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ORIGINAL ARTICLE

# Approximal plaque pH lowering after sugar intake in a periodontally infected dentition

S. G. Rüdiger[a] and A. Carlén[b]

[a]Department of Periodontology, Specialist Dental Care Centre, Public Dental Service, Lund, Sweden; [b]Department of Oral Microbiology and Immunology, University of Gothenburg, Gothenburg, Sweden

**ABSTRACT**

**Objectives:** The aim of this study was to evaluate the effect of periodontal inflammation on the approximal plaque pH after a sucrose rinse.

**Materials and methods:** Thirty-two periodontitis patients (aged 38–72 years; 9M/23F) were included. All patients were in need of periodontal surgery. Two non-adjacent interdental spaces, one healthy (no bleeding on probing [BoP] and probing pocket depth [PPD] $<4$ mm) and one periodontally diseased (BoP and PPD $\geq 5$ mm) were selected. Before and after surgery, the approximal plaque pH was measured before and after 2, 5 and 10 min after a 1-min rinse with sucrose solution.

**Results:** In periodontally diseased interdental spaces, a significant pH drop was seen 5 min after rinsing. In healthy spaces and after surgery, a significant pH drop was seen after 2 min. A multilevel regression analysis showed that greater probing pocket depths were significantly associated with pH change measured 5 min after rinsing ($p < .05$). Further on, the approximal pH drop after a sucrose rinse tended to be delayed in dentitions with $\geq$10% of PPD $\geq 5$ mm ($p = .052$).

**Conclusions:** The results suggest that an ongoing periodontal inflammation could temporarily neutralize acidic metabolic products after a sugar challenge. This may further suggest that plaque pH measured after a sugar rinse might be used to identify an ongoing periodontal disease.

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Periodontitis; pH-lowering; sucrose rinsing; periodontal infection

## Introduction

The plaque pH is widely used for caries risk assessments [1], its significance in relation to periodontal disease, on the other hand, has not been investigated as extensively. In a recent *in vitro* study, it has been shown that the metabolic activity of biofilms representing periodontal disease (including species like *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*) was highest at pH 7.0–7.5 while cariogenic bacteria (including *Streptococcus gordonii*, *Actinomyces naeslundii*, *Streptococcus mutans*) thrived best at pH 5.5–6.0 [2]. In growing supra- and subgingival biofilms *in vivo* saccharolytic species, for example, *Streptococcus* ssp. and *Actinomyces* ssp., dominate initially. From day four, however, in both healthy and periodontally diseased individuals, a subgingival shift to an asaccherolytic proteolytic microbiota, for example, *Campylobacter rectus*, *Campylobacter showae*, *Capnocytophaga ochracea* and *Prevotella nigrescens* was seen [3]. As a result, signs of inflammation occur at the gingival margin and the flow of the gingival crevicular fluid (GCF) – a serum-like fluid derived from blood vessels in the gingival sulcus – increases [4]. While only traces of GCF are detectable in gingival health [5], clinical signs of inflammation usually are accompanied by higher volumes [6–8] and higher pH of the GCF [9–11]. In contrast, bacteria in the supragingival environment metabolize typically glucose, creating an acidic environment and favouring acid-tolerant microorganisms [12].

In a series of experiments, we noted that excess of GCF oozing from the gingival margin can reach enamel surfaces far distant from the gingival margins [13,14] and, thus, possibly neutralize an acidic supragingival environment. Our hypothesis based on our previous findings is that plaque pH measured directly under an intact approximal contact point may be influenced by periodontal inflammation.

The aim of this study is to evaluate to which extent periodontal inflammation could affect approximal plaque pH (further on referred to as 'pH') after a sucrose rinse.

