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Review Article

Why Classify Further? When the Extension of Miller Classification of Gingival Recession is an Answer to Most Clinical Question Related to Diagnosis and Prognosis of Gingival Recession

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Abstract

Many attempts have been made in the past to classify gingival recession defects. Classification of gingival recession is one such condition where there is no clear-cut grouping of the gingival defects. The miller's classification is the most accepted classification of gingival recession so far. However, many classifications of gingival structural defects are popular in the literature but are of no significant clinical use. Too many classifications rather confuses the clinician about the diagnosis, treatment guidelines and prognosis of the gingival defect which may takes the attention of the clinician away into the classification terminology rather than the actual insight into the defect. The contribution of various authors in providing the classification is well appreciated and acknowledged, but it is not seemingly possible to understand the shortcomings and applications of all the classification system since no classification is complete, rather it would be better for the clinician to apply simple permutation and combinations of basic biological principals to achieve the favorable outcome. No two gingival recessions are similar in the mouth. A combination of Multiple factors is playing a role in defining the complex nature of gingival recession. Here in this paper an attempt has been made to acknowledge and simplify all clinical possibilities of gingival recession in terms of diagnosis and predetermining the prognosis.

Keywords: *Miller Classification; Gingival Recession; Prognosis; Gingival Defect*

Why do we classify something? The answer is simple; classification is grouping or segregating the objects or conditions in proper way to make it easier to work with them. Diagnosis and classification form an important part of approach to any condition or disease. But what if classification complicates the clinical situation? Classification of gingival recession is one such issue where there is no clear-cut grouping of the gingival recession defects and every clinical case is different. The miller's classification is the most accepted classification of gingival recession so far [1,2]. Further classification of gingival structural defects rather confuses the clinician about the diagnosis, treatment guidelines and prognosis of the gingival defect. It takes the attention of the clinician into the classification terminology rather than the actual insight into the defect.

So far many classification systems has been proposed in literature Sullivan and Atkins (1968), Mlinek (1973), Liu and Solt (1980), Bengue (1983), Miller (1985), Smith (1990), Nordland and Tarnow (1998), Mahajan (2010), Cairo., *et al.* (2011), Rotundo., *et al.* (2011),

Ashish Kumar and Masamatti (2013), Prashant., *et al*. (2014) and finally Nagappa and Mukta in 2019 [3]. I appreciate and acknowledge the contribution of various eminent researchers in this field, but it is not seemingly possible to understand the shortcomings and applications of all the classification system since no classification is complete, rather it would be better for the clinician to apply simple permutation and combinations of basic biological principals to achieve the favorable outcome. No two gingival recessions are similar in the mouth. A combination of Multiple factors is playing a role in defining the complex nature of gingival recession [4]. Here in this paper an attempt has been made to acknowledge and simplify all clinical possibilities of gingival recession in terms of diagnosis and predetermining the prognosis.

The etiology of gingival recession

The etiology of gingival recession can be classified into predisposing factors and precipitating factors [5].

Predisposing factors

1) Inadequate attached gingiva, shallow vestibule or high frenal attachment.
2) Malpositioning of the tooth
3) Osseous dehiscence.

Precipitating factors include

1) Toothbrush trauma
2) Recurrent inflammation
3) Lacerations
4) Iatrogenic factors.

The severity of recession defect and gingival response to trauma also gets influenced by the tissue biotype and the age of the patient [4,6].

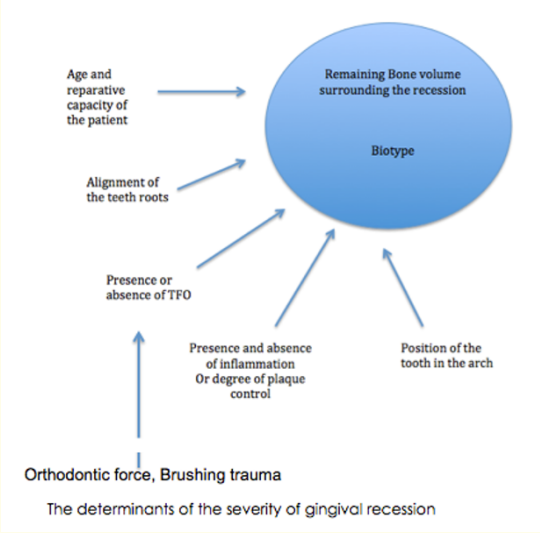


Figure 1

The pathogenesis of gingival recession

There are two possible mechanisms that could explain gingival recession of different etiological origin.

Gingival recession with minimal plaque/or with inadequate width of attached gingiva/ or recession caused by mild inflammation

Baker and Seymour in 1976 first explained the different stages in the development of gingival recession. In the initial stage there is normal or subclinical inflammation and histologically there is proliferation of epithelial rete pegs. Which in later stages shows increased epithelial proliferation resulting in loss of CT core and finally there is merging of oral and sulcular epithelium resulting in separation and recession of the gingival tissues due to loss of nutritional supply [7].

