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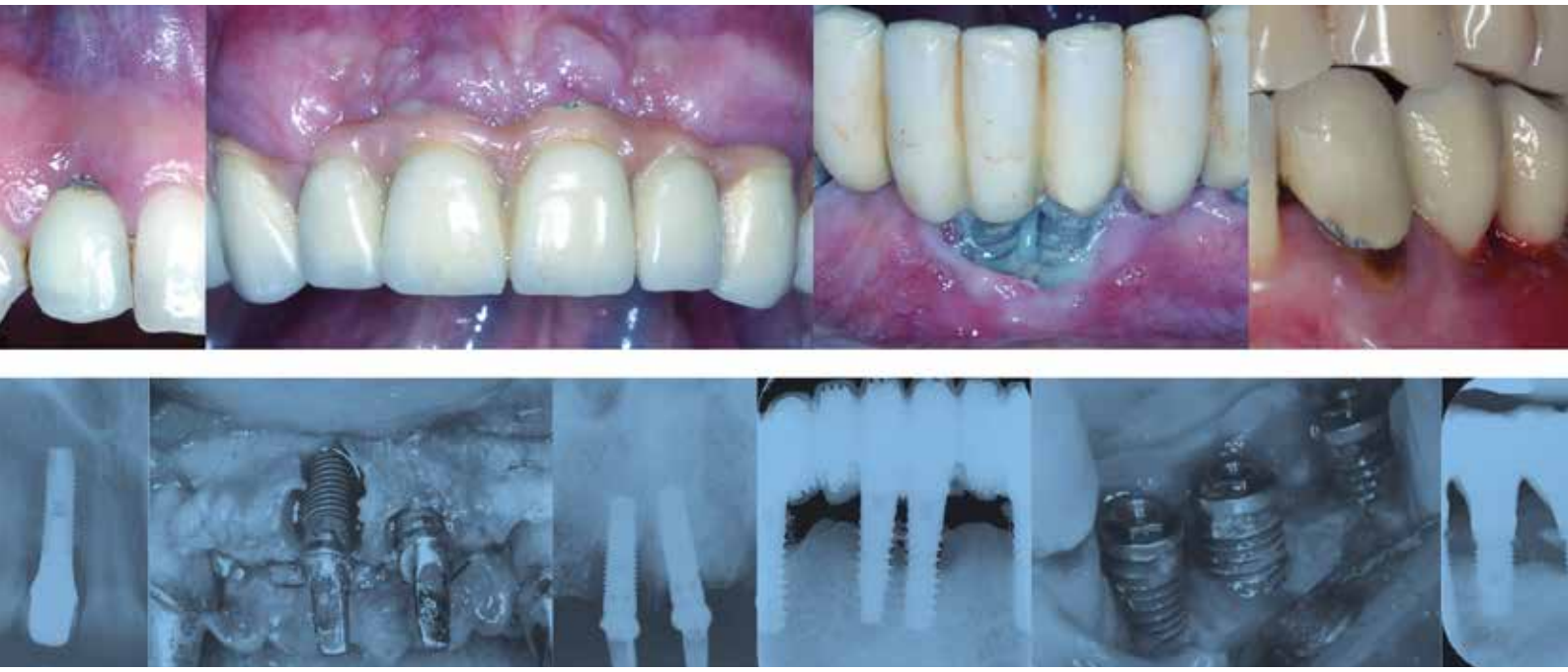

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Inside this issue:

The use of implants is currently one of the most common treatments in dentistry. At the same time, however, the number of people experiencing complications after implants is rising. Clinicians require treatment options that offer good results with a high degree of predictability and a low risk of complications.

Follow-up studies have revealed a high prevalence of infections around implant sites. Both experimental and clinical studies have identified the etiology and risk factors associated with such diseases. Diagnostic methods taken from periodontics have been adapted to this field. Furthermore, a series of different surgical and non-surgical resection and regenerative treatment methods are now available for the treatment of peri-implant diseases.

The continuous development of new diagnosis and treatment methods means that we can now avoid a clinical course of this type of disease in most cases. These Clinical Guidelines attempt to clarify the management required for peri-implant diseases, based on the literature and our clinical experience.*

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1. INTRODUCTION

1.1 DEFINITION

What is peri-implantitis?

