THEORIES OF TRAUMA FROM OCCLUSION

TMD and Occlusion Seminar
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LECTURE OUTLINE

• Definitions
• Tissue responses to increased occlusal forces
• Historical perspective
• Human studies
• Molecular mechanisms
• Conclusions
WHAT IS OCCLUSAL TRAUMA?

Definitions
DEFINITIONS

• **Occlusal Trauma**: The pathological or adaptive changes to the periodontium caused by excessive or abnormal occlusal forces

• Could be either *primary* or *secondary*:

• **Primary occlusal trauma**: Injury produced by excessive or abnormal occlusal forces acting on a normal, healthy periodontium.

• **Secondary occlusal trauma**: The injury produced by normal or excessive occlusal forces acting on a reduced / compromised periodontal attachment apparatus
TISSUE RESPONSES TO INCREASED OCCLUSAL FORCES
Under the forces of occlusion, a tooth rotates around a fulcrum or axis of rotation, which in single-rooted teeth is located in the junction between the middle third and the apical third of the clinical root and in multirooted teeth in the middle of the interradicular bone. This creates areas of pressure and tension on opposite sides of the fulcrum.
• **Occurs in 3 stages**: *(Carranza 1967)*

• **Stage 1 Injury:**
  - Temporary depression in the mitotic activity, proliferation and differentiation of fibroblasts
  - Temporary reduction in the rate of collagen and bone formation

• **With slightly excessive forces:**
  - **Pressure Side**: bone resorption with widening of PDL space. Blood vessels are numerous and reduced in size.
  - **Tension Side**: bone apposition with elongation of PDL fibers. Enlarged blood vessels.
Areas of tension and pressure in opposite sites of the periodontal ligament caused by experimentally induced orthodontic movement in a rat molar
• With greater excessive forces:
  - Compression of blood vessels → Hyalinisation
  - Necrosis of areas of the PDL
  - Disruption of blood vessel walls at 1-7 days
  - Increased resorption of the alveolar bone and root surface

• With severe excessive forces:
  - Thrombosis, haemorrhage, tearing of the PDL fibers, and resorption of alveolar bone on the tension side.
  - Pressure severe enough to force the root against bone causes necrosis of the periodontal ligament and bone. The bone is resorbed from viable periodontal ligament adjacent to necrotic areas and from marrow spaces, a process called undermining resorption
change in shape of marginal bone found in a human autopsy case resulting from chronic occlusal trauma
• **Stage 2 Repair:**
  - Repair is *constantly occurring* in the normal periodontium
  - *Forces remain traumatic only as long as the damage produced exceeds the reparative capacity of the tissues*
  - Damaged tissues are removed, new CT, cementum and bone are formed in an attempt to restore the injured periodontium to its original state

• **Stage 3 Adaptive remodeling:**
  - If repair can not keep pace with the destruction caused by the forces, the periodontium will attempt to remodel in an effort to create a structural relationship in which the forces are no longer injurious to the tissues *(Tooth Migration)*
  - This results in a widened periodontal ligament, which is funnel shaped at the crest, and angular defects in the bone, with no pocket formation. The involved teeth become loose
relative amounts of areas of bone formation and bone resorption in periodontal bone surfaces in rats as a result of occlusal trauma
HISTORICAL PERSPECTIVE
**Hypothesis:** Excessive pressure/tension due to occlusal trauma changes PDL fiber orientation allowing gingival inflammation to progress directly into the PDL

- *6 Adult Rh monkeys*
- *Test teeth:* Gold crowns to produce excessive orthodontic type forces
- All animals had plaque-induced gingivitis

**On the pressure side:**
- PDL fibers rearranged parallel to the tooth surface.
- Osteoclastic resorption of adjacent bone
- Marginal funnel shaped widening of the PDL space
GLICKMAN AND CO-WORKERS 1968
“THE ALTERED PATHWAY OF DESTRUCTION”

