

# Effect of air-polishing devices on the gingiva: histologic study in the Canine

Avital Kozlovsky<sup>1</sup>, Zvi Artzi<sup>1</sup>,  
Carlos E. Nemcovsky<sup>1</sup> and  
Abraham Hirshberg<sup>2</sup>

Departments of <sup>1</sup>Periodontology and <sup>2</sup>Oral Pathology and Oral Medicine, The Maurice and Gabriela Goldschleger School of Dental Medicine, Tel Aviv University, Tel Aviv, Israel

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## Abstract

**Objectives:** The aim of this study was to evaluate histologically the immediate effect of two types of air-polishing devices (APDs) on the gingiva.

**Material and Methods:** The buccal gingiva in four mongrel dogs was exposed to 5, 10 and 20 s of instrumentation, applying a hand-piece type of APD (HP-APD) PROPHYflex<sup>®</sup> and a stand-alone type of APD (SA-APD) Jetpolisher<sup>™</sup> in a split-mouth design. Immediately after treatment, the exposed gingiva was excised by sharp dissection and fixed in 10% buffered formalin. For each specimen, 15 sections, 20 µm apart, corresponding to the central part of the treated and control untreated gingiva were cut and stained with haematoxylin and eosin. Stained sections were examined histomorphometrically for keratin width and epithelial cell layer (ECL) (prickle and basal cell layers) width. Extent of erosion was expressed as loss of keratin and ECL compared with control.

**Results:** Microscopic examination presented changes in keratin and ECL, including keratin detachment and disruption of the normal ECL architecture. The erosive changes in the gingiva caused by both APD, positively correlated with instrumentation time ( $p < 0.001$ ) and type of instrument ( $p = 0.008$ ). Keratin loss was significantly higher for SA APD than for the HP APD in each time interval ( $p = 0.019$ ). Following exposure for 5 s, both APD caused a 25% loss of the ECL. Exposure for 10 and 20 s revealed a significantly greater ECL loss caused by the SA-APD than the HP-APD ( $p = 0.018$ ). Exposure for 20 s was the only time interval that presented the area of total epithelium erosion with SA-APD causing significantly more ( $p = 0.002$ ) areas of total epithelial erosion than the HP-APD.

**Conclusion:** Gingiva exposure to air-polishing slurry delivered by APD caused localized trauma because of epithelial erosive changes with severity, positively correlated with instrumentation time and design principles of the applied APD. The clinician should be aware of the potential insult of the gingiva when applying the APD and careful precautions should be taken.

Key words: air-polishing device; gingival erosion

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Maintenance of a healthy dento-gingival complex requires the removal of external deposits from the tooth surface to eliminate bacteria and bacterial by-products from the gingival area (Kaldahl et al. 1996, Westfelt 1996). In the early 1980s, an air-polishing device (APD) was introduced for clinical use, aimed at rapidly and efficiently removing stains and plaque from the supra-gingival tooth surface with less fatigue

and with the advantage of reaching areas of difficult accessibility (Willmann et al. 1980, Atkinson et al. 1984, Berkstein et al. 1987, Kozlovsky et al. 1989).

The APD operates by delivering a fine slurry of pressurized air, water and abrasive powder from a special reservoir against the tooth surface (Weeks et al. 1984). The abrasive powder contains finely powdered sodium bicarbonate to

which tricalcium phosphate is added (up to 0.8% by weight) to improve flow characteristics. The abrasive property of the slurry delivered from APD is determined by the particle's velocity, hardness and shape (Momber & Kovacevic 1998, Petersilka et al. 2003b), and design principles of the APD (Petersilka et al. 2002). The stand-alone type of APD (SA APD) working principle is based on mixing air and powder by

swirling, and the hand-piece type of APD (HP APD) air/powder mixture is created by the carburetor technique and swirling (Petersilka et al. 2002). The amount of powder released in different powder settings of APD depends on the way the pressurized air is led through the powder chamber, resulting in high variability in powder emission rates at different powder settings between the APDs (Petersilka et al. 2002).