## Material and methods

### Patient population

Thirty-two referred periodontitis patients (9 men; 23 women) aged 38–72 years and with a dentition of 19–32 teeth were included in this study upon completion of cause-related therapy at the Specialist Dental Care Centre, Lund, Sweden. The inclusion criteria were dichotomous full-mouth plaque (FMPS; [15]) and bleeding (FMBS) scores $<$10% after cause-related therapy; the need for periodontal surgery at at least one tooth with pocket probing depth (PPD) $\geq 5$ mm, Bleeding on Probing (BoP) and no furcation involvement; at

CONTACT S. G. Rüdiger stefan.rudiger@folktandvardenskane.se Department of Periodontology, Specialist Dental Care Centre, Public Dental Service, Sockerkokaregatan 15, Lund S-222 36, Sweden

least one interdental spaces with PPD <4 mm and no BoP. The patients should be able and willing to follow the recommendations for the treatment of periodontitis. Nine patients, of those three men, were smokers (≥ 10 cig/d). None of the patients used snuff. The patients were diagnosed with periodontitis stage III/grade B ($n = 22$), stage III/grade C ($n = 5$) and stage IV/grade C ($n = 5$) according to the classification of 2018 [16] or chronic severe localized ($n = 14$) and generalized ($n = 18$) according to the classification of 1999 [17]. Upon recruiting, PPD of ≥5 mm was seen in 15% (median); 10–28% (quartiles) of all sites in the dentition. 17 patients were medically healthy and did not take any drugs. Fifteen patients took medications for cardiovascular ($n = 7$), respiratory ($n = 2$), rheumatic diseases ($n = 2$), depression ($n = 2$), diabetes ($n = 1$) or hypothyroidism ($n = 1$). Periodontal surgery was performed as described by Sallum et al. 2005 [18]. After local anaesthesia and intrasulcular incisions, full-thickness flaps were raised both from the buccal and palatal/lingual aspect. Granulation tissue was removed with the blunt edge of a curette. Thorough debridement was performed aiming at the removal of calculus deposits using hand instruments and, in concavities, an ultrasonic device. In no case, the prerequisites for regenerative treatment were seen. If ostectomy was deemed necessary for pocket elimination a round bur was used. Care was taken to reposition the soft tissues without tension. Sutures were placed (Supramid DS 19 4/0; B. Braun Melsungen AG; Melsungen; Germany). When necessary for postoperative tissue adaptation, a periodontal dressing (Coe-pak; GC America Inc.; IL, USA) was used [19]. During the first two weeks post-surgery, the patients used an antiseptic mouth rinse (Paroex; Sunstar Americas Inc. Oral Care Schaumburg, IL). At suture removal, 10–14 days post-surgery, the patients were thoroughly instructed to brush their teeth again with a toothbrush and interdental brushes. Thereafter, they were seen by a dental hygienist for dental prophylaxes – at least twice – until full-mouth plaque scores of <10% were reached before re-evaluation was scheduled. The study was performed from spring 2017 to autumn 2019.

### Clinical measurements

All probing measurements were performed using a PCP 12 probe (Hu-Friedy Mfg. Co., LLC.

Frankfurt; Germany). BoP was read 30 s after probing and plaque was registered after disclosing (Rondels Red; Directa AB; Upplands Väsby, Sweden). Pre- and postoperative PPD, FMPS [15] and FMBS were taken at four sites per tooth.

### Experimental interdental spaces

Two interdental spaces – one healthy, one periodontally diseased – in the same jaw with intact (i.e. non-restored) contact points were selected. Upper teeth were selected in 26, lower teeth in 6 patients. Most of the experimental interdental spaces were located in the anterior sections of the dentition (Table 1). There should be at least two approximal spaces between the selected experimental spaces. In the healthy interdental spaces both approximal periodontal sites displayed PPD of <4 mm and no BoP. In the periodontally diseased interdental spaces, one of the approximal periodontal sites should display PPD of ≥5 mm and BoP (in this study called 'diseased sites'). The other approximal periodontal sites within the diseased interdental spaces showed signs of periodontal disease having PPD ≥3 mm with or without BoP (in this study called 'slightly diseased sites'; see Figure 1).

For the analysis of a possible impact of PPD and BoP on the pH after sugar rinsing, either before or after pocket elimination, the data were arranged as follows. The analysis of the impact of PPD on the pH was based on PPD of the 'diseased site' ≤6 mm vs >6 mm before and ≤4 mm vs >4 mm after surgery, as no probing depth >5 mm was noted at the 'diseased sites' after surgery.