Today, dental implants constitute a highly predictable treatment for replacing missing teeth. After determining and monitoring the factors that underpin osseointegration and overcoming the technical difficulties involved in prosthetic rehabilitation, the long-term maintenance of results requires the monitoring, anticipation, and treatment of potential biological complications stemming from the oral environment.

The term *peri-implantitis* was coined in 1987 by Mombelli.[1] We now accept the definition of the Sixth European Workshop on Periodontology (2008) that expanded and broke down the description to include peri-implant mucositis: **"Peri-implant mucositis is an inflammatory lesion that affects the mucosa, while peri-implantitis also affects the supporting bone"**[2] *(Figures 1-1 and 1-2).*

Figure 1-1 a

Figure 1-1 b

Figure 1-1a. Patient with peri-implant mucositis and marginal gingivitis because of ineffective plaque control.
Figure 1-1b. Patient with recurrent episode of peri-implant mucositis with supragingival calculus.

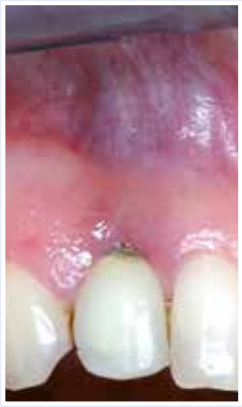


Figure 1-2 a

Figure 1-2a. Implant in the position of a maxillary lateral incisor affected by peri-implantitis with onset of mucosa retraction.

Figure 1-2 b

Figure 1-2b. The radiograph underestimates bone loss by not showing the buccal plate.

This definition of peri-implant diseases is intentionally descriptive and not highly specific as it does not include possible causes and clearly has an impact on the selection of parameters used for diagnosis.[3] This fact means that the figures relating to its prevalence are biased by the definition itself, by the bone loss threshold and probing depth used to detect them, by the differential diagnosis vis-à-vis other infectious entities, by differences in treatment and follow-up care, and by the differences in the study population.[4] However, and despite these limitations, according to our current data, peri-implantitis will occur in one out of every five patients, meaning the peri-implant tissue must be monitored for signs of inflammation that may jeopardize the results of the implant-supported rehabilitation[5] *(Figure 1-3).*

Under this approach, the aim of these guidelines is to provide clinicians with a clear and quick protocol that allows them to identify and effectively treat peri-implant diseases.

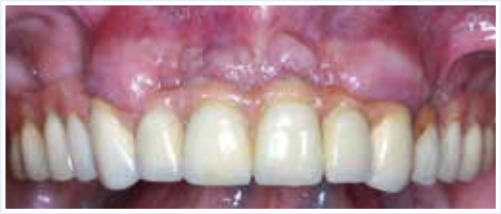


Figure 1-3 a

Figure 1-3 b

Figure 1-3 c

Figure 1-3 d

Figures 1-3. **Peri-implantitis in implants supporting a telescopic dental and implant-supported prosthesis.** The radiograph reveals major bone loss, which is confirmed after achieving surgical access.

1.2 ETIOLOGY

What is the cause?

Albeit briefly, it is important to know about the etiology of peri-implant diseases to understand the focus given to their treatment. Since the Sixth European Workshop on Periodontology (2008), it has been confirmed that peri-implant mucositis and peri-implantitis **are inflammatory infectious diseases**.[6]

Mombelli had already outlined the facts that supported the infectious hypothesis in 1999 at the Third European Workshop on Periodontology.[7]

a. An accumulation of bacterial biofilm induces peri-implant mucositis.
b. Qualitative and quantitative differences of biofilm exist between healthy implants and implants with peri-implantitis.
c. It is possible to induce experimental peri-implantitis using devices that encourage bacterial accumulation.
d. Peri-implant diseases respond positively to antimicrobials.
e. There is epidemiological evidence regarding the effect of oral hygiene on the condition of peri-implant tissue.

WHAT IS NOT CONSIDERED TO BE PERI-IMPLANTITIS?

However, clinicians should be aware that there are some clinical situations that can trigger or perpetuate peri-implant problems. The following conditions can cause bone resorption and even loss of the implant but are not included within the category of peri-implant diseases.