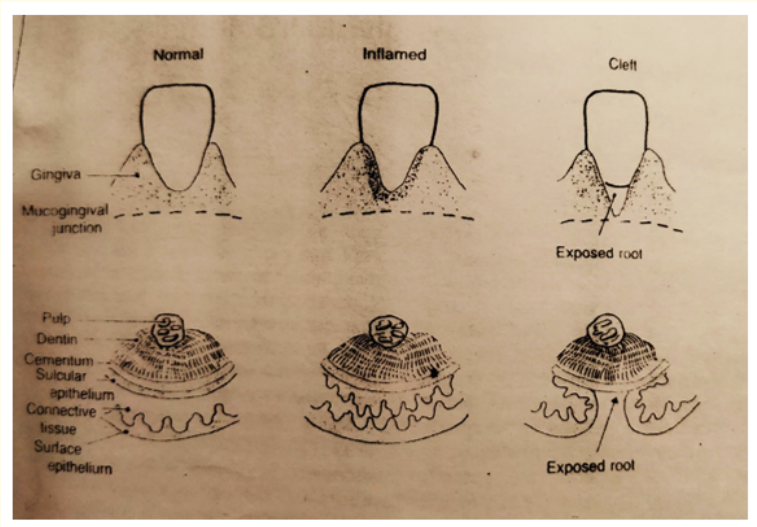


Figure 2: *Showing the pathophysiology of gingival recession.*

Gingival recession of infectious etiology

Waerhaug proposed that the distance between the periphery of plaque on the tooth surface and the labial, apical extension of the inflammatory infiltrate hardly ever exceeds 1 - 2 mm. Thus, if the free gingiva is voluminous the infiltrate will occupy only a small portion of the connective tissue however, if it is thin the entire connective tissue portion may be involved consequently there is proliferation of epithelial cells from the dento-gingival epithelium, the root dehiscence is exposed. This pathologic mechanism is called centrifugal as it acts from inside to the outside. Thus, the zone of CT decreases and it is obliterated by the fusion of these two epithelia. Finally, the epithelium loses its nutritional source, and gingival recession ensues [8].

On the other hand when brushing trauma is the cause of gingival recession the pathogenic mechanism is centripetal because it acts from outside towards the inside [9].

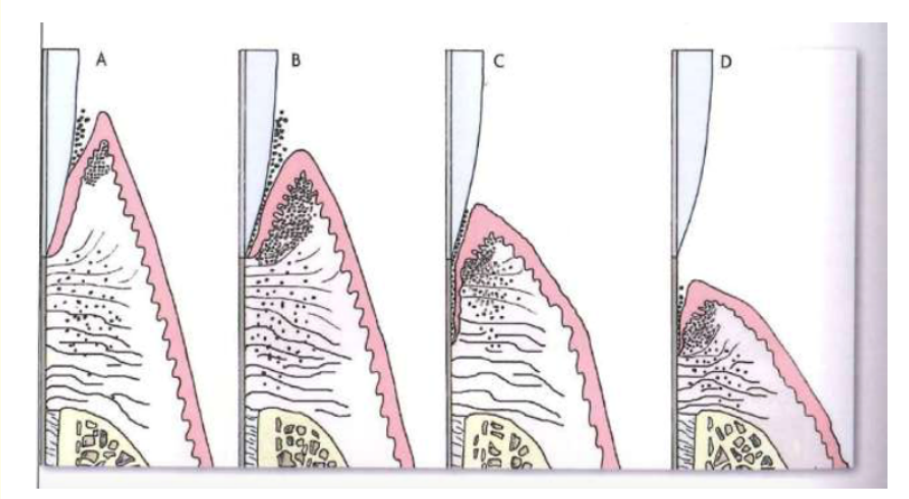


Figure 3: *Pathogenesis of recession along with pocket formation at the apical end due to bacterial plaque.*