**Table 1.** Types of teeth surrounding the respective experimental interdental spaces.

| | Interdental space | |
|---|---|---|
| Types of teeth | Healthy | Diseased |
| Incisor/incisor | 6 | 8 |
| Canine/incisor | 2 | 8 |
| Premolar/canine | 11 | 5 |
| Premolar/premolar | 6 | 7 |
| Molar/premolar | 5 | 3 |
| Molar/molar | 2 | 1 |

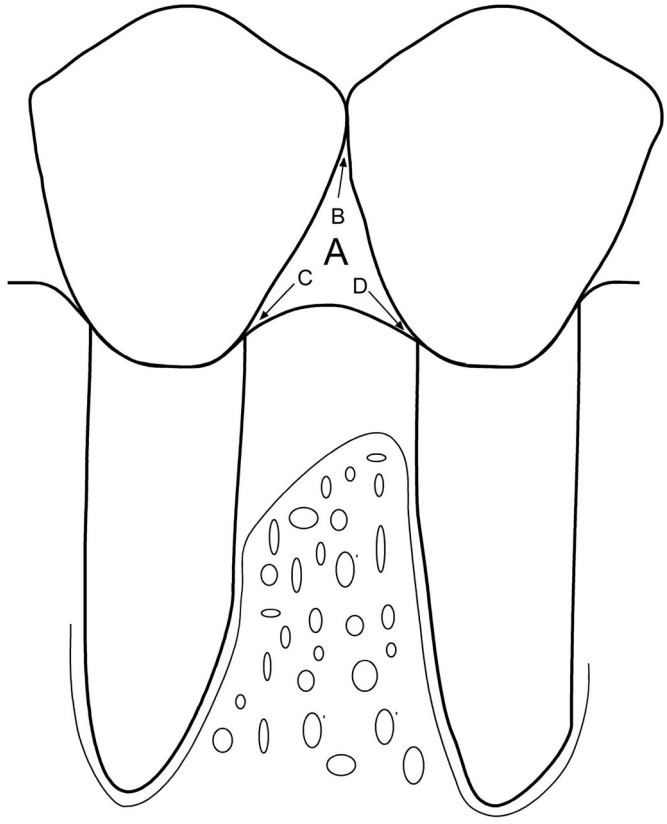


**Figure 1.** The diseased interdental space before surgery. (A) The interdental space with an intact contact point forms a biological unit on its own influenced by the two periodontal sites within the space. (B) The space directly under the intact interdental contact point was the location for pH measurement in this study. (C) The 'diseased site' and (D) the 'slightly diseased site' within the diseased interdental space both contribute to the inflammatory load of the interdental space.

Before surgery, all 'diseased sites' bled upon probing. Therefore, BoP at the 'slightly diseased sites' of interdental spaces were chosen for the analysis of having BoP pre-surgery which strictly speaking was BoP additional to BoP at all the 'diseased sites'. After surgery, none of the 'slightly diseased sites' bled on probing, therefore BoP of the 'diseased sites' was chosen for the analysis post-surgery.

In this context, the interdental space can be considered as a biological unit as the 'diseased' and the 'slightly diseased site' both contribute to the inflammatory load of the interdental space.

### pH measurements

The pH strip method was used as previously described [20], however slightly modified as the pH was measured at a distance from the gingival margin directly below the contact point (see Figure 1). This location was chosen since plaque is formed during relative seclusion and the changing imbalance of bacterial acid production and alkaline GCF components from the gingival margin was believed to be the least exposed to disturbing influences. Further on, this location was – in relation to the gingival margin – considered to be the least affected by possibly changing dimensions of the soft tissues after periodontal surgery. We used two types of pH indicator strips (range 4–7 and 6.5–10; MQuant®; Merck KGaA; Darmstadt, Germany) not to miss values in the alkaline range. The original strips were cut into three pieces (ca. 2 mm in width), which were easy to insert directly under the contact point in the interdental space.