- Similar Animal study
- The altered pathway of destruction, which occurred in the presence of excessive forces that allowed gingival inflammation to extend along the periodontal ligament, resulted in vertical bony defects where bone loss followed the PDL.
GLICKMAN AND CO-WORKERS 1963
“THE CO-DESTRUCTIVE EFFECT”

• Series of studies based on human autopsy material

• In the presence of plaque, the inflammation is confined to the *zone of irritation* (bound by marginal gingiva and transseptal fibers apically)

• With the presence of occlusal trauma, the gingival fibers degenerate and allow the inflammation to spread apically directly through the PDL (*zone of co-destruction*)
Without occlusal trauma, the gingival inflammation extends apically by involving the alveolar bone and later the PDL area.

With the presence of occlusal trauma, the inflammatory lesion spreads directly into periodontal ligament. This will create an angular bony lesion combined with an infrabony pocket.
In his classical study, Waerhaug disputed Glickman’s concept. Based on a large number of human autopsy specimens, he found that bacterial plaque in conjunction with variation in local anatomy was the primary cause of intrabony defect formation and not occlusal trauma.

The reduction in alveolar crestal height was related to the presence of subgingival plaque, ranging from 0.5 to 2.7 mm (mean 1.63 mm) from the alveolar crest. The angular defects resulted when subgingival plaque advanced to different levels on adjacent teeth, and circumferential defects formed when the alveolus was thicker than the range of bacterial influence.

Excessive occlusal forces have no relationship to the underlying bone defect.

Bone loss was always associated with the plaque downgrowth.
ANIMAL STUDIES

Rochester Group (Primate Model) vs. Gothenburg group (Dog Model)
### ANIMAL STUDIES

<table>
<thead>
<tr>
<th>Rochester Group (Polson and co workers)</th>
<th>Gothenburg Group (Lindhe and co workers)</th>
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<tr>
<td>• Used <em>squirrel monkeys</em></td>
<td>• Used <em>beagle dogs</em></td>
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<td>• Used <em>mesial-distal compression forces (orthodontic type)</em></td>
<td>• Applied <em>buccal-lingual forces using a cap splint (jiggling type)</em></td>
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<td>• Experimental times up to 10 weeks</td>
<td>• Experimental times up to one year</td>
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<td>• Occlusal trauma <em>does not</em> influence periodontal disease progression</td>
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<td>(No evidence of attachment loss in the presence of plaque and occlusal forces)</td>
<td>• Occlusal trauma <em>could accelerate</em> the progression of periodontal disease</td>
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<td>• Occlusal trauma <em>can cause bone loss</em></td>
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<td>• Adaptive changes in response to occlusal trauma can be largely <em>reversible</em> if inflammation is controlled</td>
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<td>• Bone regeneration maybe inhibited in the presence of inflammation</td>
<td>• In the presence of <em>reduced healthy periodontium</em>, occlusal trauma will <em>not</em> produce loss of attachment</td>
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<td>• The ability of the periodontium to adapt to occlusal trauma maybe inhibited in the presence of inflammation</td>
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Fig. 4. A photomicrograph of a lesion of a test tooth (Dog 2) which extends beyond apex. Note the presence of plaque and calculus in the apical portions of the mesial root (×15).

Ericsson & Lindhe 1982
ANIMAL STUDIES

BOTTOM LINES

• Trauma from occlusion alone could not cause attachment loss

• When excessive occlusal forces were removed, loss of bone density was reversible, except in the presence of periodontitis

• More bone density was lost in the presence of both, inflammation and occlusal forces

• The control of plaque and gingival inflammation would stop the periodontal disease progression in the presence or absence of excessive occlusal forces.
ANIMAL STUDIES
BOTTOM LINES

• Trauma from occlusion tends to change the shape of the alveolar crest. The change in shape consists of a widening of the marginal periodontal ligament space, a narrowing of the interproximal alveolar bone, and a shelf like thickening of the alveolar margin.

