Received: 29 June 2018 | Revised: 6 September 2018 | Accepted: 13 October 2018

DOI: 10.1002/JPER.18-0405

JOURNAL OF Periodontology

ORIGINAL ARTICLE

Periodontal reconstructive surgery of deep intraosseous defects using an apical approach. Non-incised papillae surgical approach (NIPSA): A retrospective cohort study

Jose A. Moreno Rodríguez[1] | Antonio J. Ortiz Ruiz[2] | Raúl G. Caffesse[3]

[1]Private practice, Murcia, Spain

[2]Department of Stomatology, Faculty of Medicine, University of Murcia, Spain

[3]Visiting Professor, Postgraduate Periodontics, Complutense University of Madrid, Spain

Correspondence
Dr. Jose A. Moreno Rodríguez, C/Ctra de Granada n°46, Caravaca de la Cruz, 30400, Murcia, Spain.
Email: joseantonio171087@gmail.com

Abstract

Background: The objective of this study was to compare a minimally-invasive surgical technique (MIST) and a non-incised papilla surgical approach (NIPSA) in periodontal reconstructive surgery of deep intraosseous defects.

Methods: Data on 30 patients with a deep intraosseous defect treated with MIST (n = 15) or NIPSA (n = 15) were analyzed retrospectively. All patients met the same inclusion criteria and were treated following the same protocol, except for the surgical management of soft tissue (MIST versus NIPSA). Clinical parameters at baseline and at 1-year post-surgery, early healing at 1 week, and postoperative pain were assessed.

Results: NIPSA and MIST resulted in significant clinical attachment gain (CAG) ($P < 0.001$) and probing depth reduction (PDr) ($P < 0.001$) at 1-year post-surgery. However, NIPSA resulted in significantly lower recession of the tip of the interdental papilla compared with MIST ($P < 0.001$). Smoking negatively influenced early healing in both techniques ($P < 0.05$).

Conclusions: NIPSA and MIST both resulted in significant improvements in clinical parameters. NIPSA showed significant soft tissue preservation. NIPSA may represent a promising papillae preservation technique in the treatment of intraosseous periodontal defects.

KEYWORDS

alveolar bone loss, enamel matrix proteins, periodontitis, reconstructive surgical procedure, surgical flaps

1 | INTRODUCTION

Scientific evidence indicates that teeth treated by periodontal reconstructive surgery have a good long-term prognosis, even in the case of periodontal lesions associated with deep intraosseous lesions.[1–7] New techniques based on microsurgical approaches have been presented with the objective of maximizing tissue preservation and reducing morbidity.[8–14] Throughout the history of periodontal regeneration, the design of the flap has been contingent upon the evolution of biomaterials.[15,16] However, increasingly, studies place more importance on the flap design than on the regenerative biomaterial,[3,13] with the aim of favoring healing under optimal conditions that allow the periodontal ligament cells to access and regenerate the defect.[17,18] Based on these principles, a new technique, named the non-incised papillae surgical approach (NIPSA), has recently been developed.[19,20] NIPSA is a papillae preservation technique, where an apical approach is performed, without incisions or disinsertion of tissues at the level of the papillae or marginal tissues, as opposed to current marginal access techniques,[12,21] which locate the incision intrasulcularly at the level of the marginal tissue and in the area of the papillae, with the subsequent disinsertion of these tissues for the treatment of the periodontal defect.

The objective of this study was to compare the clinical results obtained after periodontal reconstructive surgery of deep intraosseous defects by means of two regenerative techniques using a marginal approach, MIST,[12] or an apical approach, NIPSA.[19,20]

2 | MATERIALS AND METHODS

2.1 | Study design and ethical aspects

The present study is a restrospective cohort study in which, except for soft tissue management (NIPSA or MIST), all clinical procedures were identical. For each patient treated with NIPSA, a patient treated with MIST was selected with, as similar as possible, periodontal intrabony defect configuration. A database of baseline clinical parameters and intrasurgical defect configuration measurements was created with the periodontal defects of patients treated with NIPSA and MIST from January 2015 to January 2017 at a private dental office in Murcia, Spain. The inclusion criteria were: 1) patients diagnosed with periodontitis;[22] 2) a plaque index and bleeding index of < 30%;[23] 3) periodontal lesions with pocket probing depth (PD) > 5 mm; 4) intrabony defect > 3 mm; 5) intrabony defect configuration including a 1 and/or 2-wall component, always involving the buccal wall. All patients complied fully with the study protocol until the final evaluation. Exclusion criteria were: 1) patients with systemic diseases that contraindicated treatment; 2) third molars; 3) teeth with incorrect endodontic or restorative treatment. All patients were informed of the technique to be used and gave written informed consent. All clinical procedures were performed according to the Declaration of Helsinki and Good Clinical Practice Guidelines as revised in 2013. The study protocol was approved by the Research Ethics Commission of the University of Murcia (Spain) (protocol number: 1757/2018, approval date: February 5, 2018).

2.2 | Clinical parameters

Variables were measured at baseline and at 1-year postsurgery. A calibrated masked examiner (AJOR) performed all the following clinical recordings. All measurements were made using a periodontal probe:* 1) probing depth (PD), measured from the gingival margin to the bottom of the pocket; 2) Clinical attachment level (CAL), measured from the cementoenamel junction (CEJ) to the bottom of the pocket; 3) Recession (REC), measured on the buccal aspect, from the CEJ to the gingival margin zenith; 4) Location of the tip of the papillae (TP): taking as reference the level of the mid-axis of the tooth, the distance from the CEJ at the zenith of the tooth to the tip of the papilla was measured. A positive value was recorded when the tip of the papillae was located coronally to the CEJ and a negative value otherwise; 5) Keratinized tissue width (KT), measured, on the buccal aspect, from the gingival margin to the mucogingival line; and 6) Bleeding on probing (positive or negative).

Immediately after debridement of the periodontal lesion and before applying the biomaterials for regeneration, the morphology of the defect was determined intrasurgically by recording the following parameters: 1) distance from the CEJ to the bottom of the defect, 2) intraosseous component of the defect or distance from the coronal limit of the interproximal bone crest to the bottom of the defect, and 3) 3-wall component of the intraosseous defect or distance from the coronal limit of the 3-wall defect to the bottom of the defect.

At 1-week wound closure (WC) during early healing was assessed. Three types of wound closure were recorded: complete wound closure of the incision line (WC = 2); incomplete closure with fibrin clot in the incision area (WC = 1); incomplete closure with tissue necrosis in the interproximal area and exposure of the regenerative biomaterial (WC = 0) (Figure 1).