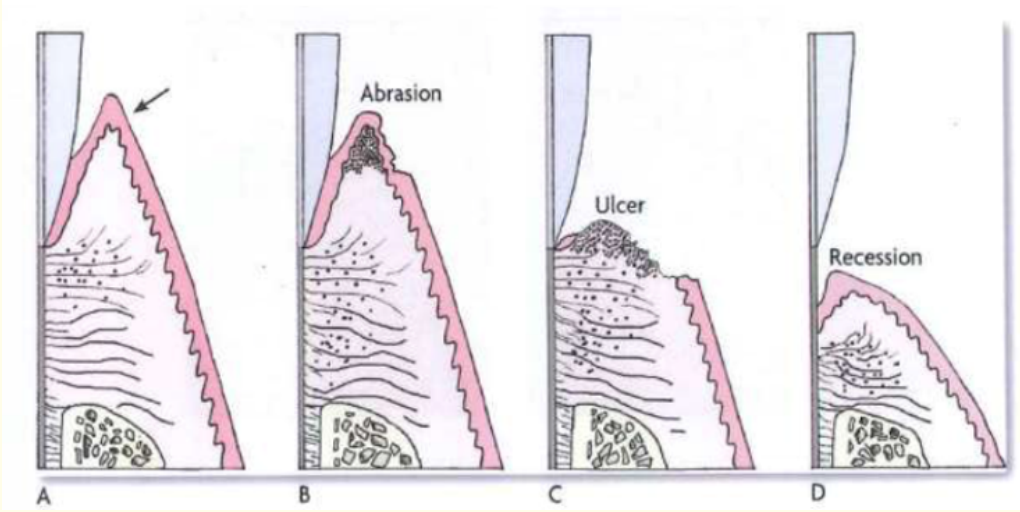


Figure 4: *Pathogenesis of recession due to toothbrush trauma.*

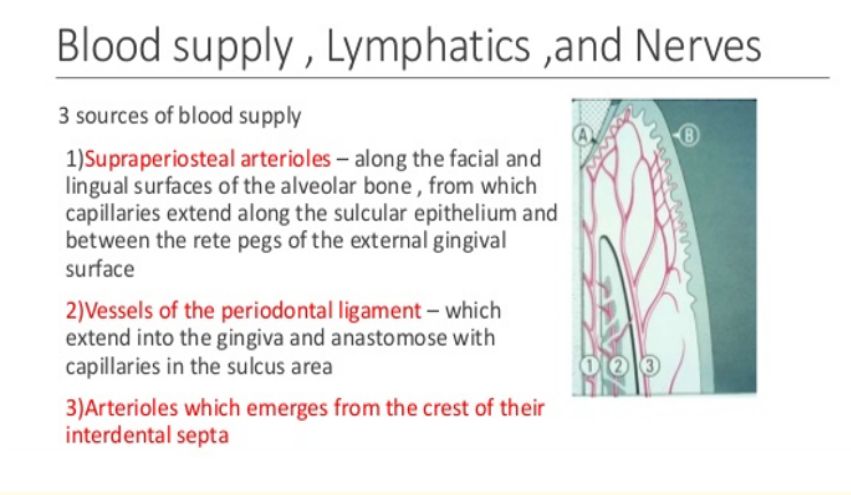
The bone as a biological tissue

Bone is a highly vascularized connective tissue [10]. Skeletal vasculature plays a significant role in the process of bone development (endochondral and intramembranous ossification), regeneration and remodeling and repair [11,12]. The skeletal system alone in the

body receives between 10 and 15% of total cardiac output [13]. Blood vessels supply the skeletal system with oxygen or nutrients also have other roles like removing the metabolites from the bone, they also provide the skeleton with specific hormones, growth factors and neurotransmitters secreted by other tissues (e.g. brain-derived serotonin) [14], maintaining the bone cells survival and stimulating their activity [15].

Mandible (an example of the irregular bone) is supplied by three arteries: the lingual, facial and inferior alveolar arteries. The first 2 arise directly from the external carotid artery [16]. The microvasculature of mandible possesses some characteristics as Panarale., *et al*. analyzed that the bone thickness strongly affects the pattern of the micro- circulation in the flat bones and that it varies in the thick and thin parts of all types of bones. At the sites thinner than 0.4 mm, only the periosteal and dural networks exist. Larger vessels, which do not form a true vascular network, connect the two sites of the bones in these regions. In thicker sites, the organization of the microvasculature is similar to that observed in the long bones. The blood flow pattern in bones has been described as primarily centrifugal: blood is supplied to the cortical bone through the nutrient arteries in the marrow cavity and returned by the periosteal veins [10,17]. In addition, there is evidence that the blood flow direction can shift from centrifugal to centripetal, de- pending on the hemodynamic conditions in the bone [18]. The vasculature of the long bone composed of three main systems: (1) the central nutrient artery, (2) metaphyseal-epiphyseal arteries, and (3) periosteal artery [19]. It is worth mentioning that in the long bones, the central nutrient artery supplies the entire medullar cavity and the 2/3 of the outer cortical bone, whereas the periosteal arteries penetrate the other 1/3 of the outer cortical bone [20]. Besides secreting different nutritional and signaling factors, e.g. VEGF-A, blood vessels localized in the bone marrow cavity orchestrate the process of hematopoiesis, and provide the Hematopoietic Stem Cells (HSCs) with the necessary niche [21].

