

# Professional implant management

## A balance between thorough but gentle cleaning

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**The treatment of** peri-implant disease remains a great challenge for the practising dentist. In spite of current guidelines, a direct therapy recommendation for treating diseased implants is still lacking. Owing to demographic change and the wide range of indications for implants, peri-implant disease is becoming an increasingly relevant problem in everyday practice. Since peri-implantitis is an irreversible disease that can lead to pain, severe aesthetic impairments and implant loss, it is necessary to adequately care for implants and treat the first signs of peri-implant inflammation at an early stage.

### On peri-implantitis and how it can occur

Peri-implant health and disease were classified in the context of periodontal and peri-implant diseases and conditions at the joint World Workshop of the American Academy of Periodontology and the European Federation of Periodontology in 2017 for the first time.<sup>1</sup> Table 1 provides an overview of the case definition of peri-implant health and peri-implant disease. Peri-implant health, on the one hand, is clinically defined as the absence of signs of inflammation such as erythema, bleeding on probing, swelling and suppuration. Peri-implant diseases, on the other hand, are classified as biofilm-associated diseases that are clinically conspicuous by inflammatory changes in peri-implant soft tissue accompanied by bleeding on probing and/or suppuration.<sup>2</sup> Compared with measurements at the time of insertion of the superstructure (baseline), which are caused by progressive bone loss that goes beyond the initial remodelling, peri-implantitis shows increased probing depth.<sup>3</sup> Given the lack of radiographs and probing depth measurements at baseline (directly after superstructure insertion), radiographic ev-

idence of a bone level of  $\geq 3$ mm and/or a probing depth of  $\geq 6$ mm connected with heavy bleeding and/or suppuration after probing are sufficient for the diagnosis of peri-implantitis. In contrast, peri-implant mucositis does not involve any decrease of the crestal bone level beyond the initial remodelling after insertion of the implant.

Similar to periodontitis, which is almost always preceded by chronic gingivitis, peri-implant mucositis exists before peri-implantitis arises. As mentioned earlier, this is marked by signs of inflammation, but does not yet involve bone resorption. Peri-implant mucositis is strongly associated with biofilm, which makes it—fortunately—reversible by adequate biofilm management. The transition to peri-implantitis is fluid and cannot be diagnosed clearly, and this must be taken into account when selecting the treatment approach. The cause of the progression of peri-implant mucositis to peri-implantitis has not been identified yet,<sup>1</sup> but the risk factors described later certainly play a role. If no elaborated therapy for peri-implantitis is provided, rapid, often non-linear progression of bone resorption and inflammation occurs,<sup>4</sup> presumably with faster spread and higher prevalence than in periodontitis. Peri-implantitis can already occur at the beginning of the maintenance phase, even shortly after the implantation. Noticeable problems can be expected after five years, and 20% of patients require peri-implantitis therapy after five to ten years.<sup>5</sup> Some experts report the start of the disease two to three years after implantation.<sup>6</sup>

### Risk factors for peri-implantitis

The aetiology of peri-implantitis is comparable to that of periodontitis. Both are multifactorial events that are modi-

	Peri-implant health	Peri-implant mucositis	Peri-implantitis
BOP and/or suppuration with gentle probing (possibly increased PD compared with baseline)	–	+	+
Bone loss	–	–	+

**Table 1:** Case definition of peri-implant health and disease according to the new classification.<sup>1</sup>

fied by co-factors multiple times. Bacterial (plaque) biofilm accumulation, which causes an initial immune response (inflammation), can be seen as the main cause. It is directly related to the oral hygiene of the patient. It is crucial to avoid restorations with difficult-to-clean niches—especially in older patients—which requires a close cooperation between dentist and dental technician.<sup>7</sup> Poor cleanability of the implant and its superstructure and thus biofilm accumulation as well as cement residue are termed as local modifying factors.