Postoperative pain was evaluated according to the anti-inflammatory drug consumption (milligrams of ibuprofen).

2.3 | Experimental protocol

2.3.1 | Presurgical procedure

All patients included were previously treated by scaling and root planing[24] and were maintained for ≥ 1 year. Active residual pockets associated with intraosseous defects not resolved with non-surgical treatment were included in the study. Two to 3 weeks before surgery, the pockets associated with the defects to be regenerated were treated by scaling and root planing with ultrasonic micro-tips† and micro-mini curets,‡ instrumenting only the exposed root surface and the first millimeters of the periodontal pocket (marginal periodontal pocket area).[19,20] Surgery was not performed until minimal or absent marginal inflammation and a fibrous tone of the marginal tissue that allowed its correct manipulation was achieved. All patients received 2 g of amoxicillin§ and 600 mg of ibuprofen¶ 1 hour before surgery.[25]

2.3.2 | Surgical procedure

All interventions were performed by an experienced periodontal surgeon (JAMR) using magnification (×3).# The

* PCP UNC 15, Hu-Friedy, Frankfurt, Germany

† After Five Piezo Scaling, Hu-Friedy

‡ Micro Mini Five Gracey, Hu-Friedy

§ Amoxicilina Normon, Laboratorios Normon, SA, Madrid, Spain

¶ Normon Ibuprofen, Laboratorios Normon

ExamVision, Galileo HD, Akura, Madrid, Spain

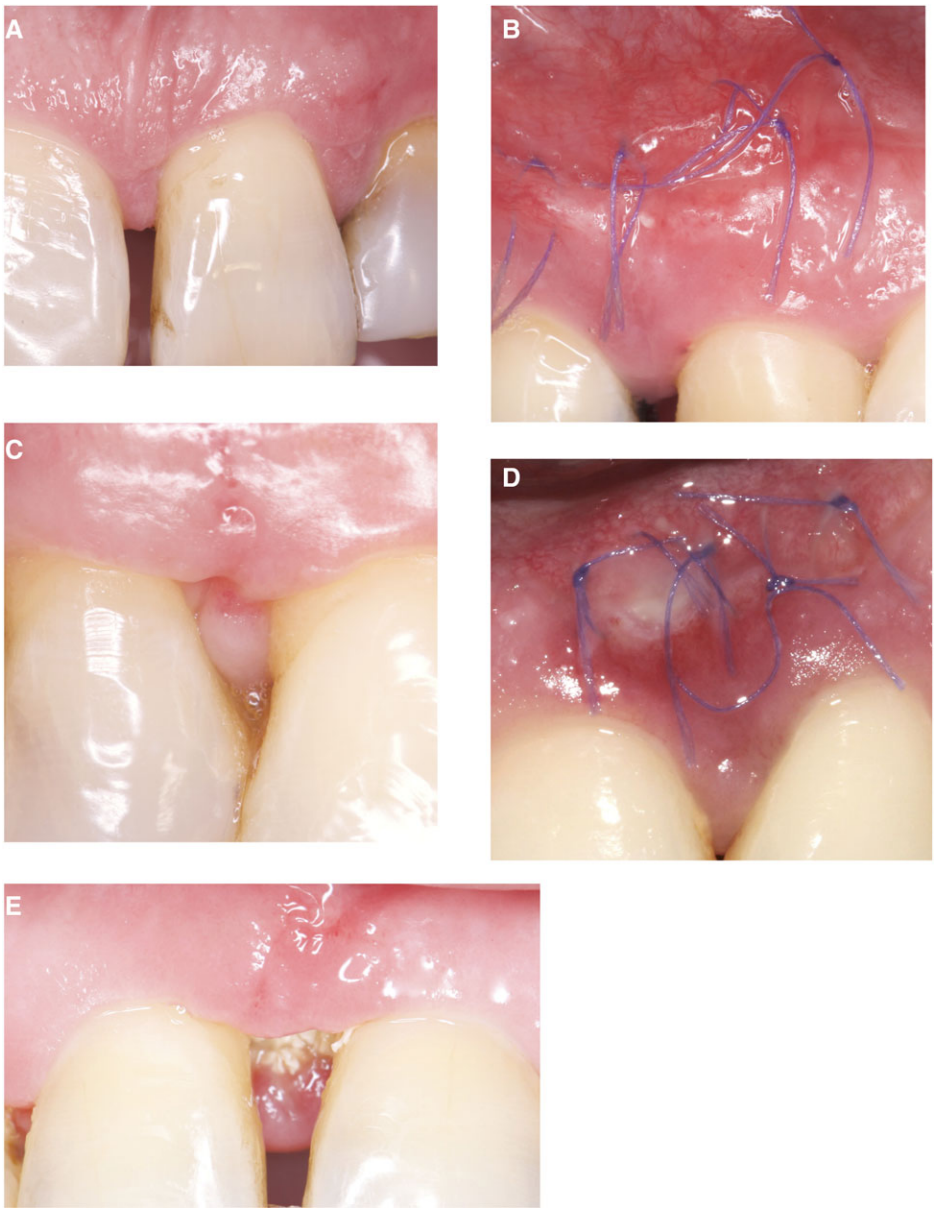

FIGURE 1 Early wound healing (WC) at 1 week. **A**) Case treated with MIST. WC = 2, complete closure; **B**) Case treated with NIPSA. WC = 2, complete closure; **C**) Case treated with MIST. WC = 1, incomplete closure; **D**) Case treated with NIPSA. WC = 1, incomplete closure with fibrin clot; **E**) Case treated with MIST. WC = 0, necrosis of papillae and exposure of biomaterial

surgical area was anesthetized with articaine/epinephrine 1:100,000.*

NIPSA group (Figures 2 and 3)[19,20]

To access the defect, a single horizontal or oblique apical incision was made in the mucosa located on the bony cortex, far from the marginal tissues and apically to the edge of the bony crest delimiting the defect. The location of the incision was determined by probing to bone, to assure its presence, (Figure 2D) and was always placed outside the esthetic zone. The incision was extended mesiodistally as necessary to allow access to the defect and correct debridement of the granulation tissue, thus creating the necessary space for stabilization of the clot. The tissue coronal to the incision was raised full thickness, trying to maintain the preoperative marginal tissue and the papillae architecture intact. The granulation tissue was disinserted from the bony walls by micro-mini curets,‡ from the base of the interproximal papillae by a scalpel micro-blade,† and the granulation tissue and epithelium of the pocket was eliminated with micro-mini curets, respecting the marginal soft tissue and residual fibers attached to cementum (Figure 3F). If the defect