Two measurements were carried out immediately before the re-evaluations after cause-related therapy and after periodontal surgery, respectively (see flow chart: Figure 2). Thus, gingival bleeding never interfered with the pH measurements. Before and after 2, 5 and 10 min after a 1-min mouth rinse with 10 ml of a 10% sucrose solution, plaque pH was measured directly under the contact point. Before each measurement, the participants were asked to swallow and then forcefully inhale through the interdental spaces to remove excess saliva from between the teeth. Subsequently, cotton rolls were placed into the vestibular fornix and in the lower jaw sublingually to keep away the cheek and tongue thus facilitating the measurements. Directly after each measurement, the cotton rolls were removed, and the patients were placed in an upright rest position (no talking) until the next pH measurement. Following successive insertion of the strips (first range from 4 to 7, then range from 6.5 to 10) for 10 s, the pH value (one decimal) was assessed by comparing the colour of the strip with the colour on the respective index scheme supplied by the manufacturer. The pH value, that was with a good margin within the measuring range of the respective pH strip (mostly the value given by the pH indicator strips of the range from 4 to 7), was considered for analysis.

Ethical approval was granted by the Regional Ethical Review Board in Lund/Sweden (Dnr 2014/164). All patients were provided with oral and written information and gave their informed consent.

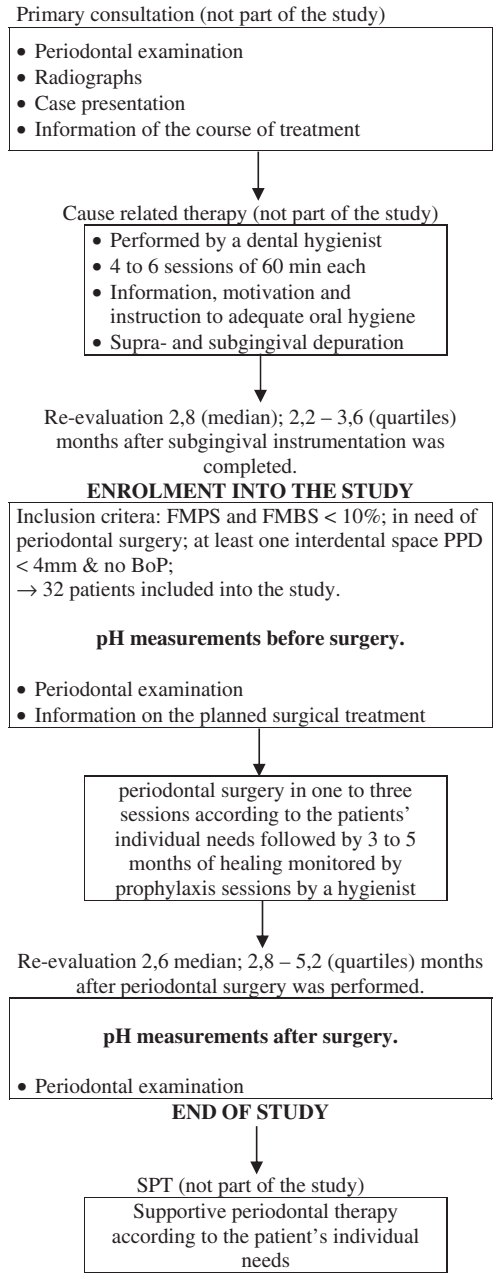


Figure 2. Flow-chart measurement occasions.

### Statistical analysis

All analyses were performed using a statistical software package analysis (IBM SPSS Statistics 25 New York, USA). 32 patient-based observations were available. The primary outcome was a changing pH value. Due to the nature of the data, median and quartiles were mostly used. For the description of some of the clinical characteristics, means and standard deviations were given. The data were grouped according to variables at the level of the interdental space and subject level respectively, and comparisons within the respective groups were performed between the pH before rinsing and the respective pH values at 2, 5 and 10 min after rinsing with sucrose solution. The data were arranged on a timeline and – except for the principal findings – the *p*-value for the difference between before rinsing and the respective time point after rinsing was given. Data on the level of the

interdental space and on the subject level were related to a changing pH value. The analyses of the individual pH values allow only for intraindividual comparisons within one measurement session. The only comparison feasible to perform of pH values taken at different time points or different interdental spaces is to see if the respective pH drop after rinsing was statistically significantly delayed or not.