The effect of APD on tooth hard tissues has been investigated in several studies. Tooth enamel is minimally affected by the abrasive powder as shown with the use of profilometre scans (Willmann et al. 1980). However, the action of the APD on the root structure can cause substantial cementum and dentin loss, which is almost linearly related to the amount of time the area is subjected to the spray (Galloway & Pashley 1987). The mode of use of the device, i.e., overlapping brush strokes or stationary position while dwelling on the tooth surface, powder setting of APD and various working parameter combinations, cause different root surface abrasion (Berkstein et al. 1987, Galloway & Pashley 1987, Jost-Brinkmann 1998, Petersilka et al. 2003b).

APD slurry is usually aimed at the tooth surface close to the gingival margin. The effect of the APD jet on gingival mucosa has been reported in a few studies based on clinical observations in humans (Weeks et al. 1984, Newman et al. 1985, Mishkin et al. 1986), scanning electron microscopy (SEM) examination of positive replica prepared from gingiva impressions following air polishing (Kontturi-Närhi et al. 1989) and microscopic examination in rabbit mucosa (Newman et al. 1985).

Clinical studies have shown that APD induces localized trauma to the gingiva that heals within 6 days (Weeks et al. 1984, Mishkin et al. 1986). SEM of replica prepared from impressions taken from gingiva following teeth cleaning with APD revealed gingival erosive changes with severity, positively correlated with the presence of gingival inflammation before instrumentation (Kontturi-Närhi et al. 1989).

The aim of the present study was to examine microscopically the influence of the abrasive slurry of APD on healthy canine gingiva and to assess its relation to time of exposure and the type of the applied APD.

## Material and Methods

The study protocol was approved by the Ethics and Institutional Animal Care and Use Committees of Tel Aviv University. Four mongrel 2-year-old dogs were used. The maxillary canine, premolar and molar regions were selected as experimental sites. Following general intravenous sodium pentobarbitone anaesthesia (25 mg/1 kg), the maxillary teeth were scaled and polished. Premedication of ketamine (5 mg/1 kg) was used to gently brush the experimental teeth every other day for 3 weeks (Tromp et al. 1986). At the end of the pre-operative period, the buccal gingiva was carefully examined for deviation from normal colour, texture and bleeding with gentle probing, and diagnosed as clinically healthy gingiva.

An SA APD Jetpolisher™ (Deldent, Petach-Tikva, Israel) and an HP APD PROPHYflex® (KaVo, Biberach, Germany) were used for the study. The powder chambers of both instruments were filled with standard NaHCO<sub>3</sub> powder as supplied by the manufacturer, to the maximum powder level to ensure maximum reproducibility of powder emission (Petersilka et al. 2002). Instruments were set up according to the manufacturers' instructions to a medium water and powder setting. The APD was applied to the gingiva in a split-mouth design under general intravenous sodium pentobarbitone anaesthesia and without local infiltration of anaesthetic solution. Each animal was treated with both types of APD. The jet was aimed at the gingiva through an orifice, 5 × 7 mm, cut in an aluminium foil protecting the untreated area (Fig. 1). The tip of the instrument was moved in constant, overlapping brush strokes,

held 5 mm from the surface. Air powder abrasion was applied for 5, 10 and 20 s using each of the APDs. Two areas of treatment were obtained for each time interval. Immediately following treatment the exposed gingiva was excised by sharp dissection. To standardize the histologic trimming of the samples, a marker of the disto-apical corner of the sample was made by a stitch. Lidocaine 2% with norepinephrine (1:100,000) was administered by local infiltration at the end of the surgical procedure to prevent haemostasis and reduce post-operative pain. A total of 14 samples from each animal were obtained: two control samples and two experimental samples for each time interval using each APD.

Tissue samples were fixed in 10% buffered formalin and embedded in paraffin. For each specimen, 5 µm wide sections, 20 µm apart, were serially cut and stained with haematoxylin and eosin. A total of 15 sections corresponding to the central part of treated and control untreated gingival sample were obtained. Sections were examined microscopically, at a magnification of × 400 using a standard 100-point scale or grid (100 grids = 250 µm). Measurements were carried out with the aid of a calibrated ocular micrometre. The keratin and epithelial cell layer (ECL) (prickle and basal cell layers) width was measured in 10 random points along each section. The mean width was calculated for each section, in each sample, and expressed in millimetres.