* Ultracain, Laboratorios Normon

† Mamadent, Tuttlingen, Germany

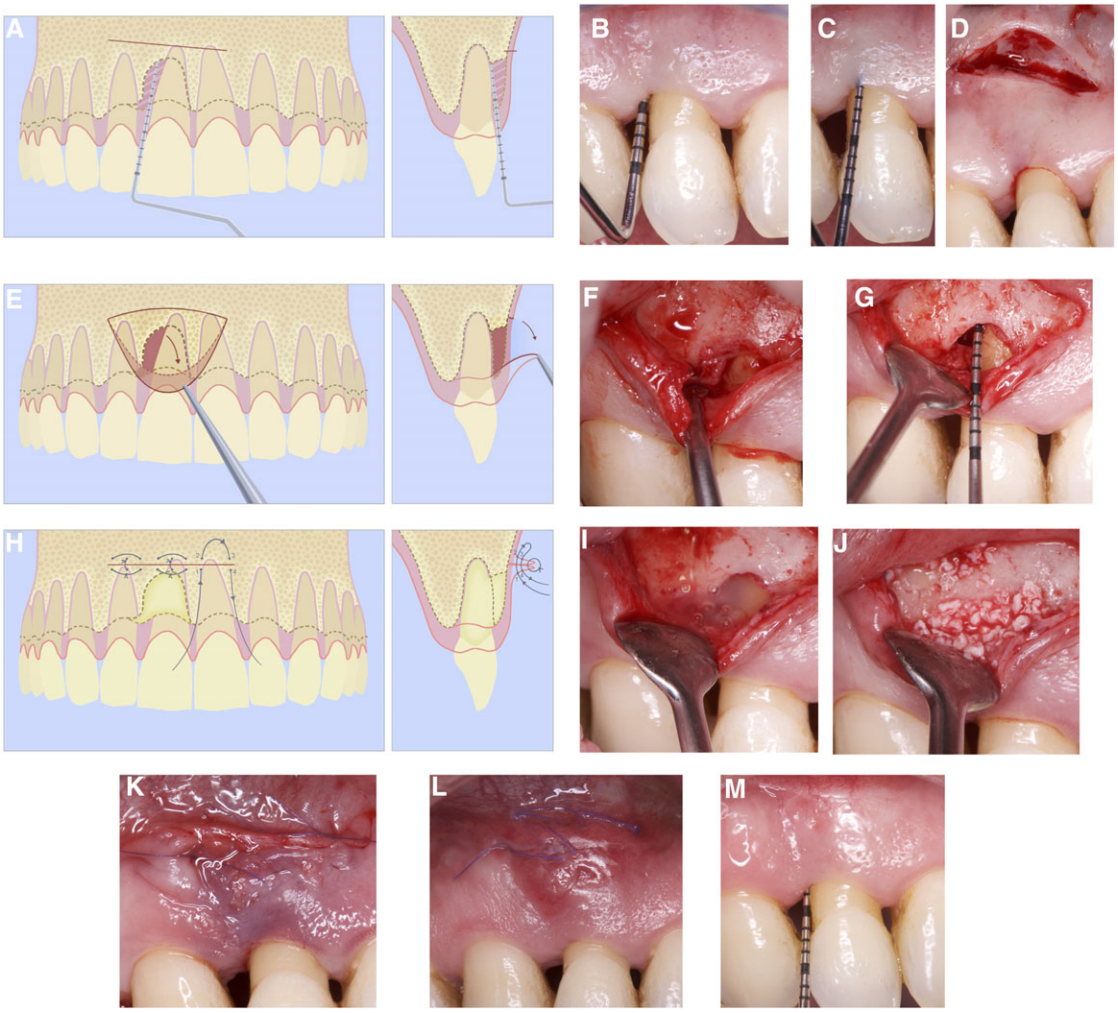

FIGURE 2 NIPSA schematic sequence. **A**) Bone probing delimiting the incision location. Frontal and sagittal views; **B**) PD before surgery and after presurgical tissue conditioning. Firm marginal tissue after non-surgical treatment; **C**) Buccal bone probing to locate the marginal bony crest. **D**) Apical mucosal incision; **E**) Flap reflected coronally exposing the defect and preserving marginal tissue attached. Schematic frontal and sagittal views; (**F**) Flap reflected coronally. Granulation tissue filling the intrabony defect. **G**) Defect after debridement: 1-wall plus 3-wall component; Bone probing showing a 5-mm 3-wall defect, and a 3-mm 1-wall. **H**) Schematic mixed HA-graft and EMD placed into the defect. Double line suturing obtaining connective tissue contact. Frontal and sagittal views; **I**) EMD application; **J**) EMD and xenograft composite application; **K**) Double suturing. Marginal tissue preservated at the end of surgery; **L**) 1 week after surgery. Complete wound closure (WC = 2); **M**) PD at 1 year

presented a lingual component, the lingual area was instrumented through the vestibular access.* The affected root (deep area of the periodontal pocket) was scaled and planed, and calculus eliminated with ultrasonic micro-tips and micro-mini curets. Once the defect was debrided, the regenerative biomaterials were applied. Then the incision line was sutured by a double suture line† to facilitate closing without tension: The first with internal horizontal mattress sutures to approximate the connective tissue of both edges of the mucosal incision, and the second with single interrupted sutures.

* Micro-papillae elevator, Mamadent

† PGA 6.0, Hu-Friedy

MIST group[12]

The defect was accessed by two papillae preservation techniques according to the anatomy of the interproximal space: simplified papillae preservation flap[26] when the width of the interproximal space was ≤ 2 mm, or modified papillae preservation technique[27] when the width was >2 mm. The interproximal incision was extended intrasulcularly in the lingual and vestibular aspect of the teeth adjacent to the defect and extended mesiodistally as necessary to allow access to the defect and for debridement. From the incision, full-thickness vestibular and lingual flaps were elevated to expose the vestibular and lingual crests delimiting the intraosseous defect. Only when access to the defect was not possible, the adjacent papillae were involved to avoid vertical releasing incisions. If a vertical releasing incision was necessary,

