Gingival Recession – Etiology and Treatment

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This article focuses on the recognition and understanding of recession defects of the oral mucosa. Specifically, which cases are treatable, how we treat these cases and why we chose certain treatments. Good evidence has suggested that the amount of height of keratinized or attached gingiva is independent of the progression of recession (Miyasato et al. 1977, Dorfman et al. 1980, 1982, Kennedy et al. 1985, Freedman et al. 1999, Wennstrom and Lindhe 1983). Such a discussion is an important consideration with recession defects but this article will focus simply on a loss of marginal gingiva.

Recession is not simply a loss of gingival tissue; it is a loss of clinical attachment and by necessity the supporting bone of the tooth that was underneath the gingiva. Recession is measured by the distance from the CEJ to the gingival margin, but the gingival margin in health typically covers 1-3mm of the crown and does not rest at the CEJ. When we measure a recession defect of 1mm, it is not simply 1mm of attachment loss, but instead 2-4mm of attachment. This is why we include both the recession and the pocket depth when calculating attachment loss. It also hopefully brings to light an important point – when we detect recession, significant attachment loss has already occurred.

Recession defects typically present to us as 3 different patient scenarios. The most common is the asymptomatic patient who often is never even aware of the attachment loss unless notified by a dental professional. The other two common scenarios are a patient with tooth or gingival sensitivity or a patient displeased with his or her esthetic appearance.

Reasons to Treat

First we need to understand why we would treat any of these patients. The latter two scenarios provide the answer in the patient's complaint: they either want to alter the appearance of the gingiva or cover the exposed root to prevent discomfort. For these cases, if there is a reasonable expectation of a successful treatment outcome, then treatment should be recommended. In asymptomatic patients the reason is often a prophylactic one; that is we want to prevent the recession from getting worse. This reasoning is also true for the esthetic and sensitivity scenarios as well. Severe recession is not only more difficult to treat, but can also be associated with food impaction, poor esthetics, gingival irritation, root sensitivity, difficult hygiene, increased root caries, loss of supporting bone and even tooth loss . To avoid these complications we would want to treat even the asymptomatic instances of recession if we anticipate them to progress. However, non-progressing recession with no signs or symptoms does not need treatment. In order to know which cases need treatment, we need to distinguish between non-progressing and progressing recession. In order to do that, we need to understand the causes of recession.

Etiology

Typical causes of recession are trauma, periodontitis, tooth position or local inflammation (Wennstrom and Prato 2003). This list is not inclusive, as diseases, cysts, non-carious cervical tooth lesions, occlusal trauma or aberrant frena may also contribute to tissue defects, however they are the most common causes of recession.

Trauma resulting in recession is typically from aggressive tooth-brushing (Wennstrom and Prato 2003). Patients should understand that the term is tooth-brushing¬ and not tooth-scrubbing. Khocht et al. (1993) showed that hard tooth-brushes are also more likely to cause recession soft tooth-brushes. Tongue and lip rings can cause trauma to the marginal gingiva as can iatrogenic damage from scaling or other dental treatment and factitious habits, such as using tooth picks inappropriately or scratching the gingiva with finger nails or other devices. Traumatically induced lesions need to be first treated by addressing the etiology. If a tooth scrubber will not stop scrubbing his or her teeth, then treating the defect will only provide a temporary benefit and the defect will continue to progress. These recession defects are almost exclusively found on the facial and sometimes lingual surfaces of teeth.

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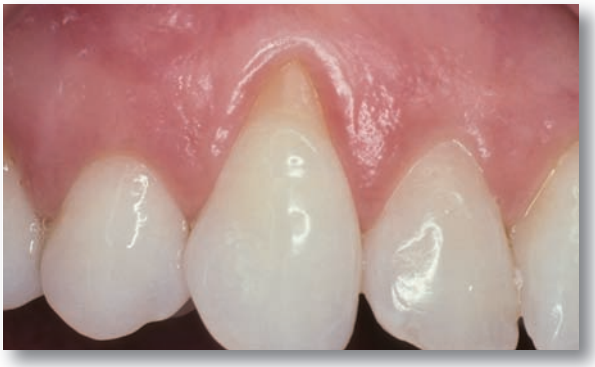


Fig. 1: 1.3 with a 2mm mid-facial recession defect in a patient who confesses to aggressive tooth-brushing.