## Statistical analysis

The measurements of keratin and ECL width in control and experimental



Fig. 1. Gingiva covered by an aluminium foil protecting the untreated area. Orifice cut in the foil enables exposure of the experimental area to the air-polishing device slurry.

samples were averaged according to the type of APD and time of exposure. The extent of erosion was expressed as loss of keratin and ECL width compared with untreated (control). Results for both devices at different time points were analysed and compared. Each dog was designed as a statistical unit and a three-way ANOVA with repeated measures (time, type of APD, width of keratin or ECL) was performed. The results and interactions using four dogs can be analysed reliably using the models of ANOVA with repeated measurements (Tal et al. 1996).

## Results

### Clinical observation

Prior to the exposure to APD, there was no gingival pathosis, i.e., no change in colour or bleeding. Immediately following application of the APD, there was noticeable gingival erosion with slight pinpoint to profuse bleeding related to time of exposure (Fig. 2).

### Histopathological findings

#### Control specimens

The epithelium of the control samples presented normal characteristics of gingival mucosa with keratinized stratified squamous epithelium with a well-defined parakeratin layer (Fig. 3).

#### Experimental specimens

The experimental specimens presented histomorphologic changes for both instruments. In the keratin layer, early changes included detachment from the underlining ECL and disruption of the normal architecture (Fig. 4). In the ECL, APD caused tears in the epithelium layer, disappearance of nuclei and disruption of the intercellular bridges resembling an acantolytic lesion (Figs. 5 and 6). Application of APD for 20 s resulted in areas of total loss of the epithelium with exposure of the underlining connective tissue (Fig. 7).

#### Histomorphometry

The mean width of keratin and ECL in the control untreated gingiva was 0.028 mm (SD 0.00071) and 0.322 mm (SD 0.0014), respectively. The mean and percent of erosion caused by each APD applied for 5, 10 and 20 s are presented in Table 1. The degree of the



Fig. 2. Following application of air-polishing device for 10 s, noticeable gingival erosion and bleeding are clearly shown.

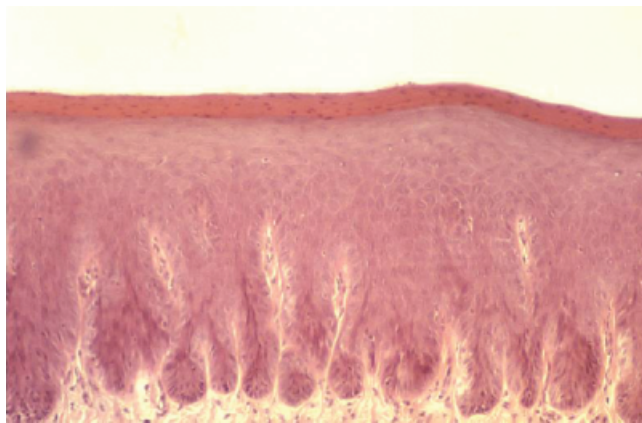


Fig. 3. Photomicrograph of the gingiva of the control sample with normal characteristics of the gingival mucosa covered with a well-defined parakeratinized layer (haematoxylin and Eosin, original magnification  $\times 100$ ).

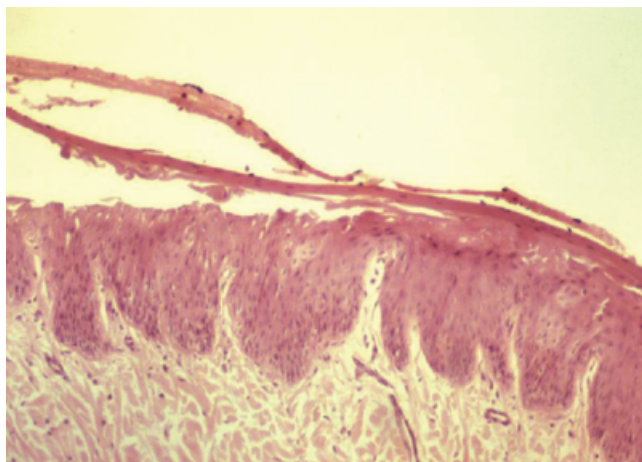


Fig. 4. Photomicrograph of the gingiva following 5 s of air-polishing device exposure. Keratin layer is disrupted and detached from the underlying epithelial cell layer (haematoxylin and eosin, original magnification  $\times 40$ ).

erosion measured in keratin and ECL for each APD positively correlated with the time of exposure ( $p < 0.001$ ). Kera-

tin loss was significantly higher for SA APD than for the HP APD ( $p = 0.019$ ) in each time interval. Following 5 s of



