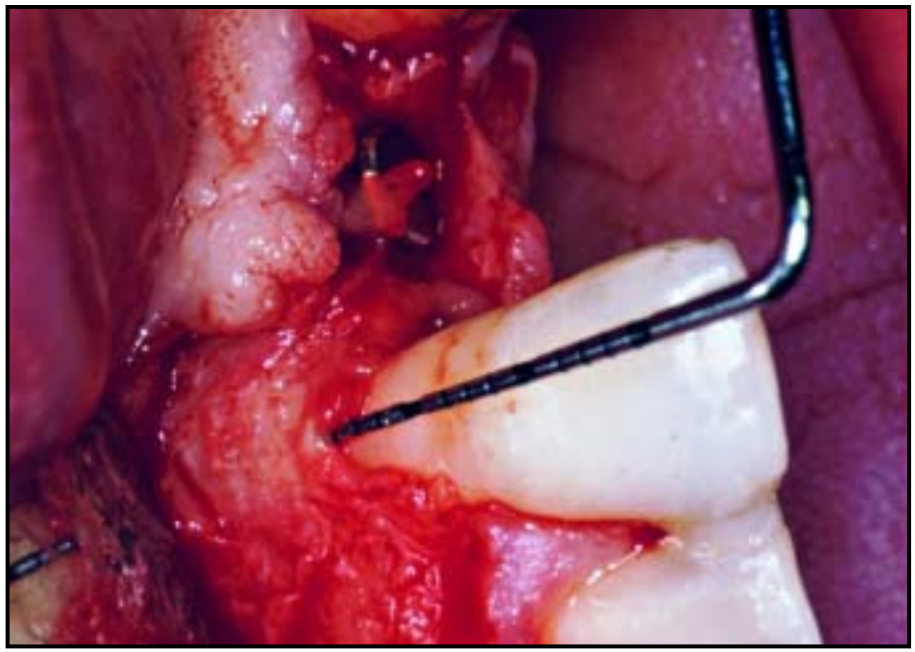

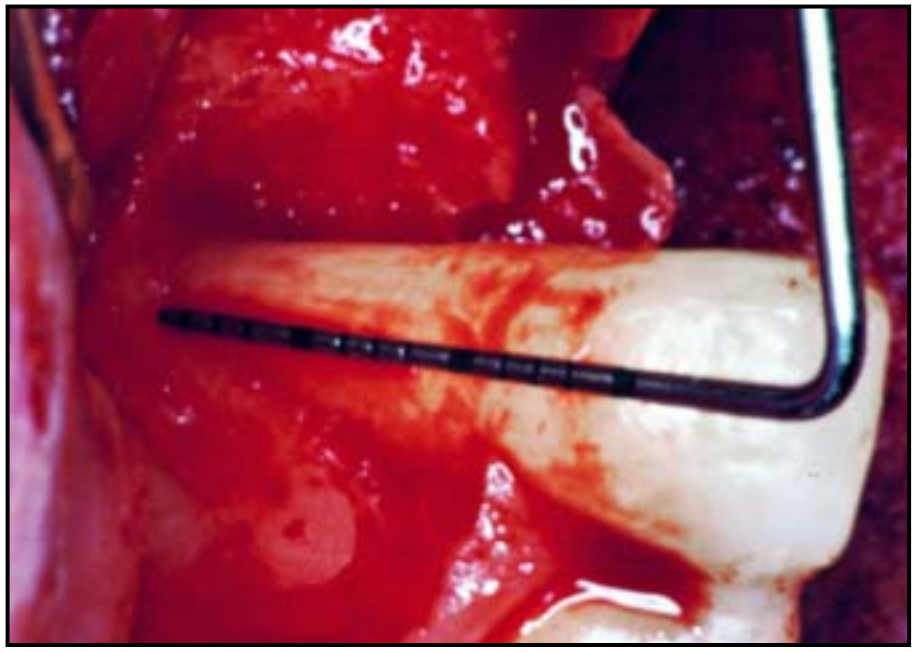

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The Use of Enamel Matrix Derivative in the Treatment of Periodontal Osseous Defects: A Clinical Decision Tree Based on Biologic Principles of Regeneration

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An investigation of the periodontal literature reveals four critical factors necessary for a regenerative response in the treatment of intrabony defects. These include root preparation to remove toxins and altered cementum, space creation by the graft or membrane barrier for migration of progenitor cells, stabilization and flap coverage of the graft, and use of a membrane barrier or enamel matrix derivative (EMD). This article reviews the literature supporting this concept and presents a clinical decision tree to determine when to use EMD alone or with autogenous grafts and membrane barriers in the treatment of defects of varying morphologies. The clinical decision tree is designed to increase the predictability of a positive clinical response. Cases are shown to demonstrate the indication for each treatment. (Int J Periodontics Restorative Dent 2001;21:437–449.)