- **Osseointegration failure** *(Figure 1-4)*. Premature loss of the implant after loading with no initial obvious signs of mucosal inflammation. It usually presents as pain when masticating or when tightening the prosthetic screw or the transmucosal element and is associated with mobility although occasionally

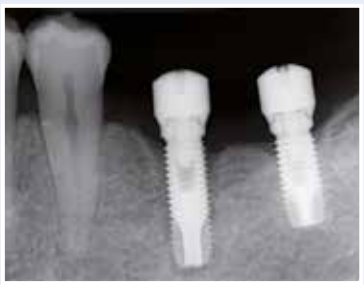


Figure 1-4

Failure of osseointegration. The implant in the mesial position presents a low-radiographic density halo of more or less regular thickness before implementation of the prosthesis.

no movement is perceptible. Radiographically, bone levels tend to be preserved, and the bone surrounding the implant may not show any disorders. Depending on clinical course time, a lower density (radiolucent halo) band with quite a homogeneous thickness may appear throughout the implant. This thickness, along with the mobility, may increase over time.

- **Physiological bone remodeling** *(Figures 1-5).* The connection between the implant and the oral environment involves an inevitable bacterial *translocation* to the peri-implant sulcus from adjacent microbiological niches (teeth, mucosa, tongue). This entails a specific adaptation of the tissue to restore the principle of hemostasis (termed the recovery of biological width by some authors). The resulting bone morphology places the bone profile between 1.5 and 2mm from the shoulder of the implant and is influenced by the position and morphology of the shoulder of the implant in relation to the alveolar process, the teeth and adjacent implants and should be taken into account when scheduling the treatment.[8,9]

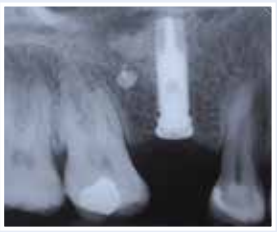


Figure 1-5 a

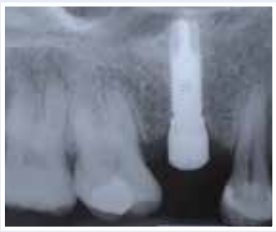


Figure 1-5 b

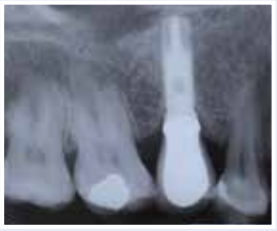


Figure 1-5 c

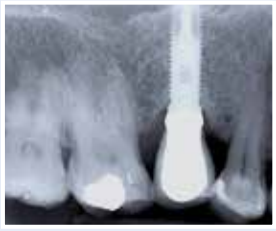


Figure 1-5 d

Figures 1-5. **Physiological bone remodeling.** In an implant placed at the juxta-bone level (Figure 1-5 a), the connection to the oral environment (Figure 1-5 b) causes bone remodeling with loss of coronal bone which forms the characteristic crater (Figure 1-5 c). After several years of clinical course, bone morphology is maintained and interproximal cortical bone indicates the integrity of the bone volume (Figure 1-5 d).

- **Loss of osseointegration** *(Figures 1-6)*. It has been shown under experimental conditions and at the clinical, histological, and radiographic level that occlusal overloading may trigger a loss of osseointegration. On a clinical level, mucositis is not usually present, but mobility and pain when masticating are observed. On a radiographic level, it progresses as a failure of osseointegration (radiolucent halo) although it can be associated with some bone loss due to peri-implantitis with a crater-like pattern (also known as a patellar defect). However, it has yet to be demonstrated that occlusal overloading can, on its own cause gradual coronal marginal bone loss.[10]

