

Ulrich Schlagenhauf

## Significance of personal plaque control for therapy and prevention of periodontal disease – do established concepts still reflect current knowledge?



**Ulrich Schlagenhauf**  
University of Würzburg  
Clinic and Polyclinics  
of Dentistry,  
Department of  
Periodontology,  
Pleicherwall 2,  
97070 Würzburg,  
Germany  
Email: schlagenhauf@  
klinik.uni-wuerzburg.de

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Establishment of effective personal oral hygiene is a widely accepted prerequisite for therapy and prevention of periodontal disease. Long-term clinical studies have, however, to date not been able to deliver definitive proof of a preventive periodontal effect of good oral hygiene beyond gingivitis reduction. Data derived from more recent studies have fundamentally challenged the validity of the central role of oral hygiene in periodontal prevention. Contrary to former concepts, the driving force behind the development of pro-inflammatory dental plaques is not a lack of oral hygiene, but rather a malfunction or overreaction of the oral immune system. Good oral hygiene can only, to a very limited extent, alleviate the effects of such immunological deficiencies on the integrity of the periodontium, and it is therefore essential in periodontal patients to supplement it with regularly performed professional preventive measures.

### ■ Introduction

Establishment of effective personal oral hygiene is today still generally considered an essential prerequisite for the success of any kind of systematic periodontal therapy, and it is thus one of the few basic periodontal concepts which have survived the sometimes stark changes of paradigm in the treatment of periodontal disease that have taken place during the last few years. Supported by the results of a large number of experimental clinical studies conducted over four decades, there is to date still unity of opinion amongst nearly all who work in periodontology, that

effective personal oral hygiene with removal of bacterial plaques is an absolutely fundamental requirement for life-long dental health. 'Well cleaned teeth are not prone to disease' is, furthermore, the message dentists pass on to their patients, which suggests that reliable prevention of periodontal disease is not a scientific problem any more, but is merely being prevented by a lack of willingness in the population to implement personal oral hygiene.

A whole series of more recent study results have, however, called into question even the established concepts on the aetiological and preventive significance of personal oral hygiene. Due to the fact that