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Since Melcher[1] first advanced the biologic principles associated with guided tissue regeneration, clinicians and researchers have sought techniques to predictably regenerate periodontal structures lost to the disease process. Successful regeneration, which can only be evidenced histologically, consists of a functionally oriented periodontal ligament (PDL) inserting into newly formed cementum and alveolar bone. A review of the periodontal literature suggests that surgical procedures using autografts, allografts, xenografts, and/or barrier membranes can contribute to a successful regenerative response.[2–5] Because of the obvious difficulties in obtaining human posttreatment histology, other measurement parameters have become the standard for evaluating a healing response. From a clinical viewpoint, measurements such as gain in probing attachment level, decrease in probing depth, fill of the osseous defect, and percentage fill of the osseous defect have been used to measure and compare the results of various "regenerative" therapies. In cases

where the clinical response is positive and histologic evidence (human block sections) shows that regeneration is possible, the term "regenerative therapy" may then be applied to that specific material or technique.

Analysis of published regenerative studies shows that four critical factors are consistent requirements. First, the root surface must be prepared so that bacterial cells and other cytotoxic products are removed.[6–9] Second, space must be created so that progenitor cells from the PDL can selectively repopulate the root surface.[10–12] Third, stability of the maturing fibrin clot, graft, implant, and/or membrane under the tissue flap is essential.[13–15] Fourth, the flap must be designed to maintain coverage of the barrier membrane and/or graft for containment of the material to prevent bacterial contamination and to minimize recession (Fig 1).[16–20]

Enamel matrix derivative (EMD) (Emdogain, Biora) was introduced into the periodontal literature in 1997 as a tissue-healing modulator to mimic events that occur during root development and to help stimulate periodontal regeneration.[21–23] The use of EMD in the treatment of intrabony defects has been shown to result in significant improvement in probing depth, clinical attachment level (CAL), and bone fill.[24–29] Histologic evidence of regeneration of new periodontal attachment has been demonstrated in human and animal model systems.[30–35] Two separate studies compared the benefits of EMD to those obtained with the use of bioabsorbable and nonabsorbable barrier membranes.[27,33] The results showed no significant difference in probing depth reduction, CAL gain, and gingival recession between the membrane- and EMD-treated sites. Furthermore, all test groups (EMD and membrane treatment) showed a superior result when compared to the open-flap debridement control. At the time of this writing, there were no data on the use of EMD in conjunction with barrier membranes and/or bone grafts and only one published study on the use of EMD in combination with a xenograft.[36]

The recommended surgical technique and postoperative care for using the EMD material have been previously described.[29,32] In our experience, and in a review of the periodontal literature of EMD-treated cases, the recommended technique has demonstrated variable clinical and histologic outcomes.[34] Clinical results are often dependent upon the dimension and morphology of the intrabony defect. Deeper intraosseous lesions result in greater gains in CAL and bone fill than shallower defects. Pontoriero et al[27] and Heden et al[26] have documented this difference in the healing response of intrabony defects treated with EMD. Previous studies with other regenerative techniques showed variable healing responses dependent upon the number of walls in an osseous defect.[37–39] Three-walled defects have a greater potential to fill than one- and two-walled defects. Others have proposed that the potential for fill of an osseous defect is directly related to the number of surrounding osseous walls, the amount of exposed root surface, and the ability to obtain flap coverage of the treated area.[40] Following surgery, it has also been shown that there is a greater gain of attachment when the angle of the defect to the long axis of the root is less than 45 degrees.[41]

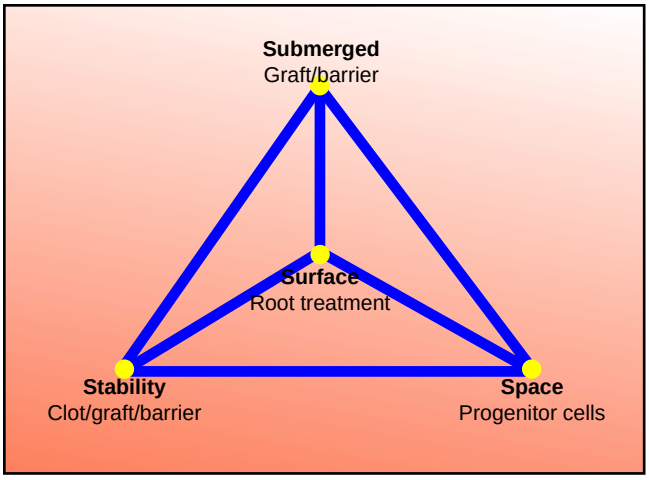

Fig 1 *"4S pyramid" of regenerative requirements.*

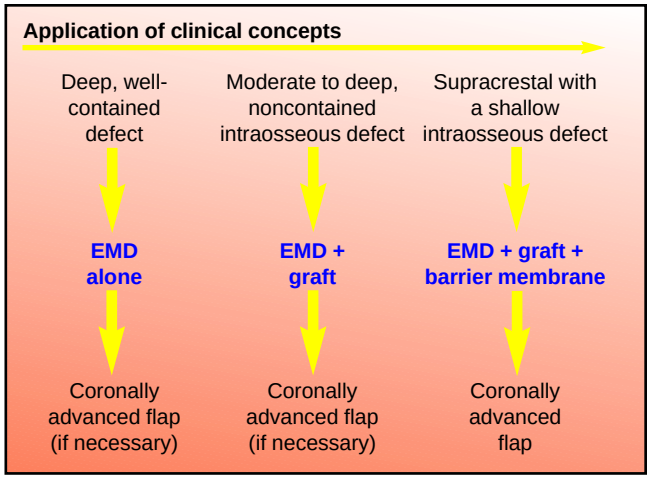

Fig 2 *Clinical decision tree for treatment of periodontal defects with EMD.*

Clinical decision tree and criteria for treatment selection

The basis for successful use of the EMD material has been the precipitation of the protein along the root surface approximating the osseous defect and the resulting stimulation of new cementum. It is therefore logical to assume that a limiting factor for success would include the ability to contain the EMD adjacent to the affected root surface. A clinical decision tree (CDT) (Fig 2) combines the biologic principles of regeneration and considerations of defect morphology in an effort to improve clinical success. Modifications of the surgical approach are presented to facilitate the use of EMD for defects of varying morphologies.