The alveolar bone and the basal bone beneath it is highly vascular and in such an enriched environment there are a significant number of avascular paving stones or hindrances called teeth. Cementum over the root has a unique structure and it is completely avascular [22]. It is just that the arteries supplying the bone also supplying the tooth pulpal tissue to maintain the tissue integrity. The Root canal treated teeth does not even carry dental artery and are absolutely avascular [23]. Not only this main supply to the buccal side of gingiva place where gingival recession is common also comes from the underlying bony tissue namely the interdental and supra-periosteal and the arteries originating from periodontal ligament also supply a part of the gingiva (Figure 5) the triple blood supply of the gingival cuff clearly does not permit the vasculature of the gingival cuff to become embrassed by occlusal trauma or orthodontic tooth movement [24].



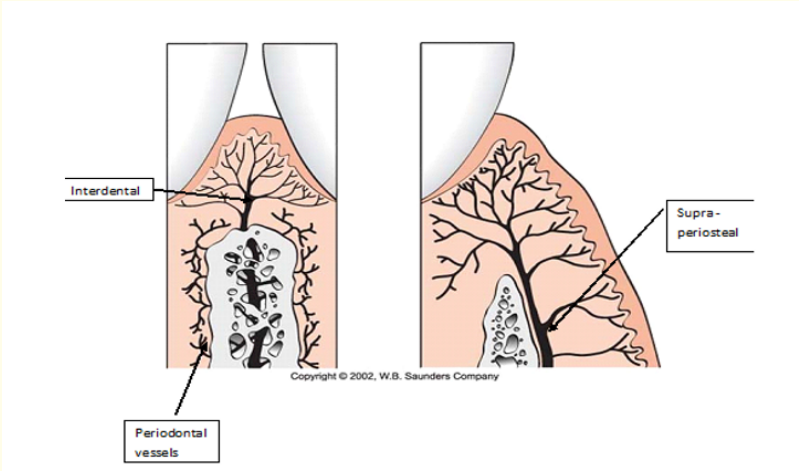


Figure 5

To analyze a gingival recession defect it is evident that the part of gingiva which is missing must have had lost its vascularity due to a number of reasons. The underlying tooth cementum is avascular. So the main source of nutrition to the remaining gingiva is coming from surrounding bone or adjacent periodontal ligament housing its vasculature [25].

As the etiology of gingival recession is multifactorial [4] the treatment is highly technique sensitive and there is a large discrepancy in the treatment outcomes worldwide [26].

Vascularity place a major role in etiology and repair of gingival recession. Decreased perfusion concurrent with the increased metabolic demands of repair leads to hypoxia within the wound site [4]. As a result, restoration of blood flow through angiogenic mechanisms is a key component of wound healing [10,27].

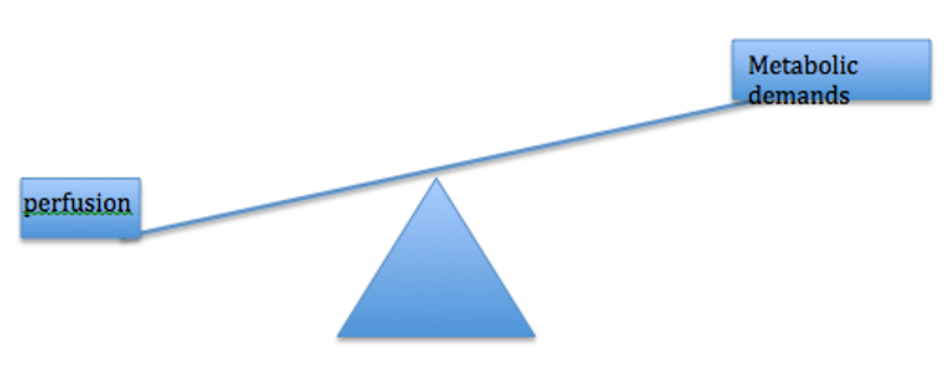


Figure 6

The regulation of blood flow is complex, involving a balance of both pro- angiogenic and anti-angiogenic factors. An endogenous anti-angiogenic factor, endostatin, was recently examined following fracture healing. Administration of exogeneous endostatin decreased vascularity and hard callus formation, but soft callus formation was increased [28].

The outcome of the recession surgery depends on the 3 dimensional volume of the remaining bone, presence of the amount of periodontal ligament and the soft tissue biotype [29], other factors which will influence these three factors or have indirect effect on the treatment outcome are: The alignment of the teeth roots, tooth position like rotation, extrusion, abrasions. The presence or absence of plaque [30] and inflammation, shallow vestibule, aberrant frenum [31], width of the attached gingiva [32] the presence or absence of Trauma from Occlusion [33,34], age of the patient which is a count of the regenerative capacity and position of the tooth in the arch [35].