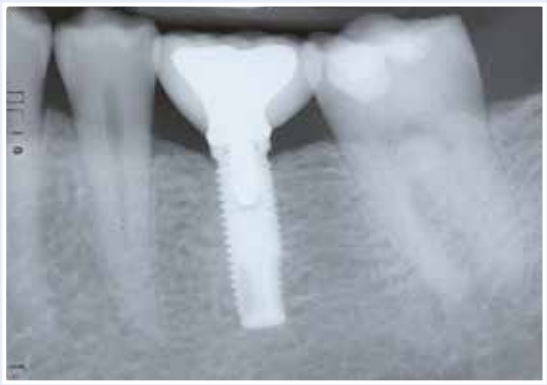


Figure 1-6 a

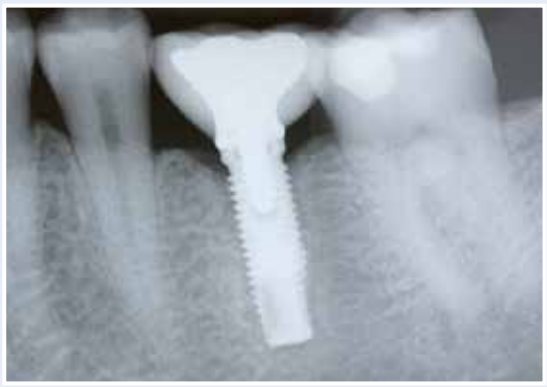


Figure 1-6 b

Figures 1-6. **Loss of osseointegration.** The thin radiolucent halo surrounding the implant (Figure 1-6 b) indicates fibrous tissue, a sign of loss of osseointegration.

• **Iatrogenic factors.** Certain factors, such as submucosal cement remnants in cemented prostheses *(Figures 1-7)*, a poor fitting of the prosthetic abutment (which causes contamination of the internal implant chamber), overhanging prostheses, or poorly positioned implants *(Figures 1-8)*, may favor bacterial accumulation, the onset of mucositis and its potential development into peri-implantitis when unresolved. Special attention must be paid to the incorrect positioning of implants (proximity to another implant or tooth, excessive inclination, non-anatomical emergence) not only due to the difficulties involved in any correction, but, in common with certain types of periodontitis (Group VIII of the Armitage classification),[11] these acquired conditions predispose the development of peri-implant mucositis and peri-implantitis.

Figure 1-7 a

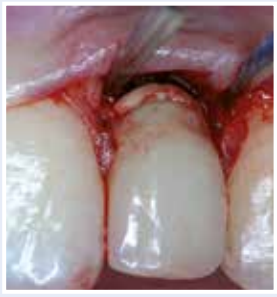


Figure 1-7 b

Figures 1-7. **Peri-implant mucositis**, resistant to non-surgical treatment caused by submucosal cement.

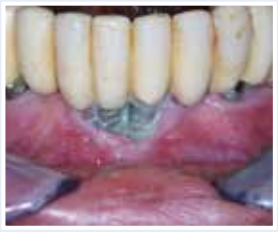


Figure 1-8 a

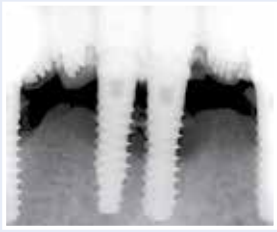


Figure 1-8 b

Figure 1-8. **Malpositioning.** (Figure 1-8 a) The proximity of the implants in the areas of the lower incisors has favored the emergence and clinical course of peri-implantitis. (Figure 1-8 b) The radiographic image shows a bone defect in the form of a crater characteristic of peri-implantitis and bridge of subgingival calculus.

2. DIAGNOSIS

2.1 CLINICAL EXAMINATION

As with any disease, a correct diagnosis of peri-implant disease is critical for its proper treatment.
After placing the prosthesis and sealing or temporarily cementing it, we recommend a two- to four-week period of acclimatization, which will enable us to verify whether the patient feels any discomfort and whether the oral hygiene is adequate. After this period, we move into the so-called ***baseline period***, which is when we seal or perform final cementing and take a number of records that will provide data on the initial condition of the implant. Data recorded during the baseline period will constitute a reference for future re-evaluations *(Figure 2-1)*. We must retain this data and continue to monitor it during the ***Follow-up Program*** that we recommend for all implant patients *(see the section on risk factors and prevention)*.
The records to be taken during the *baseline period* are:

a. **Probing depth** at 6 points (mesiovestibular, vestibular, distovestibular, mesiolingual, lingual, and distolingual). Unlike teeth, healthy probing depth of implants ranges far more since it depends on the position of the shoulder of the implants in relation to the bone level, the amount and condition of the surrounding keratinized tissue, the restoration, and the pressure when probing.

b. **Detecting and recording plaque and calculus**. We will verify whether patients can clean their prostheses. We will remind patients again about oral hygiene using a manual or power toothbrush and the appropriate interdental technique for each case.

c. **Final radiographs and photographs**: to be used as a reference for future check ups.