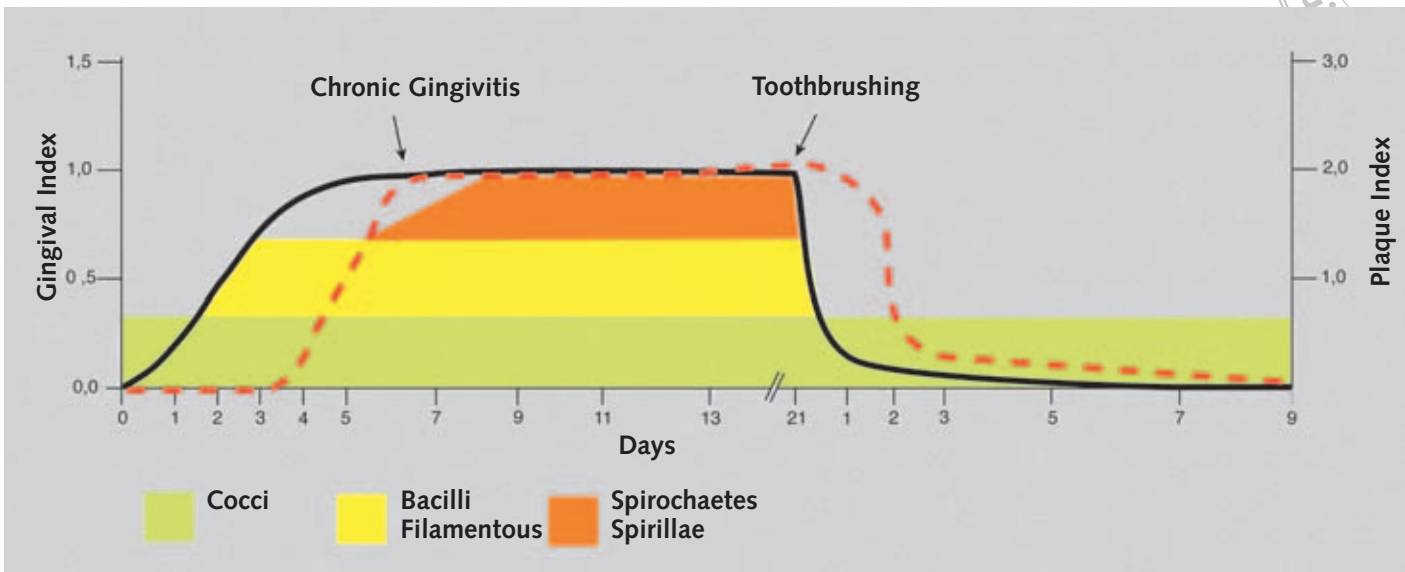


Fig 1 Diagram of gingivitis development upon absence of oral hygiene (according to Løe et al<sup>1</sup>).

professional preventive strategies, in which implementing effective oral hygiene plays a central role, are evidently crowned with clinical success, these new research findings have to this day been largely ignored.

The facts presently available concerning the scientifically proven benefit of personal plaque control are therefore presented in detail, and an attempt is made to discuss the resulting consequences for clinical practice.

■ **Personal oral hygiene – present state of knowledge**

Investigations conducted by Løe and coworkers<sup>1</sup> on the origin of gingivitis have shown that a lack of oral hygiene leads to the teeth being overgrown with bacterial plaques that in turn cause an inflammatory reaction in the surrounding gingiva. When the study participants again took up oral hygiene measures however, the chronic gingival inflammation could be eliminated rapidly and reliably, so that gingivitis was identified as a disease that can be reliably controlled by personal oral hygiene (Fig 1).

As early forms of periodontitis are clinically indistinguishable from chronic gingivitis, the idea that periodontitis can only develop on the basis of preceding gingivitis has for a long time been held almost as

a dogma of periodontology. If the periodontium can, by means of good oral hygiene, be kept free of gingivitis from early youth onward, development of periodontitis is, according to this theory, prevented. The validity of this assumption was tested in a clinical study conducted by Albandar et al<sup>2</sup>. In this study, 16 Brazilian schoolchildren were randomly assigned either to a test group or to one of two control groups. The subjects in the test group were given detailed instructions on how to keep their teeth and gums free of plaque and gingivitis and on the significance of oral hygiene for long-term oral health. The outcome of personal oral hygiene was monitored on a regular basis and continuing oral hygiene deficits were successively eliminated by continued instruction, until the test group showed optimal conditions of freedom from plaque and gingivitis. The subjects in the first control group were given identical instructions, the practical day to day implementation was, however, not monitored further. The second control group did not receive any information or instructions. The main outcome parameter of the study was alveolar crest height that was assessed by means of bitewing radiographs in each subject at the start of the study and subsequently at yearly intervals.

Analysis of the radiological findings at the end of the study, which had been conducted over a period of 3 years, yielded a rather sobering result. In all three trial groups, subjects were observed who at the age



of 16 to 19 years already showed significant loss of alveolar crest height as a sign of progressive periodontitis. Comparison of the three groups with respect to loss of bone revealed no significant differences among the test group that had received intensive supervision and the control groups that had not been supervised. This showed that even the best oral hygiene, achieving optimal conditions of freedom from plaque and gingivitis, had no significant influence on the likelihood of early occurrence of periodontitis in the study population under investigation.

These data are supported by longitudinal observations conducted by Hugoson et al<sup>3</sup> in Swedish patients. Thus, the subjects' quality of oral hygiene during the investigation period of 20 years did have a significant impact on the development of gingivitis; the presence or absence of gingivitis did not, however, significantly influence the frequency of periodontitis.