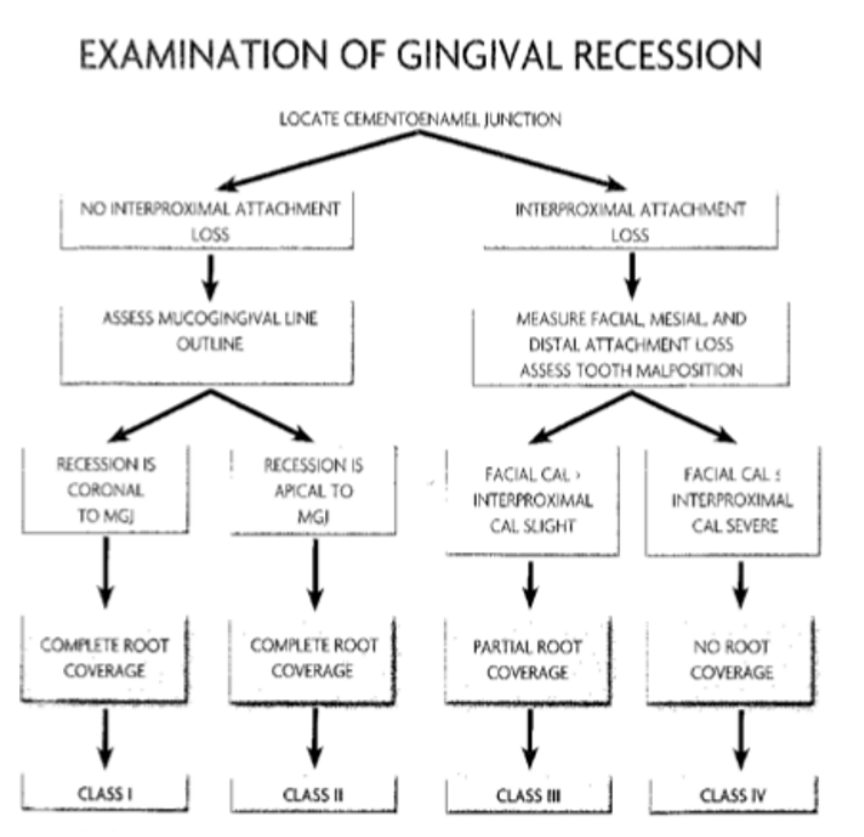


Figure 7

The Millers classification: Miller classified gingival recession in 1985 according to the prognosis of root coverage. Class I and ii include gingival recession affecting teeth with intact interdental periodontal support. In class I the gingival recession is confined to the facial keratinized tissue while in class ii the recession extends apical to the muco-gingival junction. Class iii gingival recession affects teeth whose interproximal attachment loss is less than their facial attachment loss. Class iv recession affects teeth with interproximal attachment loss is equal to or greater than the facial attachment loss [36,37].

Figure 8: *The complete root coverage is possible.*

Figure 9: *The complete root coverage is again possible.*

Class II is further classified in IIa and IIb.

Class IIa

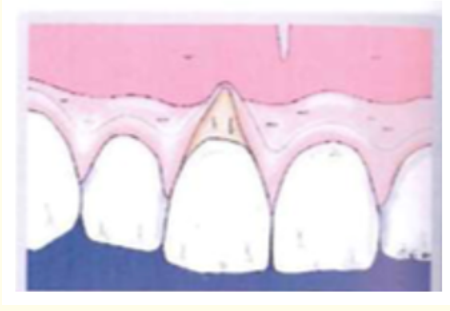


Figure 10

Clinically there exist forms of miller class II recession in which the most apical extension is apical to the mucogingival outline of the adjacent teeth but there is still a margin of keratinized tissue apical to the root area. The full coverage is still possible.

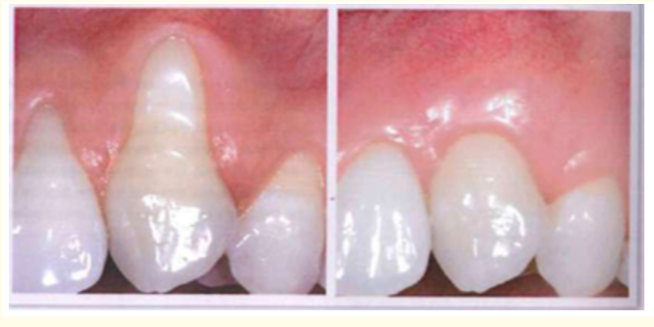


Figure 11

Class IIb

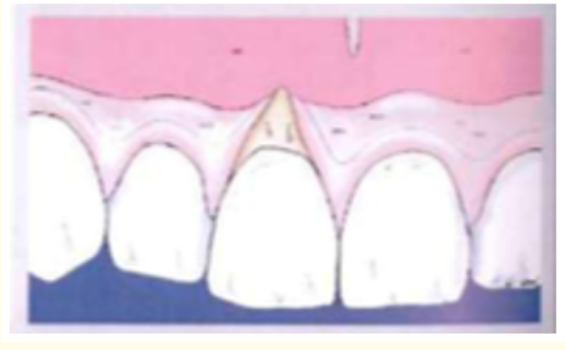


Figure 12

In this case there is no keratinized tissue apical to the exposed root area.