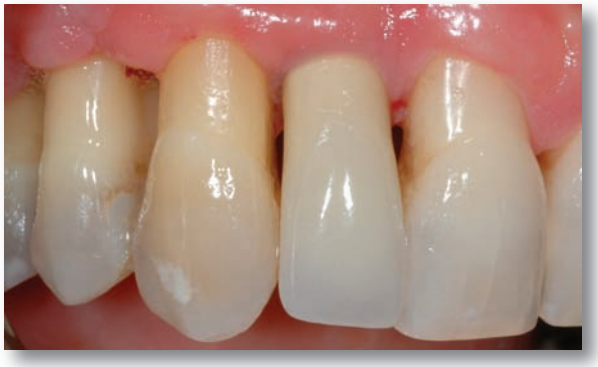
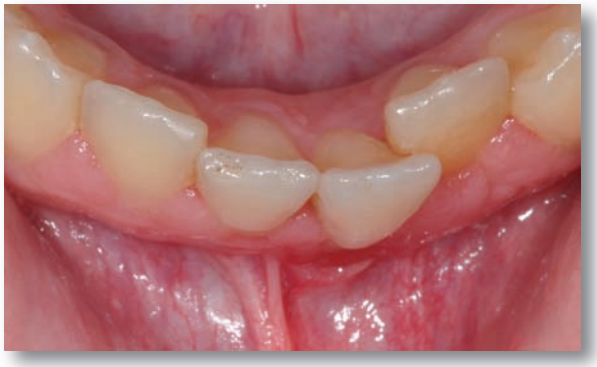
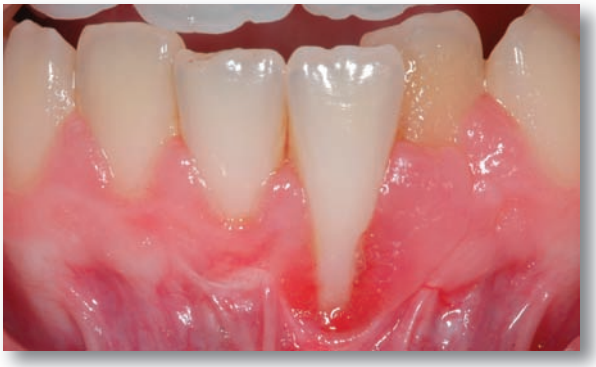


Fig. 2: Circumferential loss of marginal gingiva due to periodontitis.





Figs. 3a and 3b: A prominently positioned 3.2 moved outside of the maxillary alveolus with associated severe recession.

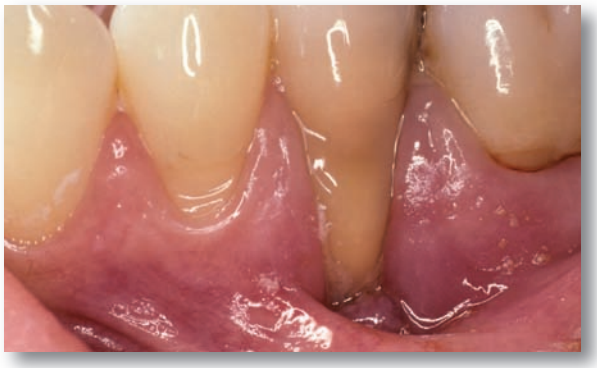


Fig. 4: Severe recession at 3.5 caused by traumatic injury.

Periodontitis associated recession defects are caused because the alveolar bone supports the gingiva. When the bone is lost, the gingiva becomes unsupported. Sometimes the gingiva remains in place due to intrinsic gingival fibers, but when recession occurs, it is difficult to regenerate because of the lack of underlying bone. These types of defects can be found on any surface of the tooth.

Tooth position is also a cause of recession defects. As said earlier, the bone supports the gingiva. If a tooth is moved outside of its alveolar housing, as in some orthodontic cases, then the tooth will often lose bone on the surfaces that extends outside. This can easily be detected by assessing root prominences. This can occur on any tooth that is moved outside the alveolus, either facially or lingually.

