence:</b></p> <p>Occurs in the brain:</p> <ol style="list-style-type: none"> <li>1. Brain cells in the <b>hypothalamus</b> secrete <b>releasing factors</b> in the cell body. (epigenetic regulation)</li> <li>2. Cytoplasmic transportation of the releasing factors along the axon to a richly vascular area at the base of the hypothalamus near the pituitary gland.</li> <li>3. Passage into capillaries, carried by blood to the pituitary.</li> <li>4. Releasing factors stimulate in the anterior part of the <b>pituitary gland</b> the cells to produce the pituitary <b>gonadotropin hormones</b>.</li> <li>5. Gonadotropin hormones stimulate endocrine cells in the <b>adrenal glands</b> and the developing <b>sex organs</b> to produce <b>sex hormones</b>. In both genders, male and female sex hormones are produced.</li> <li>6. Sex hormones cause the development of the secondary sexual characteristics, accelerate growth of the genitals and general body growth, shrinkage of lymphoid tissues.</li> </ol> <p>The control signal is amplified in each step.</p> <ul style="list-style-type: none"> <li>- <b>Boys:</b> Production of male &amp; female sex hormones in the testes + some female hormones in the adrenal cortex.</li> <li>- <b>Girls:</b> Ovaries produce estrogen and later progesterone. The adrenal cortex produces male sex hormones.</li> <li>- The earlier the onset of puberty, the smaller the adult size: (and the more intense the growth spurt) Sex hormones stimulate the cartilage in the long bone to grow faster, but increase even more the skeletal maturation → cartilage transform into bone, the epiphyseal plates close.</li> <li>- Obese children: Puberty starts earlier → body length ↓</li> </ul>

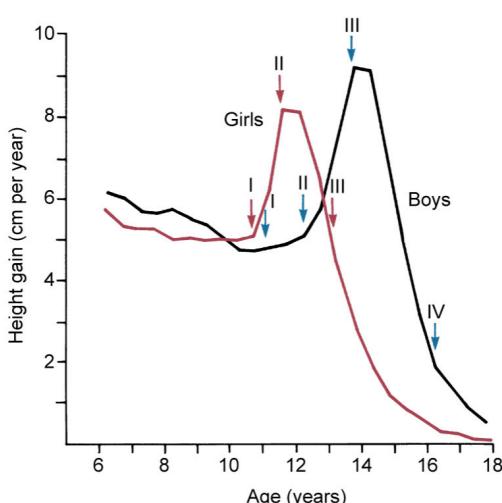


Growth curves for the maxilla and the mandible shown against the background of Scammon's curves. Growth of the jaws is intermediate between the neural and general body curves, with the mandible following the general body curve more closely than the maxilla. The acceleration in general body growth at puberty, which affects the jaws, parallels the dramatic increase in development of the sexual organs. Lymphoid involution also occurs at this time.

### Timing of Puberty

- Girls 2 y earlier than boys → Orthodontic tx must be done earlier for girls than for boys.
- Stage of development of secondary sexual characteristics is a good indicator for the stage of adolescence that correlates well with physical growth status.

Girls: Adolescent Growth total 3.5 y, 3 stages	
<b>Stage 1</b> <i>Beginning of adolescence growth</i>	Appearance of breast buds Initial pubic hair
<b>Step 2 (12m later)</b> <i>Peak velocity in height</i>	Breast development Axillary hair Darker/more abundant pubic hair
<b>Stage 3 (12-18m later)</b> <i>Growth spurt ending</i>	Menstruation Broadening of the hips with adult fat distribution Breasts completed
Boys: Adolescent Growth total 5 y, 4 stages	
<b>Stage 1</b> <i>Beginning of adolescent growth</i>	Fat spurt weight gain Feminine fat distribution
<b>Stage 2 (12m later)</b> <i>Height spurt is beginning</i>	Redistribution / reductions in fat Pubic hair Growth of penis
<b>Stage 3 (8-12m later)</b> <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
<b>Stage 4 (15-24m later)</b> <i>Growth spurt ending</i>	Facial hair on chin and lip Adult distribution / color of pubic and axillary hair Adult body form



- Girls:
- Pubertal growth spurt **10.8 -13.3 y**
  - Peak of growth: **12.0 y**
  - Menarche **13.3 y** (CH **13.7 y**, Mullis 2009)
  - Maximum growth per year: **8.2 cm**
  - **10.7 y** = Start of puberty = breast development (Mullis, 2009)

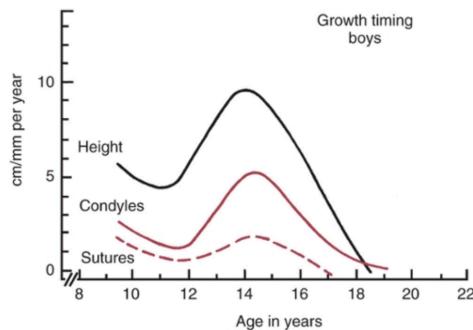
- Boys:
- Pubertal growth spurt **12.4 - 15.2 y**
  - Peak of growth: **14.0 y**
  - Maximum growth per year: **9.2 cm**
  - **11.7y** = Start of puberty = testis size > **4ml** (Mullis, 2009)

- 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:

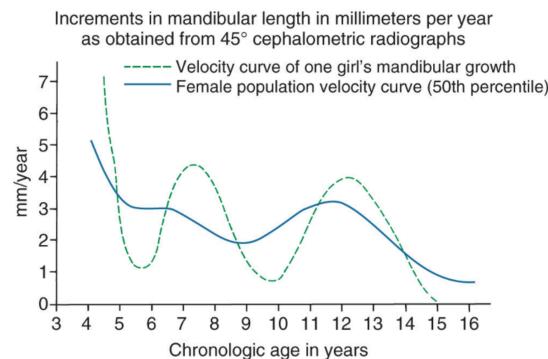
Adolescent growth spurt in length of the mn and to a smaller extent in the mx → Face becomes less convex.