There are three branches on the EMD decision tree. Each allows the clinician to evaluate the depth and morphology of a specific defect and plan the appropriate treatment sequence. The first branch involves well-contained defects, ie, two- and three-walled intrabony defects and craters. The morphology of these defects permits the EMD to be well contained against the affected root surface.

Case 1 showed a 6-mm-deep two-walled defect on the distal aspect of the mandibular right first molar. The patient was a 24-year-old nonsmoking man with a negative medical history. The flap was reflected, and the soft tissue defect was debrided. At the time of photographing the initial osseous defect, it was noted that blood had pooled within the walls. A photograph was taken of the level at which the defect was able to contain the blood (Fig 3a). The root was planed, the defect irrigated and dried, and the smear layer removed with citric acid applied for 15 seconds. The root was then rinsed with sterile saline for 1 minute and again dried. The EMD was syringed along the root surface, starting from the base of the defect and extending coronally. At the time of flap closure, the EMD was well contained within the entire 5.5-mm intraosseous defect. This was similar to the level of the pooled blood in the initial osseous defect as seen in Fig 3a. Twelve months postsurgery, the surgical site was reentered and the residual depth of the defect was recorded. Bone had filled to a level equal to the most coronal position of the EMD at the time of closure. Fill of the defect measured 3.4 mm (approximately 62% of the original defect) (Fig 3b). Presurgical and postsurgical radiographs recorded the defect fill (Fig 3c).

The second branch of the CDT involves deep, wide intraosseous defects. The morphology of these defects would permit only limited containment of the EMD. With these types of lesions, an autogenous bone graft is advised in an effort to hold the EMD material against the

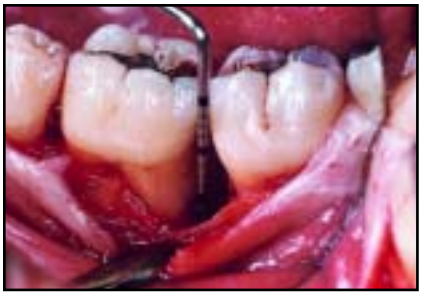

Fig 3a *Case 1. A 6-mm two-walled defect on the distal aspect of the mandibular right first molar after debridement and prior to EMD treatment. Note the level of the pooled blood.*

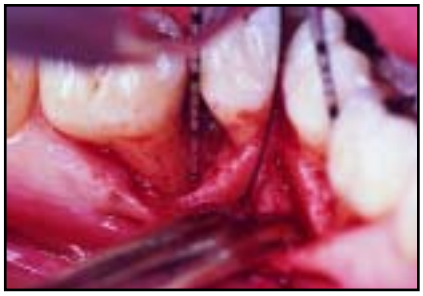

Fig 3b *Same site at 12-month reentry. The defect filled to the level that the EMD filled the defect, the same level as the pooled blood.*

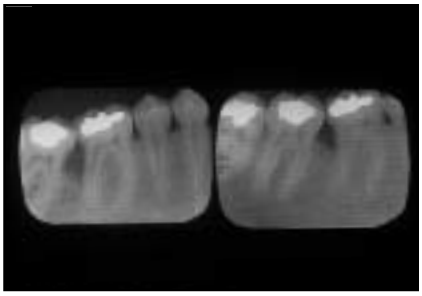

Fig 3c *Presurgical radiograph of the distal aspect of the first molar* (left) *and 1-year postsurgical radiograph prior to reentry surgery* (right).

affected root surface and help maintain space by supporting the flap. Following placement of the EMD, an autogenous graft obtained from intraoral edentulous areas (or ramus) is placed into the defect. No attempt is made to dry or condense the graft material. The surgical flaps are repositioned or positioned coronally in an attempt to achieve primary closure.

Case 2 illustrates treatment of this type of defect. The patient was a healthy, nonsmoking 46-year-old woman who presented with clinical and radiographic evidence of severe, chronic periodontitis. Initial therapy consisted of a review and reinforcement of personal plaque control procedures, root planing, and selective occlusal equilibration. Eight weeks following the initial treatment, a 10-mm defect was measured on the mesiopalatal aspect of the maxillary right canine. Eight mm of probing depth was measured on the distopalatal aspect of the maxillary right lateral incisor. Upon debridement of the osseous defects, intrabony depths were measured at 6 mm on the mesial aspect of the canine and 5 mm on the distal aspect of the lateral incisor (Fig 4a). The anatomy of the defects would not permit full containment of the EMD against the root surface. An autogenous graft was taken from the ridge and maxillary tuberosity distal to the right second molar. After the application of EMD, the graft was placed. The EMD was then reapplied over the bone graft (an EMD "sandwich") (Fig 4b). One year following the grafting procedure, the probing depths were 3 mm. A reentry procedure showed that the 5 mm of osseous fill on the mesial aspect of the canine and 4 mm on the distal aspect of the lateral incisor had eliminated the intrabony defect (Fig 4c). Presurgical and postsurgical radiographs reflected these changes (Fig 4d).