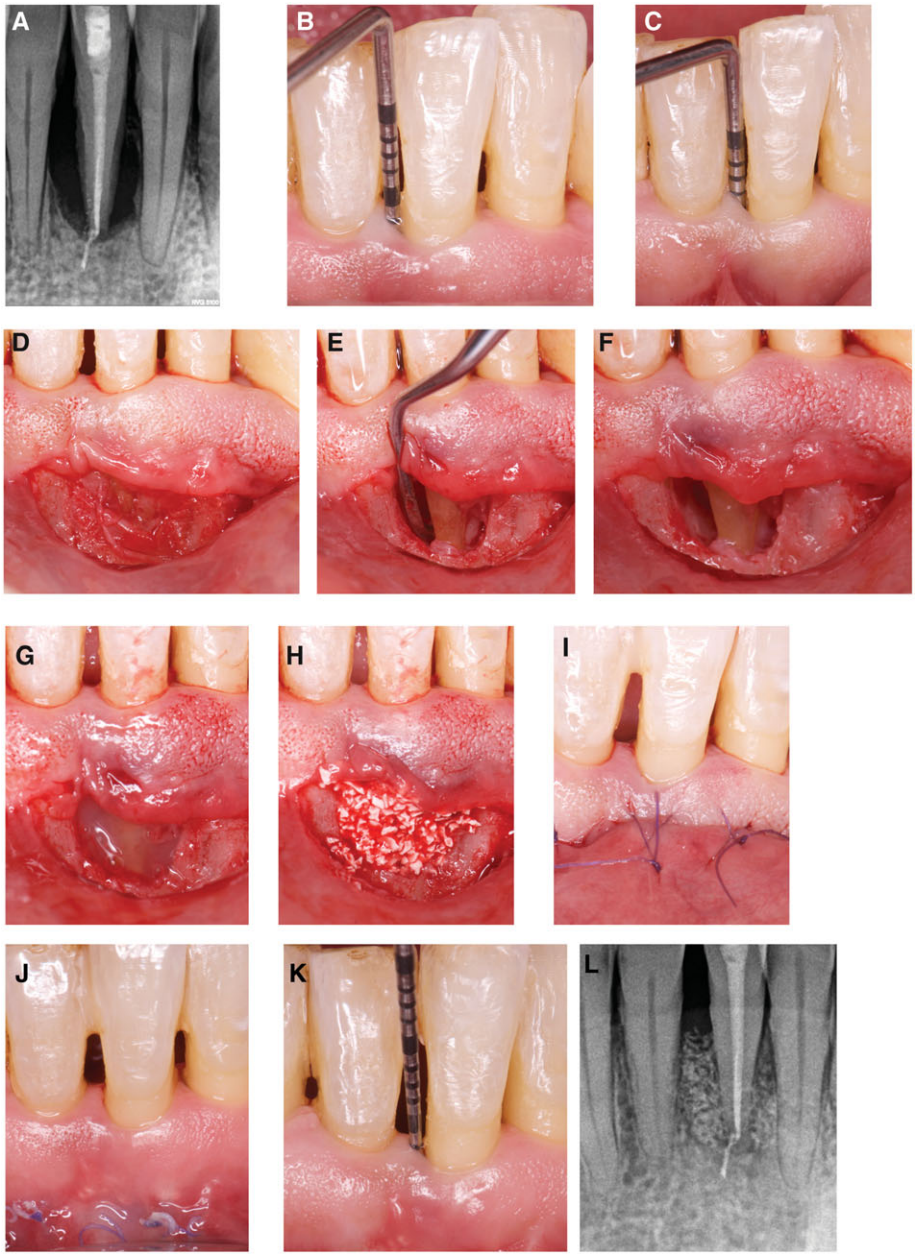

FIGURE 3 Treatment of a periodontal deep intraosseous defect according to NIPSA. **A**) Presurgery radiograph: endodontic therapy was done at the time of the non-surgical treatment; 1 year later, the periodontal lesion had not resolved; **B**) Suppuration on probing before presurgery tissue conditioning; **C**) PD after presurgery treatment and immediately before surgery. Fibrous tone of the marginal tissue and no supuration on probing; **D**) Apical incision on mucosa and apically to the edge of the bony crest. The tissue coronal to the incision was raised full thickness. Soft tissue filling the intrabony defect; **E**) Defect debridement of the lingual aspect; (**F**) View of the bony lesion after debridement. Residual tissue attached to the root surface in the apical aspect; **G**) EMD application; **H**) Xenograft plus EMD mixture application; **I**) Double line sutures: horizontal internal mattress and single sutures. Marginal tissue unaltered at the end of the surgery; **J**) Complete wound closure after 1 week; **K**) PD after 1 year; **L**) 1-year periapical radiograph

it was always minimal, did not exceed the mucogingival line and was placed outside the esthetic zone. Debridement of the defect and root instrumentation was performed in a similar way to the NIPSA group. After application of the regeneration materials, the edges of the incision were repositioned and sutured. The edges of the incision were sutured with a double suture line: internal horizontal mattress sutures at the base of the papillae and internal vertical mattress sutures in the coronal area of the papillae. Vertical incisions, if any, were sutured by single interrupted sutures.

2.3.3 | Application of biomaterial

The application of the biomaterial was identical in the two groups. After defect debridement and instrumentation of

JOURNAL OF Periodontology

the root surface, 24% ethylenediaminetetraacetic acid,* was applied to the root surface. After 2 minutes, it was irrigated with abundant saline. Enamel matrix-derived (EMD) proteins† were applied to the root surface, followed by filling of the intraosseous defect using a mixture of xenograft of bovine origin‡ and enamel matrix-derived proteins.†

2.3.4 | Post-surgical procedures

Postoperative pain and inflammation were controlled using ibuprofen.§ The dose was self-administered and recorded by the patient according to the need for pain control. Patients rinsed with 0.2% chlorhexidine, twice a day for 4 weeks, without performing mechanical hygiene measures on the operated area. The sutures were removed 1 week later. After the first 4 weeks, the patient was instructed in the mechanical cleaning of the area using an ultra-smooth toothbrush and an apico-coronal brushing technique. Control visits were made at 1, 2, 3, and 4 weeks and at 3, 6 months, and 1 year. At all visits, professional maintenance cleaning of the surgical area was performed.

2.4 | Statistical analysis

Patients contributed one defect site. Therefore, the patient was considered as the statistical unit. The sample size (n = 15 per group) was calculated a posteriori for two paired means, repeated in two groups, using CAL values, and accepting an alpha risk of 0.05, a beta risk of 0.20 (power 0.8) in a two-sided test, to recognize a difference of ≥ 1.6 units as statistically significant. A common standard deviation of 1.6 and a correlation coefficient between the baseline and final measurements of 0.552 was assumed. A dropout rate of 0% was anticipated.