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:

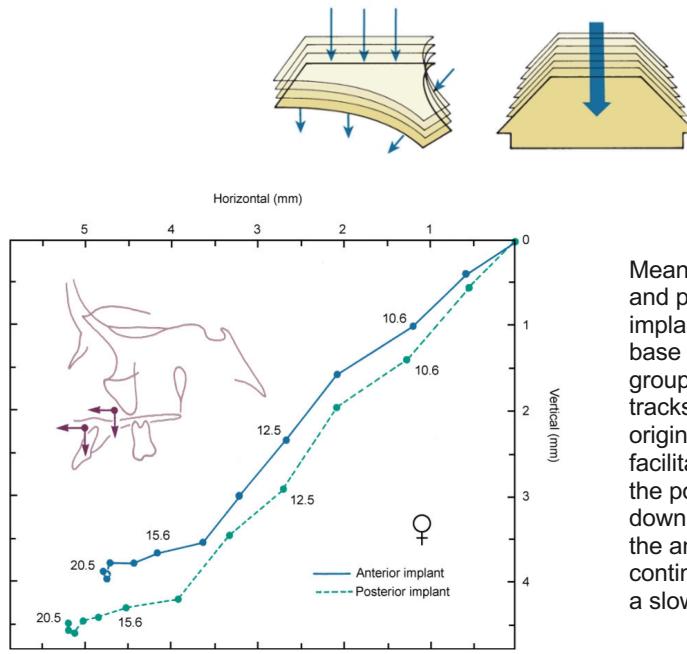
- If a juvenile growth spurt of the jaw occurs, it is mostly less intense than the growth acceleration at puberty.
- A considerable amount of physical growth remains in the permanent dentition in slowly maturing boys.

- **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.

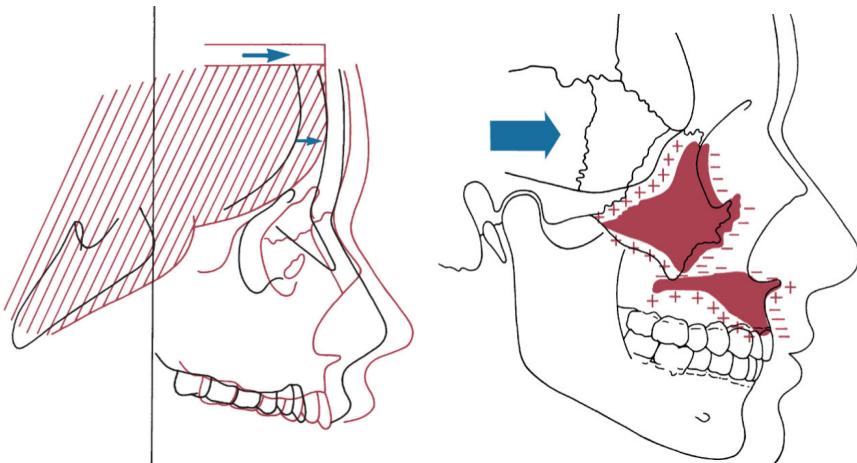
## Growth of the nasofacial complex

- 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:  $\Delta +4.8 \text{ mm}$
  - Increasement of the intermolar width correlates with the sutural growth.  
Increasement 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 1<sup>st</sup> 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.

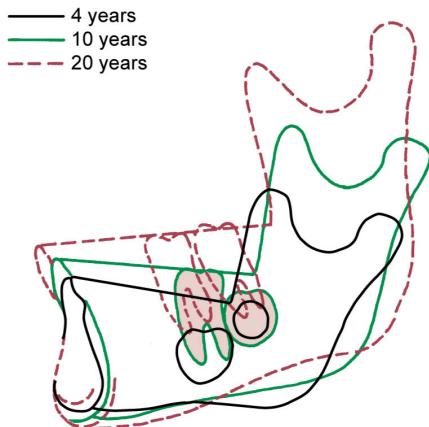
Mx: Width across the 2<sup>nd</sup> and 3<sup>th</sup> molars (if they erupt).

Mn: Molar and bicondylar width.

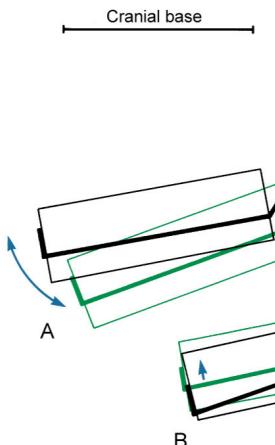
## Rotation of the jaws during growth

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.