The final two cases illustrate the third branch of the decision tree and involve treatment of horizontal bone loss or shallow infrabony defects. Similar to the treatment of lesions associated with the other two branches of the decision tree, full-thickness mucoperiosteal flaps were elevated, the osseous defects debrided, and the root surfaces planed. The smear layer in one case was removed with 24% ethylenediaminetetraacetic acid (EDTA) applied for 2 minutes and in the other case citric acid pH 1 applied for 15 seconds. The roots were then irrigated and dried, and EMD was applied. The autogenous graft material was placed, and EMD was again applied over the bone graft. For these horizontal or shallow defects, the graft material is covered with a bioabsorbable membrane to further contain the material. The membrane was stabilized with bone tacks.

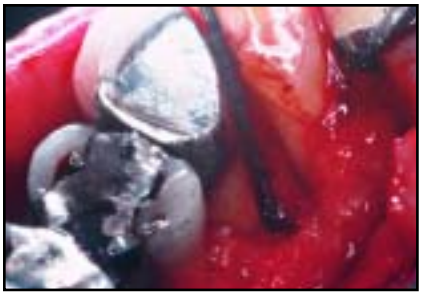

Fig 4a (left) *Case 2. The debrided osseous defects measure 6 mm on the mesiolingual aspect of the maxillary right canine and 5 mm on the distolingual aspect of the lateral incisor.*

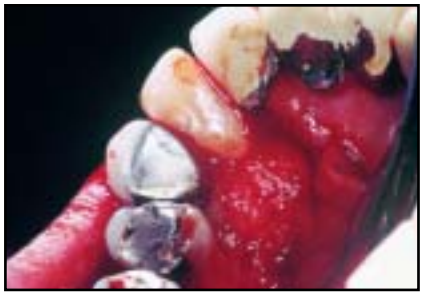

Fig 4b (right) *Defects are filled with autogenous bone and covered with EMD.*

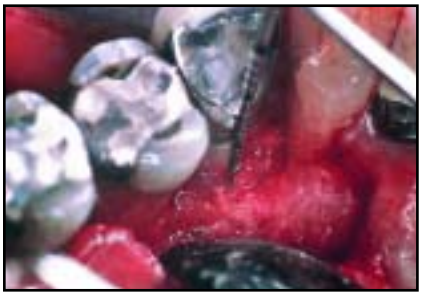

Fig 4c (left) *One-year reentry reveals a 5-mm fill on the mesiopalatal aspect of the canine and 4 mm of fill on the distopalatal aspect of the lateral incisor.*

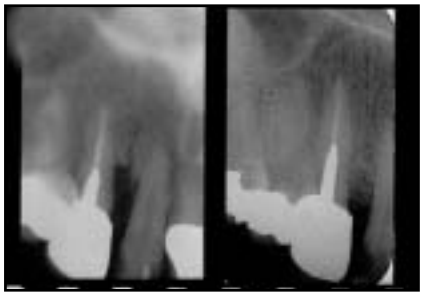

Fig 4d (right) *Presurgical radiograph of the canine and lateral incisor* (left) *and the 1-year postsurgical radiograph* (right).

Case 3 involved the maxillary right canine of a healthy, nonsmoking 65-year-old woman who had a negative medical history. The canine served as the terminal abutment for an anterior fixed partial denture, which supported a bilateral, distal extension, semiprecision, removable partial denture. The trough-like osseous defect extended from the distobuccal to the distal of the palatal aspect of the tooth, creating a shallow intrabony defect with an extensive supracrestal component (Fig 5a). Following placement of the EMD material along the root surface adjacent to the osseous defect, an autogenous graft was placed into the site. The graft was obtained with a trephine from the maxillary right edentulous ridge, milled, and carefully placed to fill the defect. EMD was then placed on the graft surface. The graft was covered with a bioabsorbable membrane (BioGide, Osteohealth) and stabilized on the buccal and palatal aspects with nonresorbable bone tacks (Ace Surgical Supply). The tissue flaps were coronally positioned to obtain primary closure over the membrane. At 1 year, the probing depth had been reduced from 11 to 4 mm. The 7-mm osseous defect had completely filled. There was 3 mm of supracrestal bone apposition (Fig 5b). Presurgical and postsurgical radiographs demonstrated the osseous changes (Fig 5c).