In the descriptive analysis, values were expressed as mean ± SD. The Kolmogorov-Smirnov normality test and Levene test for equality of variances were used for quantitative variables. Between-group comparisons were made using the Student *t* test when there was normality and equality of variances and the Mann–Whitney test when there was not.

Values at baseline and at 1 year were compared using the paired *t* test for normally distributed values with equal variances and Wilcoxon test for non-normally distributed values and/or those with unequal variances.

Qualitative variables were compared using contingency tables and Fisher exact test or Pearson Chi-square test. A value of $P < 0.05$ was considered statistically significant. The statistical analysis was performed using a statistical package.¶

* PrefGel, Straumann, Basel, Switzerland

† Emdogain, Straumann

‡ Bio-Oss, Geistlich Pharma, Wolhusen, Switzerland

§ Normon Ibuprofen, Laboratorios Normon

¶ Systat Software, Point Richmond, CA

3 | RESULTS

3.1 | Study population and characteristics of the defects

Patient characteristics and the bone defects of each group are shown in Table 1. Thirty patients (19 men and 11 women, mean age 44.36 ± 5.9 years, range 30 to 60 years), 14 of whom were smokers (>10 cigarettes/day) were included. The two groups were homogeneous, with no significant differences according to age, sex, smoking, location or the severity and morphology of the intraosseous defect.

3.2 | Clinical parameters

Clinical characteristics at baseline and 1 year are shown in Tables 2 and 3. At baseline there were no significant between-group differences in PD, CAL, REC, TP, or KT, and there was positive bleeding on probing in all cases. At 1 year, a significant reduction in PD was observed ($P < 0.001$), without significant between-group differences, and a significant gain in CAL ($P < 0.001$), with significant between-group differences ($P < 0.05$). No significant differences were observed in PD or CAL between smokers and non-smokers in either group (Table 4). At 1 year there were significant differences between the two groups in REC, TP, or KT ($P = 0.05$, $P < 0.001$, and $P < 0.05$ respectively), while bleeding on probing was negative in both groups.

WC = 2 was present in 11 cases of NIPSA and WC = one in four cases, and there was no WC = 0. The MIST group presented WC = 2 in six cases, WC = one in four cases and WC = zero in five cases. One week after surgery, there were significant between-group differences ($P < 0.05$, Chi square test) in WC = 2 and WC = 0. However, early healing did not affect the clinical results achieved with the two techniques at 1 year ($P > 0.05$) when WC = 2 and WC < 2 in PD reduction (PDr) (NIPSA, $P = 0.53$ and MIST, $P = 0.15$, *t* test), CAG (NIPSA, $P = 0.88$ and MIST, $P = 0.21$, t test) and TP (NIPSA, $P = 0.65$ and MIST, $P = 1.00$, Mann-Whitney test) were compared. Smoking significantly worsened WC in both groups ($P = 0.008$) (Table 4).

The mean total dose of anti-inflammatories (ibuprofen), was 2,360 ± 2,059 mg in the MIST group and 2,323 ± 2,013 mg in the NIPSA group, without significant differences ($P = 0.96$, Mann–Whitney test).

4 | DISCUSSION

This study compared two different approaches to address the periodontal defects: marginal approach (MIST) versus apical approach (NIPSA). The results showed that periodontal reconstructive surgery in teeth with advanced periodontal loss and deep periodontal pockets associated with

TABLE 1 Patient-related and periodontal defect characteristics

	MIST group (n = 15)	NIPSA group (n = 15)	*P* value
Patient-related characteristics			
Sex (male/female)	9/6	10/5	1.00[a]
Age (years) (mean ± SD)	42.9 ± 4.8	45.9 ± 9.4	0.52[b]
Smoking (non-smokers/smokers)	8/7	8/7	0.71[c]
Periodontal defect characteristics			
Dental arch (maxillary/mandibular)	10/5	9/6	1.00[a]
Tooth type (incisors/canines/premolars/molars)	8/3/1/3	7/5/1/2	0.86[c]
CEJ-defect bottom (mm) (mean ± SD)	9.87 ± 2.56	10.40 ± 3.50	0.89[b]
Intraosseous component (mm) (mean ± SD)	5.27 ± 2.02	5.13 ± 2.42	0.75[b]
3-wall component (mm) (mean ± SD)	3.00 ± 2.59	2.53 ± 1.55	0.55[d]
Defect configuration distribution			
1/3-wall	4	7	
2/3-wall	8	5	
1/2-wall	0	1	
1-wall	3	2	

[a]Fisher Exact test.
[b]Mann–Whitney test.
[c]Chi square test.
[d]*t* test.

deep intraosseous defects was achievable with both techniques, with a reduction in the periodontal pocket and a significant gain in clinical attachment at 1 year. Other studies have shown that teeth affected by advanced periodontal disease are susceptible to successful regenerative treatment as long as there is correct diagnosis and treatment, adequate maintenance, and the collaboration and motivation of the patient.[1–4]

The results obtained for MIST were similar to previous studies.[12,26,27] For NIPSA there is only one preliminary study,[20] which showed results similar to the present ones. Although techniques using an apical approach have been widely developed and used in mucogingival surgery with good results,[28–30] only a few preliminary studies describe the apical approach for periodontal reconstructive surgery.[20,31]

Comparison of the two techniques showed no significant differences in PDr, but significant changes in CAG ($P < 0.05$), with more favorable results for the NIPSA group at 1 year. These results are due to a different response of the soft tissues (REC, TP, KT) according to the approach used. The NIPSA design seems to minimize surgical trauma in the marginal tissues, with REC increasing by only 0.2 ± 0.41 mm, while with MIST the increase was 0.73 ± 0.88 mm. With respect to TP, in the MIST group there was a papillae recession of 1.06 ± 0.96 mm, compared with no recession in the NIPSA group ($P < 0.001$). With NIPSA, the incision is moved away from the gingival margin and the area of the papillae, without incising or detaching these tissues, so the incision is far from the periodontal defect to be treated, accessing the defect from the apical aspect and maintaining a firm soft tissue roof which acts as a "dome" protecting the underlying interproximal defect and thus avoiding collapse of the papillae and recession of the marginal soft tissue. However, as shown in other studies on marginal access to the defect that obtained similar results to those reported here, the marginal location of the incision and the detachment of the papillae and marginal tissues seem to significantly increase postoperative recession of soft tissues.[32,33]