Analytical statistics included dependent (Wilcoxon matched-pairs signed-rank test) and independent (cross-tabulation) tests using subject-based observations. A logistic multilevel regression model analysis was performed with the difference of pH before rinsing and pH at 2 min (pH lowering of >0 at 2 min) as a dependent variable in relation to variables at the level of the interdental space and at the subject level.

## Results

### Clinical characteristics

In the whole dentition, the percentage of remaining pocket probing depths (PPD) and dichotomous FMPS and FMBS decreased statistically significantly ($p < .05$ for FMPS; otherwise $p < .01$) between re-evaluations before and after surgery. After surgery, a significant ($p < .05$) reduction of PPD and BoP at both sites of the diseased interdental space could be achieved (Table 2).

### Principal findings

In the periodontally healthy interdental spaces, a statistically significant ($p < .01$) pH drop was seen 2 min after rinsing with sucrose; at 5 and 10 min the pH remained on this lowered level. In the periodontally diseased interdental spaces, a statistically significant ($p < .01$) lowering of the pH was seen first at 5 min after the sucrose rinse. This deviant pattern was no longer observed after surgery. Like for the healthy interdental spaces, the lowered pH remained on this level at 10 min (Table 3).

Table 2. Clinical description of the changes at patient and at site level within the diseased interdental space after cause-related therapy and periodontal surgery.

| Re-evaluation after … | … Cause-related therapy | … Periodontal surgery |
|---|---|---|
| FMPS | 6%, 3–9%[a] | 2%, 1–5% |
| FMBS | 7%, 5–12% | 5%, 2–8% |
| PPD 5 or 6 mm | 13%, 8–22% | 3%, 0–5% |
| PPD >6 mm | 3%, 1–5% | none |
| Diseased interdental spaces | | |
| 'diseased site' | | |
| BoP | 100% | 18.8% |
| PPD | 6.9 ± 1.1 mm[b] | 4.1 ± 1.1 mm |
| 'slightly diseased site' | | |
| BoP | 19% | no BoP |
| PPD | 3.6 ± 1.0 mm | 3.0 ± 0.7 mm |

[a]Median and quartiles; [b]mean and standard deviation.

### Level at the interdental space – impact of probing depth and bleeding on the pH

If – before surgery – either a PPD >6 mm or BoP was noted within the diseased interdental space, no pH drop was seen over the whole 10 min observation period. If – after surgery – either a PPD >4 mm or BoP was observed, a statistically significant pH drop was seen 5 min after rinsing with sucrose, but it was lost again in the group with PPD >4 mm 10 min after rinsing. When PPD was ≤6 or ≤4 mm, respectively, or no BoP was noted, the pH was significantly reduced already at 2 min both before and after surgery (Table 4).

### Subject level

A comparison of dentitions with either <10% or ≥10% of PPD ≥5 mm before surgery showed that the pH drop before surgery followed the pattern of the principal findings presurgery in both groups. After pocket elimination, five patients, still having ≥10% of PPD ≥5 mm, showed a pattern deviant of the principal findings post-surgery. Here, a statistically significant pH drop was not recorded before 5 min after rinsing with sucrose and lost again at 10 min after the rinse. This was found although both sites in the diseased interdental spaces used for the pH measurements had healed at the re-evaluation after surgery showing neither PPD >5 mm nor BoP in these five patients (Table 5).

A separate analysis (Pearson's Chi-square for cross-tabulation) showed that the distribution of PPD and BoP within the groups with <10% or ≥10% of PPD ≥5 mm did not differ significantly before and after pocket elimination.

An analysis of FMBS of ≥10% and of <10% did not disclose any differences with respect to pH lowering after sucrose rinsing. In both groups, a significant pH drop was reached at 5 min before and at 2 min after surgery (data not shown).

Data from diseased interdental spaces before surgery showed no statistically significant approximal pH lowering up to 10 min after rinsing with sucrose, in smokers and patients taking medications (Table 5).

At healthy and diseased interdental spaces, after pocket elimination, a statistically significant pH drop at 2 min and lowered pH of 10 min according to the principal findings were observed, irrespective of the patients' smoking habit or medications (data not shown).