• In the absence of inflammation, the response to trauma from occlusion is limited to adaptation to the increased forces. In the presence of inflammation, however, the changes in the shape of the alveolar crest may be conducive to angular bone loss, and existing pockets may become intrabony.
HUMAN STUDIES

Does this all apply to humans?

Big Controversy !!

Ethical Issues

REEM AL KATTAN
HUMAN STUDIES

- Some studies have reported that patients who have occlusal discrepancies have no more severe periodontal destruction than do patients without occlusal discrepancies (Knowles 1967, Philstrom et al. 1986, Ramfjord 1981, Rosling et al. 1976)

- **Philstrom et al.** examined maxillary 1st molars clinically and radiographically in 300 individuals for signs of occlusal trauma and severity of periodontitis. Teeth with signs of occlusal trauma did not show greater severity of periodontitis than teeth without these signs.

- **Rosling et al.** found that regeneration of bone within osseous defects after surgical treatment will occur in patients maintained on an ideal standard of oral hygiene. **Tooth mobility had no influence on the healing.**
HUMAN STUDIES

- Other studies reported that tooth mobility can affect the response to periodontal treatment (Fleszar et al. 1980). In an 8-year longitudinal study of 82 patients, there was a mean difference of 1.57 mm in attachment gain between grades 0 and 3 mobility for teeth with 7 to 12 mm pockets, 2 years after treatment. Pockets of clinically mobile teeth did not respond as well to treatment as firm teeth with comparable initial disease severity.

- A series of reports on risk factors for periodontal disease indicated that mobility and parafunctional habits that are not treated with a biteguard are associated with increased attachment loss, worsening prognosis, and tooth loss (McGuire & Nunn 1996).
HUMAN STUDIES

- A recent retrospective study \textit{(Harrel et al. 2001)} showed that:
  - Teeth with occlusal discrepancies had deeper PDs and worse prognosis
  - Occlusal adjustment improved the prognosis
  - Occlusal discrepancies can be viewed as a risk factor that contributes to more rapid periodontal destruction

- However, the individual tooth was used as the experimental unit
- There were issues with the sample size!!
- Teeth with occlusal discrepancies were identified (Not occlusal trauma)
Figure 1. Change in probing depth over time for all subjects. General estimating equation regression model with median follow-up of 2.7 to 8.7 years; range of follow-up, 0.8 to 21.2 years. mm: Millimeters. Adapted with permission of the American Academy of Periodontology from Harrel and Nunn.\textsuperscript{38}
MOLECULAR MECHANISMS
MOLECULAR MECHANISMS

- RANKL is expressed by osteoblastic lineage cells and stimulates its specific receptor RANK to promote differentiation, survival, fusion, and activation of osteoclasts and to prevent osteoclast apoptosis.

Receptor activator of NF-κB ligand (RANKL), receptor activator of NF-κB (RANK), and osteoprotegerin (OPG) in the regulation of osteoclast cell biology.
A recent study on rats has demonstrated that inflammation and traumatic occlusion enhance the expression of RANKL on endothelial, inflammatory and periodontal ligament cells (Yoshinaga et al. 2007), using Escherichia coli LPS as the stimulant and gold inlays to raise the bite and induce occlusal trauma.

Expression of RANKL could be a more definitive marker than the clinical parameters of occlusal trauma.
MOLECULAR MECHANISMS

- Regulation of osteoclastogenesis is determined by the balance between RANKL and osteoprotegerin (OPG).

- Reduced expression of OPG has been demonstrated in PDL cells in vivo in response to a combination of LPS and mechanical stress (Tsuji et al. 2004).
CONCLUSIONS
- Trauma from occlusion does not initiate connective tissue attachment loss.

- Occlusion may play a secondary role in the progression of periodontal disease.

- Inflammation should be removed initially and potential occlusal factors subsequently re-evaluated.

- Healing following surgical treatment of periodontal disease may be more advantageous in non-mobile than in mobile teeth.
thank you