Patients who already have a severe form of periodontitis prior to implant placement, have poor biofilm control and are not integrated into a regular aftercare system (supportive periodontal therapy) can be classified as a high-risk group.<sup>8</sup> Patients with periodontitis have been shown to have a significantly higher rate of peri-implantitis occurrence within ten years (28.6% vs 5.8%) and thus a significantly lower success rate (71.4% vs 94.5%).<sup>9</sup> Therefore, healthy periodontal conditions through systematic periodontitis therapy and a high-frequency recall system must be guaranteed, even before implant placement.<sup>10</sup> In other words, only if both conditions are met is the patient ready for implants. Reducing the accumulation of bacteria immediately prior to implant placement is recommended, for example mucosal antiseptics with chlorhexidine rinsing solution. Subsequently, wound healing must be optimised.<sup>11</sup> In addition, smoking cessation should take place before implant placement.<sup>8,12</sup> The development of peri-implantitis has thus far been considered to be particularly favoured by the combination of pre-existing periodontal disease and smoking.<sup>5,13–15</sup> Diabetes mellitus and interleukin-1 polymorphism, especially, have been systemic risk factors so far.<sup>8,16–18</sup> A recent paper evaluates excess cement as a potential risk factor/indicator, but states that data identifying “smoking” and “diabetes” as risk factors are so far inconclusive.<sup>4</sup>

## Differences in the inflammatory response

Whether the bacterial spectrum in peri-implantitis is different from that in periodontitis, which would also result in a slightly different immune response, is matter of much discussion. Implants of titanium or ceramic have a biocompatible surface, but no biological surface. For osseointegration, they should have a large-volume, sponge-like surface. However, these surfaces, if they are exposed or become accessible to bacteria, offer perfect conditions for bacterial proliferation. A Swiss research group compared the inflammatory reaction to 21 days of plaque accumulation on the tooth and implant in an experimental gingivitis/mucositis model using plaque and bleeding indices and inflammatory markers.<sup>19</sup> While no significant differences in plaque index between tooth and implant were revealed, significant differences were found for the gingival index and inflammatory markers (active matrix metalloproteinase-8 and interleukin-1 $\beta$ ). Both were sig-



**Fig. 1:** Interdental brushes have to be selected individually—even for implants. The fitting should be part of the instruction during prophylaxis sessions.

nificantly higher for implants than for teeth despite very similar plaque accumulation. This is probably due to the lack of a periodontal ligament on implants.<sup>19</sup>

## On peri-implantitis prophylaxis

The most important pillar should be the avoidance of peri-implant disease. Problematically, just as with periodontitis, peri-implant disease is rarely conspicuous at the initial inspection, is largely painless and shows few symptoms. For this reason, the patient is not able to make a self-diagnosis, which often leads to a delayed diagnosis and, in particular, a significantly late start of therapy. The irreversibility of tissue loss explains the poor prognosis. For this reason, dentists and prophylaxis staff must prioritise precaution, that is, optimum maintenance care of the inserted implant and its superstructure. Prophylaxis for the implant does not only mean prophylaxis sessions every three to six months but also optimal instruction and motivation for good oral hygiene at home for the whole year.<sup>12</sup>

### Home care prophylaxis measures

Motivating patients by staining the teeth with a plaque disclosing agent is a proven method. This enables the dentist to specifically show the patient where an improvement in home biofilm management is necessary. The use of interdental brushes and the explanation of their application should be strongly recommended here (Fig. 1). Alternatively, soft picks are offered on the market. These are quite practical and usually cheaper, but the scientific data for an equivalence to interdental brushes is not yet available. In addition to mechanical biofilm control at home, chemical biofilm management can support measurements—especially for patients who cannot perform adequate cleaning of their implants.<sup>20</sup> This S3 level guideline<sup>20</sup> on “home care, chemical biofilm management” highlighted patients with implants and implant-supported dentures as those with a particularly high risk of inflammatory changes (gingivitis or mucositis). While 0.1–0.2% chlorhexidine digluconate solutions are recommended



**Fig. 2:** Powder air polishing systems with low-abrasive powder (glycine and erythritol powder) clean gently.

for short-term (14-day) intensive bacterial reduction and therewith reduction of an acute inflammatory event, mouthrinses containing 0.06% chlorhexidine, a special formulation of essential oils, a formulation with amine fluoride or stannous fluoride, or a formulation with cetylpyridinium chloride can support insufficient mechanical oral hygiene for a variety of reasons. For implants, the specific application of a 1% chlorhexidine gel is also suitable. Regular professional mechanical biofilm removal by trained persons as well as an improvement of biofilm management at home are the basis for the success of the therapy, both for prevention and in the case of already existing peri-implantitis.<sup>7, 17, 21–23</sup>

#### Professional prophylactic measures

In addition to these prophylactic measures, the practitioner must identify the systemic and local risk factors already mentioned and at least provide the impetus to remove them, which should be done before implant placement if possible.<sup>8, 17</sup> In order to confirm success, but also to be able to recognise the necessity of further therapy measures, regular check-ups including measurements are also indispensable for the dentist throughout the patient's life. Measurements, supragingival and, where necessary, subgingival cleaning (scaling and root planing) should be performed up to four times a year and should be carried out at regular intervals. Checking the complete periodontal status is recommended at least once a year in the case of six-monthly follow-up intervals and at least twice a year in the case of three-monthly intervals.