Table 3. Approximal pH lowering after rinsing with sucrose before and after surgical pocket elimination; pH values (median; quartiles).

| | Status of interdental space | Before rinsing with sucrose | 2 min | 5 min | 10 min |
|---|---|---|---|---|---|
| After rinsing | | | | | |
| Before surgery | Diseased | 6.5; 5.9–7.0 | 6.1[a]; 5.3–6.5 | 5.7; 5.3–6.5 | 5.5; 5.3–6,4 |
| | Healthy | 6.5; 6.1–7.0 | 5.5; 5.0–6.1 | 5.5; 5.3–6.1 | 5.3; 5.2–6.1 |
| After surgery | Diseased | 6.3; 5.8–6.5 | 5.5; 5.3–6.1 | 5.5; 5.3–6.1 | 5.5; 5.3–5.8 |
| | Healthy | 6.5; 5.8–6.5 | 5.5; 5.3–6.0 | 5.5; 5,1–5.7 | 5.4; 5.1–5.8 |

[a]Not significant different from pH before rinsing ($p = .06$); all other pH values after rinsing were significantly different from the respective pH before rinsing ($p < .01$).

**Table 4.** Analyses at the level of the interdental space.

| | | | After rinsing | | |
|---|---|---|---|---|---|
| | | Before rinsing with sucrose | 2 min | 5 min | 10 min |
| | PPD | | | | |
| Before surgery | PPD > 6 mm ($n = 15$) | 6.5; 6.1–7.0 | 6.5; 5.8–6.5<br>$p = .683$ | 5.8; 5.5–6.5<br>$p = .102$ | 5.5; 5.3–6.5<br>$p = .055$ |
| | PPD ≤ 6 mm ($n = 17$) | 6.5; 5.8–6.9 | 5.5; 5.3–6.5<br>$p = .020$ | 5.5; 5.3–6.5<br>$p = .018$ | 5.3; 5.3–6.3<br>$p = .003$ |
| After surgery | PPD > 4 mm ($n = 9$) | 6.1; 5.8–6.5 | 5.5; 5.3–6.5<br>$p = .107$ | 5.5; 5.3–6.0<br>$p = .018$ | 5.5; 5.4–6.1<br>$p = .091$ |
| | PPD ≤ 4 mm ($n = 23$) | 6.5; 5.8–7.0 | 5.5; 5.3–6.1<br>$p = .000$ | 5.5; 5.3–6.1<br>$p = .001$ | 5.5; 5.3–5.8<br>$p = .000$ |
| | BoP | | | | |
| Before surgery | BoP[a] ($n = 6$) | 6.3; 5.8–6.6 | 6.8; 6.1–7.2<br>$p = .498$ | 6.5; 5.5–7.0<br>$p = .893$ | 6.0; 5.3–7.1<br>$p = .588$ |
| | No BoP[a] ($n = 26$) | 6.5; 6.0–7.0 | 5.8; 5.3–6.5<br>p = .008 | 5.5; 5.3–6.5<br>$p = .002$ | 5.5; 5.3–6.2<br>$p = .002$ |
| After surgery | BoP[b] ($n = 6$) | 6.2; 5.8–6.6 | 5.7; 5.2–6.2<br>$p = .058$ | 5.5; 4.9–5.8<br>$p = .027$ | 5.5; 5.2–5.9<br>$p = .042$ |
| | No BoP[b] ($n = 26$) | 6.3; 5.8–6.6 | 5.5; 5.3–6.2<br>$p = .000$ | 5.5; 5.3–6.1<br>$p = .000$ | 5.5; 5.3–5.9<br>$p = .000$ |

Approximal pH lowering after rinsing with sucrose before and after pocket elimination in relation to PPD (≤6 mm vs >6 mm before and ≤4 mm vs >4 mm after surgery) and BoP (at 'slightly diseased sites' before[a] and 'diseased sites' after surgery[b]); pH values (median; quartiles); *p*-values for the comparison of pH before rinsing and at 2, 5 and 10 min after rinsing.