Case 4 involved buccal and mesial defects adjacent to the maxillary right central incisor of a healthy, nonsmoking 50-year-old man with no significant medical history. Probing depths measured 13 mm on the buccal and 14 mm on the mesial aspect of the maxillary right central incisor (Fig 6a). The osseous defect was found to be 10 mm on the buccal and 11 mm on the mesial aspects (Fig 6b). The tooth also evidenced Miller Class III mobility. Prior to undertaking the surgery, the tooth was stabilized with a composite splint joining it to the adjacent lateral incisor. The source of the autogenous intraoral graft was bone removed during an osteotomy for the placement of a dental implant in

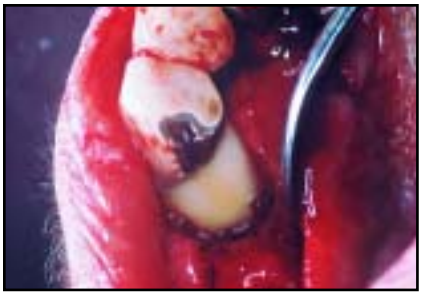

Fig 5a *Case 3. The distal osseous defect on the maxillary right canine following debridement demonstrates an intrabony and suprabony defect. The defect was filled with EMD, autogenous intraoral bone, and a bioabsorbable membrane barrier. The barrier was stabilized with bone tacks.*

Fig 5b *One-year reentry reveals the 7-mm defect filled completely and 3 mm of supracrestal bone apposition.*

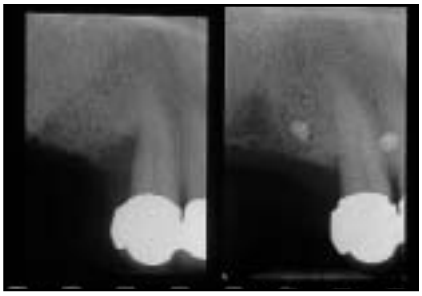

Fig 5c *Presurgical radiograph of the distal defect on the maxillary right canine* (left) *and 1-year postsurgical radiograph* (right).

the left central incisor position. Additional bone was obtained from the right tuberosity. A bioabsorbable membrane (BioGide) was placed over the bone graft and stabilized with bone tacks (Fig 6c). The flap was coronally positioned to submerge the membrane. At the 6-month postoperative evaluation, probing depth had been reduced to 3 mm. The reentry photograph showed 5 and 6 mm of supracrestal bone growth on the buccal and mesial aspects, respectively (Fig 6d).

Discussion

Each of the cases presented illustrates the application of the CDT for treatment of periodontal defects with EMD. While this decision tree is based on defect size and morphology, it also adheres to the critical requirements for attaining successful clinical results. These factors—surface treatment, space, stability, and submerged material (flap coverage)—have been documented in the literature as contributing factors to a positive healing response.

Root surface preparation is necessary to minimize the presence of bacteria and endotoxins. Contamination of the root surface of periodontally compromised teeth has been well documented in the literature. Scaling and root planing have been shown to remove the endotoxins contaminating a periodontally involved root surface.[42] The use of root-surface modifiers remains controversial. Citric acid[43] and an etching agent at neutral pH (24% EDTA)[44] have demonstrated successful root demineralization and the creation of a root surface that favors fibroblastic attachment. However, a meta-analysis of guided tissue regeneration studies showed no clinical advantage with the use of citric acid conditioning.[45] With EDTA, there is 10% less recession, 10% greater reduction of pocket depth, and more total histologic attachment when compared to citric acid and a control.[46] The use of root conditioning with EMD follows the manufacturer's recommendation. This protocol remains to be tested to determine if and when conditioning is a necessary requirement following root planing and prior to the EMD application.

A primate study[47] and a dog model[48] with Class III furcation defects demonstrated the importance of space maintenance and showed that little or no regeneration occurs if the membranes collapse into the defect sites. Articles reviewing evidence-based approaches to the successful regeneration of intrabony defects[18] and mandibular Class II furcation

Fig 6a *Case 4. Buccal probing of maxillary right central incisor reveals an 11-mm pocket depth.*

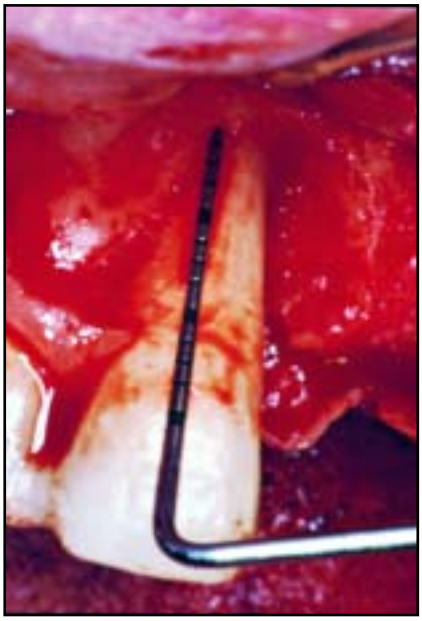

Fig 6b (right) *Exposed and debrided defect measures 10 mm on the buccal and 11 mm on the mesial aspects.*

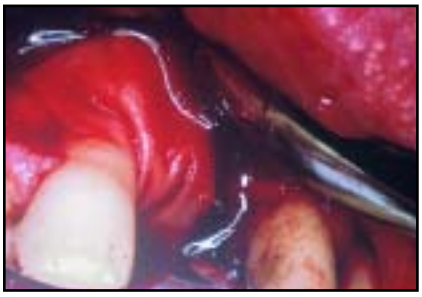

Fig 6c *Autogenous bone obtained during the osteotomy to prepare the left central incisor implant site is placed in the defect on the right central incisor after treatment with EMD and covered with a bioabsorbable membrane barrier.*

