The etiology of orthodontic problems
Course Outline (5 sessions)

- Specific causes of malocclusion
- Genetic Influences
- Environmental Influences
- Etiology in contemporary perspective
Specific causes of malocclusion

Disturbances in Embryologic Development
Skeletal Growth Disturbances
Muscle Dysfunction
Acromegaly and Hemimandibular Hypertrophy
Disturbances of Dental Development
Environmental Influences

- Equilibrium Theory and Development of the Dental Occlusion
- Functional Influences on Dentofacial Development
Etiology in contemporary perspective

- Changing Views of Etiologic Possibilities
- Etiology of Crowding and Malalignment
- Etiology of Skeletal problems
Malocclusion is a developmental condition. In most instances, malocclusion and dentofacial deformity are caused, not by some pathologic process, but by moderate distortions of normal development.
Etiology of malocclusion

- Etiological assessment of malocclusion is an important aspect in orthodontics, as the genesis of the deformity provides keys to the planning treatment. The developmental process of the dentition and craniofacial growth takes place over a period of approximately 20 years, whereby the environment has a modeling impact on the genotype, being an integral part of the factors of heredity. Due to this interaction, it is difficult to classify the etiology of malocclusion exactly, as the causes are often multifactorial and prevent exact differentiation between endogeneously and exogeneously induced changes.
Etiology of malocclusion

Diagram showing the etiology of malocclusion, with two main categories: Hereditary factors and Environmental effects.
# Causes of Malocclusion

<table>
<thead>
<tr>
<th>Cause</th>
<th>Time</th>
<th>Tissue</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Heredity</td>
<td>1. Continual, intermittent, or only once</td>
<td>1. Neuromuscular tissue</td>
<td>1. Malfunction</td>
</tr>
<tr>
<td>2. Anomalies due to maldevelopment</td>
<td>2. Various age levels (pre- or postnatal)</td>
<td>2. Teeth</td>
<td>2. Malocclusion</td>
</tr>
<tr>
<td>5. Habits</td>
<td></td>
<td>5. Soft tissue, except muscles</td>
<td></td>
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<td>6. Diseases</td>
<td></td>
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<td>7. Malnutrition</td>
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</tbody>
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Specific Causes of Malocclusion
Specific causes of malocclusion

Disturbances in Embryologic Development
Skeletal Growth Disturbances
Muscle Dysfunction
Acromegaly and Hemimandibular Hypertrophy
Disturbances of Dental Development
Disturbances in Embryologic Development (Developmental Damage)

- Those malocclusions which, etiologically speaking, were caused by developmental damage during the fetal period, are considered *Congenital Anomalies*.

  (Moss 1962, Enlow 1982)
Congenital anomalies

- In many of these cases the exact causal pathogenesis cannot be determined. The following are included among the causes of these dysplasia proven to date:
  - Embryopathies caused by virus diseases in the mother (e.g. measles, toxoplasmosis, rubella ...)
  - Ionizing radiation
  - Poisonous effects (e.g. medication) or other teratogenic harmful substances.
Congenital anomalies

- Fetal damages with this type of genesis and the following craniofacial abnormalities have been proven to be closely connected with one another:
  - Maldevelopment of the first and second branchial arches
  - Micrognathism
  - Oligodontia
  - Anodontia
Congenital anomalies

- The majority of cases with lip-jaw-palate clefts are included in this etiological group.

- Hereditary embryonal defects only account for approximately 20% of these patients.

  (Schilli et al., 1970)
## Disturbances in Embryologic Development

<table>
<thead>
<tr>
<th>Stage</th>
<th>Time (humans) Post fertilization</th>
<th>Related Syndromes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Germ layer formation and initial organization of structures</td>
<td>Day 17</td>
<td>Fetal alcohol Syndrome (FAS)</td>
</tr>
<tr>
<td>Neural Tube formation</td>
<td>Days 18-23</td>
<td>Anencephaly</td>
</tr>
<tr>
<td>Origin, migration, and interaction of cell populations</td>
<td>Days 19- 28</td>
<td>Hemifacial microsomia Mandibulofacial dysostosis (Treacher Collins syndrome) Limb abnormalities</td>
</tr>
<tr>
<td>Formation of organ systems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary palate</td>
<td>Days 28-38</td>
<td>Cleft lip and/ palate, other facial clefts</td>
</tr>
<tr>
<td>Secondary palate</td>
<td>Days 42-45</td>
<td>Cleft Palate</td>
</tr>
<tr>
<td>Final differentiation of Tissues</td>
<td>Day 50 - birth</td>
<td>Achondroplasia Synostosis syndromes (Crouson’s, Apert, s, etc.)</td>
</tr>
</tbody>
</table>
Fetal alcohol syndrome
Fetal alcohol Syndrome
Lip and Palate Cleft
Median Mandibular Cleft
Median Mandibular Cleft
Median Mandibular Cleft
Crouson’s disease (Craniofacial dysostosis)
Hemifacial Microsomia
Hemifacial Microsomia
## Teratogens affecting Dentofacial Development