**Table 5.** Analyses on subject level.

| | | | After rinsing | | |
|---|---|---|---|---|---|
| | | Before rinsing with sucrose | 2 min | 5 min | 10 min |
| | %PPD ≥ 5 mm | | | | |
| Before surgery | <10% ($n = 8$) | 6.5; 5.9–6.9 | 5.8; 5.3–6.5<br>$p = .075$ | 5.5; 5.3–6.1<br>$p = .012$ | 5.3; 5.3–5.7<br>$p = .012$ |
| | ≥10% ($n = 24$) | 6.5; 5.9–7.0 | 6.1; 5.4–6.5<br>$p = .161$ | 5.8; 5.5–6.5<br>$p = .049$ | 5.7; 5.3–6.9<br>$p = .025$ |
| After surgery | <10% ($n = 27$) | 6.5; 5.8–6.5 | 5.5; 5.3–6.1<br>$p = .000$ | 5.5; 5.3–6.1<br>$p = .000$ | 5.5; 5.3–5.8<br>$p = .000$ |
| | ≥10% ($n = 5$)a | 6.1; 5.7–6.8 | 5.5; 5.3–6.5<br>$p = .225$ | 5.5; 5.4–5.8<br>$p = .043$ | 5.5; 5.4–6.2<br>$p = .066$ |
| | Smoking/medication | | | | |
| Before surgery | Non-smoker ($n = 23$) | 6.5; 6.1–7.0 | 6.1; 5.3–7.0<br>$p = .176$ | 5.8; 5.3–6.5<br>$p = .023$ | 5.5; 5.3–6.5<br>$p = .011$ |
| | ≥10 cig ($n = 9$) | 6.5; 5.8–6.8 | 5.8; 5.5–6.5<br>$p = .106$ | 5.5; 5.4–6.2<br>$p = .106$ | 5.5; 5.3–6.3<br>$p = .046$ |
| Before surgery | No medication ($n = 17$) | 6.5; 6.1–6.7 | 5.5; 5.3–6.3<br>$p = .020$ | 5.5; 5.3–6.0<br>$p = .004$ | 5.3; 5.3–5.8<br>$p = .003$ |
| | Taking medication ($n = 15$) | 6.5; 5.8–7.0 | 6.5; 5.5–7.0<br>$p = .844$ | 6.5; 5.5–6.5<br>$p = .299$ | 5.8; 5.3–7.0<br>$p = .195$ |

Approximal pH lowering after rinsing with sucrose before and after surgery in the diseased interdental spaces in relation to the percentage of PPD ≥5 mm in the dentition, and before surgery in relation to smoking habits ≥10 cig/day and medications; pH values (median; quartiles); *p*-values for the comparison of pH before rinsing and at 2, 5 and 10 min after rinsing.
[a]Both sites in the diseased interdental spaces used for pH measurements in these five patients, had healed; neither PPD > 5 mm nor BoP were registered.

### *Logistic multilevel regression model*

A logistic multilevel regression model analysis (Table 6) showed a tendency that 2 min after rinsing with sucrose in dentition with ≥10% of PPD ≥5 mm the approximal pH was likely to remain unchanged or to increase ($p = .052$). At the level of the interdental space, greater probing pocket depths were significantly associated with stable or increasing pH 2 min after a sucrose rinse ($p = .032$).

**Table 6.** Results from data analysis of all observations using a logistic multilevel regression model analysis for association of factors on subject level and the level of the interdental space with pH lowering of > 0 at 2 min after rinsing with sucrose as dependent variable.

| | | pH lowering of > 0 at 2 min | | |
|---|---|---|---|---|
| Outcome | Estimate | *t*-Value | *p*-Value | 95% confidence interval |
| Intercept | 1.119 | 7.751 | .001 | 0.833 to 1.405 |
| Subject level | | | | |
| Smoking | −0.0115 | −0.123 | .903 | −0.203 to 0.180 |
| Medication | 0.0314 | 0.408 | .684 | −0.121 to 0.184 |
| ≥10% of PPD ≥5 mm | −0.800 | −1.996 | .052 | −1.607 to 0.0074 |
| FMBS ≥ 10% | 0.0324 | 0.370 | .712 | −0.141 to 0.206 |
| Level of interdental space | | | | |
| PPD | −0.0772 | −2.174 | .032 | −0.147 to −0.0068 |
| BoP | 0.0519 | 0.376 | .707 | −0.221 to 0.325 |

## Discussion

This study investigated the impact of periodontal inflammation on the approximal plaque pH. In our model of bacterial

challenge by a sucrose rinse, we compared a healthy with a periodontally diseased interdental space and the latter also before and after periodontal surgery. We found that a pH drop after the sugar rinse was delayed when the periodontium was infected.