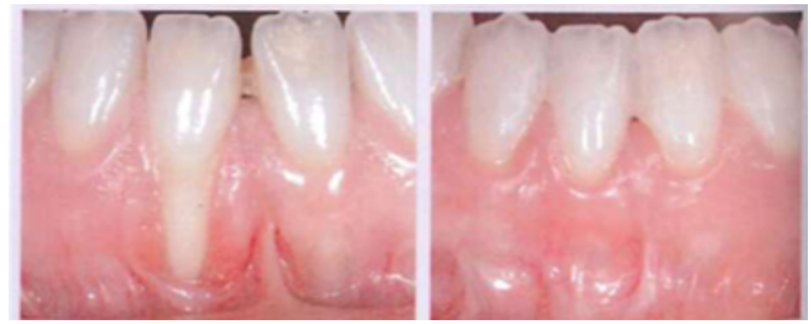


Figure 13

The complete root coverage is still possible.

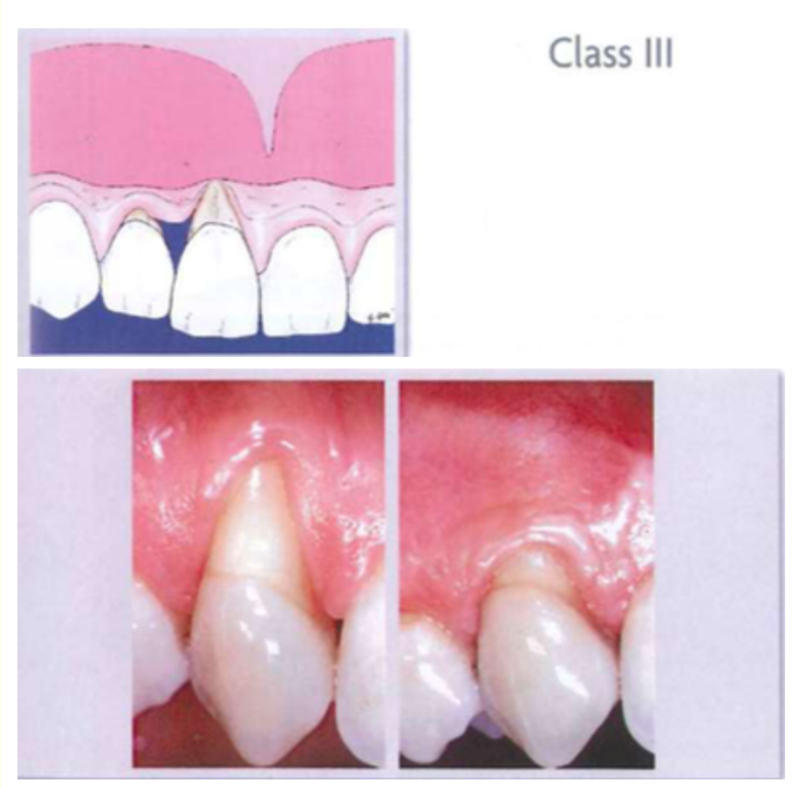
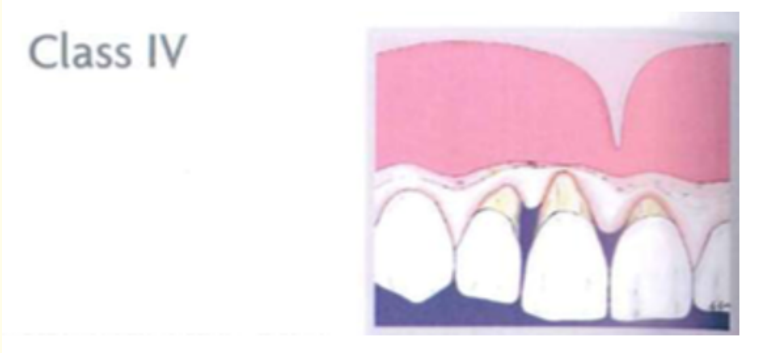


Figure 14

Only partial root coverage is possible.



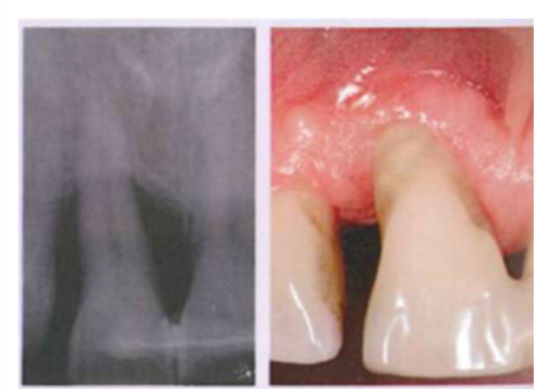


Figure 15

No root coverage is possible [9].