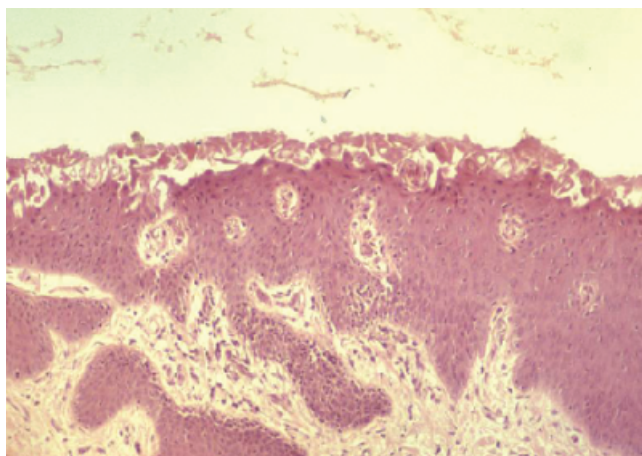


Fig. 5. Photomicrograph of the gingiva following 10 s of air-polishing device application. Note loss of keratin and tears in the epithelial cell layer (haematoxylin and eosin, original magnification  $\times 100$ ).

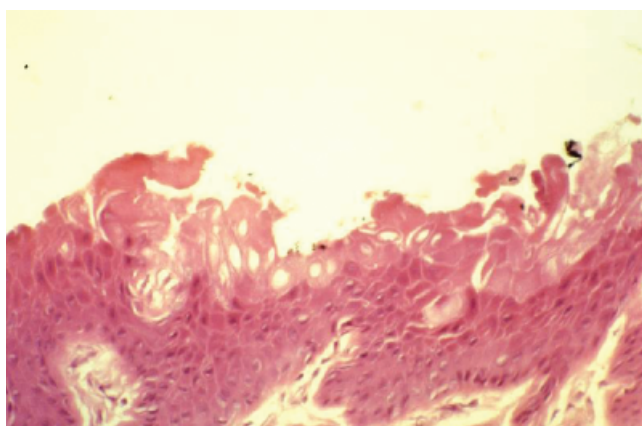


Fig. 6. Higher magnification of Fig. 5. Note disappearance of nuclei and disruption of intercellular bridges (haematoxylin and eosin, original magnification  $\times 200$ ).



Fig. 7. Photomicrograph of the gingiva following 20 s of air-polishing device exposure. Note extensive epithelium loss and the exposed underlining connective tissue (haematoxylin and eosin, original magnification  $\times 40$ ).

instrumentation, the SA APD caused a mean keratin erosion of 77% as compared with 46% by HP APD. Statistical

analysis revealed a significant interaction ( $p = 0.008$ ) between the extent of keratin erosion, time of exposure and

type of instrument. With regard to the ECL, erosion increased significantly with time ( $p < 0.001$ ) for both instruments (Table 1, Fig. 8). A significant interaction was noted between the extent of ECL erosion, time and type of instrument ( $p = 0.018$ ). There was a non-significant difference between the damage caused to the ECL by the abrasive slurry delivered for 5 s by both APDs (26.3% for SA APD Jetpolisher<sup>TM</sup> and 25.1% for HP APD PROPHYflex<sup>®</sup>) but exposure for 10 and 20 s revealed significantly greater ECL erosion caused by the SA APD Jetpolisher<sup>TM</sup> as compared with HP APD PROPHYflex<sup>®</sup> (Table 1, Fig. 9). A 60% mean loss of ECL width was observed following exposure to 20 s using the SA APD Jetpolisher<sup>TM</sup> and 47% using the HP APD PROPHYflex<sup>®</sup>. A 20 s exposure was the only time interval that presented the area of total erosion of epithelium, with the SA APD Jetpolisher<sup>TM</sup> causing significantly greater ( $p = 0.002$ ) areas of total epithelial erosion than the HP APD PROPHYflex<sup>®</sup>.

