INVESTIGATIVE REPORT

Distribution of Mucosal pH on the Bucca, Tongue, Lips and Palate

A Study in Healthy Volunteers and Patients with Lichen Planus, Behçet's Disease and Burning Mouth Syndrome

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Saliva coating all oral surfaces has a buffering capacity that neutralizes bacterial and cariogenic acids. The aim of our study was to determine the surface pH in different regions of the oral cavity in healthy volunteers and in patients with diseases affecting the oral mucosa. Oral pH was measured with a flat glass electrode on the anterior third of the ventral surface of the tongue, middle hard palate, buccal mucosa and inner lips in 32 healthy volunteers, 12 patients with Behçet's disease, 23 patients with oral lichen planus, and 11 patients with burning mouth syndrome. The present study showed that there was an uneven distribution of oral surface pH. The palate had a higher pH than most other sites in all groups, and in patients with lichen planus, the palate pH was higher than that in healthy controls. Those with dentures had lower pH values in the hard palate than dentate patients. The relatively high pH in the palate region in all patient groups as well as healthy volunteers needs to be further studied to clarify its mechanisms and clinical relevance. Key words: oral mucosal pH; hard palate.

(Accepted March 26, 2001.)

Acta Derm Venereol 2001; 81: 178-180.

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It is well known that saliva coating all oral surfaces has a buffering capacity which neutralizes bacterial and cariogenic acids, and has a pH range of 6.5-7.5 (1, 2). It is considered an important factor in preventing dental caries and encouraging mineralization (3). However, there is no literature pertaining to topographic measurements of oral pH in healthy controls or patients. Although the entire oral cavity is coated with saliva, there are different "ecological niches" at different locations within the oral cavity, in which the conditions may differ in such parameters as relative oxygen pressure, bacterial and fungal population, penetration of food and drink substances and exposure to thermal or chemical irritants. All these factors may affect the local pH of oral mucosa. The main compartments of the oral mucosa are buccal mucosa, palate, tongue, floor of mouth and lips. It is of interest to compare the pH values between these sites. The buccal mucosa, tongue and lips are prime targets in inflammatory diseases, such as Behçet's disease (BD), and lichen planus (LP). Burning mouth syndrome (BMS) is an ill understood condition in which symptoms may involve primarily the tongue, lips or, less frequently, the entire oral cavity. The etiology of this syndrome is yet unknown, but has been related to several factors among which are xerostomia, neurological and metabolic disorders, hormonal disturbances, psychological factors, and ill-fitting dentures (4). As changes in pH can cause irritation and affect neural receptors, it seems important to evaluate the oral pH in these patients as a possible factor that could explain the symptomatology.

The purpose of this study was twofold: (a) to measure the baseline oral mucosal pH in the hard palate, tongue, inner lips and buccal mucosa in healthy volunteers, and (b) to examine whether oral mucosal pH shows any abnormalities in LP, BD and BMS.

MATERIAL AND METHODS

The study group comprised 32 healthy adult volunteers, 12 patients with established BD according to the International Criteria for Classification of BD (5), 23 patients with biopsy-proven oral LP and 11 patients with BMS in which no abnormality was found in examination of the oral mucosa. The duration of symptoms was 6 months or more. All individuals with dentures had dentures in both the upper and lower jaw. Characteristics of the study groups are shown in Table I.

All subjects were requested to refrain from eating, drinking and brushing their teeth 12 h prior to the test. All measurements were performed between 8 a.m. and 12 a.m. Healthy controls did not use any medication. Ten of the patients with BD were on colchicine treatment. All patients with erosive oral LP were treated with topical corticosteroids (dexamethasone 0.01% solution, or clobetasol 0.5% ointment), and were asked to refrain from using the medication 12h prior to testing. Ulcerated areas in LP and BD were not tested.

Oral surface pH was measured with a flat, glass electrode pH meter (HANNA instruments HI 8424). Three measurements were taken at each site, in the hard palate, buccal mucosa, anterior third of ventral tongue, and lower lip mucosa.

The Kruskal–Wallis test was performed to compare the differences in oral surface pH at each site among the different groups; and also to compare the differences among the 4 sites within each group. In the event that a significant difference was found, a Mann–Whitney U test was carried out to detect the differences. A multiple regression analysis was performed to correlate age, gender and use of dentures to oral mucosal pH within each site. The threshold for significance was set at p < 0.05.

RESULTS

The palatal surface pH was significantly higher than pH in most other sites in both healthy controls and patients (Table II). In healthy controls, palatal pH was higher than that on the tongue (p = 0.008), bucca (p = 0.009) and lips (p = 0.006). In the patients with LP, the pH was higher on the palate than on the tongue (p < 0.001), bucca (p < 0.001) and

Table I. Characteristics of the study group

	Healthy controls $n = 32$	Oral lichen planus $n = 23$	Behçet's disease $n = 12$	Burning mouth syndrome $n = 11$
Age (mean ± SD)	46.3 ± 16.7	59 ± 12.3	43.7 ± 11.4	71.2 ± 5.8
Range	(23–83)	(38–79)	(28—63)	(58–77)
Men/Women	10/22	7/16	6/6	2/9
Dentures	6	9	3	9

Table II. Surface pH (mean ± SD) in tongue, bucca, palate and inner lip in healthy controls and patients with oral mucosal disease