Millers classification could help the clinician segregate cases where root coverage is possible and where it is not possible. Borrowing the basic concepts from millers classification the line of root coverage can be predetermined [38]. In class I and class II gingival defects a number of clinical variations could be seen. The clinical conditions in which ideal anatomical structures are lacking like teeth with loss of interdental papilla height, tooth rotations or tooth extrusions it is possible to predetermine the root coverage by determining the MRC (maximum coverage of root which is achievable) [38].

The procedure to find MRC is simple. First of all we have to find the facial line angle of the tooth. Facial line angle is an imaginary line where facial surface of teeth meet the proximal surfaces. At the neck of the teeth where CEj meet this line is called as CEJ point angle (CPA). This point is easy to identify even in the tooth with cervical abrasions.

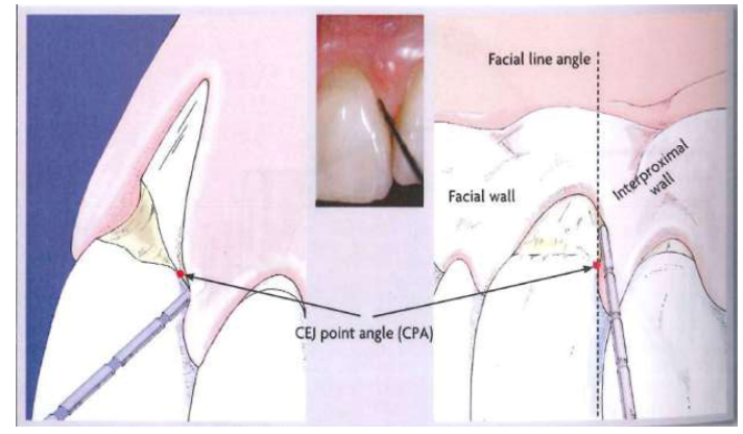


Figure 16

The next step is to find the ideal papilla height. The ideal papilla height is the vertical distance between the horizontal line joining the CPAs of two adjacent teeth and the tip of papilla donated by 'x'.

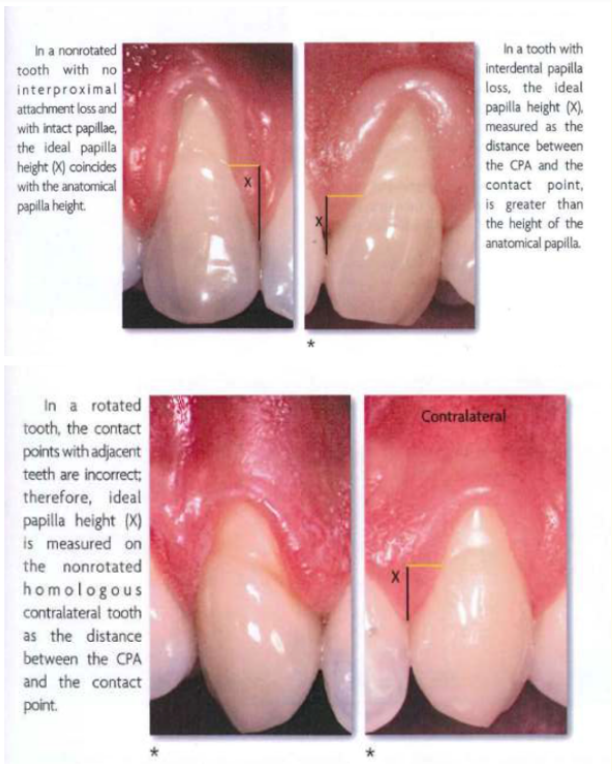


Figure 17

Once the ideal papilla has been measured, this dimension is reported apically starting from the tip of both papillae mesial and distal to the tooth with the recession defect. The projections on the recession margin of these measurements allow identification of two points that are connected by a scalloped line, the outline of which varies according to the patient's bio- types and the shape of the anatomic CEJ of other adjacent teeth. This line represents the line of root coverage or the so-called clinical CEJ [38].