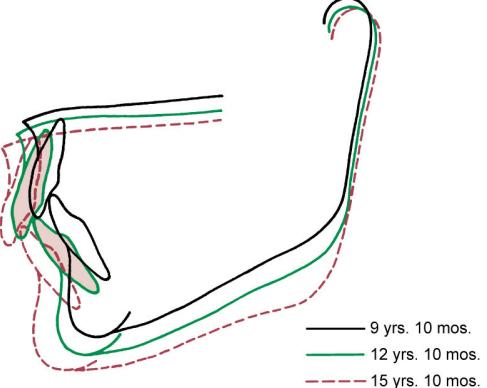
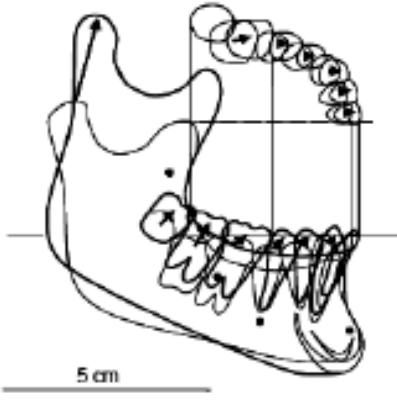
- **Internal rotation:** = Rotation that occurs within the core of each jaw.  
Average 15°, 25% matrix rotation, 75% intramatrix rotation.
  - **External rotation:** = Due to surface changes (remodeling)
  - Internal rotation tends to be masked by surface changes and alterations in the rate of tooth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

- 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.

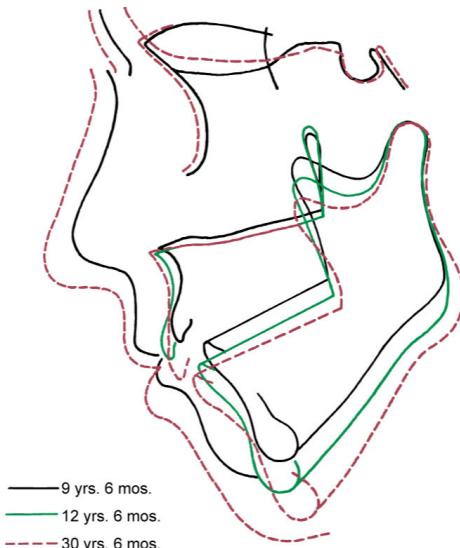


Internal rotation of the mandible (i.e., rotation of the core relative to the cranial base) has two components: **A**, Rotation around the condyle, or matrix rotation. **B**, Rotations centered within the body of the mandible, or intramatrix rotation.

<b>Maxilla</b>	<ul style="list-style-type: none"> <li>- Small and variable degree of internal rotation forward (usual pattern) and backward (also frequent).</li> <li>- Remodeling of the palate.</li> <li>- 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 &gt; posterior)</li> </ul> <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"> <li>- <i>Björk and Skieller, 1972:</i> <ul style="list-style-type: none"> <li>• Maxilla rotation is normally in the same direction like the mn rotation, but only <math>\frac{1}{3}</math> of the extent.</li> <li>• Apposition and resorption compensate the rotation, so that there is net no change of inclination for the orbit and the nasal floor.</li> </ul> </li> <li>- <i>Björk and Skieller, 1977: Changes age 4 y - adulthood:</i> <ul style="list-style-type: none"> <li>• Sutural lowering: <b>11.2 mm</b></li> <li>• Apposition at the orbital floor: <b>6.4 mm</b></li> <li>• Resorption at the floor of the nose: <b>4.6 mm</b></li> <li>• Apposition at the alveolar process: <b>14.6 mm</b></li> </ul> </li> </ul>
<b>Face types: short face type</b>	<ul style="list-style-type: none"> <li>- Excessive forward rotation of the mandible during the growth. (increase in internal rotation + decrease in external compensation)</li> <li>- Low mandibular plane angle, large (? Rather small) gonial angle.</li> <li>- Deep bite.</li> <li>- Crowded incisors through uprighting and lingual positioning. (molars move more mesial than the incisors which are uprighted by the rotation → arch length ↓ = crowding)</li> <li>- Nearly horizontal palatal plane.</li> <li>- 20<sup>th</sup> 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.</li> <li>- <i>Björk 1969:</i> <ul style="list-style-type: none"> <li>• Condyle growth in vertical direction → ramus length ↑ or</li> <li>• Middle cranial fossa displaced caudal (bending of the cranial base) → Fossa condylaris displaced caudal.</li> <li>• = Mn lowering &gt; Mn forward displacement But: attachments and ligaments transform the caudal displacement into an anterior rotation.</li> </ul> </li> </ul>

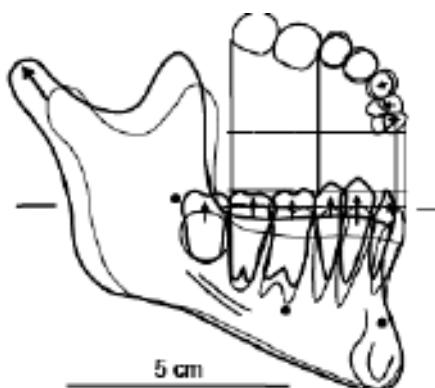
	<ul style="list-style-type: none"> <li>○ Type 1: center of rotation = condyles (loss of teeth)</li> <li>○ Type 2: center of rotation = incisors</li> <li>○ Type 3: center of rotation = premolars (if OJ &gt; 3 mm)</li> <li>● Incisor are directed forward to compensate the rotation (the incisors inclination within the face stays always <math>\pm</math> the same)           <ul style="list-style-type: none"> <li>○ <math>\rightarrow</math> interincisal angle ↓</li> <li>○ <math>\rightarrow</math> anterior crowding</li> <li>○ Angle incisors - chin = <math>\Delta</math> -1 mm/y <math>\rightarrow</math> mentolabial fold more pronounced</li> </ul> </li> <li>● All teeth are anterior directed <math>\rightarrow</math> crowding</li> <li>● Posterior teeth more upright in relation to the mx plan:           <ul style="list-style-type: none"> <li>○ <math>\rightarrow</math> intermolar angle ↑</li> <li>○ <math>\rightarrow</math> interpremolar angle ↑</li> </ul> </li> </ul>  <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>
<b>Face types: long face type</b>	<ul style="list-style-type: none"> <li>- Zusammenfassung der Wachstumsbereiche (Apposition, Resorption) des Unterkiefers</li> <li>- Anteriore Wachstumsrichtung der Kiefergelenke (<i>Berner Skript</i>)       <ul style="list-style-type: none"> <li>● Ausgeprägte Resorption im Angulusgebiet.</li> <li>● Ausgeprägte Apposition unter dem Kinn.</li> <li>● Keine Resorption am vorderen Ramusrand.</li> <li>● Wenig Apposition am hinteren Ramusrand.</li> </ul> </li> </ul> 