## Discussion

Active periodontal treatment and the following supportive treatment that continues at varying intervals for the life of the dentition (Wilson 1996) include removal of bacterial plaque and extrinsic staining. The use of APDs is time saving and more convenient compared with other conventional modes of debridement procedures (Weeks et al. 1984). Minor or negligible alterations of the enamel-treated surface have been shown, thus encouraging its use (Willmann et al. 1980, Jost-Brinkmann 1998). Nevertheless, it is not completely harmless. Several studies report that an application of an air-abrasive jet to the root surfaces results in root substance removal (Atkinson et al. 1984, Berkstein et al. 1987, Horning et al. 1987), and a wide range of defect depth and defect volumes, which are highly influenced by working conditions (Petersilka et al. 2003a, b).

Contact between the abrasive slurry and the marginal gingiva is inevitable when the APD is used on the cervical part of the clinical crown. Complete plaque removal from the tooth surface can be achieved within 5–20 s of APD application. Therefore, these time intervals are the working parameters in most studies that evaluate the APD (Berkstein

Table 1. Mean width (mm) and percentage ( $\pm$  SD) of gingival erosion after APD application

Time (s)	SA APD	HP APD
Keratin: control $0.028 \pm 0.00071$		
5	$0.022 \pm 0.0034$ ( $77 \pm 12\%$ )	$0.013 \pm 0.0063$ ( $46.5 \pm 22\%$ )*
10	$0.024 \pm 0.0022$ ( $84 \pm 7.8\%$ )	$0.017 \pm 0.0083$ ( $57 \pm 29\%$ )*
20	$0.025 \pm 0.0020$ ( $88 \pm 7.1\%$ )	$0.021 \pm 0.0058$ ( $68 \pm 20\%$ )*
Epithelium: control $0.322 \pm 0.0014$		
5	$0.085 \pm 0.0012$ ( $26 \pm 3.75\%$ )	$0.081 \pm 0.0073$ ( $25 \pm 2.26\%$ )
10	$0.149 \pm 0.0098$ ( $46 \pm 3.0\%$ )	$0.106 \pm 0.0014$ ( $33 \pm 4.3\%$ )*
20	$0.195 \pm 0.0011$ ( $61 \pm 3.4\%$ )	$0.150 \pm 0.0031$ ( $44 \pm 2.6\%$ )*

\*Significant difference ( $p < 0.001$ ).

HP APD, hand-piece type of APD; SP APD, stand-alone type of APD.

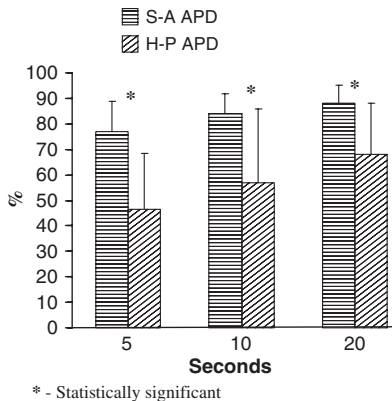


Fig. 8. Percentage of keratin erosion in different time intervals as exposed to two types of air-polishing device (APD).

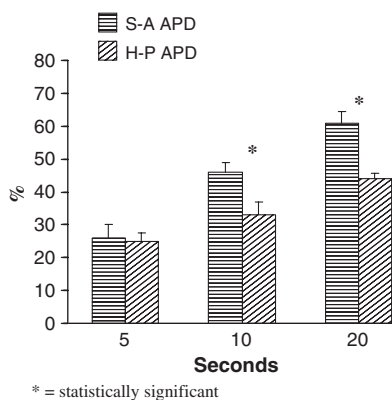


Fig. 9. Percentage of epithelial cell layer erosion in different time intervals as exposed to two types of air-polishing device (APD).

et al. 1987, Petersilka et al. 2003a–c). The present study characterized the histological influence of the APD on the gingiva following exposure to minimal time intervals, up to 20 s. This is in contrast to the dog study of Van de Velde et al. (1982), in which the APD was applied for 30 s–2 min, a period of

time that is rarely used in a clinical situation.

In the present study, the histological examination of healthy dog gingiva following an application of an air-abrasive jet with standard  $\text{NaHCO}_3$  powder revealed erosive changes in the keratin and ECL. The extent of the damage correlated positively with the time of exposure. APD slurry contacting the gingiva for no more than 5 s causes significant erosion mainly of the keratin layer (44–77%) with minimal extension (25%) to the ECL. Nevertheless, exposure to the slurry lasting 10 and 20 s resulted in mean 33–61% ECL erosion. A 20 s exposure was the only time interval to present the area of total erosion of the epithelium.