With respect to WC, complete closure of the incision line (WC = 2) was achieved more frequently (73%), and significantly ($P < 0.05$) with NIPSA than with MIST (40%), where a result similar to other studies was obtained.[11,34] In addition, with NIPSA, there was no case of interproximal tissue necrosis, compared with 33% with MIST. The location of the incision intrasulcular and in the area of the papillae may condition the mechanical stability of the marginal tissues that cover the periodontal defect, compromising stable clot adhesion to the root surface.[35,36] Furthermore, incising, raising, and suturing the marginal tissues may act as a complicating factor in areas of terminal blood supply.[37] Therefore, the prognosis of regeneration may be affected, in addition to compromising early healing[34] and increasing the risk of contamination of the area to be regenerated.[38]

We also analyzed the influence of smoking on clinical outcomes and found no significant differences between smokers and non-smokers in terms of PDr and CAG, as did in another study of intraosseous defects[39] but unlike others.[40,41] However, the WC results were significantly worse in smokers ($P < 0.05$), as reported by Trombelli et al. (2018)[39] and unlike the results found by Farina et al. (2013), who analyzed WC after a marginal approach to the defect.[34] They found,

JOURNAL OF Periodontology

TABLE 2 Clinical parameters (mm)

	Baseline	1 year	1-year change	*P* value
PD			**PDr**	
NIPSA	8.27 ± 2.22	2.73 ± 0.80	5.53 ± 2.56	<0.001[c]
MIST	7.73 ± 1.28	3.4 ± 0.98	4.33 ± 1.45	<0.001[c]
P value	0.69[a]		0.17[a]	
CAL			**CAG**	
NIPSA	9.07 ± 3.17	3.73 ± 1.22	5.33 ± 2.47	<0.001[c]
MIST	8.73 ± 1.94	5.13 ± 1.46	3.6 ± 1.40	<0.001[b]
P value	0.86[a]		0.03[a]	
REC				
NIPSA	0.80 ± 1.20	1.00 ± 1.36	−0.20 ± 0.41	0.71[c]
MIST	1.00 ± 1.60	1.73 ± 1.75	− 0.73 ± 0.88	0.13[c]
P value	0.92[a]		0.05[a]	
TP				
NIPSA	2.40 ± 0.73	2.47 ± 0.74	−0.07 ± 0.26	0.78[c]
MIST	1.87 ± 2.17	0.80 ± 2.00	1.06 ± 0.96	0.08[c]
P value	0.93[a]		<0.001[a]	
KT				
NIPSA	3.60 ± 1.59	3.47 ± 1.51	0.13 ± 0.35	0.83[c]
MIST	4.53 ± 0.91	3.87 ± 1.06	0.67 ± 0.72	0.09[c]
P value	0.1[a]		0.02[a]	

PD, probing depth; PDr, probing depth reduction; CAL, Clinical attachment level; CAG, clinical attachment gain; REC, recession; TP, Tip of papillae; KT, keratinized tissue.

Negative value in REC indicates increased recession. Positive value in TP indicates papillae apical displacement.

[a]Mann–Whitney test.

[b]Paired *t*-test.

[c]Wilcoxon test.

as we did, that the type of WC did not have a significant impact ($P > 0.05$) on the clinical outcome at 1 year. However, scientific literature shows that favoring optimum healing conditions, such as maintaining complete closure during healing, seems to be an absolute requirement to achieve periodontal regeneration with restoration of the periodontal ligament, cementum and alveolar bone, thus avoiding exposure of immature neoformed tissue, interruptions in tissue maturation, and healing by a long junctional epithelium.[17,18,35,42]

In the present study, the marginal area of the periodontal pocket was conditioned 2 to 3 weeks before surgery, with the aim of reducing inflammation and improving the tone of marginal soft tissues. Histological studies show that, after 14 to 21 days of healing, the connective tissue presents mature collagen fibers with a similar appearance to healthy tissue.[43,44] In the presurgical treatment, the marginal part of the pocket is treated without invading the deep areas, to minimize shrinkage of the tissues and detachment of residual fibers inserted on the root surface in deeper areas of the pocket.[45] Subsequently, with surgical treatment through an apical access, the intrabony periodontal defect is treated and space for the establishment of the clot is created, maintaining a watertight and stable area, sealed by a previously-conditioned firm soft tissue roof that favors optimal conditions for the stability of the clot adhesion to the root surface and to maintain wound stability during the maturation process.[18,35,42] NIPSA is indicated when vestibular access to the periodontal defect is possible and therefore, it must be a situation where part or all of the vestibular bony wall of the defect is absent. This clinical situation is the most frequent in intraosseous periodontal defects.[3,46,47] NIPSA may be considered a blind and sensitive technique for the defects involving a palatal/lingual site. In these clinical situations a computed tomography (CT) scan may offer a more complete assessment of the defect morphology.[19] Clear mapping of the defect by bone probing is required to place the horizontal incision on the cortical bone. Furthermore, a CT scan may be required for this purpose.[19] The apical incision is made as apically as necessary to preserve the maximum collateral blood supply to the supra-incision soft tissue, but not so apical to hinder the access to the periodontal lesion and requires a longer horizontal extension that may damage the apical blood

TABLE 3 Frequency distribution for probing depth reduction, gain of clinical attachment level and residual probing depths in both groups

	MIST			NIPSA		
mm	**PDr**	**CAG**	**rPD**	**PDr**	**CAG**	**rPD**
2	1 (6.7%)	4 (26.7%)	3 (20.0%)	1 (6.7%)	1 (6.7%)	7 (46.7%)
3	4 (26.7%)	4 (26.7%)	5 (33.3%)	1 (6.7%)	1 (6.7%)	5 (33.3%)
4	3 (20.0%)	3 (20.0%)	5 (33.3%)	4 (26.7%)	5 (33.3%)	3 (20.0%)
5	5 (33.3%)	2 (13.3%)	2 (13.3%)	4 (26.7%)	3 (20.0%)	–
6	–	2 (13.3%)		1 (6.7%)	2 (13.3%)	–
7	2 (13.3%)			1 (6.7%)	–	–
8	–			1 (6.7%)	2 (13.3%)	–
9	–			1 (6.7%)	–	–
12	–			1 (6.7%)	1 (6.7%)	–
TOTAL	15	15	15	15	15	15

PDr, probing depth reduction; CAG, clinical attachment gain; rPD, residual probing depth.