- $= M_n$  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

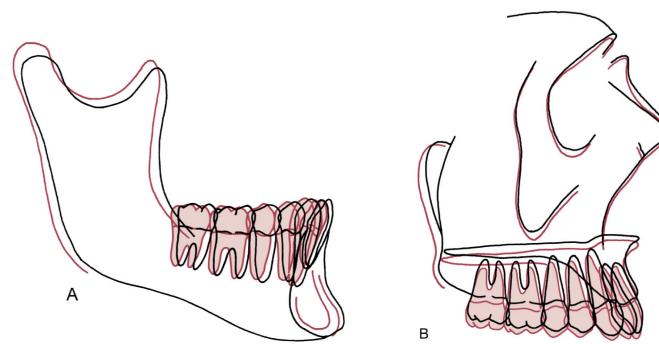
- Rotational pattern of jaw growth influences the magnitude and direction of tooth eruption as well as the ultimate anterior-posterior position of the incisor teeth.
- Maxilla:  
Normal tooth eruption is downward and forward.  
Movement of the teeth in relation to the cranial base as a combination of 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 upright and their prominence decreased.

	<p>- <b>Mandible:</b>          Normal eruption path is upward and forward.</p> <ul style="list-style-type: none"> <li>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)</li> <li>o Posterior rotation:            Incisors are carried forward → dental protrusion.            Anterior open bite, if the incisors do no erupt for an extreme distance.</li> </ul> <p>- 20<sup>th</sup> 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

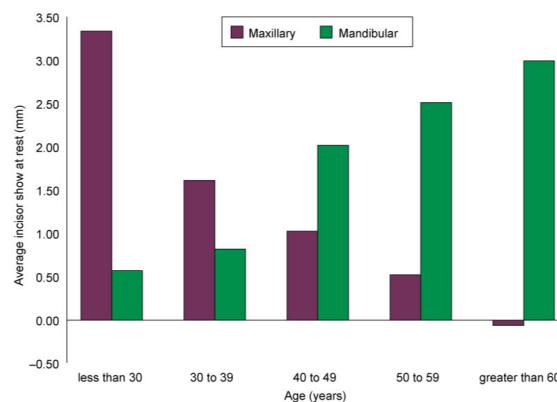
- Facial growth in adults**
- Growth processes decline to basal level after the attainment of sexual maturity and continue to show a cephalocaudal gradient.
  - Size and shape of the craniofacial complex change due to slow continuous growth:
    - Soft tissues > jaw relationship.
    - Vertical > anterior-posterior > 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

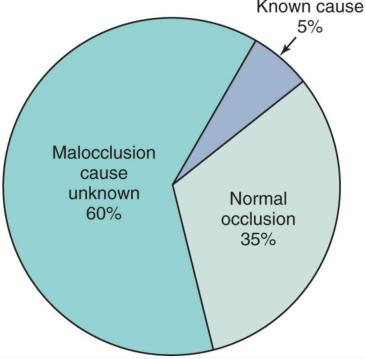


Incisor display at rest as a function of age. With aging, both men and women show less of their upper incisors and more of their lower incisors, so display of upper incisors is a youthful characteristic.

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

## Proffit Chapter 5:

### The Etiology of Orthodontic Problems

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

### 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

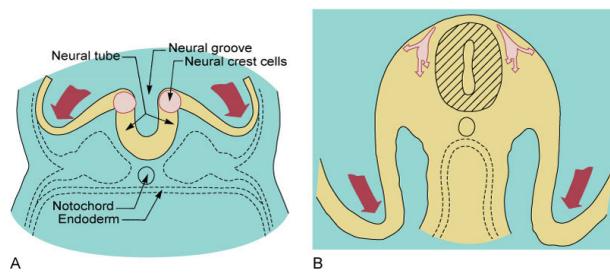
- 5 principal stages in craniofacial development:

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 <sup>th</sup> week Secondary palate 11 <sup>th</sup> 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)