<table>
<thead>
<tr>
<th>Teratogens</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aminopterin</td>
<td>Anencephaly</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Cleft lip and palate</td>
</tr>
<tr>
<td>Cigarette smoke(hypoxia)</td>
<td>Cleft lip and palate</td>
</tr>
<tr>
<td>Cytomegalovirus</td>
<td>Microcephaly, hydrocephaly, microphthalmia</td>
</tr>
<tr>
<td>Dilantin</td>
<td>Cleft lip and palate</td>
</tr>
<tr>
<td>Ethyl alcohol</td>
<td>Central mid-face deficiency</td>
</tr>
<tr>
<td>6-Mercaptopurine</td>
<td>Cleft Palate</td>
</tr>
<tr>
<td>13-cis Retinoic acid (Accutane)</td>
<td>Retinoic acid syndrome: malformations virtually same as hemifacial microsomia, Treacher Collins syndrome</td>
</tr>
<tr>
<td>Rubella virus</td>
<td>Microcephaly, hydrocephaly, microphthalmia</td>
</tr>
<tr>
<td>Thalidomide</td>
<td>Malformations similar to hemifacial microsomia, Treacher Collins syndrome</td>
</tr>
<tr>
<td>Toxoplasma</td>
<td>Microcephaly, hydrocephaly, microphthalmia</td>
</tr>
<tr>
<td>X-radiation</td>
<td>Microcephaly</td>
</tr>
<tr>
<td>Valium</td>
<td>Cleft lip and palate</td>
</tr>
<tr>
<td>Vitamin D excess</td>
<td>Premature suture closure</td>
</tr>
</tbody>
</table>
Skeletal Growth Disturbances

- Fetal molding and birth injuries
  - Intrauterine molding
  - Birth trauma to mandible
- Childhood fractures of the jaw
Intrauterine molding

- Arm pressed across the face → Severe maxillary deficiency at birth
- Head flexed tightly against the chest → Pierre Robin anomaly or sequence (1/3 Stickler syndrome)
Pierre Robin Syndrome
Birth trauma to mandible

- Heavy pressure to TMJ → Internal hemorrhage, loss of tissue, under development of the mandible.
- It is rare and children with deformities involving the mandible are much more likely to have a congenital syndrome.
Childhood Fractures of the jaw

- 75% of the children with early fractures of the mandibular condylar process have normal mandibular growth.
- When problem arise it usually is asymmetric growth with the previously injured side lagging behind.
- 5% severe mandibular deficiency is due to early fracture of the jaw. (Dentofacial Clinic university of North Carolina)
- Problem arises when there is enough scarring in the area to restrict the normal growth movements of maxilla and mandible.
Ankylosis of TMJ
Ankylosis of TMJ
Childhood Fractures of the jaw

- The best therapy is conservative management at the time of injury and early mobilization of the jaw to minimize any restriction on movement.

- An old fracture is the most likely cause of asymmetric mandibular deficiency in a child but it might be due to rheumatoid arthritis or congenital absence of tissue as in hemifacial microsomia.
Muscle Dysfunction

- Torticollis
- Major decrease in tonic muscle activity
  - Muscular dystrophy
  - Cerebral palsy
  - Muscle weakness syndromes
Muscular Dysfunction
Acromegaly and Hemimandibular Hypertropy

- Acromegaly  ➔ Anterior Pituitary tumor
- Hemimandibular Hypertropy
  - Likely in girls between 15-20
  - Condylar hyperplasia prominent
  - Possible onset from age of 10 in either sex
Any Questions?