TABLE 4 Change in clinical parameters at 1 year and early wound healing in non-smokers and smokers

	NIPSA			MIST		
	NS (n = 8)	S (n = 7)	*P* value	NS (n = 8)	S (n = 7)	*P* value
PDr	5.37 ± 2.82	5.71 ± 2.43	0.54[b]	4.37 ± 1.85	4.29 ± 0.95	0.91[c]
CAG	5.37 ± 2.82	5.29 ± 2.21	0.95[c]	3.75 ± 1.58	3.43 ± 1.27	0.67[c]
WC = 2	8	3	0.008[a]	5	1	0.008[a]
WC < 2	0	4	0.008[a]	3	6	0.008[a]
WC = 1	0	4		2	2	
WC = 0	0	0		1	4	

S, smoker; NS, non-smoker; WC, wound closure; PDr, Probing depth reduction.
[a]Chi square test.
[b]Mann–Whitney test.
[c]*t* test.

supply.[37] In addition, depending on the location of the defect an oblique, instead of a horizontal incision may favor the disto-lateral blood supply support.[37]

EMD is a widely documented approach whose objective is to biomodify and improve healing in periodontal regeneration.[48] In 1- to 2-wall defects, the capacity to contain the clot and the regenerative material against the collapse of the soft tissue is diminished.[49] In this type of intraosseous non-contained defects, EMD may not be sufficient to prevent flap collapse and maintain space for periodontal regeneration.[49] In this type of defects, studies show better results when applying EMD together with a xenograft that acts as a vehicle to improve the physical properties of EMD.[50]

NIPSA seems to favor healing through complete closure, maintenance of space for regeneration and the stability of marginal tissues, primordial conditions for the success of periodontal regeneration,[17,18] in addition to minimizing postoperative soft tissue contraction.

5 | CONCLUSIONS

The results of this study show that NIPSA and MIST both provide good clinical results. However, NIPSA resulted in improvements in soft tissue preservation.

ACKNOWLEDGMENT

The authors report no conflicts of interest related to this study.

REFERENCES

1. Sculean A, Kiss A, Miliauskaite A, Schwarz F, Arweiler NB, Hannig M. Ten-year results following treatment of intra-bony defects with enamel matrix proteins and guided tissue regeneration. *J Clin Periodontol*. 2008;35:817–824.
2. Pretzl B, Kim TS, Steinbrenner H, Dörfer C, Himmer K, Eickholz P. Guided tissue regeneration with bioabsorbable barriers III 10-year results in infrabony defects. *J Clin Periodontol*. 2009;36:349–356.
3. Cortellini P, Tonetti MS. Clinical and radiographic outcomes of the modified minimally invasive surgical technique with and without regenerative materials: a randomized-controlled trial in intrabony defects. *J Clin Periodontol*. 2011;38:365–373.
4. Pini Prato G, Cortellini P. Thirty-year stability after regeneration of a deep intrabony defect: a case report. *J Clin Periodontol*. 2016;43:857–862.
5. Cortellini P, Stalpers G, Mollo A, Tonetti MS. Periodontal regeneration versus extraction and prosthetic replacement of teeth severely compromised by attachment loss to the apex: 5-year results of an ongoing randomized clinical trial. *J Clin Periodontol*. 2011;38:915–924.
6. Cortellini P, Buti J, Pini Prato G, Tonetti MS. Periodontal regeneration compared with access flap surgery in human intra-bony defects 20-year follow-up of a randomized clinical trial: tooth retention, periodontitis recurrence and costs. *J Clin Periodontol*. 2017;44:58–66.
7. Schwendicke F, Stolpe M, Plaumann A, Graetz C. Cost-effectiveness of regular versus irregular supportive periodontal therapy or tooth removal. *J Clin Periodontal*. 2016;43:940–947.
8. Cortellini P, Tonetti MS. Improved wound stability with a modified minimally invasive surgical technique in the regenerative treatment of isolated interdental intrabony defects. *J Clin Periodontol*. 2009;36:157–163.
9. Harrel SK. A minimally invasive surgical approach for periodontal regeneration: surgical technique and observations. *J Periodontol*. 1999;70:1547–1557.
10. Harrel SK, Wilson TG, Nunn ME. Prospective assessment of the use of enamel matrix proteins with minimally invasive surgery. *J Periodontol*. 2005;76:380–384.
11. Wachtel H, Schenk G, Böhm S, Weng D, Zuhr O, Hürzeler MB. Microsurgical access flap and enamel matrix derivate for the treatment of periodontal intrabony defects: a controlled clinical study. *J Clin Periodontol*. 2003;30:496–504.
12. Cortellini P, Tonetti MS. A minimally invasive surgical technique (MIST) with enamel matrix derivate in the regenerative treatment of intrabony defects: a novel approach to limit morbidity. *J Clin Periodontol*. 2007;34:87–93.
13. Trombelli L, Simonelli A, Pramstraller MW, Farina R. Single flap approach with and without guided tissue regeneration and a

hydroxyapatite biomaterial in the management of intraosseous periodontal defects. *J Periodontol*. 2010;81:1256–1263.