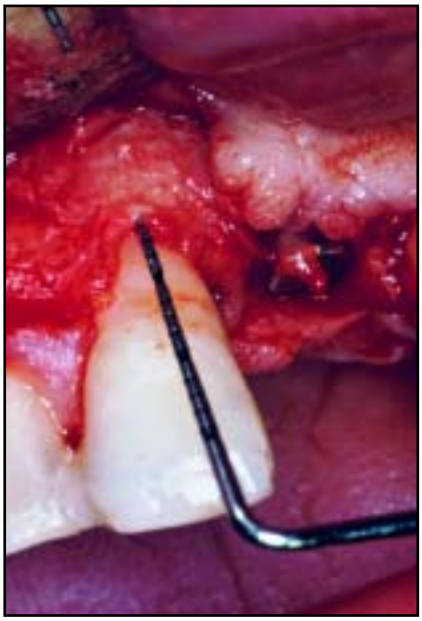

Fig 6d (right) *Six-month reentry reveals 5 and 6 mm of supracrestal fill on the buccal and mesial aspects, respectively.*

defects[49] and a study on Class III furcation repair in dogs[50] cited the importance of space maintenance. Regenerative studies[12] concluded that the greatest gains of "newly formed granulation tissue" are found in deeper defects because there is greater space that could be filled with a blood clot.

Stability of the clot and bone graft has been shown to be a basic requirement in all wound-healing studies.[51–53] In the periodontal literature, stability of the clot, membrane, and tooth have been discussed as components of a successful healing response. Wikesjö et al[14] reviewed the significant events in the early healing of a periodontal wound and stressed the importance of a stable clot and wound if regeneration is to succeed. The 1996 World Workshop in Periodontics[54–56] reiterated that mobility of a wound margin may be a potential cause of disruption of the fibrin clot and a reason for procedure failure. Dog models demonstrated that root surfaces conditioned with heparin lack wound stability, resulting in minimal connective tissue repair along the root surface.[15,48] An 8-year longitudinal study[57] found that mobile teeth do not respond as well as nonmobile teeth to periodontal treatment. A 2-year study[58] reported that "occlusal adjustment in conjunction with conventional periodontal therapy resulted in a more favorable clinical attachment level for the adjusted than the non-adjusted patients."

Lastly, flap coverage of the membrane barrier has been cited as essential for optimum healing. Murphy[59] stated that 85% of all membranes used in his study became exposed and that exposure time was approximately 2 weeks after placement. Membrane exposure has been associated with a reduction in attachment gain.[60,61] The greater the exposure of the membrane, the less the gain of clinical attachment. It has been demonstrated that tissue flaps with a thickness of 1 mm or less are associated with 3.5 times more flap recession than those with flaps thicker than 1 mm.[62] Coronal flap repositioning has been shown to increase the chances of supracrestal apposition of new bone.[63]

The CDT presented in this article is designed to increase the predictability of obtaining a positive clinical result when using EMD to treat osseous defects with varying morphologies. EMD, without the addition of bone substitutes and/or membrane barriers, has been demonstrated to be clinically successful in reducing probing depth, gaining CAL, and volumetrically increasing the defect fill in the treatment of intraosseous defects (Table 1). However, analysis of the results of clinical studies using EMD alone demonstrates the indications of this approach.

Heijl et al[24] evaluated the treatment of one- or two-walled osseous defects. They noted that the "mean intrabony component" was "close to 5 mm, with about one quarter of the defect having a 3 wall component." Apparently, approximately half of the reported fill occurred in the well-contained part of the defect. This is in accordance with the first branch of the CDT.

A study of 145 defects treated with EMD reported a CAL gain of 4.6 mm and probing depth reduction of 5.2 mm.[26] The recorded fill was 2.9 mm on average, which represented 69% of the original defect. Sixty-six percent of the treated defects were two walled, two and three walled, or three walled. Although these types of defects offer excellent containment of the EMD gel and respond well to EMD alone (as indicated by the CDT), the authors concluded that neither the type nor depth of the intrabony defect reached statistical significance when correlating them with the clinical results.

Another study of 72 consecutively treated intrabony defects of one- or two-walled morphology[28] reported a mean pocket depth reduction of 4.7 mm and a mean CAL gain of 4.2 mm at 12 months postsurgery. It also reported a mean radiographic fill of 3.1 mm and mean defect fill of 70%. Although at 1 year postsurgery only smoking and bleeding on probing significantly correlated with defect fill, 69% of the treated defects were two walled and showed a slightly greater bone gain than the one-walled defects (3.3 vs 2.6 mm).