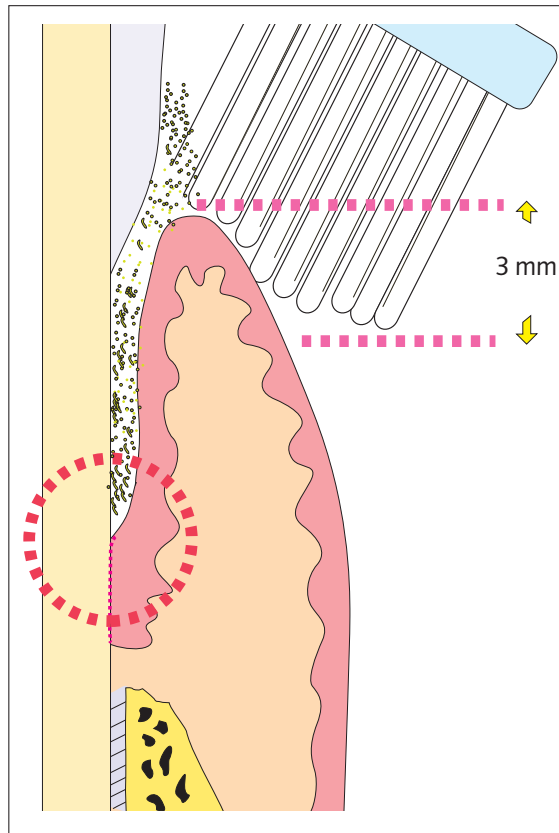
In another longitudinal study conducted by the same group<sup>4</sup>, patients who had shown an extraordinarily high standard of oral hygiene over a period of more than 20 years were analysed. Even among these subjects, all of whom maintained an intensive level of care, 80% suffered minor but significant losses of alveolar crest height during the period under investigation; 17% of the investigated subjects even showed marked progressive periodontitis. This corresponds to the frequency of severe periodontal disease observed in populations in developing countries who lack the means necessary for personal oral hygiene and do not have access to professional dentistry services<sup>5</sup>.

A study conducted by Merchant et al<sup>6</sup> on periodontal health in American dentists and dental assistants yielded equally surprising results. It could be shown that this group enjoyed significantly better periodontal health than age-correlated controls who did not practise a profession in the dental field. Quality and frequency of oral hygiene were also above average compared to the controls: 70% cleaned their teeth at least twice a day. More than half used dental floss on a daily basis. Nevertheless, a significant correlation between periodontal status and quality of oral hygiene could not be demonstrated. Dentists and dental assistants who reported cleaning their teeth less than once a day and who never used dental floss did not, on average, exhibit inferior peri-

odontal health compared with their colleagues who cleaned their teeth intensively. The dentists' superior periodontal status in comparison with the control group could only be explained in terms of significantly less common systemic periodontal risk factors amongst the subjects in the test group such as, for instance, smoking.

Do these results, which indicate only moderate or less effectiveness of oral hygiene in preventing periodontal disease, not diametrically oppose those of well-known clinical studies conducted in the 1980s, such as, for example, those of Axelsson and Lindhe<sup>7</sup>? Did these studies not prove beyond a doubt that establishment of effective oral hygiene, together with professional dental cleaning on a regular basis, effectively diminished the progression of periodontal disease? The seeming contradiction can however be explained in terms of the obvious differences in study design. In practically all successful periodontal preventive studies, the patients were not only trained in effective personal oral hygiene but were also subjected to a scheme of regularly performed professional dental cleaning. The preventive value of improved oral hygiene by itself cannot in such cases be distinguished from the preventive effectiveness of professional subgingival cleaning. In a study designed to clarify this question, Bakdash<sup>8</sup> demonstrated in a collective of periodontally compromised patients who took part at three-monthly intervals in a professional recall scheme, that the quality of oral hygiene had no significant influence on the likelihood of recurrence of periodontal disease within the 26-month investigation period. In other words, patients without plaque or gingivitis who regularly underwent professional subgingival cleaning of deep periodontal pockets had the same likelihood of suffering disease progression as those who did not carry out effective oral hygiene between recall sessions. A study conducted by Ramfjord et al<sup>9</sup> in 1982 further supports these findings. A relationship between quality of personal oral hygiene and long-term success of periodontal therapy in a collection of recall patients observed over a period of 8 years could not be demonstrated in this study either.

A possible explanation for this phenomenon could be that effective removal of bacterial biofilms out of the depth of the gingival sulcus is possible only to a very limited extent, if at all, with personal



**Fig 2** Depth of penetration of personal oral hygiene into the gingival sulcus. The circle marks the zone of plaque growth critical for development of periodontitis.

oral hygiene and even with professional dental cleaning, if this is limited to the supragingival dental surface (Fig 2). Only these deep biofilms, however, due to their closeness to the junctional epithelium, are responsible for progressive, irreversible destruction of the dental supportive tissue. This theory is supported by clinical studies that demonstrate a significantly diminished periodontal preventive effect for exclusively supragingival professional dental cleaning compared with professional cleaning that included the subgingival root surfaces. A patient with obviously deficient oral hygiene, but who regularly undergoes efficient subgingival cleaning of diseased periodontal pockets by a dentist or dental hygiene professional, will therefore as a rule be less in danger of suffering disease progression than a periodontal patient with excellent personal oral hygiene who, after completing systematic periodontal therapy, either does not undergo professional dental cleaning at all, or where only the supragingival surfaces are professionally cleaned (Fig 3).