#### Designing supportive peri-implant therapy

Good oral hygiene of the patient as well as regular, life-long maintenance care sessions at intervals of three to six months are the key to long-term success. The regular recording of findings in order to determine both the oral hygiene status and the attachment level to implants and to diagnose changes at an early stage are the basis for this. Part of each session of supportive peri-implant therapy should include supragingival measures as well as regular motivation and instruction of the patient on good home biofilm management. An essential part of these maintenance sessions should, if necessary, be devoted to subgingival instrumentation of the implants. Necessary

cleaning must not be omitted owing to fears of possible surface damage. A compromise must be found between protecting the implant by gentle instrumentation to avoid deep scratches on its surface and thorough cleaning. Rough implant surfaces show not only more biofilm but also a more pathogenic flora, whereas surfaces that are too smooth disrupt soft-tissue attachment and fibroblast attachment. Hence, a good balance between bacterial adhesion and soft-tissue adhesion must be found.<sup>23</sup> The practitioner has various therapy options for subgingival cleaning. Recently, Schmidt et al. conducted a series of studies to examine cleaning options for their balance between bacterial adhesion and soft-tissue adhesion.<sup>24–26</sup> The following conclusions were drawn:

- a) If curetting is necessary (i.e. radiographically visible deposits), titanium curettes should be used instead of the conventional steel curettes, as they are much gentler on titanium surfaces.
- b) Ultrasonic instruments with a plastic coating hardly change the surface roughness, but should be reserved for the removal of hard deposits.
- c) Air-powder prophylaxis units with low-abrasive powder (glycine and erythritol powder; air polishing) are ideal for biofilm removal. At probing depths of up to 5 mm, it is even possible to blast into the sulci. At higher probing depths, nozzle attachments should be used (Fig. 2). The spray jet of the nozzles is deflected laterally so that it does not radiate apically and the risk of emphysema formation is avoided.

The mentioned approaches (titanium curettes, ultrasonic instruments, air polishing with low-abrasive powder) are gentle on the implant surfaces, show good clinical results and do not differ significantly from each other. Steel curettes lead to greater surface roughness and should therefore be avoided.<sup>24–26</sup> Considering teeth, the clinical and microbiological results of subgingival air polishing for moderately deep pockets are similar to those of ultrasonic treatment.<sup>27</sup> Compared with conventional scaling and root planing, subgingival air polishing actually performs better in terms of its effectiveness in subgingival biofilm removal.<sup>28</sup> Good results for subgingival therapy with air polishing have also been demonstrated for implants with peri-implant disease.<sup>8</sup>

The effectiveness of hand instruments, adjuvant air polishing (glycine powder) and ultrasonic scalers has been proved by clinical studies on implants with a significant improvement in clinical parameters (especially bleeding on probing).<sup>29, 30</sup> The elimination of inflammatory signs should be the primary goal of all procedures.<sup>17, 31</sup> In addition to cleaning, an individual risk analysis and, if possible, the elimination of risks must also be part of maintenance care if they have developed after implant placement. This includes advice on quitting smoking but also an exchange with the attending physician or internist to optimise the control of any diabetes that may be present. Subgingival