After this, and on an annual basis, we will take clinical records and Radiographs to detect any deviation from the "healthy" condition of the implants, characterized by:

- No signs of inflammation: bleeding or suppuration.
- Stable probe depth (PD) compared with the baseline period.
- No bone changes on the radiographs (the reference is the shoulder of the implant).
- No mobility.
- No pain.

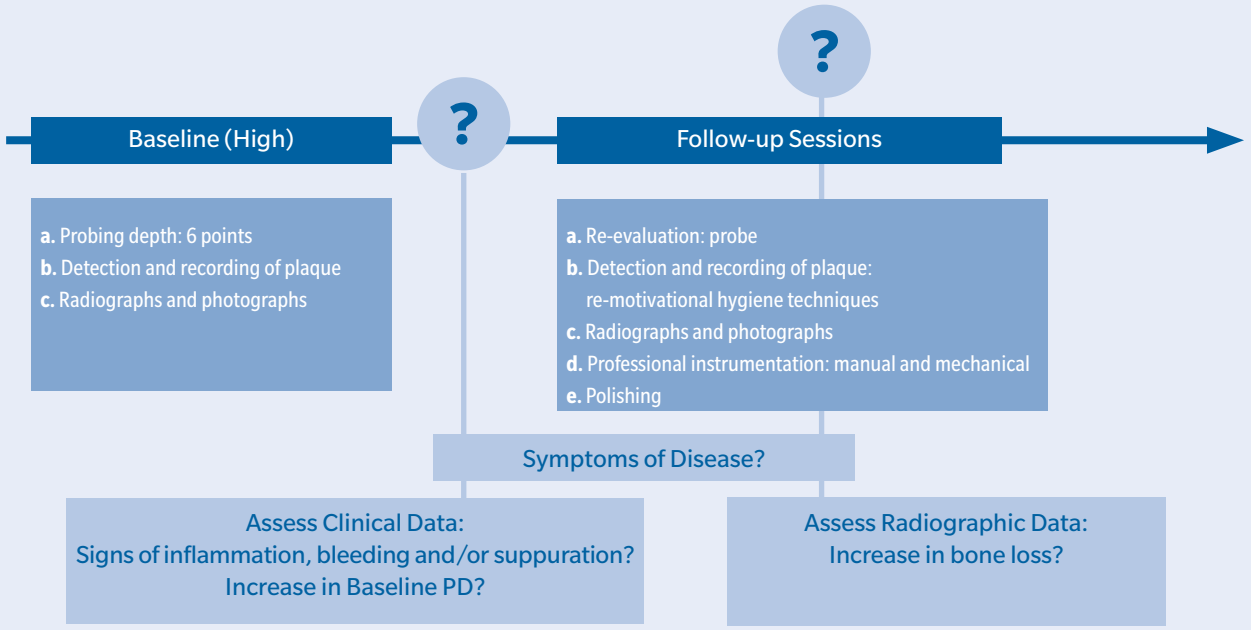


Figure 2-1 **Monitoring protocol for patients with implants.** After the baseline period, in which we perform closure or final cementing and take records representing the initial health status of the implant, the patient shall be referred periodically to monitor the condition of the peri-implant tissue, verify the level of oral hygiene, and remove, if necessary, supra and subgingival biofilm. At any time, the patient may go for a consultation to see whether signs of disease in the tissues are detected.

Out of all of the above, the existence of signs of inflammation and gradual bone loss are the best indicators of peri-implant inflammatory disease.[12] We are therefore able to detect signs of disease in patients included in the monitoring programs. When it is detected, we can use a series of records to assess the appropriate treatment. Before performing additional tests, we will begin the **initial diagnosis of peri-implant disease**, based on the symptoms reported by the patient (subjective) and the signs identified by the professional (objective). It is important to state that a diagnosis does not rely solely on a visual examination or radiographs, as these are unable to reveal the early stage of disease. For this reason, we rely on peri-implant indices, in compliance with the following order of procedures:

1. INSPECTION

A visual observation will detect the problem and raise alarm signals. **We will assess the clinical signs of inflammation:** redness, swelling, contour and consistency abnormalities or the form of the mucosa, bleeding and suppuration *(Figure 2-2)*. Early detection of these signs is key.