**Fig 3** 24-year-old female patient with excellent oral hygiene. Pus drains from a 9 mm deep gingival pocket.

### ■ Possible explanations

Is it then pointless from the point of view of periodontal prevention to optimise personal oral hygiene in periodontal patients? Certainly not – but it is high time to take leave of another simplistic dental truth: clinically plaque free is not automatically healthy, and aggressiveness of periodontal disease is equally not in proportion to the thickness of the remaining plaque layer. In order to understand this, it is advisable to take a look at the presently accepted model of how periodontal disease develops<sup>10</sup>.

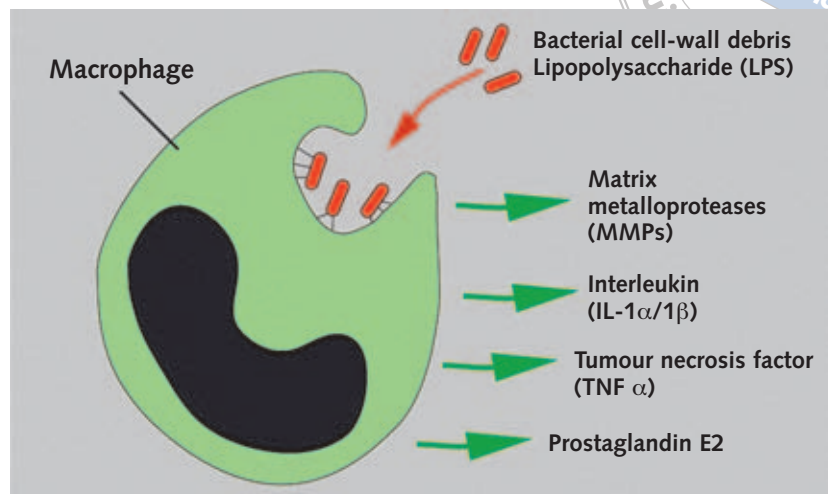
Bacteria, bacterial fragments and products of bacterial metabolism are all physiological constituents of the oral cavity. Nature has, in the course of evolution, developed a whole array of mechanisms to effectively limit growth of microorganisms and the inflammatory conditions to which they give rise. The growth of bacterial biofilms in the vicinity of the gingival sulcus is decisively kept in check by migrant neutrophil granulocytes, antibodies in the sulcus fluid and other natural local defense mechanisms. If bacteria or bacterial fragments still reach the connective tissue underneath the protective epithelium, they give rise to localised inflammation which leads to a partial dissolution of connective tissue structures and elimination of infiltrated bacterial antigens by immune cells (Fig 4).

In most humans, penetration of small amounts of bacterial antigens into the gingival connective tissue leads to gingivitis with reversible consequences. In



patients with marked risk of periodontal disease, on the other hand, penetration of bacterial antigens into the connective tissue often induces an exaggerated inflammatory response that is out of proportion to the bacterial threat, and in which a whole number of tissue dissolving inflammatory mediators are released. If the inflammation intensity exceeds a certain threshold, this leads to small-area but nevertheless irreversible destruction of the periodontium. Many minor periodontal inflammatory events add up in the course of time and subsequently lead to clinically visible damage. Aggressiveness of periodontal disease is, by this aetiological model, determined by the frequency of inflammatory episodes that lead to irreversible damage. The exaggerated inflammation may be hereditary, but it is often decisively modified by external factors such as smoking and emotional stress, or systemic illness. Inflammation intensity is also modified by specific virulence of periodontally pathogenic microorganisms. These microorganisms are, however, not in every case pathogenic. In some instances, periodontal patients pass them on via smear infection to their periodontally healthy partners, without them, as a rule, contracting clinically significant periodontal disease<sup>11</sup>.