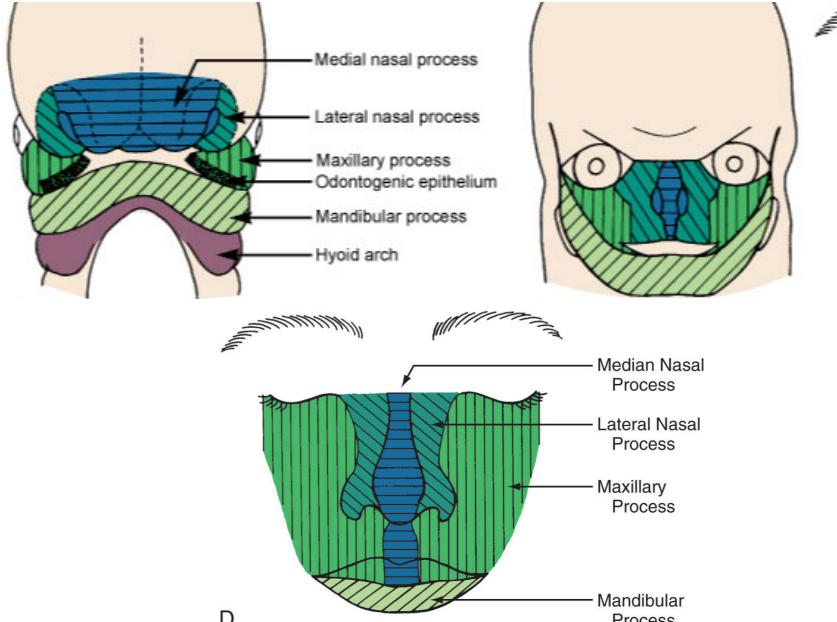
- Neutral crest cells:
    - Bone
    - Cartilage
    - Dermis
  - Paraxial mesoderm:marathon
    - Bloodvessels
    - Muscles
  - Ectoderm:
    - Epidermis
- Neural crest cells:
- Formation at the edge ectoderm / neural tube
  - Are originally ectoderm cells, but they undergo a mesenchymal transformation.
  - 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.

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

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

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

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

Growth disturbances in the fetal (9-40 w intrauterine) and perinatal period	
<b>Intrauterine Molding</b>	<ul style="list-style-type: none"> <li>- Pressure against the developing face prenatally can lead to distortion of rapidly growing areas: <ul style="list-style-type: none"> <li>• Arm pressed across the face in utero → maxillary deficit.</li> <li>• Head flexed tightly against the chest in utero → mandible inhibited to grow.</li> </ul> </li> <li>- Possibility of normal growth after birth and eventually a complete recovery.</li> </ul>
<b>Birth trauma to the mandible</b>	<ul style="list-style-type: none"> <li>- Use of forceps on the head in difficult births can damage the TMJ: → subsequent underdevelopment of the mandible.</li> </ul>
<b>Pierre Robin anomaly / sequence</b>	<ul style="list-style-type: none"> <li>= Extreme mandibular deficiency at birth.</li> <li>- Multiple causes can lead to the deformity, not a syndrome with a defined cause.</li> <li>- Possible cause: Head tightly flexed against the chest in utero (related to a decreased volume of amniotic fluid) → mn cannot grow forward.</li> <li>- Often related with CLP: The restriction forces the tongue upwards and prevents normal closure of the palatal shelves.</li> <li>- Birth problems possible at birth due to the reduced volume of the oral cavity.</li> <li>- Early mandibular advancement via distraction osteogenesis has been used recently to provide more space for the airways.</li> <li>- 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.</li> <li>- Catch up growth is most likely when the cause was mechanical growth restriction.</li> <li>- <math>\frac{1}{3}</math> of the affected subjects have a defect in cartilage formation = <b>Stickler syndrome</b></li> </ul>

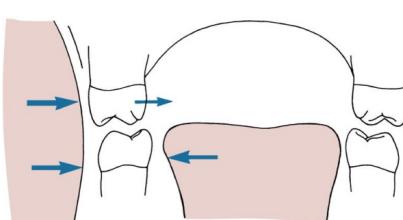
Progressive deformities in childhood	
= Deformities which steadily become worse.	
<b>Childhood fractures of the jaw</b>	<ul style="list-style-type: none"> <li>- 75% of the children with early fractures of the mandibular condylar process have normal mandibular growth afterwards.</li> <li>- Diagnosis is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

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

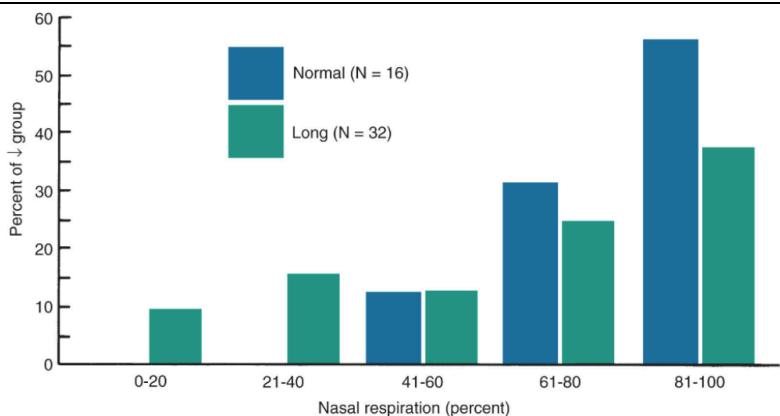
	<ul style="list-style-type: none"> <li>- Distribution: <ul style="list-style-type: none"> <li>o Mostly mesiodens in the maxillary midline.</li> <li>o Sometimes extra premolars, lateral incisors, 4<sup>th</sup> molars.</li> </ul> </li> </ul>
<b>Traumatic displacement of teeth</b>	<ul style="list-style-type: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