Although we are unaware of the importance of the stability of soft-tissue margins for the survival of implants, we do know that it is essential to prevent and control the onset of shrinkage to prevent the surface of implants from becoming exposed to the oral environment, which generates more accumulation of plaque. At this stage of the diagnosis, we must **teach patients to observe their tissue** and be able to distinguish between healthy and unhealthy tissue. This training is particularly important if we assume

Figure 2-2

Clinical image of signs of peri-implant inflammation. Inspection of peri-implant tissues is used to detect clinical signs of inflammation: redness, swelling, abnormal contour and consistency or form of the soft tissue, bleeding, and suppuration.

that, in most cases, mucositis is the precursor of peri-implantitis. In fact, the absence of marked variations between the microbiology of both lesions may indicate that, in most cases, the disease develops from mucositis to peri-implantitis.[12]

2. PALPATION

After inspecting the situation, we then turn to palpation *(Figure 2-3)*. The following may appear during this stage:

Figure 2-3

Palpation can be performed digitally or by using equipment such as a gauze, swab, or cotton roll. Essentially, this maneuver enables us to verify the absence or presence of bleeding or suppuration.

a. Suppuration. The existence of suppuration is related to bone loss and clinically associated with advanced lesions.[14, 15] However, this is often not easy to detect, and its sensitivity/specificity as an initial marker of peri-implantitis or its clinical course has not been established *(Figure 2-4)*.

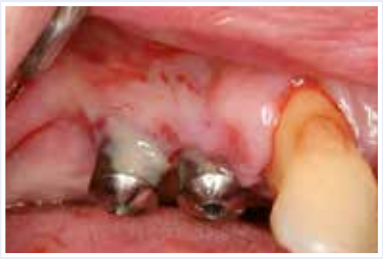


Figure 2-4

Palpation may reveal the presence of suppuration. It is normally the case for implants to have gradual bone loss.

b. Percussion. Pain or a dull non-metallic sound upon percussion can be a sign of osseointegration. Therefore, *it cannot be used for the early detection of peri-implant diseases*.

c. Mobility. This is a key factor for establishing the viability of an implant. It can be assessed manually or using devices such as Osstell™ (Integration Diagnostics Ltd., Gothenburg, Sweden) or Periotest™ (Siemens AG, Bensheim, Germany). Any degree of mobility is associated with a complete loss of osseointegration and, therefore, requires the removal of the implant *(Figure 2-5)*. Clearly, mobility is not useful for the early diagnosis of peri-implant diseases, as it is a sign of non-reversible bone loss.

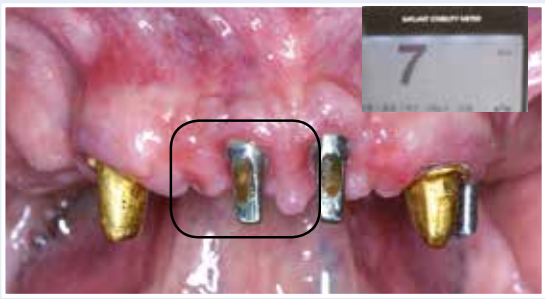


Figure 2-5

Measurement of implant mobility in position 11 measured using Osstell. ISQ value 7 indicates that the implant has little stability.

For its detection, a number of considerations must be taken into account:

- It should be evaluated on an individual basis for each implant.
- Full or partial arch prostheses should be removed for an adequate evaluation (this is one of the reasons why it is more advisable to screw in prostheses rather than cementing them).
- Single prostheses are considerably easier to assess but, when any movement is detected, a differential diagnosis is required involving the possible loosening or debonding of the dental prosthetics.

3. PROBING

After inspection and palpation, we move on to the probing stage, which is not without limitations, such as access problems *(Figure 2-6)*. This parameter is therefore entirely determined by the emergence of the prosthesis, and it will often be necessary to remove it to obtain accurate and reliable measurements *(Figures 2-7)*. A conventional periodontal probe can be used since no data has shown that special materials or designs are required.

a. Depth. The probing depth and clinical insertion levels are both basic tools for the diagnosis of peri-implant diseases. It is well

known that healthy peri-implant tissue offers resistance to probing[16], while if the disease is present, the periodontal probing depths increase *(Figures 2-8)*. As discussed above, the probing depths of healthy peri-implant tissue ranges far more than that of natural teeth, meaning that healthy tissue probing can exceed 3 to 4mm without implying the involvement of disease, although this does not pose a greater risk for patients.