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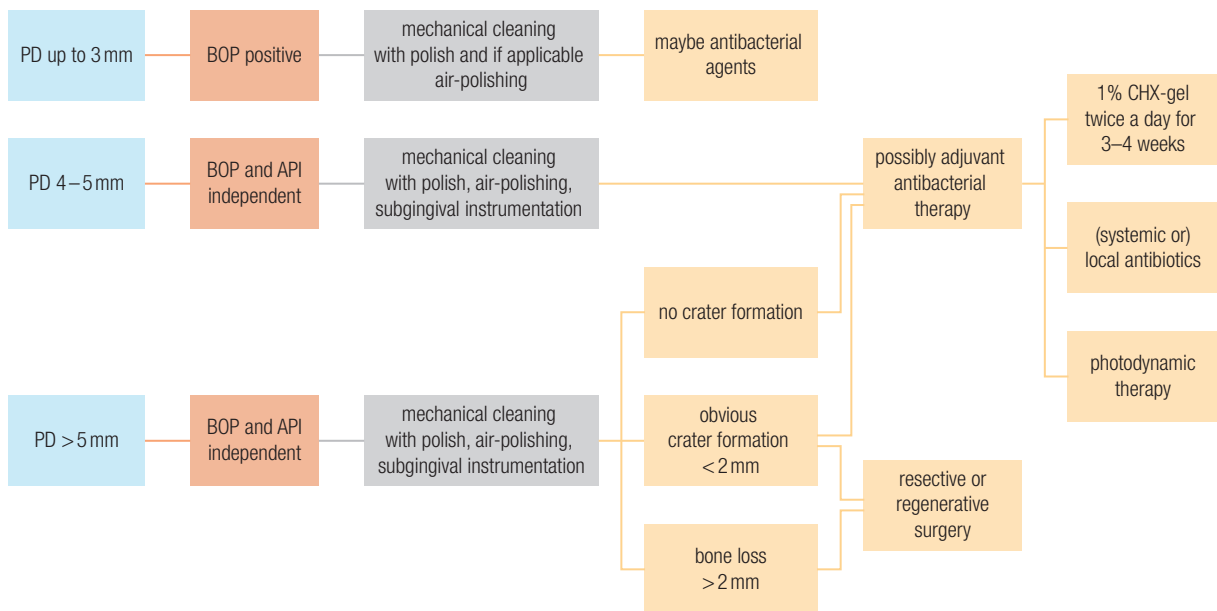
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**Fig. 3:** Therapy options for peri-implantitis therapy. PD = probing depth; BOP = bleeding on probing; API = approximal plaque index.

cleaning can also be supplemented in the context of a re-evaluation or supportive peri-implant therapy by antibacterial therapy measures such as local antibiotics, photodynamic therapy and laser as part of re-evaluation.

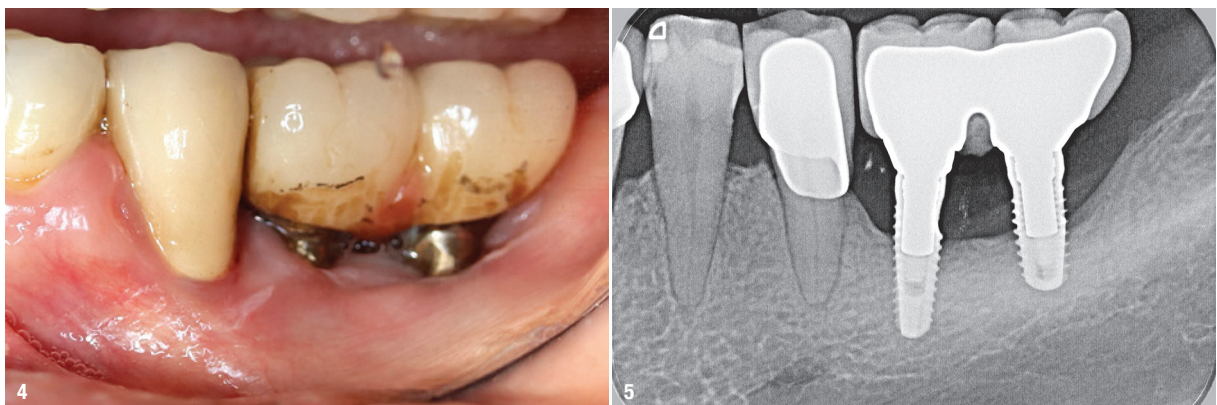
### What if peri-implantitis occurs nevertheless?

There is no standardised therapy scheme for peri-implantitis, but there are therapy suggestions that can be used to decide on the further course of action on an individual basis. These are shown in Figure 3 by a decision tree and are briefly explained in the following.

