Coronally Positioned Flap for Root Coverage: Poorer Outcomes in Smokers

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Background: Gingival recession is significantly more common among smokers, while the relative outcome of various root coverage procedures in smokers, compared to non-smokers, is debatable. The objective of this study was to evaluate the influence of cigarette smoking on the outcome of coronally positioned flap (CPF) in the treatment of Miller Class I gingival recession defects.

Methods: Ten current smokers (≥10 cigarettes daily for at least 5 years) and 10 non-smokers (never smokers), each with one 2- to 3-mm Miller Class I recession defect in an upper canine or bicuspid, were treated with CPF. At baseline and 6 months, clinical parameters, probing depth (PD), clinical attachment level (CAL), recession depth (RD), and apico-coronal width of keratinized tissue (KT) were determined.

Results: Intragroup analysis showed that CPF was able to reduce RD and improve CAL in both groups (P<0.05). Inter-group analysis demonstrated that smokers presented greater residual RD at 6 months and lower percentage of root coverage (69.3% versus 91.3%; P<0.05). No smokers obtained complete root coverage compared to 50% of non-smokers (P<0.05).

Conclusions: Within the limits of the present study, it can be concluded that CPF provides benefits for both smokers and non-smokers in terms of root coverage of shallow Miller Class I recession defects. However, cigarette smoking negatively impacts the clinical outcomes, specifically residual recession, percent root coverage, and frequency of complete root coverage. J Periodontol 2006;77:81-87.

KEY WORDS
Flap; gingival recession/therapy; root; smoking/adverse effects.

Gingival recession, i.e., the apical shift of the gingival margin in relation to the cemento-enamel junction (CEJ), is a condition affecting a large portion of the adult population, regardless of country of origin or ethnic background. Because gingival recession may lead to esthetic concerns, dentine hypersensitivity, and root caries, several surgical approaches have been used over the years to achieve root coverage. One of the most widely used surgical techniques is the coronally positioned flap (CPF), a procedure particularly indicated for the treatment of Miller Class I gingival recession defects. Many factors, such as operative technique, defect characteristics, and patient attributes, have been related to periodontal plastic surgery healing responses. Nevertheless, recent systematic reviews of root coverage procedures have concluded that there is a need for 1) further efficacy studies and 2) identification of factors most associated with better outcomes.

Besides age, the factor most significantly associated with buccal gingival recession in epidemiological studies is smoking. Smokers, compared to age-matched groups of non-smokers, exhibit greater prevalence, extent, and severity of recession. The strong association between smoking and gingival recession appears independent of the overall severity of interproximal attachment loss and dependent on the level of tobacco exposure. Therefore, smokers, and especially heavy smokers, represent a population with greater root...
Smoking is also associated with poorer periodontal therapy outcomes, with heavy smokers being more likely to experience poor results.\textsuperscript{16-19} Although the potential negative impact of smoking on the outcome of root coverage procedures was recognized by Miller\textsuperscript{20} almost 20 years ago, there are few studies specifically designed to address the role of smoking in periodontal plastic surgery outcomes, and the resulting evidence is conflicting.\textsuperscript{19} This led Johnson and Hill\textsuperscript{19} to conclude that there is a need for additional controlled studies to investigate treatment outcomes for root coverage in smokers. The aim of this prospective, controlled clinical trial was to evaluate the influence of cigarette smoking on CPF outcomes in the treatment of Miller Class I gingival recession defects.

MATERIALS AND METHODS

Subject Selection and Experimental Design
Twenty systemically healthy subjects, 11 males and nine females, average age 34.5 years (\pm 10.3 years; age range, 22 to 53 years), were recruited between February 2003 and September 2003. Ten of the subjects were smokers and 10 were non-smokers (Table 1). Subjects were considered smokers if they reported smoking at least 10 cigarettes per day for at least 5 years prior to the beginning of the study. Smokers with lesser current cigarette use or lesser cumulative tobacco exposure and former smokers were excluded. Individuals who had never smoked composed the non-smoker group.

All study participants met the study inclusion criteria: one Miller Class I recession defect (2 to 3 mm in depth) involving maxillary canine or premolar, presence of identifiable CEJ, periodontally healthy (a minimum of 18 teeth in good periodontal status), no occlusal interferences, systemically healthy, no contraindications for periodontal surgery, and no medications known to interfere with periodontal tissue health or healing. Recession defects associated with caries or restorations and teeth with evidence of pulpal pathology were excluded.