Site	Healthy controls $n = 32$	Lichen planus $n = 23$	Behçet's disease $n = 12$	Burning mouth syndrome $n = 11$	Kruskal–Wallis test between the different groups
Tongue	6.65 ± 0.61	6.84 ± 0.51	6.31 ± 0.73	6.69 ± 0.41	n.s.
Bucca	6.68 ± 0.55	6.95 ± 0.58	6.60 ± 0.53	6.81 ± 0.62	n.s.
Palate	7.23 ± 0.88	8.14 ± 0.69	7.05 ± 0.71	7.27 ± 0.64	p < 0.001
Lip	6.61 ± 0.6	6.77 ± 0.47	_	6.46 ± 0.53	n.s.
Kruskal–Wallis test between different sites	p = 0.012	<i>p</i> < 0.001	p = 0.009	n.s.	

lips (p < 0.001). In patients with BD, the pH on the palate was higher than that on the tongue (p = 0.005) and bucca (p = 0.024), and in patients with BMS it was higher than the pH on the tongue (p = 0.047) and lips (p = 0.019).

Healthy males had a significantly lower palatal pH than females $(6.55 \pm 0.74 \text{ vs } 7.53 \pm 0.76; p = 0.002)$, but there were no gender differences in the patient groups. Age did not have any significant effect on oral pH in healthy controls or patients.

Oral pH and disease: palatal pH was significantly higher in LP than in healthy controls (p < 0.001). Oral pH in other sites in LP did not, however, differ significantly from that in healthy controls. There was a tendency toward lower pH in patients with BD compared with healthy controls. (The lip was not examined in patients with BD). Using multiple regression on oral pH, the LP group was found to have an average of 0.86 units higher pH compared with healthy controls (p < 0.001) with significant age and dentures effects (p = 0.03 and p = 0.015, respectively).

Differences between denture users and non-users were found only in the oral LP group, where patients with dentures had lower pH values in the palate (7.83 ± 0.68 vs 8.41 ± 0.62 ; p = 0.046 for non-users). In the healthy group and in patients with BMS, the mean pH values in the palate tended to be lower in denture users, but the differences did not reach statistical significance.

DISCUSSION

The results of this study clearly demonstrate that significant differences in pH exist between different oral sites in healthy controls and patients with LP, BD and BMS. The surface pH of the hard palate was significantly higher than that of both the bucca and tongue. As yet, the biological role of the higher pH of the hard palate is not clear. A high salivary secretion is associated with a high pH value (2), but gland secretions in the palate have the lowest flow rates (6), implying that saliva is not the cause of the high palatal pH. Recently, Christie et al. (7) showed that carbonic anhydrase, an important

buffering enzyme, is abundant in all oral epithelial cells including those of the oral palate. We therefore speculate that the high palatal pH may be attributable to an endogenous mucosal buffering system in the palate in addition to that of the salivary buffering system. Future studies are required to clarify this hypothesis.

Many of the oral mucosa diseases, such as aphthous ulcers, BD and LP, are not evenly distributed on all surfaces of the oral cavity, but have certain predilection sites. Therefore, we investigated the pH values in 4 different oral locations in both patients and controls. It appears that in BMS and BD, the disease process does not alter the oral pH in any of the sites. However, in LP, the mean pH value of the hard palate was significantly higher than that of healthy controls. The hard palate is not a major target organ in this disease; in fact, it is rarely involved. Lundstrom et al. (8) have found a low secretion of saliva in oral LP with changes in salivary pH. Currently, no satisfactory explanation can be given for these differences. A possible explanation could be the administration of oral topical corticosteroids to patients with LP. This explanation is not feasible, however, as measurements taken in sites other than the palate did not differ from those of healthy controls. Another explanation could be related to age differences between the study groups, since the mean age of patients with LP was higher than the control cases (59 \pm 12 vs 46 ± 16 years). However, age-related changes were not statistically significant in either controls or patients.

It remains to be determined in future studies what factors are responsible for the uneven pH distribution in the oral cavity, and especially the higher palatal pH.

Candidal infections commonly involve the hard palate, especially in patients with dentures, and it is well established that candidal organisms adhere to the denture surfaces (9). Several studies have found that oral *Candida* carriers have a lower salivary pH (9–10). Although the examined patients did not have clinical signs of candidiasis, the relatively lower palatal surface pH found in LP patients with dentures versus

LP patients without dentures supports the previous findings (9–10).

BMS is still an enigma to dentists and dermatologists, and many precipitating factors have been implied, including xerostomia, contact hypersensitivity, neuropathy, metabolic factors, hormonal disturbances, mineral deficiencies, psychological disturbances and ill-fitting dentures (4). Patients with BMS often describe a stinging-burning sensation as if they have scalded the mucosa, or a feeling of eating unripe fruit, in the absence of any detectable changes in mucosa. Changes in pH could have been one possible explanation. However, oral pH in patients with BMS did not differ significantly from that in the normal control group, indicating that pH changes are not involved in the etiology of BMS.

In conclusion, the present study has shown for the first time that there is an uneven distribution of oral mucosal surface pH. Palatal pH was significantly higher than the pH of other locations examined. In BD and BMS, pH was unaltered, but in LP, palatal pH was significantly higher than normal.

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