The relationship between bacterial plaque and gingival inflammation is very often misunderstood. It is by no means the case that every person who neglects personal oral hygiene develops marked gingivitis within a short period of time, as the classic study conducted by Løe et al<sup>1</sup> on the development of gingivitis would suggest. Brex et al<sup>12</sup> discovered substantial individual differences in a histological clinical study on subjects who had abstained from personal oral hygiene for a period of six months. While some study participants showed rapid plaque growth and quickly developed clinically visible gingivitis, fully-fledged gingivitis could not be histologically determined in others even after six months without dental cleaning (Fig 5). This in turn shows that the intensity of inflammation is not primarily determined by the developing bacterial plaque, but rather is directly dependent on systemically determined factors which influence the oral immune system. A recent study conducted by Rowshani et al<sup>13</sup> supports this explanatory model. They observed that six months after discontinuing personal oral hygiene, development of bacterial plaque was significantly slower



**Fig 4** Release of cytokines and tissue-dissolving enzymes from a macrophage upon contact with cell wall debris of Gram-negative bacteria.

and less marked in periodontally healthy subjects than in subjects with periodontal disease. Success of periodontal therapy could even be measured in terms of the rate of formation of bacterial dental plaques. Patients who had completed systematic periodontal therapy developed significantly less dental plaque upon subsequent lack of personal oral hygiene than before the start of periodontal therapy. The reason for this is that the protein rich inflammatory fluid present in the gingival pocket and sulcus is the primary source of nutrition for bacteria. If the flow of sulcus fluid is greatly reduced due to elimination of the inflammation, the microorganisms populating the pocket lose an important part of their primary source of nourishment and their reproductive capacity is greatly diminished (Figs 6 and 7).

Quantitative reduction of dental plaque therefore is not necessarily due to improved implementation of oral hygiene on the part of the patient. There are probably many more people with only rudimentary oral hygiene in Europe than hitherto assumed. In the majority of people who have an intact oral immune system, however, this most often merely leads to clinically unspectacular dental plaques and comparatively moderate gingivitis. Only people with a malfunctioning, hyper-reactive immune system, resulting in strongly increased, protein-rich sulcus fluid, show the well-known and at times impressive plaque quantities and inflammation symptoms when oral hygiene is insufficient. However, complying with the demand for the establishment of good oral hygiene

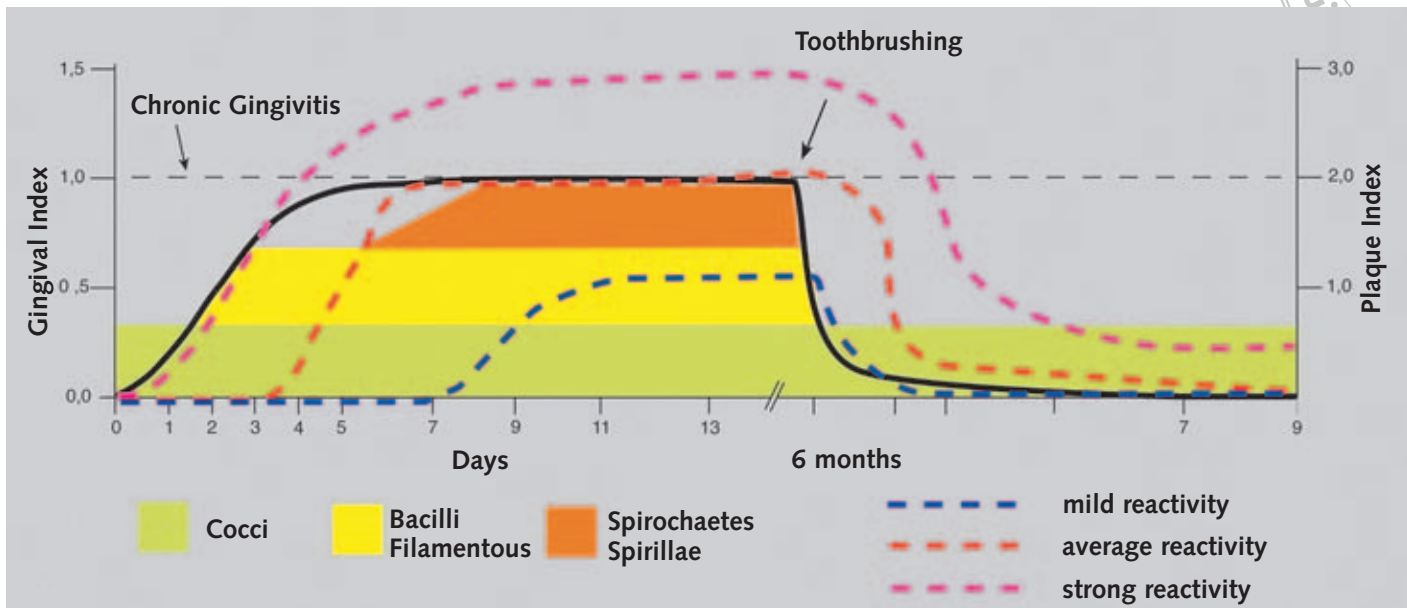
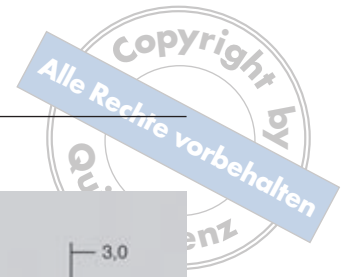