Subjects were selected from patients referred for regular dental treatment at the School of Dentistry at Piracicaba, University of Campinas, Brazil. Predictable risks and anticipated benefits were explained to all subjects by an investigator not directly related to this study. Informed consent was signed by each of the subjects after explanations were provided. The University’s Ethical Committee approved the experimental protocol and consent form.

The study protocol involved a screening appointment to verify eligibility, followed by initial therapy to establish optimal plaque control and gingival health conditions, surgical therapy, postoperative professional plaque control, and final evaluation 6 months after the surgical intervention (Fig. 1).

Initial Treatment
All subjects received initial treatment consisting of oral hygiene instructions and prophylaxis, removal of plaque-retentive factors such as decay, and replacement of faulty restorations at least 1 month prior to the study procedures. Immediately prior to baseline recordings, impression of the maxilla was obtained and duplicate casts were made and used for fabrication of stents for clinical measurements.

Clinical Parameters
At baseline and throughout the study, the full-mouth gingival bleeding index (GBI) and visible plaque index (VPI)\textsuperscript{21} were used to monitor oral hygiene and gingival health conditions.

The following clinical parameters were assessed to the nearest 0.2 mm on the mid-buccal aspect of the study teeth using a standard pressure electronic probe\textsuperscript{8} and a custom stent for probe positioning: probing depth (PD), measured as the distance from the gingival margin (GM) to the bottom of the gingival sulcus; clinical attachment level (CAL), measured as the distance from the CEJ to the bottom of the sulcus; recession depth (RD), measured as the distance from the CEJ to the GM; and the apico-coronal width of...
keratinized tissue (KT), measured as the distance from
the mucogingival junction to the GM. For KT determi-
nation, the position of the mucogingival junction was
assessed using the visual method.

In addition, bleeding on probing (BOP) was recorded. All the
measurements were collected again 6 months after
the operation. A sole trained and calibrated examiner,
masked as to the status of the patient, assessed all
clinical parameters.

Percent root coverage (%), a derived parameter,
was calculated at the end of the study according to
the following formula:

\[(\text{preoperative RD} - \text{postoperative RD}) \times 100/\text{(pre-
operative RD)}\]

Presurgical Preparation and Surgical Procedure
One hour prior to surgery, each patient was given 750
mg acetaminophen\(i\) for pain management. Extraoral
antisepsis was performed with a 2.0% chlorhexidine
solution and intraoral with 0.12% chlorhexidine\(¶\) rinse
for 1 minute. Local infiltration with lidocaine 2.0% with
1:100,000 epinephrine\(#\) was used for anesthesia.

Prior to flap elevation, the exposed root surface was
instrumented with hand instruments to minimize root
convexity, and conditioned with tetracycline** (50
mg/ml in sterile saline solution). The tetracycline
was applied by light-burnishing action using cotton pellets for
90 seconds, followed by copi-
ous saline irrigation. Immedi-
ately after the saline rinsing,
flaps were elevated. The flap
(Figs. 2 and 3) was performed
as previously described.\(23\) The
flap design started with an intra-
sulcular incision at the vestibu-
lar aspect of the involved teeth
and extended horizontally to
the center of the interdental gin-
giva, at CEJ level, mesial and
distal to the defect. Two oblique, apically divergent re-
 laxing incisions, extending beyond the MGJ, com-
pleted the flap design. A full-thickness trapezoidal
mucoperiosteal flap was elevated until the crest of
the marginal bone was reached on the mid-buccal
aspect of the tooth under treatment. Then a split-
thickness flap was extended further apically to allow
the flap to be positioned coronally at the CEJ without
 tension.\(24\) The vestibular epithelium of the interdental
papillae was removed to provide a proper wound bed
for healing. Then the flap was coronally advanced so
that the tissue margin slightly covered the CEJ and
was fixed with 6.0 nylon monofilament†† mattress
sutures (Fig. 4). Thorough saline irrigation was per-
formed during the surgery and periodontal dressing
was not used. One operator performed all surgeries.