Genetic influences	
2 Theories	<ol style="list-style-type: none"> <li>1. Inherited disproportion between the size of the teeth and the size of the jaws: → Crowding / spacing.</li> <li>2. Inherited disproportion between the size or shape of the upper and lower jaws: → Improper occlusal relationships.</li> </ol>
<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"> <li>- 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.</li> <li>- 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.</li> <li>- <u>Studies with family members:</u> <ul style="list-style-type: none"> <li>○ Heritability of craniofacial (skeletal) characteristic is relatively high and increases with age.</li> <li>○ Heritability of dental (occlusal) characteristics is low and decreasing with age. → Shows an increasing environmental contribution to the dental variation.</li> </ul> </li> <li>- <u>Twin studies: Hereditary component for:</u> <ul style="list-style-type: none"> <li>○ Variations in spacing and tooth position within the dental arch = 69-89%</li> <li>○ Overbite 53%</li> <li>○ Overjet 28%</li> </ul> </li> <li>- Effect of interracial crosses appears to be only additive, but not multiplicative.</li> <li>- Knowing the type of growth associated with different genetic patterns can help to chose the type and timing of orthodontic and surgical treatment.</li> <li>- 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.</li> </ul> <p><b>→ Bone more related to genes</b> <b>→ Teeth more related to environmental factors</b></p> <ul style="list-style-type: none"> <li>- <u>Mn prognathisme:</u> <ul style="list-style-type: none"> <li>○ 0.316 heritability.</li> <li>○ Autosomal dominant with incomplete penetration.</li> <li>○ One major gene was determined as cause, but not only one single gene responsible for the malocclusion.</li> </ul> </li> <li>- <u>Puri, 2007:</u> <ul style="list-style-type: none"> <li>○ Crowding: Patients have in general larger teeth</li> <li>○ Spacing: Patients have in general smaller teeth</li> </ul> </li> </ul>
Environmental influences	
<ul style="list-style-type: none"> <li>- Consist largely of pressures and forces related to physiologic activity.</li> </ul> <p><b>1. Equilibrium considerations</b></p> <p>→ Changes of the equilibrium = changes of tooth and jaw position</p>	<ul style="list-style-type: none"> <li>- 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 1<sup>st</sup> law)</li> <li>- <u>Tooth:</u> <ul style="list-style-type: none"> <li>○ Experiences normally forces from masticatory effort, swallowing and speaking, but does not move.</li> <li>○ Continuous force from an orthodontic appliance applied: → Equilibrium altered → tooth moves.</li> </ul> </li> <li>- <u>PDL:</u> <ul style="list-style-type: none"> <li>○ 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.</li> <li>○ An impact on the soft tissues occurs only, if the pressure is maintained long enough to squeeze out the fluid.</li> </ul> </li> </ul> <p>→ Only light forces of long duration (<math>\geq 6</math>h 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"><b>Tooth Contacts</b></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"><b>Soft tissue pressures of lip, cheek and Tongue</b></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><b>Resting</b></td><td>Very light</td><td>Long</td><td><b>Important</b></td></tr> <tr> <td colspan="4"><b>External Pressures</b></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"><b>Intrinsic Pressures</b></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	<b>Tooth Contacts</b>				Mastication	Very heavy	Very short	No significant impact	Swallowing	Light	Very short	No significant impact	<b>Soft tissue pressures of lip, cheek and Tongue</b>				Swallowing	Moderate	Short	No significant impact	Speaking	Light	Very short	No significant impact	<b>Resting</b>	Very light	Long	<b>Important</b>	<b>External Pressures</b>				Habits	Moderate	Variable		Orthodontics	Moderate	Variable		<b>Intrinsic Pressures</b>				PDL fibers	Light	Long		Gingival fibers	Variable	Long	
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<b>Function and dental arch size</b>  → Influence yes, but little effect.	<ul style="list-style-type: none"> <li>- Muscle size and activity is reflected in the size and shape of the muscular processes of the jaws: <ul style="list-style-type: none"> <li>○ → Vertical jaw relationship is affected.</li> <li>○ → Enlargement of the mandibular gonial angles can be seen in humans with hypertrophy of the mandibular elevator muscle</li> </ul> </li> <li>- 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.</li> <li>- 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.</li> </ul>																																																								
<b>Biting force and eruption</b>  → 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"> <li>- 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"> <li>○ → 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.</li> <li>○ The difference is rather an effect than a cause for the malocclusion. (failure of long-face individual to gain strength)</li> </ul> </li> <li>- Bite forces do not determine dental arch size, tooth eruption or vertical dimensions in the absence of syndromes.</li> <li>- Muscular dystrophy and related syndromes can affect growth.</li> </ul>																																																								
<b>Sucking and other habits</b>  → Can have an impact on the dentition, if long enough applied.	<ul style="list-style-type: none"> <li>- Sucking habits during the primary dentition: little if any longterm effect.</li> <li>- 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.</li> <li>- <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"> <li>• Flared and spaced maxillary incisors.</li> <li>• Lingually positioned lower incisors.</li> <li>• 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)</li> <li>• Narrow upper arch (tongue lowered + increased cheek pressure).</li> <li>• Mx arch can become V-shaped because cheek pressure due to contraction of the m. buccinator is greatest at the corners of the mouth.</li> <li>• Changes in symmetry if the thumb is placed on one side instead of the midline.</li> </ul> </li> </ul>																																																								