Fig 5 Diagram of gingivitis development upon absence of oral hygiene with respect to individual reactivity of the subject (according to Brex et al<sup>12</sup>).

as the essential primary measure eliminates only the clinically visible symptoms of the problem in this patient group, without significantly influencing the actual causes. The alveolar bone losses observed in spite of excellent oral hygiene in the study conducted by Albandar et al<sup>2</sup> give clear proof of this.

Optimal personal oral hygiene is therefore not an alternative to integrating periodontally compromised patients into a professional follow-up scheme and specifically eliminating avoidable systemic risk factors (smoking, stress, diabetes mellitus, etc.).

Overall it must be stated that the validity of many clinical studies on the effectiveness of mechanical plaque removal in preventing periodontitis is by now questionable on account of the facts presented. Due to study designs in which individual speeds of plaque growth and development of gingivitis in the absence of personal oral hygiene was not assessed, subjects with a higher inflammatory propensity and extensive dental plaque were often compared with subjects for whom it was no problem to remain free of plaque and gingivitis even with only moderate oral hygiene.

Refusal to grant systematic periodontal therapy on grounds of inadequate oral hygiene, as was, for example, again laid down in the German health insurance guidelines which have only recently been revised, thus tends above all to leave affected patients

with high inflammatory activity untreated. Furthermore, this has no proven scientific foundation, especially in young patients with aggressive periodontitis. Limiting access to systematic periodontal therapy was, for economic reasons, evidently considered essential by the financing institutions. Such refusal to grant treatment on grounds of assumed lack of long-term therapeutic effectiveness would however be more appropriate for active smokers or especially for people with whom regular attendance of recall sessions is questionable. As lacking motivation to optimise oral hygiene and lacking reliability in complying with professional preventive measures often have the same psycho-social causes, the quality of personal oral hygiene is in practice, at least in young, physically non-handicapped patients, quite a useful predictor of future compliance.

### Consequences

What are the consequences for clinical practice that follow from presently available data?

1. Periodontitis is not a disease that can be attributed primarily to insufficient oral hygiene. Refusal to administer treatment solely on the grounds of insufficient oral hygiene is therefore unjustified from a medical point of view.



**Fig 6** Untreated periodontitis; poor oral hygiene; strong plaque formation; distinctly visible, marked inflammatory reaction.



**Fig 7** View following successful periodontal therapy; poor oral hygiene; extensive areas covered with plaque; only minor or even absent gingival inflammatory reaction.



**Fig 8** Extensive supragingival calculus rendering cleaning of approximal dental surfaces impossible.



**Fig 9** Ciclosporin-A-induced gingival hyperplasia upon insufficient personal oral hygiene.

2. Rapid development of bacterial dental plaque in the gingival sulcus paired with rapidly developing gingivitis is an indicator of a high level of periodontal inflammatory activity and an urgent need for professional care in a patient.
3. Even with optimal oral hygiene, life-long professional aftercare is essential for periodontally compromised patients.