Figure 2-6

Clinical image of peri-implant sulcus probing. Bleeding on probing and changes in the baseline probing depth are basic data for diagnosing and monitoring peri-implant problems.

Figure 2-7 a

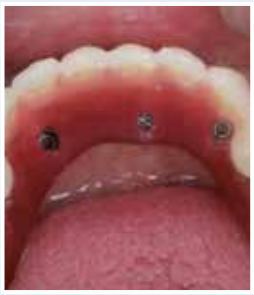


Figure 2-7 b

Figure 2-7. Probing is determined by the emergence of the prosthesis. In many cases it is necessary to remove it to achieve accurate and reliable measurements.

Figure 2-8 a

Figure 2-8 b

Figures 2-8. Probing is a basic clinical examination used to detect peri-implant problems since we know that healthy peri-implant tissues provide resistance to such problems (Figure 2-8a), while when disease is present (inflammation) the depths increase (Figure 2-8b).

When probing, the following must be taken into account:

- Gentle force must be used (0.25 Ncm).[17, 18] These forces yield probing values that accurately reflect the location of the apical extension of the junctional epithelium, both in healthy conditions and in mucositis and peri-implantitis. It was traditionally thought that probing around implants could damage the perimucosal seal and, therefore, should not be used routinely. However, we now know that it does not imply any trauma to or infection of the peri-implant tissue, provided that gentle force is used, since the mucosal seal heals fully after five days.[19] Controlled pressure probes, such as the Florida Probe® (Florida Probe Corporation, Gainesville, Florida, USA), are available on the market and make it easier to perform probing at adequate pressure *(Figure 2-9)*.e

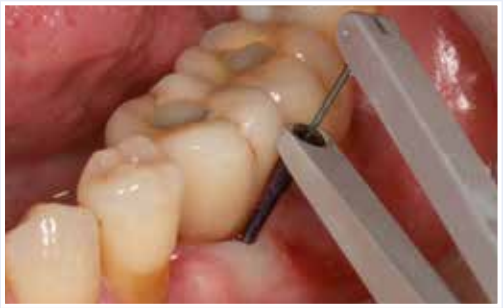


Figure 2-9

Gentle force must be used (0.25 Ncm). For this reason, there are controlled pressure probes on the market such as the Florida Probe® (Florida Probe Corporation Gainesville, Florida, USA).

b. Bleeding on probing *(Figure 2-10)*. This is a valid clinical sign used for monitoring.[20] The existence of bleeding on probing indicates instability of the peri-implant tissue and offers more positive predictive value than with teeth.[21, 22]

Figure 2-10 a

Figure 2-10 b

Figure 2-10. Clinical diagnosis using a manual probe, PCP-12. An essential clinical sign in monitoring the health of peri-implant tissues is the absence (Figure 2-10a) or presence (Figure 2-10b) of bleeding on probing.

Because of the specific anatomical characteristics of the seal around the implant, if the probing is not performed carefully it can reach connective tissue, even when healthy, and cause bleeding. However, if the probing is performed correctly, the bleeding can be extremely useful for diagnosis. It is also useful for follow-up: we must probe and evaluate bleeding on probing before and after our treatments to verify there is no bleeding.

To conclude, the appearance of peri-implant mucosa, mobility, stability of probing depths, bleeding on probing, and suppuration all need to be regularly evaluated to detect peri-implant diseases.[23]

2.2 RADIOGRAPHIC EXAMINATION

Bone loss is the key diagnosis

Radiographic examination is an essential test for detecting and monitoring peri-implantitis. To take advantage of all its diagnostic capability, it is important to know which technique to use and when to use it, as well as its limitations *(Figure 2-11)*.

The aim is to record the initial position *(baseline period, Figure 2-1)* of the interproximal bone crest from a point that is stable over time (implant shoulder) and to use this distance to monitor changes to the bone over time *(Figure 2-12)*.

Figure 2-11 a

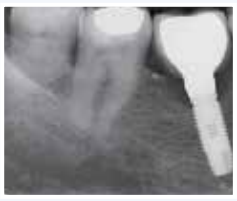


Figure 2-11 b

(Figure 2-11 a) Bone defect in crater or patellar typical of peri-implantitis. The resulting U-shaped arch places the alveolar bone profile at a distance of several millimeters from the shoulder of the implant.
(Figure 2-11 b) Bone morphology in the form of a crater as a result of physiological bone remodeling. Unlike figure 2-11a, the more coronal bone profile is situated 1 to 1.5mm from the implant shoulder.