	<ul style="list-style-type: none"> <li>- Different sucking pattern:           <ul style="list-style-type: none"> <li>• Short duration, but very intensive: May not a risk for malocclusion.</li> <li>• Long duration, little force (during sleep) → Increased risk for a malocclusion.</li> </ul> </li> <li>- <u>Music instruments:</u> Duration too short to make any difference except for highly professional musicians (only minor changes).</li> </ul>
<b>3. Tongue thrusting</b>  → <i>Tongue thrusting = result of displaced incisors, but NOT the cause.</i>	<ul style="list-style-type: none"> <li>- Populations with tongue thrusting:           <ul style="list-style-type: none"> <li>• Young children:               <ul style="list-style-type: none"> <li>▪ Normal transitional stage in swallowing.</li> <li>▪ Delay, if a sucking habit is present.</li> </ul> </li> <li>• Individuals of any age with displaced incisors: Adaption to seal the space between the teeth = result of displaced incisors, but NOT the cause.</li> </ul> </li> <li>- 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.</li> <li>- 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).</li> <li>- Tongue thrusting is often present in children with good occlusion.</li> <li>- Too short duration to have an impact on tooth position: 1000 x / 24 h swallowing   1 swallowing ~ 1s.</li> <li>- 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.</li> <li>- Only the postural resting position of the tongue is important and can have an effect on the teeth.</li> </ul>
<b>1001 Respiratory pattern</b>  → <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"> <li>- Respiratory need = primary determinant of the posture of the jaws and the tongue.</li> <li>- Human:           <ul style="list-style-type: none"> <li>○ = Primarily nasal breather.</li> <li>○ Mouth breather in certain physiologic conditions: exercise, heavy mental concentration, conversation.</li> <li>○ Greater effort needed to breathe through the nose than through the mouth (warming, cleaning, and humidifying the air).</li> </ul> </li> <li>- 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.</li> <li>- BUT: Not all individuals in the long face population have an evidence of nasal obstruction (other etiologic factor!).</li> <li>- Mouthbreathers:           <ul style="list-style-type: none"> <li>• Change in posture if nasal obstruction occurs.</li> <li>• Mandible and tongue must be lowered → face height increases.</li> <li>• Posterior teeth overerupt.</li> <li>• Mandible rotates downward and backward → anterior OB ↓ + increased OJ.</li> <li>• Narrow maxilla due the increased pressure from the stretched cheeks.</li> </ul> </li> <li>- Long-face and normal-face children are predominantly nasal breathers.</li> <li>- Some long-face children have &lt;40% nasal breathing (no normal-face children with such low values).</li> <li>- More long-face children have a nasal obstruction compared to normal-face children, but the majority are not predominantly mouth breathers.</li> <li>- → Impaired nasal respiration may contribute to the development of the long-face condition, but is not the sole or even the major cause.</li> </ul>



- 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 O<sub>2</sub> 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"> <li>Collection of information and creation of a database.</li> <li>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)</li> <li>Consider a solution for each single problem and evaluate afterwards the interactions between the solutions.</li> <li>Goal = truth, scientific inquiry.</li> <li>No room for opinion or judgment. Factual arise of the situation.</li> </ul>
2. Treatment planning	<ul style="list-style-type: none"> <li>Goal = wisdom.</li> <li>Plan, which a good clinician will follow to maximize the benefit for the patient.</li> <li>Tx is usually comprehensive: → Goal = The best possible occlusion, facial esthetics and stability.</li> <li><u>Optimal tx-plan:</u> <ol style="list-style-type: none"> <li>Timing of tx: Mostly adolescence: Sufficient growth remaining, self-motivation develops, short course of tx. Timing depends on the specific problem.</li> <li>Complexity of the tx required.</li> <li>Predictability of success with a given tx approach: Prefer an evidence based choice!</li> <li>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.</li> </ol> </li> </ul>
	<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"> <li>Interview from questions (written and oral) from the patient and parents.</li> <li>Clinical examination of the patient.</li> <li>Evaluation of diagnostic records: Dental casts, x-ray, photographs.</li> </ol> </p>

Questionnaire / interview	
<b>Patient's chief concern</b>	<ul style="list-style-type: none"> <li>- Mostly more than one reason to seek orthodontic tx. → Establish their relative importance to the patient:           <ul style="list-style-type: none"> <li>• "Do you think you need braces?"</li> <li>• "What bothers you most about your teeth or your appearance?"</li> <li>• "What do you want tx to do for you?"</li> </ul> </li> </ul>
<b>Medical and dental history</b>	<ul style="list-style-type: none"> <li>- 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.</li> <li>- Provide a background for understanding the patient's overall situation and evaluation of specific concerns.</li> <li>- Growth deficiencies from an old condyle fracture = most common reason for real face asymmetry.</li> <li>- Chronic medical problems are no contraindication for orthodontic tx if they are under control.           <ul style="list-style-type: none"> <li>• Diabetes: periodontal breakdown more likely</li> <li>• Arthritis / Osteoporosis: tooth movement maybe difficult because of bisphosphonates</li> </ul> </li> </ul>
<b>Physical growth evaluation</b>	<ul style="list-style-type: none"> <li>- Growth spurt facilitates tooth movement.</li> <li>- "How rapidly grown recently? Clothes sizes changed? Sexual maturation onset yet? Time of sexual maturation in siblings?"</li> <li>- Observe sexual characteristics.</li> <li>- Consider height-weight records.</li> <li>- Calculating bone age from the vertebrae in the lateral ceph.</li> <li>- Serial cephalometric x-rays = Most accurate way to determine whether facial growth has stop or is continuing.</li> </ul>
<b>Social and behavioral evaluation</b>	<ul style="list-style-type: none"> <li>- Motivation for tx:           <ul style="list-style-type: none"> <li>• External: Pressure from another individual.</li> <li>• 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.</li> </ul> </li> <li>- Cooperation: More likely to be a problem with children than adults.           <ul style="list-style-type: none"> <li>• Depends on the extent to which the child sees the tx as a benefit opposed to something he is required to do.</li> <li>• Degree of parental control.</li> </ul> </li> <li>- Treating an unwilling child is hardly a good professional judgment, even if the parents force an apparent assent (Zustimmung).</li> </ul>