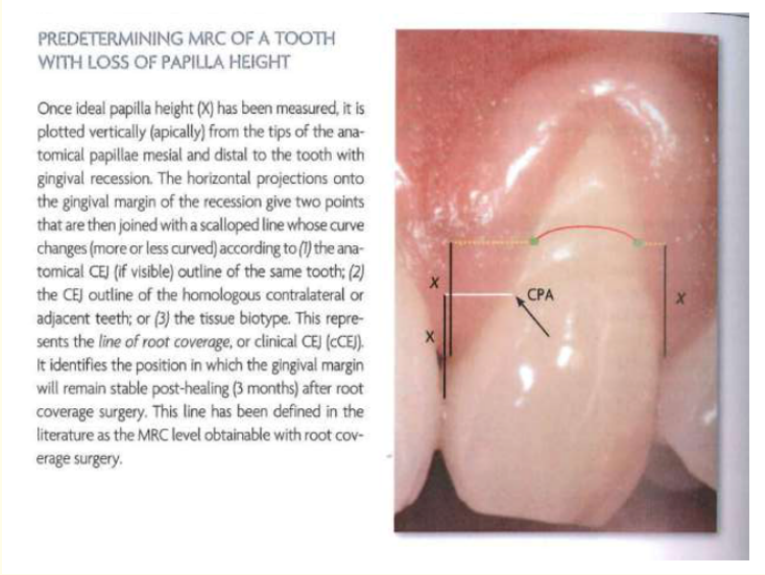


Figure 18

Hence it can be said that Extrapolating from the Miller classification, a tooth with gingival recession and with no loss of interdental attachment and bone requires a definite papilla height so that complete root coverage can be accomplished; if some papilla(e) is lost, coverage up to the CEJ cannot be achieved [36].

The surgical treatment options for recession coverage

In general, the surgical procedures can be broadly classified in pedicle flap procedures, free graft procedures and guided tissue regeneration procedures [21,39].

Pedicle flap surgical techniques offer long-term predictability and satisfying aesthetic results in anterior shallow, narrow defects. (< 5 mm) where there is adequate keratinised tissue (attached gingiva) close to the recession defect [40].

The laterally positioned flap procedure was the first pedicle graft procedure that was used for the treatment of gingival recessions [35]. It was first introduced by Grupe and Warren in 1956 and later modified by Grupe whereby sparing the cervical cuff of donor gingiva [35]. It was a full-thickness flap prepared from the adjacent site on the side of the recession and repositioned to cover the defect. This was later modified by Hattler who used a split- thickness flap repositioned in a similar way to cover multiple exposed root surfaces [41]. Pfeifer and Heller advocated the use of this split-thickness flap to minimize the potential risk for development of dehiscence at the donor's tooth [42].

The success rate reported for this technique was 69% - 72% [37] as per Zucchelli., *et al*. the adequate height and width of the keratinized tissue lateral to the recession the wide dimensions of the pedicle and the adequate tissue thickness of the flap are critical in order to

achieve predictable root coverage and good aesthetic results [43]. The procedure is relatively easy and not time-consuming, it produces excellent aesthetic results and avoids the need for a second surgical site [44]. Although the possible risk of gingival recession, dehiscence, or fenestration at the adjacent donor site has to be considered.

Coronally advanced flap (CAF)

This procedure was first presented by Bernimoulin., *et al*. and it involves the coronal repositioning of the gingival tissue that lies apical to the recession defect [45]. In cases of a thick gingival biotype and a sufficient amount of keratinised gingiva, it can be performed as a one-stage procedure [40]. In other cases, there is the need to increase the thickness and the amount of the gingiva using a free gingival graft, a connective tissue graft or a resorbable/non-resorbable membrane (guided tissue regeneration). At a second surgery is then needed after three months of healing, the coronal advancement of the tissue for recession coverage. [43]. The coronally advanced flap can be used with great reliability and predictability for the treatment of Miller Class I and II recession defects [40]. Zuchelli and de Sanctis have also proposed a modified approach for the treatment of multiple recession defects in cases with high aesthetic demands [46]. The mean root coverage achieved with a single stage coronally repositioned flap varies between 55 - 99% and complete root coverage ranges from 24 - 95% of sites [47]. According to Huang., *et al*. the height of the interdental papilla, the amount of keratinised gingiva, the presence of gingival cleft extending in the alveolar mucosa, the deep cervical wear, the frenulum attachment, and the vestibular depth-might have an impact in the outcome [43].

Subepithelial connective tissue grafts were first introduced by Langer and Calagna in 1980 and further described in detail by Langer and Langer in 1985 [42]. It was presented as an alternative that overcame the limitations of the free gingival graft. It offered excellent predictability of the results. Overall comparisons allow us to consider the subepithelial connective graft in combination with the overlying flap as the golden standard procedure in the treatment of recession-type defects [42].

Various modifications of the original technique have been proposed, including connective tissue graft with or without an epithelial collar, partially or covered by a pedicle flap, with an envelope or tunnel design preparation [48,49]. VISTA technique has recently been proposed by Dr Homa Zadeh for maxillary anterior teeth as a modification of tunnel technique [50,51].

Conclusion

The Millers classification is so far the most encompassing of the gingival recession conditions. It would be more prudent to further evolve the miller classification to cover the diverse dynamics of gingival recession conditions. The Miller classification should be evaluated and extended carefully with sound clinical trials on gingival recessions and root coverage. Further attempts to classify gingival recession would rather confuse then help the clinicians.

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