14. Aslan S, Buduneli N, Cortellini P. Entire papilla preservation technique: a novel surgical approach for regenerative treatment of deep and wide intrabony defects. *Int J Periodontics Restorative Dent*. 2017;37:227–233.
15. Trombelli L, Heitz-Mayfield LJ, Needleman I, Moles D, Scabbia A. A systematic review of graft materials and biological agents for periodontal intraosseous defects. *J Clin Periodontol*. 2002;29:117–135.
16. Needleman IG, Worthington HV, Giedrys-Leeper E, Tucker RJ. Guided tissue regeneration for periodontal infra-bony defects. *Cochrane Database Syst Rev*. 2006:CD001724.
17. Polimeni G, Xiropaidis AV, Wikesjö UME. Biology and principles of periodontal wound healing/regeneration. *Periodontol 2000*. 2006;41:30–47.
18. Susin C, Fiorini T, Lee J, De Stegano JA, Dickinson DP, Wikesjö UM. Wound healing following surgical and regenerative periodontal therapy. *Periodontol 2000*. 2015;68:83–98.
19. Moreno Rodríguez JA, Caffesse RG. A new papilla preservation technique for periodontal regeneration of severely compromised teeth. *Clin Adv Periodontics*. 2018;8:33–38.
20. Moreno Rodríguez JA, Caffesse RG. Nonincised papillae surgical approach (NIPSA) in periodontal regeneration. Preliminary results of a case series. *Int J Periodontics Restorative Dent*. 2018;38(Suppl):s105–s111.
21. Trombelli L, Farina R, Franceschetti G, Calura G. Single-flap approach with buccal access in periodontal reconstructive procedures. *J Periodontol*. 2009;80:353–360.
22. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: framework and proposal of a new classification and case definition. *J Periodontol*. 2018;89(Suppl 1):S159–S172.
23. Cortellini P, Tonetti M. Clinical concepts for regenerative therapy in intrabony defects. *Periodontol 2000*. 2014;65:1–27.
24. Ribeiro FV, Casarin RCV, Palma MG, Júnior FN, Sallum EA, Casati MZ. Clinical and patient-centered outcomes after minimally invasive non-surgical or surgical approaches for the treatment of intrabony defects: a randomized clinical trial. *J Periodontol*. 2011;82:1256–1266.
25. Jepsen K, Jepsen S. Antibiotics/antimicrobials: systemic and local administration in the therapy of mild to moderately advanced periodontitis. *Periodontol 2000*. 2016;71:82–112.
26. Cortellini P, Pini Prato G, Tonetti M. The simplified papilla preservation flap. A novel surgical approach for the management of soft tissues in regenerative procedures. *Int J Periodontics Restorative Dent*. 1999;19:589–599.
27. Cortellini P, Pini Prato G, Tonetti M. The modified papilla preservation technique. A new surgical approach for interproximal regenerative procedures. *J Periodontol*. 1995;66:261–266.
28. Azzi R, Etienne D, Sauvan JL, Miller PD. Root coverage and papilla reconstruction in Class IV recession: a case report. *Int J Periodontics Restorative Dent*. 1999;19:449–455.
29. Nemcovsky CE. Interproximal papilla augmentation procedure: a novel surgical approach and clinical evaluation of 10 consecutive procedures. *Int J Periodontics Restorative Dent*. 2001;21:553–559.
30. Zadeh HH. Minimally invasive technique of maxillary anterior gingival recession defects by vestibular tunnel access and platelet-derived growth factor BB. *Int J Periodontics Restorative Dent*. 2011;31:653–660.
31. Najafi B, Kheirieh P, Torabi A, Cappetta EG. Periodontal regeneration treatment of intrabony defects in the esthetic zone using modified vestibular incision subperiosteal tunnel access (M-VISTA). *Int J Periodontics Restorative Dent*. 2018;38(Suppl):e9–e16.
32. Schincaglia GP, Hebert E, Farina R, Simonelli A, Trombelli L. Single versus double flap approach in periodontal regenerative treatment. *J Clin Periodontol*. 2015;42:557–566.
33. Trombelli L, Simonelli A, Minenna L, Rasperini G, Farina R. Effect of a connective tissue graft in combination with a single flap approach in the regenerative treatment of intraosseous defects. *J Periodontol*. 2017;88:348–356.
34. Farina R, Simonelli A, Rizzi A. Early postoperative healing following buccal single flap approach to access intraosseous periodontal defects. *Clin Oral Invest*. 2013;17:1573–1583.
35. Wikesjö UM, Claffey N, Egelberg J. Periodontal repair in dogs.Effect of heparin treatment of the root surface. *J Clin Periodontol*. 1991;18:60–64.
36. Burkhardt R, Ruiz Magaz V, Hämmerle CHF, Lang NP. Interposition of a connective tissue graft of a collagen matrix to enhance wound stability—an experimental study in dogs. *J Clin Periodontol*. 2016;43:366–373.
37. McLean TN, Smith BA, Morrison EC, Nasjleti CE, Caffesse RG. Vascular changes following mucoperiosteal flap surgery: a fluorescein angiography study in dogs. *J Periodontol*. 1995;66:205–210.
38. De Sanctis M, Zucchelli G, Clauser C. Bacterial colonization of barrier material and periodontal regeneration. *J Clin Periodontol*. 1996;23:1039–1046.
39. Trombelli L, Farina R, Minenna L, Toselli L, Simonelli A. Regenerative periodontal treatment with the single flap approach in smokers and nonsmokers. *Int J Periodontics Restorative Dent*. 2018;38:59–67.
40. Tonetti MS, Pini-Prato G, Cortellini P. Effect of cigarette smoking on periodontal healing following GTR in infrabony defects. A preliminary retrospective study. *J Clin Periodontol*. 1995;22:229–234.
41. Stavropoulos A, Mardas N, Herrero F, Karring T. Smoking affects the outcome of guided tissue regeneration with bioresorbable membranes: a retrospective analysis of intrabony defects. *J Clin Periodontol*. 2004;31:945–950.
42. Polimeni G, Albandar JM, Wikesjö UME. Prognostic factors for alveolar regeneration: effect of space provision. *J Clin Periodontol*. 2005;32:951–954.
43. Selvig KA, Bogle G, Claffey NM. Collagen linkage in periodontal connective tissue reattachment. An ultrastructural study in Beagle dogs. *J Periodontol*. 1988;59:758–768.
44. Sculean A, Gruber R, Bosshardt DD. Soft tissue wound healing around teeth and dental implants. *J Clin Periodontol*. 2014;41:6–22.
45. Saglie R, Johansen R, Flotra L. The zone of completely and partially destructed periodontal fibres in pathological pockets. *J Clin Periodontol*. 1975;2:198–202.

46. Tal H. The prevalence and distribution of intrabony defects in dry mandibles. *J Periodontol*. 1984;55:149–154.

47. Vrotsos JA, Parashis AO, Theofanatos GD, Smulow JB. Prevalence and distribution of bone defects in moderate and advanced adult periodontitis. *J Clin Periodontol*. 1999;26: 44–48.

48. Miron RJ, Sculean A, Cochran DL, et al. Twenty years of enamel matrix derivative: the past, the present and the future. *J Clin Periodontol*. 2016;43:668–683.

49. Siciliano VI, Andreuccetti G, Siciliano AI, Blasi A, Sculean A, Salvi GE. Clinical outcomes after treatment of non-contained intrabony defects with enamel matrix derivative or guided tissue regeneration: a 12-month randomized controlled clinical trial. *J Periodontol*. 2011;82:62–71.

50. Matarasso M, Iorio-Siciliano V, Blasi A, Ramaglia L, Salvi GE, Sculean A. Enamel matrix derivative and bone grafts for periodontal regeneration of intrabony defects. A systematic review and meta-analysis. *Clin Oral Investig*. 2015;19:1581–1593.

How to cite this article: Moreno Rodríguez JA, Ortiz Ruiz AJ, Caffesse RG. Periodontal reconstructive surgery of deep intraosseous defects using an apical approach. Non-incised papillae surgical approach (NIPSA): A retrospective cohort study. *J Periodontol*. 2019;90:454–464. https://doi.org/10.1002/JPER.18-0405