Finally, localized inflammation is theorized to be another major cause of recession (Baker and Seymour 1976). Patients who have a thin biotype or sensitive tissues are especially susceptible to this type of recession. It is theorized that localized inflammation, whether due to plaque or trauma, can sometimes involve the entire width of the gingiva, more commonly with thin and sensitive tissue. The epithelium may then proliferate and overcome the connective tissue, resulting in a subsidence of the epithelium that results in recession. This is likely the primary etiology for recession commonly found around supragingival calculus and restorations where the plaque accumulation at such sites can easily exacerbate local inflammation.

Treatment

Treatment of recession depends on its etiology. Recession due to periodontitis cannot be easily treated because there is no bone for grafted tissue to be supported by. It will continue to progress if the periodontitis is not stabilized.

Recession due to a tooth being positioned outside of the alveolus can be treated either before the tooth positioning occurs, by thickening the gingiva and making it more resistant to recession, or after it has occurred, by either tissue grafting, tissue grafting with root reduction or simply repositioning the tooth back into the alveolar housing (Wennstrom 1996). It is important to note that recession on roots outside of the alveolus may not be as predictably treated by tissue grafting alone because of the lack of bone to support the tissue graft. This etiology is self-limiting; once the tooth has lost the bone and gingiva covering the root prominence it tends to cease. However, if the recession has only occurred on a part of the root prominence, it may progress the entire length of the prominence unless treated.

Recession due to trauma should only be treated if the etiology is first arrested and it will tend to progress until the etiology is removed. Once removed, the recession typically does not progress.

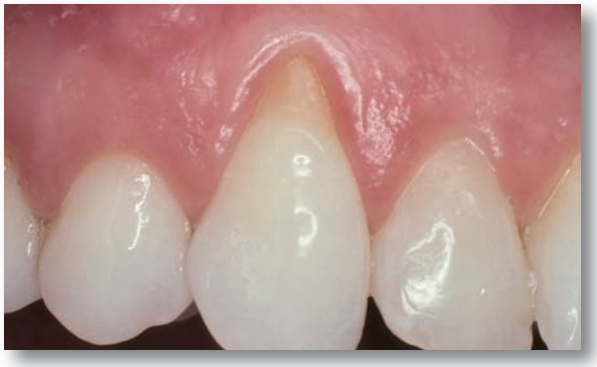


Fig. 5a: Same picture as in Figure 1, demonstrating recession due to what was believed to be traumatic tooth-brushing.

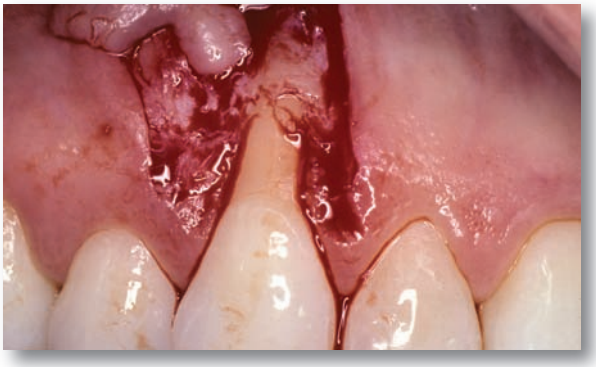


Fig. 5b: Preparation of the connective tissue graft recipient site.

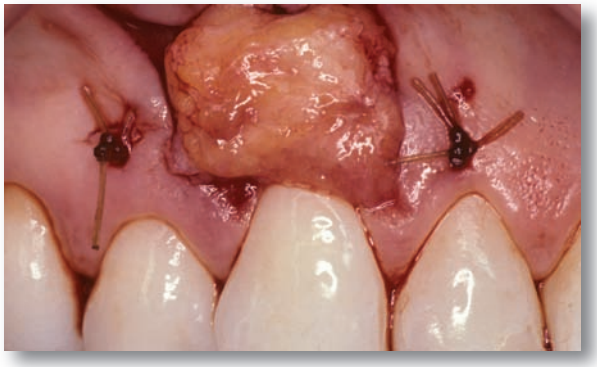


Fig. 5c: Connective tissue graft sutured in place.

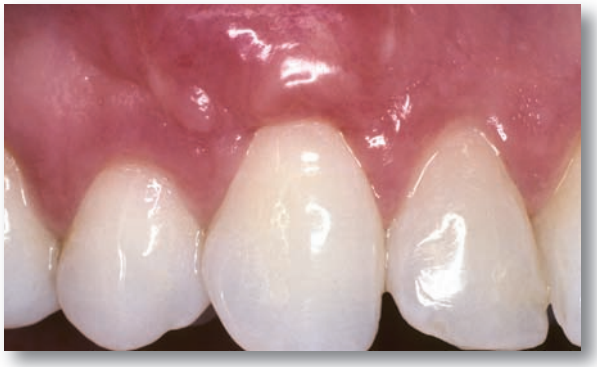


Fig. 5d: Connective tissue graft after 6 weeks.

