Unanswered Questions:
Can Bone Lost from Furcations Be Regenerated?

Based on DCNAChapter by
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University at Buffalo

Furcation defects present unique challenges

- Furcation defects present unique challenges.
- Furcation defects are bordered by both alveolar bone and root surfaces colonized by oral bacteria and contaminated by bacterial toxins.
- The furcation forim may be so narrow that patient oral hygiene and professional debridement is impossible.

Similar to periodontal osseous defects, cortical bone lines periodontal furcation defects. The procedure for osseous grafts frequently includes cortical (intramarrow) penetration. This causes bleeding from subcortical cancellous bone, clot formation, and migration of osteoblasts into the defect.
- Intramarrow penetration has been shown to increase clinical bone gain in infrabony defects treated by open flap debridement.
Three types of furcation defects were described by Hamp et al based on clinical probing measurements:

• degree I = horizontal loss of periodontal tissue in the furcation less than 3 mm
• degree II = horizontal loss of support in the furcation exceeding 3 mm but not encompassing the total width of the furcation area
• degree III = horizontal through-and-through loss of the tissue in the furcation.

Diagnosis

• Standard periapical, bitewing, and panoramic radiographs are used to diagnose furcation defects.
• A significant amount of alveolar bone loss must occur before a furcation defect can be seen on a radiograph.
• Initial degree I furcation defects are only detectable by probing.
• More severe degree II and III defects may be evident on radiographs but even then the defects may be hidden by superimposition of roots.
• Furcation defects might be evident as slight alterations in trabecular radiodensity or they might demonstrate gross radiolucencies.
• Consequently, the sensitivity of standard radiographs in detecting furcation defects is low but the specificity is high.

Probing

• Furcation defects may be misclassified by probing.
• Zappa and colleagues compared probing with surgical exposure of furcation defects.
• 7% of degree 1, 24% of degree 2 (Hamp index) and 27% of degree 3 involvements were not recognized.
• Pistorius and colleagues found probing to underestimate 31% of all furcation defects.
Furcation anatomy

Fig. 1. Electron microscopic view of a furcation. This defines a potential opening equal to the coronal root proportion that can facilitate root bleeding and lead to progression of furcation defects. (C) coronal aspect proportion. (From Blocki GL, Goldstein GA, Jemt T, et al. Critical aspect projections and associated pathologic opening in mandibular furcations. J Periodontal 2012;83(9):198-202, with permission.)

Traditional approaches for treatment of furcations

- resective surgery
- tunneling
- root resection/amputation
- bicuspidization.
The cells that repopulate the exposed root surface after periodontal surgery define the nature of the attachment that will form.

- Epithelial cells: Long junctional epithelium
- Gingival connective tissue cells: Connective tissue attachment followed by root resorption
- Bone cells: Ankylosis and root resorption
- Periodontal ligament mesenchymal cells: New cementum, periodontal ligament and alveolar bone (preferred—tissue regeneration).

Melcher, 1976

Regeneration

A number of approaches can result in tissue regeneration

- Physical prevention of epithelial migration over a root surface will favor repopulation of the root surface by periodontal ligament cells—guided tissue regeneration (GTR)
- Bone grafts
- Growth factors that promote growth of PDL cells.

Proof of regeneration

- Histologic evidence is required for proof of regeneration—however, not possible for clinical evaluation as it requires tooth extraction.
- Circumstantial evidence
  - Surgical Re-entry
  - Radiographs
  - Probing
Table 1

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Effect</th>
<th>Treatment</th>
<th>Findings</th>
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<tr>
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Fig. 3. A histological section of a human molar stained with hematoxylin and eosin. The sections were cut at 4 µm and stained with hematoxylin and eosin. The sections were then mounted on glass slides and viewed under a microscope. The images were captured using a digital camera.
Guided Tissue Regeneration (GTR): placement of physical barriers that prevent apical migration of epithelial and gingival connective tissue cells, and provides a secluded space for the inward migration of periodontal ligament mesenchymal cells.

Clinical responses of furcation defects to regeneration depend on tooth anatomy, anatomic features of the furcation area, and morphology of the furcation defect.
TYPES OF BARRIER MEMBRANES:

Bioabsorbable Membranes: collagen, degradable polymers (polylactic acid, polyglycolic acid, and polylactate/polyglycolate)
- Excellent clinical results
- Lower frequency of early spontaneous exposure
- Membranes don’t need to be surgically removed
- Early membrane degradation

No-resorbable and bioabsorbable membranes are equally effective in the treatment of periodontitis and furcation defects.

CLINICAL OUTCOMES

Additional details on the use of absorbable and non-absorbable barriers in periodontal surgery have been published in several studies.

| Method | N | GTR | BC | GTR+BC | PEG
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<td>Bone Gain</td>
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TREATMENT OF INTRABONY LESIONS

Treatment of intrabony defects with GTR results in greater probing depth reductions and clinical attachment gains compared with open flap debridement alone.

Clinical efficacy of GTR in intrabony defects depends on the morphology of the defect.

Narrow defects are associated with increased amounts of probing attachment level gains.

Number of residual bone walls has a minimal effect on the clinical outcomes of GTR.