#### Non-surgical therapy for implants

The non-surgical removal of biofilm is the basis for any therapeutic approach to peri-implant disease and in the case of peri-implant mucositis in particular. For peri-implantitis therapy, the non-surgical approach is the important basic therapy and can be supplemented by further measures.<sup>17,32</sup> Nevertheless, the success in pockets

with an initial depth of > 7 mm is considered to be low.<sup>17</sup> In addition to an antiseptic home therapy to accompany biofilm control with chlorhexidine digluconate, it can be used as a complement to local mechanical debridement as pocket irrigation or as an application in gel form, which should lead to a significant improvement in probing depth after a control period of three or eight months. However, bleeding on probing is not affected by the use of chlorhexidine.<sup>33,34</sup> Further antibacterial procedures are, as mentioned before, local antibiotic therapy and photodynamic disinfection.<sup>12</sup> Significant reductions in probing depth, recession and plaque in cases of initial peri-implantitis have been proved for both local antibiotic therapy and photodynamic therapy (–0.15 mm).<sup>35</sup> For systemic antibiotic administration, no improvement in clinical and microbiological parameters has been found.<sup>36</sup> Figures 4 and 5 show a heavy smoker with moderate oral hygiene who— with exclusively non-surgical therapy measures—could be kept stable as far as possible because professional intervention was carried out early on. An elimination of the



**Fig. 4:** In this peri-implantitis patient (heavy smoker, moderate oral hygiene), small successes could be achieved through early scaling and root planing, and thus the situation was kept largely stable. Nevertheless, smoking cessation and an improvement in oral hygiene should be aimed for in order to achieve greater success. **Fig. 5:** Radiographic image of the same patient as in Figure 4.



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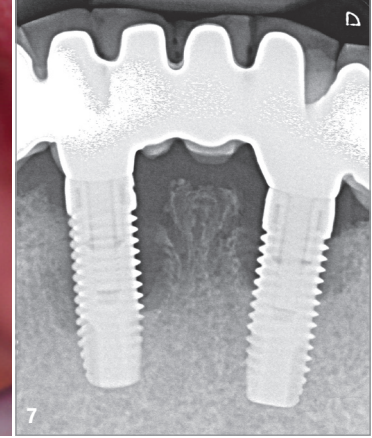
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**Fig. 6:** Patient with severe advanced peri-implantitis whose superstructures in regions #32 and 42 were difficult to clean, but with satisfactory oral hygiene.  
**Fig. 7:** Radiographic image of the same patient as in Figure 6. The bowl-shaped bone resorption around the implants is clearly visible.

## Defect type

## Surgical technique

Deep bowl-shaped defect

- Thorough cleaning and disinfection of the implant surface
- Defect reconstruction

No clear bone wall or significant horizontal bone resorption

- Thorough cleaning
- Reduction of the marginal mucosa with the aim of enabling effective oral hygiene by the patient

**Table 2:** Selection of the surgical approach based on the bone defect (modified from Schwarz et al.<sup>21</sup> and Renvert & Polyzois<sup>39</sup>).

above-mentioned risk factors should be striven for in order to further improve the situation around the implants.

### Surgical therapy for implants

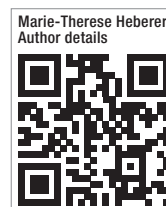
If peri-implantitis has progressed to such an extent that these conservative approaches with scaling and root planing no longer allow inflammation control and no improvement can be achieved (Figs. 6 & 7), resective or regenerative surgery or, in the worst case, explantation must be considered. When an implant has become loosened, explantation is the only therapy option.<sup>17</sup> The surgical technique should be selected based upon the bony lesion (Table 2). A recommendation for the exact technique cannot be given at this stage.<sup>17</sup> Studies however show a correlation between the success of regenerative therapy and the defect configuration.<sup>37</sup> Similar to a non-surgical approach, debridement and cleaning of the implant surface are the goals of the surgical procedure, which should be supplemented by defect reconstruction, reduction of probing depth and improvement of hygiene capability.<sup>31,32</sup> Additional surface decontamination with, for example, a 980nm diode laser does not show any improvement in the clinical or radiographic outcome of implants.<sup>17</sup> Unfortunately, as numerous publications emphasise, aesthetic losses must be accepted to prevent the progression of peri-implantitis.

### Summary

While a definitive concrete therapy recommendation for peri-implantitis therapy cannot be given at present, professional biofilm management in the practice as well as oral hygiene at home are the basis for the resolution of inflammation around the implant (as well as the tooth) and must therefore be practised regularly and well taught. Supplementary therapeutic measures such as local antibiotics,

photodynamic therapy and surgical approaches are possible and should be carried out depending on the degree of progression of the disease. Measures that significantly change the implant surface, such as the use of steel curettes or implantoplasty, should be avoided if possible.

*All images: © Marie-Therese Heberer & Nicole B. Arweiler*



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