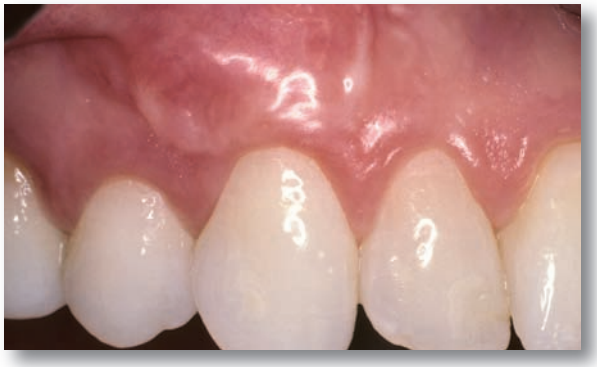


Fig. 5e: Connective tissue graft after 6 months.

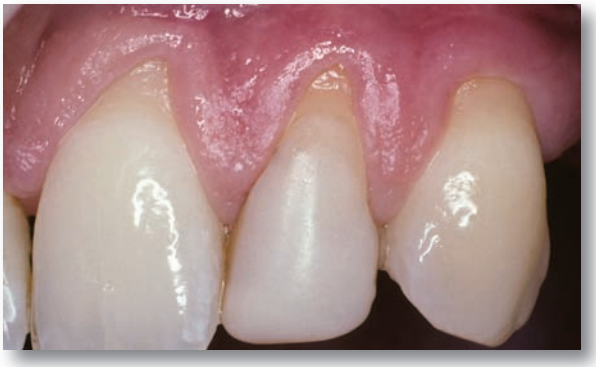


Fig. 6a: Recession before treatment with a connective tissue graft.

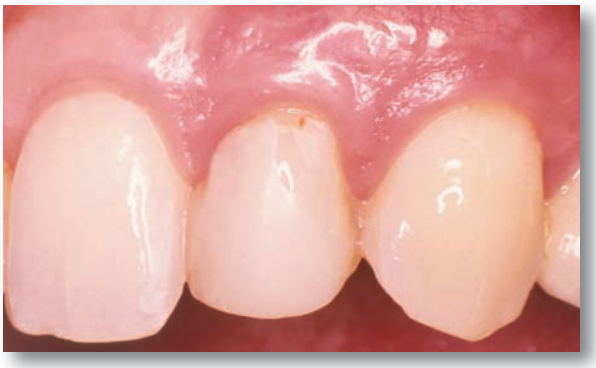


Fig. 6b: Connective tissue graft after 6 months.

Recession due to local inflammation can be treated two ways. First, if a restorative margin is at or below the gingival margin, the tissue can by prophylactically thickened in order to resist recession (Koke et al. 2003). Second, if the recession has already occurred, the tissue can be regenerated and thickened at the same time, although not as predictably as treatment prior to the occurrence of the recession. In both scenarios, consideration should be made towards limiting any suspected etiology in the area. If an overhung margin was the initial cause, regenerated tissue may suffer the same fate if it remains after treatment.

Finally, we must understand that the etiologies of recession are not always clear. It is not rare to have recession due to a factitious habit which appears to be due to an overhung restorative margin. Nor is it rare to have multiple etiologies, such as recession on a facially positioned tooth with a bulky crown margin in a patient who scrubs his teeth because he has periodontitis. These etiologies can be elusive and simultaneous. We should be cautious in discerning which etiologies we believe to be relevant to specific recession defects.

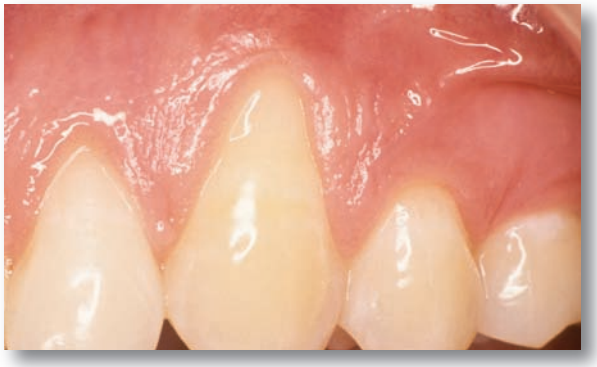


Fig. 7a: Recession before connective tissue graft.