The type of APD applied was another parameter that significantly influenced the extent of epithelial erosion. Significantly deeper keratin and ECL erosion was measured in all time intervals following exposure to the SA APD as compared with HP APD. Also the SA APD caused significantly greater areas of total epithelial erosion following 20 s of exposure as compared with HP APD.

The design principles of the APD dictate the powder emission rates, the ratio of powder and water in the slurry and the velocity of the particles (Mombert & Kovacevic 1998, Petersilka et al. 2003b), i.e., the abrasiveness of the slurry released by the APD. Therefore, according to the present histomorphometric evaluation, the working principle based on mixing air and powder by swirling, as in the SA APD Jetpolisher™ (Petersilka et al. 2002), produces slurry that is more aggressive than the slurry created by the carburetor technique and swirling, as in the HP APD PROPHYflex® (Petersilka et al. 2002). The operator should be aware of the variability in the abrasiveness of the

devices because of the technical specification of each APD, such as the drive, water pressure and powder emission rates in different powder settings, and not only to the time of exposure and properties of the applied powder. Since the HP APD showed significantly less gingival abrasiveness than the SA APD in all tested time intervals, it is a better choice, clinically.

In this study, the normal gingiva of the experimental dogs presented keratinized stratified epithelium as human gingival epithelium (Weiss et al. 1959, Squier & Finkelstein 2003). The mean width of the oral gingival epithelium as measured in the dog in the present study is comparable with the width of oral gingival epithelium in humans (Tal & Dayan 2000):  $0.350 \pm 0.0021$  and  $0.345 \pm 0.129$  mm, respectively. This similarity enables implication of the histologic results of the present study in canine to the clinical situation in humans. Thus, it can be concluded that the APD should be used with overlapping strokes for no more than 5–10 s per surface to minimize the extent of epithelial erosion and prevent the possibility of total exposure of the underlying connective tissue. The gingival erosive changes because of the application of APD, even slightly, can cause bleeding, sensitivity and difficulty in tooth brushing with a healing sequence of 6–12 days (Kontturi-Närhi et al. 1989). Therefore, it should be used with caution. These negative aspects of the use of the APD can be of major clinical significance while treating tooth surfaces close to a thin-scalloped type of periodontium. The susceptibility to trauma of thin gingiva characterized by marginal gingiva composed of epithelial cells with minimal core of connective tissue between the oral and sulcular epithelium is higher than that of thick-flat gingiva. Therefore, an application of APD to thin marginal gingiva for more than 5 s can cause deep erosion of the epithelium, which, even in a mild form, may be followed by irreversible damage, such as gingival recession, and change in the shape and height of interdental papilla. Extensive extrinsic tooth discolouration close to the gingival margin (because of smoking or use of antiseptic solution) and stained crowded teeth may force the clinician to prolong the time of instrumentation. In these cases, protection of the marginal gingiva by an aluminium foil, as used in the present study, is recommended.

Future studies should address the use of novel APD powder (Petersilka et al. 2003a), which presents low abrasiveness to root cementum, and evaluate its effect on gingiva while applied by different APDs. Use of the APD intra-sulcular for subgingival deplaquing, as suggested by Petersilka et al. (2003c, d), should consider the correlation between instrumentation time and the effect of the abrasive slurry on the sulcular non-keratinized epithelium and the junctional epithelium. It can be assumed that both the slurry-directed intrasulcular and that reflected from the root surface to the soft wall of the sulcus/pocket may result in significant epithelial erosion.

In the present study, the effect of the APD-polishing technique on healthy gingiva was examined. Further studies aimed at evaluating the correlation between the severity of the microscopic epithelial erosion because of exposure to APD slurry and gingival inflammation should be conducted. Also, the healing process of the erosive gingival lesion caused by APD in healthy and inflamed gingiva should be studied.

It can be concluded that to prevent injury of the gingiva with the APD, the device should be used with caution while taking into consideration that the time of exposure and the design principle of the APD influence the severity of the gingival erosion.

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## Address:

Dr. Avital Kozlovsky  
Department of Periodontology  
School of Dental Medicine  
Tel-Aviv University  
Tel-Aviv, Israel  
E-mail: kavital@post.tau.ac.il

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