Our model was based on early studies on acid production in dental plaque. The so-called Stephan Curve shows a drop of the approximal pH after a sucrose rinse reaching a minimum value at around 10 min after rinsing before rising again to just below the initial value at 60 min [21,22].

Several studies [5,8–10] but not all [11] reported that the pH of the GCF is somewhat higher than the one of the supragingival environment and rises with inflammation. Further on, an increased GCF flow during inflammation has been observed [7,23]. In naturally occurring gingivitis at sites showing slight, moderate or severe signs of inflammation as well as in experimental gingivitis, the GCF flow increases with increasing inflammation [6,7] and decreases during healing [5,24]. Numbers being reported for GCF flow in gingivitis were in the vicinity of those for untreated moderate periodontitis (just below 1 µl/30s) whereas a twice as high GCF flow was measured in cases of untreated severe periodontitis [25]. All patients in this study suffered from severe periodontitis and may, thus, fall into the category of patients with a high GCF flow possibly being responsible for an unchanging approximal pH after sugar intake.

In previous studies, we found components deriving from the GCF in dental pellicles on the enamel surface distant from the gingival margins [13,14]. Thus, it can be anticipated that the GCF reaches up to the dental plaque just below the approximal contact point neutralizing the acid formed by saccharolytic bacteria. The deeper the PPD the higher the GCF flow and, thus, the longer is the time period of neutralization. BoP as a further sign of inflammation is strongly correlated to deep PPD [26]. However, BoP was not significantly related to a delayed pH-drop in the present study.

The presence of pocket probing depths of $\geq$ 5 mm after surgery may in clinical practice often be considered acceptable given a severe status pre-surgery. A reduced though strictly speaking remaining inflammatory load in the dentition as a whole – irrespective of the status of the specific interdental space – delayed in our study the pH lowering at the specific interdental space. A significant impact of remaining pockets on the periodontal disease progression and tooth loss has previously been shown [27,28]. These present and previous observations may be seen as an early sign and late consequences, respectively, of a general shift of the pH balance towards an alkaline environment and an inflammatory stage [29].

The finding of a delayed pH drop in smokers corroborates recent data of a significantly higher GCF flow in smokers than in non-smokers [30]. However, even a number of contradictory data have been published on the GCF flow in smokers [31–33].

A pH drop failed to appear in patients who had to take medications from medical diagnoses. A number of general diseases have been shown to be associated with periodontal disease [34]. A general disease may increase the body's inflammatory burden, get the pH out of balance towards the alkaline range and, thus, thwart the pH drop at the specific interdental space [29].

The strengths of this study lie in the design and setting. A new approach to assess the status of periodontal inflammation using the model of bacterial stimulation with a sucrose rinse has been explored. The prospective nature of the study – albeit a short-term one – and the intraindividual comparisons allow founded conclusions to be drawn. The data of all post-surgery follow-up examinations were available. A multilevel regression model considered factors on the level of the interdental space and on the subject level. A single therapist and examiner ensures reproducibility of case selection and data collection, however, carries a potential risk of bias. Other limitations are the relatively small sample size, thus a sometimes-unequal distribution of patients in group comparisons and the lack of long-term follow-up. Larger studies with more participants will be needed to ensure more robust data regarding the detailed analyses.

In conclusion, the present study may suggest that the plaque pH value in an interdental space varies with the periodontal status. Further on, there are indications that the inflammatory status of the entire dentition may have an impact on the pH of a particular interdental space, regardless of the actual inflammatory status of this space. Measuring the pH is quick, completely non-invasive and causes no discomfort. Future research may further investigate the experimental model including a possible pH rise up to 1 h after sucrose rinse (i.e. the entire Stephan Curve) and the significance of single (i.e. no sucrose rinse) pH measurements at multiple interdental spaces in dentition in relation to the periodontal status to find a potential predictive value.

## Disclosure statement

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