In a 12-month clinical study,[27] osseous defects that were treated with three different membrane barriers or EMD alone were compared to flap debridement controls. The authors noted no significant difference between the membrane and EMD groups, while there was a statistically significant superiority of

Table 1 Clinical studies using EMD in the treatment of intrabony defects

Study	No. of defects	Type of defect	Time of evaluation	Probing depth reduction (mm)	CAL gain (mm)	Defect fill, in mm (% defect fill)
Heijl et al[24]	27 EMD	1-2 wall	36 mo	3.1	2.2	2.6 (66) radiographic
	27 control	1-2 wall	36 mo	2.3	1.7	0
Heden et al[26]	145 EMD	1 wall (13%); 1 + 2 wall (21%); 2 wall (60%); 2 + 3 wall (5%); 3 wall (1%)	12 mo	5.2	4.6	2.9 (69) radiographic
Pontoriero et al[27]	10 EMD	3 mm	12 mo	4.2	2.9	NA
	30 MB	3 mm	12 mo	4.5	3.1	NA
	40 OD	3 mm	12 mo	3.4	1.7	NA
Heden[28]	72 EMD	1 wall, 3 mm; 2 wall, 3 mm	12 mo	4.7	4.2	3.1 (70) radiographic
Froum et al[29]	53 EMD	1, 2, and 3 wall and combination	12 mo reentry	4.94	4.26	3.83 (74)
	31 OD	1, 2, and 3 wall and combination	12 mo reentry	2.24	2.75	1.47 (22.7)
Lekovic et al[36]	21 EMP	15 two wall; 6 three wall	6 mo reentry	Buccal 1.91, lingual 1.85	1.72 1.75	1.33 (29) 1.41 (31)
	21 EMP + BPBM	14 two wall; 7 three wall	6 mo reentry	Buccal 3.43, lingual 3.36	3.13 3.11	3.82 (81) 3.74 (80)

MB = membrane barrier; OD = open debridement; EMP = enamel matrix protein; BPBM = bovine porous bone mineral.

both of these groups compared to the flap debridement control. The authors further noted that "clinical improvements were better at sites with deep rather than at sites with shallow intrabony defects." This held true for all test and control groups and is in accordance with the indications for use of EMD alone as described by the CDT.

A meta-analysis was conducted to evaluate studies that treated intrabony defects with open-flap debridement with and without autogenous and allogeneic bone grafts, and with guided tissue regeneration with barrier membranes.[64] Regardless of the treatment modality, bone fill correlated significantly with the depth of the defect. Deeper initial lesions were associated with clinically and statistically improved results.[64] The correlation that the deeper the initial intraosseous defect, the more improved the healing result (ie, gain in CAL and defect fill) has been shown to be true with a variety of treatment techniques. Studies using citric acid root conditioning and replaced-flap surgery[65] with nonresorbable membrane barriers,[66] with bioabsorbable membrane barriers,[67] and with a combination of root conditioning, bone graft, and membrane barriers[68] have all demonstrated a positive relationship between defect fill and initial osseous defect depth. An evidence-based literature review of factors that influence clinical and histologic regeneration also concluded that defect morphology can directly affect the predictability of regeneration. The authors noted that deep and narrow defects are the most predictable in achieving a positive regenerative response.[49] Considering that almost all defects, regardless of predominant classification, are combination defects with three walls at the base, this correlation may reflect the predisposition for fill of the three-walled component of the osseous defect.[35] The fill rate reflects the defect proximity to PDL precursor cells and the osteoconductive

nature of the three surrounding osseous walls. In addition to the factors mentioned, the ability of the three-walled component of the defect to contain the EMD against the root surface should promote greater percentage of fill.

Although EMD has been shown in numerous reports to improve clinical parameters and to mediate a regenerative healing response, the mechanism by which EMD exerts its influence on cells has only recently been evaluated. An in vitro investigation of the effects of EMD on the behavior of human PDL cells (HPLF) and gingival fibroblasts (HGF) indicated that both cell types release significantly higher levels of transforming growth factor (TGF) under the influence of EMD than in control cultures. However, whereas HGF was shown to attach poorly and not spread on EMD-coated substrata, HPLF attaches and spreads within 24 hours. The authors concluded that this difference might account for the ability of EMD to promote selective cell repopulation during early stages of periodontal healing.[69] Several in vitro studies have reported the effect of EMD on osteoblast precursors and differentiated osteoblasts. One such study indicated that EMD increases proliferation of these two cell types.[70] However, while EMD stimulates activity of preexisting osteoblasts, it does not stimulate differentiation of the precursor cells. In a second study, EMD was found to stimulate release of TGF-β1 and increase the expression of a differentiated osteoblastic phenotype.[71] These results can be related to another study that showed that cultured PDL cells stimulated by EMD provoke a strong and lasting interleukin-6 expression that precedes TGF-β1 release.[72] Thus, EMD may provoke signaling molecules, which result in TGF-β1 expression 72 hours after exposure to EMD.[72] Another in vitro study demonstrated that "PDL cell wound fill rates" are increased significantly compared to those of gingival fibroblasts when EMD is added to a medium containing both cell types. The authors concluded that enhancement of periodontal regeneration may be a result of modifying PDL cell proliferation and migration.[73]

Two other in vitro studies documented the effects of EMD on osteoblastic cells. In the first, EMD was cultured with bovine PDL cells and rat bone marrow cells. In these cultures, EMD stimulated mineralized nodule formation in a dose-dependent manner. This corresponded to an increase in alkaline phosphatase activity.[74] In the second study, EMD enhanced the proliferation and differentiation of human osteoblasts.[75]

These documented effects of EMD on PDL and osteoblast cells, as well as on TGF-β1 release and alkaline phosphatase, may explain the enhanced osteogenesis and PDL regeneration seen in human periodontal defects. From a clinical standpoint, this molecular activity requires space for these cells to proliferate and mediate regeneration. If the defect is not naturally space maintaining, a bone graft should be used. This is described by the second branch of the CDT. The bone graft prevents the flap from collapsing and maintains the EMD in the defect site. This allows the previously described molecular reactions to take place. Although the cases shown here representing the second branch of the CDT used only autogenous bone as the grafting material, clinical and histologic trials are necessary to determine whether allografts and/or bone-replacement grafts (xenografts and alloplasts) in conjunction with EMD may result in similar clinical results.