Postoperative Protocol
Subjects were prescribed analgesics (acetaminophen
750 mg qid) for 2 days, were instructed to abstain
from brushing and flossing the maxillary teeth until su-
ture removal (14 days), and to use twice daily 0.12%
chlorhexidine rinse for 4 weeks. They were also in-
structed to consume only soft foods during the first
week and to avoid any other mechanical trauma to
the treated sites. No specific instruction was given
to smokers to avoid or reduce smoking after surgery.

Subjects were enrolled in a professional plaque
control program, performed by the same operator
who provided the surgical treatment, scheduled
weekly for the first 4 weeks and then monthly until
the end of the study period (Fig. 5).

Statistical Analysis
Descriptive statistics were expressed as mean ± SD.
For all parametric variables, repeated measures AN-
OVA was used for examination of mean differences

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\(i\) Tylenol, Cilag Farmacêutica, São Paulo, Brazil.

\(¶\) Proderma Farmácia de Manipulação, Piracicaba, Brazil.

\(#\) Lidocaína: Alphacaina, Adrenalina 1:100.000, DFL Indústria e Comércio, Rio de Janeiro, Brazil.

** Bunker Indústria Farmacêutica, São Paulo, Brazil.

†† Nylon monofilament, Ethicon, Johnson & Johnson, São José dos Campos, Brazil.
between baseline and 6 months within groups, and factorial ANOVA was used for examination of mean differences between groups at each time point. Wilcoxon signed rank test was used to compare proportions of complete root coverage between the two groups. The Fisher exact probability test was used to compare the frequency of complete root coverage between the two groups. The significance level for rejection of the null hypothesis was set at $\alpha = 0.05$.

**RESULTS**

All patients tolerated the study procedures well, experienced no postoperative complications, and complied with the study protocol. Full-mouth GBI and VPI were kept below 20%. Teeth of interest were free of plaque and gingival inflammation prior to surgery, during, and at the end of the study.

Descriptive statistics for the clinical parameters at baseline and 6 months after surgery, for both groups, are presented in Table 2. At baseline, no statistically significant differences were found between the two groups for any of the parameters evaluated (Table 2).

In the smokers group, statistically significant changes from baseline were found for RD, CAL, and KT (Table 2). RD decreased by $1.90 \pm 0.45$ mm, which represents average root coverage of 69.3%. Complete root coverage was not obtained in any case. CAL increased by $2.44 \pm 1.20$ mm, while KT decreased by $0.72 \pm 0.73$ mm.

In the non-smokers group, statistically significant changes from baseline were found for RD and CAL (Table 2). RD decreased by $2.32 \pm 0.60$ mm, which represents average root coverage of 91.3%. Complete root coverage was achieved in five cases. CAL increased by $2.20 \pm 1.20$ mm, while KT decreased by $0.72 \pm 0.73$ mm.

At 6 months, RD in the smokers group was significantly greater than the non-smokers group (Table 2). The intergroup comparison of KT values at 6 months approached, but did not reach statistical significance ($P = 0.051$).
When the average root coverage percentage was compared, smokers had a significantly lower percentage than non-smokers ($P<0.05$). When within group changes in PD, CAL, and KT were compared between groups, the KT change in smokers was significantly different than the KT change in non-smokers ($P<0.05$). The frequency of complete root coverage was significantly greater in the non-smokers group ($P=0.016$).

**DISCUSSION**

The objective of this prospective clinical trial was to compare the results of root coverage using the CPF in smokers and non-smokers. The results indicated that, under the high oral hygiene standards maintained throughout the study period in both groups, smokers exhibited poorer outcomes at 6 months, when RD at 6 months, percent root coverage, and frequency of complete root coverage were the outcomes considered.

The results of the present study indicated that use of CPF for root coverage of Miller Class I gingival recession defects provided benefits for smokers and non-smokers alike. However, cigarette smoking negatively impacted the clinical outcome of root coverage. At 6 months post-surgery, the percentage of root coverage was statistically significantly smaller in smokers (69.3%) than in non-smokers (91.3%). In addition, residual recession (RD) was greater in smokers (0.84 mm) compared to non-smokers (0.22 mm).

Perhaps more importantly, no smoker experienced complete root coverage as a result of the procedure, while 50% of non-smokers obtained complete root coverage. These results suggested that smoking was a negative factor for root coverage outcomes in Miller Class I recession defects, when CPF was the chosen treatment modality.