According to present understanding, even though insufficient oral hygiene is not aetiologically related to development of periodontal disease, optimising personal oral hygiene in addition to professional periodontal maintenance therapy will nevertheless continue to be worthwhile in the future for various reasons:

1. Persistent dental plaque in the vicinity of the efferent ducts of the large salivary glands often leads to rapid formation of supragingival tartar. This in

turn renders effective dental cleaning more difficult and hampers natural self-cleansing mechanisms (Fig 8).

2. Only in a gingivitis-free oral cavity can bleeding on probing (BOP) be assessed without disturbance. In spite of all its limitations, BOP is still the most important clinically usable parameter to assess periodontal stability in a patient<sup>14</sup>. When, due to poor oral hygiene, the mouth is affected by gingivitis, it is often not possible anymore to distinguish mostly harmless gingival bleeding from potentially critical bleeding out of the depths of a pocket upon probing.
3. Chronic gingivitis due to insufficient oral hygiene can induce extensive gingival hyperplasia upon intake of medication such as calcium channel blockers (e.g. nifedipin), ciclosporin A or hydantoin preparations. These can be surgically removed.



**Fig 10** Inflammation-free, previously periodontally damaged teeth with plaque-covered interdental retention recesses.

However, recurrence of the disease is almost unavoidable if conditions of insufficient oral hygiene persist (Fig 9).

4. Patients who have already suffered periodontal damage often have exposed root surfaces and interdental retention recesses that are especially prone to caries (Fig 10). Careful cleaning of these recesses from impacted food residues using fluoride toothpaste is therefore in most cases essential in order to prevent dental root caries. This should be supplemented by professional application of fluoride to the areas at risk during follow-up sessions.

## References:

1. Løe H, Theilade E, Jensen SB. Experimental gingivitis in man. *J Periodontol* 1965;36:177–187.
2. Albandar JM, Buischi YA, Oliveira LB, Axelsson P. Lack of effect of oral hygiene training on periodontal disease progression over 3 years in adolescents. *J Periodontol* 1995;66:255–260.
3. Hugoson A, Norderyd O, Slotte C, Thorstenson H. Distribution of periodontal disease in a Swedish adult population 1973, 1983 and 1993. *J Clin Periodontol* 1998;25: 542–548.
4. Hugoson A, Laurell L. A prospective longitudinal study on periodontal bone height changes in a Swedish population. *J Clin Periodontol* 2000;27:665–674.
5. Neely AL, Holford TR, Loe H, Anerud A, Boysen H. The natural history of periodontal disease in man. Risk factors for progression of attachment loss in individuals receiving no oral health care. *J Periodontol* 2001;72:1006–1015.
6. Merchant A, Pitiphat W, Douglass CW, Crohin C, Joshupura K. Oral hygiene practices and periodontitis in health care professionals. *J Periodontol* 2002;73:531–535.
7. Axelsson P, Lindhe J. Effect of controlled oral hygiene procedures on caries and periodontal disease in adults. Results after 6 years. *J Clin Periodontol* 1981;8:239–248.
8. Bakdash B. Oral hygiene and compliance as risk factors in periodontitis. *J Periodontol* 1994;65:539–544.
9. Ramfjord SP, Morrison EC, Burgett FG, Nissle RR, Shick RA, Zann GJ, Knowles JW. Oral hygiene and maintenance of periodontal support. *J Periodontol* 1982;53:26–30.
10. Page RC. Milestones in periodontal research and the remaining critical issues. *J Periodont Res* 1999;34:331–339.
11. Van der Velden U, van Winkelhoff AJ, Abbas F, Arief EM, Timmerman MF, van der Weijden GA, Winkel EG. Longitudinal evaluation of the development of periodontal destruction in spouses. *J Clin Periodontol* 1996;23:1014–1019.
12. Brex MC, Fröhlicher I, Gehr P, Lang NP. Stereological observations on long-term experimental gingivitis in man. *J Clin Periodontol* 1988;15:621–627.
13. Rowshani B, Timmerman MF, van der Velden U. Plaque development in relation to the periodontal condition and bacterial load of the saliva. *J Clin Periodontol* 2004;31:214–218.
14. Lang N, Adler R, Joss A, Nyman S. Absence of bleeding on probing. An indicator for periodontal stability. *J Clin Periodontol* 1990;17:714–721.