A recent 6-month reentry study[36] comparing 42 bilateral defects—21 treated with EMD alone and 21 with EMD and bovine-derived anorganic bone (xenograft)—reported a significant advantage in probing depth reduction, CAL gain, and defect fill in the EMD-xenograft–treated defects. Defect fill averaged 30% in the defects treated with EMD only, versus 81% in the combination EMD-xenograft–treated sites. The majority of the defects in both groups were two walled, which would explain the results. These are consistent with the second branch of the CDT.[36]

Finally, in shallow, horizontal, or furcation defects, an additional benefit may be derived by placing a membrane barrier over the EMD–bone graft combination. This combination (third branch of the CDT) requires coronal flap positioning to cover the membrane. The efficacy of coronal flap positioning over a membrane barrier was demonstrated in a human histology study.[63] The

authors showed supracrestal apposition of new bone, ligament, and cementum. A recent study of surgically created Class II furcation defects in mongrel dogs showed no advantage to the use of EMD in conjunction with modified Widman flap surgery.[76] According to the CDT, the treatment that was used followed the first branch (EMD alone). However, the defects being treated required a combination approach, as noted in the second or third branch of the CDT. Further investigation is needed to determine the response of EMD in the treatment of furcation defects with or without the use of bone grafts and membrane barriers.

Adherence to the four critical factors discussed and the therapeutic approach described in the CDT should allow the clinician to observe improved results with greater predictability. Although the CDT approach uses the critical factors involved in the regeneration of a variety of osseous defects, controlled clinical data are still necessary to test the CDT hypothesis.

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@article{Mellonig1999EnamelMD, title={Enamel matrix derivative for periodontal reconstructive surgery: technique and clinical and histologic case report.}, author={J. Mellonig}, journal={The International journal of periodontics \& restorative dentistry}, year={1999}, volume={19 1}, pages={. 8-19 } }. J. Mellonig. Published 1999. Medicine. The International journal of periodontics & restorative dentistry. This paper describes a step-by-step technique for the application of Emdogain, a new enamel matrix derivative (EMD) graft material, for periodontal reconstructive surgery.Â The use of enamel matrix derivative in the treatment of periodontal osseous defects: a clinical decision tree based on biologic principles of regeneration. Treatment of intrabony periodontal defects with enamel matrix derivative: a literature review. J Periodontol. 2002;73:1360â€“76.Â Froum S, Lemler J, Horowitz R, Davidson B. The use of enamel matrix derivative in the treatment of periodontal osseous defects: a clinical decision tree based on biologic principles of regeneration. Int J Periodontics Restorative Dent. 2001;21:437â€“49. CAS PubMed Google Scholar. 23. Losada M, GonzÃ¡lez R, Garcia Ã€P, Santos A, Nart J. Treatment of non-contained infrabony defects with enamel matrix derivative alone or in combination with biphasic calcium phosphate bone graft: a 12-month randomized controlled clinical trial. J Periodontol. 2017;88:426â€“35. Topic Â«COMPLEX TREATMENT OF PERIODONTAL DISEASES.PHARMACOTHERAPYÂ». Topic Â«ORGANIZATION OF THE PERIODONTAL DISEASESPREVENTIONÂ». Contents. Periodontal diseases.Â The method allows to detect the initial forms of caries, secondary caries around the filling material and cracks in the enamel of the tooth. Caries diagnosis using laser devices. This device allows to detect areas of demineralization difficult for diagnosing, fissure caries, process on the proximal surfaces of the teeth and the level of necrotomy during the cavity preparation. A clinical decision tree for the treatment of periodontal intraosseous defects has been published by Froum et al.9 This tree (Figure 1) recommends that Emdogain should be used in periodontal osseous defects to promote the regeneration of the tissues in the periodontium. The addition of other materials is based on defect dimensions and the need to have additional support during the healing period. Application of Clinical Concepts.Â 9. Froum S, Lemler J, Horowitz R, Davidson B. The use of enamel matrix derivative in the treatment of periodontal osseous defects: a clinical decision tree based on biologic principles of regeneration. Int J Periodontics Restorative Dent. 2001;21(5):437-449. the treatment of periodontal osseous defects: a clinical decision tree based on biologic principles of regeneration. Int J Periodontics Restorative Dent 2001 Oct;21(5):437â€“49. [7] Hammarstrom L. Enamel matrix, cementum development and regeneration. J Clin Periodontol 1997; 24: 658â€“668. [8] Heijl L. Periodontal regeneration with enamel matrix derivative in one human experimental defect.A case report.J Clin Periodontol 1997; 24: 693â€“696. [9] Heard R, Mellonig J. Regenerative materials: an overview. Alpha Omegan 2000; 93: 51â€“58. [10] Lindhe J, Karring T, Lang N. Clinical periodontology and implan...