The results of the present study using CPF in smokers and non-smokers were consistent with the findings of other investigators who used this procedure to treat isolated Miller Class I recession defects. The 69.3% and 91.3% average root coverage for smokers and non-smokers, respectively, obtained in the present study is within the range of reported CPF results of 61%, 64.2%, 69%, 69.4%, 97.1%, 97.8%, and 98.8%.9

Unfortunately, for smoker patients in need of root coverage, results of the present study are expanding the list of periodontal plastic surgery procedures for which smokers have been shown to experience poorer outcomes. Significant differences between smokers and non-smokers were found in the treatment of gingival recession with subepithelial connective tissue graft (SCTG) and guided tissue regeneration (GTR).30

Martins et al.29 treated by SCTG 15 patients, seven smokers and eight non-smokers, initially presenting with Miller Class I or II recession defects (RD ≥3 mm). At 4 months postoperatively, the authors observed mean root coverage of 74.7% for non-smokers and 58.8% for smokers, while smokers had significantly greater residual RD. Complete root coverage was observed in 35% of the non-smokers and apparently not in the smokers.

Trombelli and Scabia30 analyzed the results of 22 patients presenting with Miller Class I or II recession defects (RD ≥4 mm), nine smokers and 13 non-smokers, treated by GTR. At 6 months, mean root coverage was 57% for smokers and 78% for non-smokers, while the estimated residual recession was 2 mm for smokers and 1.1 mm for non-smokers. Complete root coverage was observed in one smoker (11%) and five non-smokers (38%).

From the information reviewed above, it is evident that the three University clinic-based studies being discussed at this point (present study on CPF, SCTG study,29 and GTR study30) found similar differences between smokers and non-smokers. The fact that smokers have poorer root coverage outcomes under such different treatment and defect circumstances strengthens the association between smoking and poor results. It should be noted that the three studies, besides differences in surgical procedure used and preoperative RD of the treated defects, were different in design; the Trombelli and Scabia30 study was a retrospective analysis, while the Martins et al.29 and the present study were prospective trials. Results of the above studies add support to the original observation of Miller,20 who reported that 100% of the heavy smokers (defined as smoking more than 10 cigarettes daily) treated using free gingival graft procedure for root coverage, experienced failure.

In contrast to the above studies, Harris and Harris,31 Tolmie et al.,32 and Amarante et al.27 did not find an association between smoking and poor root coverage outcomes using a CTG procedure, free gingival graft, and CPF, respectively. More specifically, Harris and Harris31 found no difference between non-smokers, light smokers (≤10 cigarettes daily), and heavy smokers (>10 cigarettes daily), while Tolmie et al.32 obtained complete root coverage in almost 92% of the smokers (not defined further) they treated. Amarante et al.27 found that 62% of heavy smokers (≥20 cigarettes per day) had complete root coverage compared to 42% of non-smokers. It should be noted that none of these three studies was specifically designed to test the effect of smoking on root coverage outcomes. Reasons for the discrepancy between results of the various studies are not clear at this point. The lack of definition of the smoking exposure32 and the specific surgical procedure used31 are the most obvious differences between these studies and the ones where smoking had a negative impact on outcomes.
Precise mechanisms by which tobacco use interferes with root coverage procedures are not completely understood, whereas several physiological and cellular functions altered by smoking may account for the compromised periodontal wound healing in smokers. Regarding physiological functions, smoking negatively impacts the gingival blood supply, a factor critical for proper periodontal flap healing. Regarding cellular functions, nicotine inhibits the proliferation, adhesion, and chemotaxis of periodontal ligament cells, alters the interaction between epithelial cells and gingival fibroblasts, inhibits the production of collagen and non-collagen matrix proteins by gingival fibroblasts, increases gingival fibroblast collagenolytic activity, and inhibits the adhesion of fibroblasts to root surfaces. These changes could all contribute to the poorer healing of smokers.

CONCLUSIONS
Within the limits of the present investigation, CPF provided benefits for both smokers and non-smokers in terms of root coverage of shallow Miller Class I recession defects. However, cigarette smoking negatively impacted the clinical outcomes, specifically residual recession, percent root coverage, and frequency of complete root coverage.

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