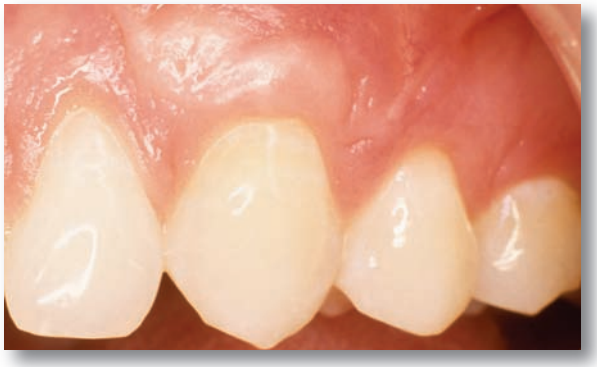


Fig. 7b: 6 months after connective tissue graft.

Fig. 7c: 5 years after connective tissue graft.

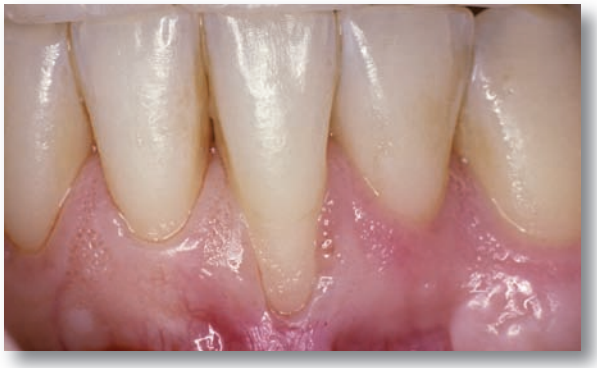


Fig. 8a: Recession before connective tissue graft.

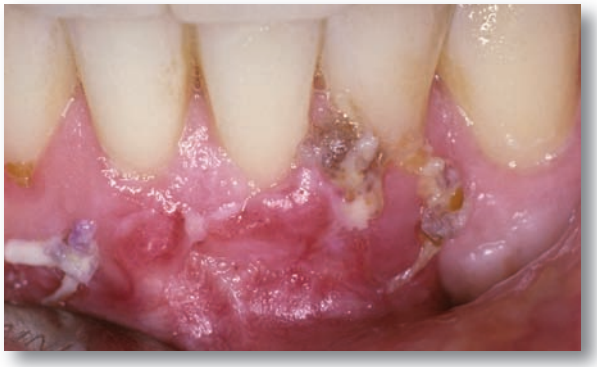


Fig. 8b: 2 weeks after connective tissue graft.

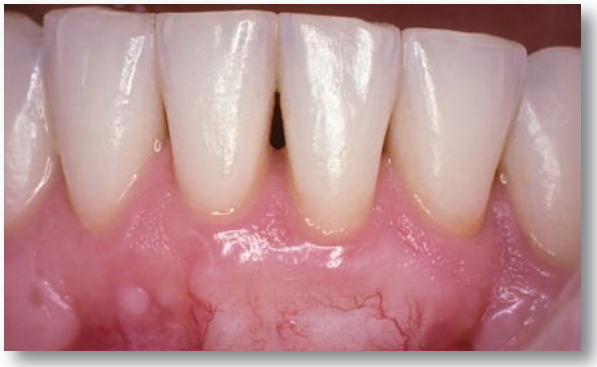


Fig. 8c: 2 years after connective tissue graft.

Summary

Recession defects can create several oral health issues and in extreme cases tooth loss. Just because a defect is asymptomatic does not mean it should not be treated. If recession is expected to progress we should attempt to prevent it from doing so. Recession is predominantly caused by tooth brushing trauma or periodontitis, but can also be caused by other types of trauma, prominent tooth positioning, local inflammation or other more uncommon conditions. Teeth with recession and no bone to support tissue grafting, such as in periodontitis and prominently positioned teeth, are difficult to predictably treat. Other recession defects can be more easily treated but the etiologies should be assessed and controlled prior to treatment.

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