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

## Macro-esthetics: Facial proportions

TABLE 6.5 Checklist of Facial Dimensions to Evaluate During Clinical Examination

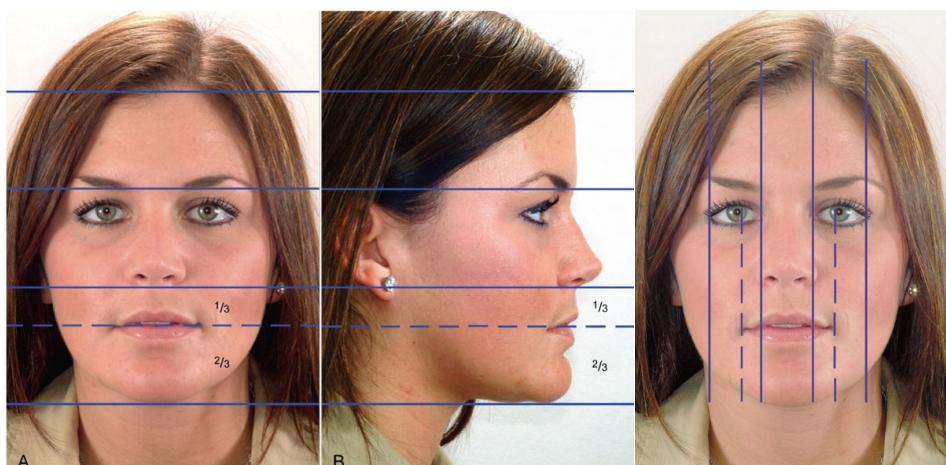
Frontal at Rest	Frontal Smile	Frontal Widths	Profile
<b>To Midsagittal Plane</b> <ul style="list-style-type: none"> <li>Nasal tip</li> <li>Maxillary dental midline</li> <li>Mandibular dental midline</li> <li>Chin (midsymphysis)</li> </ul>	<ul style="list-style-type: none"> <li>Maxillary incisor display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

### Girls: Adolescent Growth total 3.5 y, 3 stages

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

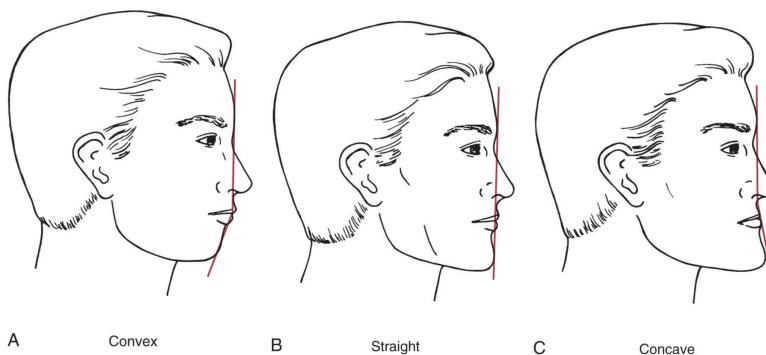
- Assessment of development age: Physical development is more important than chronologic age.
- Look for recognizable secondary sexual characteristics.

- Assess facial proportions, detect disproportions:  
Distorted and asymmetric facial features are a major contributor to facial esthetic problems, whereas proportionate facial features are generally accepted in all cultures even if they are not beautiful.
  - Usually the right side of the face is a little larger than the left side.
- Frontal:**
- Small degree of bilateral facial **asymmetry** is normal.
  - **Proportion of face height / width (the facial index):**  
→ Establishes the overall facial type and the basic proportions of the face.
  - **Vertical facial thirds:**  
Distance hairline to the base of nose = base of the nose to the bottom of nose = bottom of nose to the chin.
    - Lower third is slightly longer than the central third in modern Caucasians (*Farcas, 1987*).
    - Lower third:  $\frac{1}{3}$  above the mouth to  $\frac{2}{3}$  below.
  - **Proportion of the eyes / nose / mouth.**
  - **Midline deviation.**  
*D. Mirabella:* The inclination of the midline is rated more important than a midline shift



An ideally proportional face can be divided into central medial, and lateral equal fifths. The separation of the eyes and the width of the eyes, which should be equal, determine the central and medial fifths. The nose and chin should be centered within the central fifth, with the width of the nose the same as or slightly wider than the central fifth. The interpupillary distance (*dotted line*) should equal the width of the mouth.

- Profile**
- Jaw proportionately positioned in the anterior posterior plane → Assess convexity of the face.  
Normal = straight or slightly convex profile.  
In case of a disproportion, it does not indicate which jaw is wrong.



- Lip posture and incisor prominence:**
- Lip prominence is strongly influenced by racial and ethnic characteristics and age related.
  - The lip relationship with the nose and chin affects the perception of the lip fullness.  
To camouflage a big nose, the chin must be more prominent.
  - Major indicators of excessive lip support by the dentition:
    - Lip separation at rest.
    - Lip strain on closure.
  - Bimaxillary dentoalveolar protrusion:  
= Both lips are protruding due to important incisor proclination to gain space.