OTHER FACTORS AFFECTING GTR CLINICAL OUTCOMES

<table>
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<tr>
<th>Patient Factors</th>
<th>Local Factors</th>
<th>Surgical Considerations</th>
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<tbody>
<tr>
<td>Poor oral hygiene</td>
<td>Gingival inflammation</td>
<td>Intracrestal incisions with preservation of interproximal gingival tissue</td>
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<tr>
<td>Vertical releasing incisions (only if nec.)</td>
<td>Vertical enamel projections</td>
<td>Vertical releasing incisions (only if nec.)</td>
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<tr>
<td>Incisions are placed apically away from the anticipated membrane placement</td>
<td>Enamel pearls; tooth mobility</td>
<td>Incisions are placed in areas away from the anticipated membrane placement</td>
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<tr>
<td>Full-thickness flaps are then elevated</td>
<td>Local factors that favor plaque accumulation (calculus, overhanging restorations)</td>
<td>Flap thickness varies</td>
</tr>
<tr>
<td>Flap is coronally displaced</td>
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<tr>
<td>Mucoperiosteal flaps are apically sutured</td>
<td>Temporary stability achieved</td>
<td>Mucoperiosteal flaps are apically sutured</td>
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<tr>
<td>Mucoperiosteal flaps are sutured around the neck of the tooth</td>
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</tr>
<tr>
<td>Resorbable membranes are fixed by resorbable sutures or simply adapted in place</td>
<td>Periosteal releasing incisions are made whenever needed</td>
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GTR INTRABONY DEFECTS

Dr. Cristina Villa, UT HS CS A

GTR INPACIFICATION LESIONS

Dr. Michael Mills, UT HS CS A

POSTOPERATIVE CARE AND MAINTENANCE

INFECTION CONTROL: Doxycycline (100 mg orally twice daily for 1 week)

PAIN CONTROL: nonsteroidal analgesics

Mechanical tooth cleaning in the surgical site for 12 weeks

0.12% chlorhexidine digluconate (12 weeks)

Weekly recall visits for monitoring and professional plaque debridement

Suture removal: 2 to 3 weeks after surgery

Nonresorbable barriers should be removed after 6 to 8 weeks

Probing and subgingival instrumentation: contraindicated in the first 6 months

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# COMPLICATIONS

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<tr>
<td>Membrane exposure</td>
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<td>Membrane exfoliation</td>
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<td>Sloughing or perforation of the flap</td>
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<td>Bleeding</td>
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<td>Swelling</td>
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<td>Asymmetry</td>
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<td>Suppuration</td>
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<td>Hematoma</td>
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<td>Postoperative pain</td>
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## Clinical Reality

- Regeneration techniques – can predictably add 1-2 mm of new attachment compared to traditional debridement surgery in furcations.

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**Table 4**

<table>
<thead>
<tr>
<th>Category</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>Class I defects</td>
<td>Most defects successfully treated with periodontal regenerative surgical therapy, regenerative therapy beneficial in certain cases</td>
</tr>
<tr>
<td>Class II defects</td>
<td>Regenerative treatment predictable; periodontal regenerative demonstrated histologically and clinically</td>
</tr>
<tr>
<td>Class III defects</td>
<td>One case report demonstrates periodontal regeneration histologically</td>
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<tr>
<td>Class IV defects</td>
<td>Regenerative therapy not predictable; shown only in clinical case reports</td>
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Does Gingival Recession Require Surgical Treatment?

Based on DCNA Chapter by
Hsun-Liang Chan, Yong-Huei Patricia Chun, Mark Madfiseth, Thomas W. Oakes
University of Texas Health Science Center at San Antonio

Gingival recession

• Gingival recession is defined as “the location of the gingival margin is apical to the cemento-enamel junction (CEJ).”

• About 23% of adults in the United States have one or more tooth surfaces with 3 mm or more of gingival recession.

• The cause of gingival recession is multifactorial, confounded by poorly defined contributions from predisposing and precipitating factors.
The role of keratinized mucosa

- Inadequate keratinized mucosa (KM) (equal or less than 2 mm) is commonly seen along with gingival recession.
- Inadequate KM might simply be a consequence of gingival recession, rather than a cause of gingival recession (supported by an interventional, longitudinal study that concluded that attachment level could be maintained with control of gingival inflammation, even without adequate KM. Poor oral hygiene may be considered a precipitating factor for gingival recession.

Etiology

- Resulting from periodontitis (poor hygiene).
- Most common on the facial side of canines and premolars, and associated with overzealous brushing habits possibly the result of excessive toothbrushing.
- Local gingival tissue trauma or irritation as found with tobacco chewing and oral piercing, which can lead to inflammatory changes in the tissues resulting in gingival recession.
- Orthodontic tooth movement in the mandibular incisors.
- Repeated scaling and root planning or periodontal surgeries on shallow pockets may induce clinical attachment loss, partially manifested by gingival recession.
Gingival recession

- Surgical correction of a gingival recession is often considered when (1) a patient raises a concern about esthetics or tooth hypersensitivity, (2) there is active gingival recession, and (3) orthodontic/restorative treatment will be implemented on a tooth with presence of predisposing factors.

- The benefits of these treatment approaches are not well supported in current literature relative to alternative approaches with control of possible etiologic factors.
Box 2
Diagnosis/prognosis for gingival recession

Miller classification

- Class I: recession not beyond MGJ; no interproximal tissue loss; 100% coverage expected
- Class II: recession extend to or beyond mucogingival junction (MGJ); no interproximal tissue loss; 100% coverage expected
- Class III: recession extend to or beyond MGJ; presence of loss of interproximal tissue and/or tooth malposition; partial coverage expected
- Class IV: recession extend to or beyond MGJ; presence of loss of interproximal tissue and/or tooth malposition; coverage not expected


Fig. 7. Demonstration of a root coverage procedure with an autograft. (A) Gingival recession was found on teeth 1B and 4B. (B) A coronally advanced flap was planned. (C) An autograft was placed on 4B. (D) Results after 12 months showed satisfactory root coverage. (Courtesy of Dr. Jack Miller, DDS, MS, director of Graduate Periodontics Program, University of Michigan, Ann Arbor, MI.)