Figure 2-12 a: Jun 2006

Figure 2-12 b: Sep 2007

Figure 2-12 c: May 2008

Figure 2-12 d: Apr 2009

Figure 2-12 e: May 2010

Figure 2-12 f: Jun 2011

Figure 2-12 a) Patient with peri-implantitis which begins to manifest prior to insertion of the prosthesis (Figure 2-12a: Jun 2006). (Figure 2-12 b) After one year of clinical course (Figure 2-12b: Sep 2007), bone loss affects the coronal third of the implant. (c-d-e-f) After non-surgical etiological treatment, the bone level remains stable after 4 years of follow-up.

Longitudinal studies and experts have emphasized that **the insertion of the prosthesis is the ideal time to measure the initial reference points**. However, for immediate implants with an immediate prosthesis, this timing would be premature, as no physiological tissue remodeling has yet occurred in response to the insertion of implants.

The recommended frequency is once a year. However, follow-up studies have also indicated that patients with probing depths greater than 5mm have an increased risk of bone loss when presenting with bleeding on probing. Therefore, radiographs may need to be performed more frequently.[24]

In terms of orthopantomography *(Figure 2-13a)* versus intraoral periapical radiography *(Figures 2-13b; 2-13c)* we recommend using the latter as it offers greater diagnostic accuracy and lower doses of radiation. To minimize distortions and comparison difficulties due to the variations in the geometry of the image, a standardized parallel technique is required using positioning devices. Experimental studies have revealed that **it is extremely important to take periapical radiographs parallel to the axis of the implant** (i.e. perpendicular to the x-ray beam) as deviations exceeding 10 degrees can make the image unidentifiable.[25, 26]

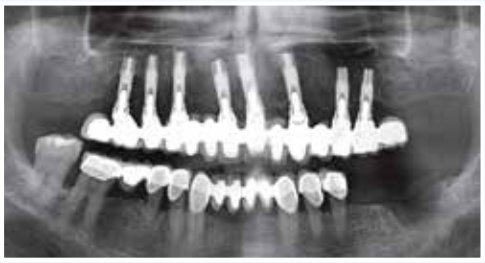


Figure 2-13 a

Figure 2-13 b

Figure 2-13 c

Figure 2-13. a) Orthopantomography of a patient with peri-implantitis. b-c) Periapical images of the same patient reveal a different bone morphology to that captured by the orthopantomography as well as subgingival calculus.

The following circumstances must be taken into account when interpreting the radiographic image:

- Conventional **radiographs have a limited capacity** to detect early bone changes. They are not a sensitive test to detect early peri-implantitis and, therefore, do not replace a clinical examination.

- When using two radiographs to diagnose peri-implantitis, the **quality of the image** must be established (contrast analogy and geometric matching). To this end, the implant itself can be used: the internal chamber of the implant must be visible, and it must also match the morphology of the implant's profile (threads) between both images *(Figure 2-14)*.

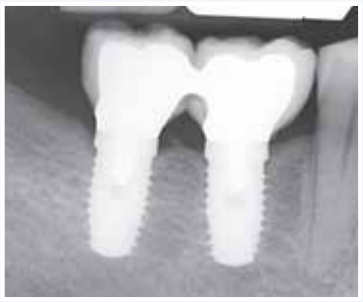


Figure 2-14 a

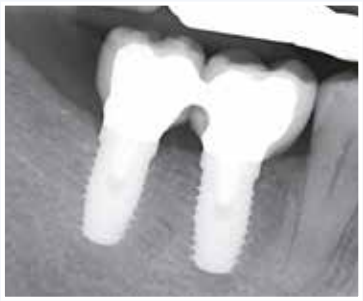


Figure 2-14 b

Figures 2-14. **Verification of the image quality.** Figure 2-14b presents a radiographic contrast similar to Figure 2-14a, but the difference in the morphology of the coils reveals a variation in the geometry that does not enable comparison. Notice the effect on the radiographic bone profile, which has "increased" on referencing it to the implant shoulder.

• We should not overlook the fact that **only the interproximal areas are detected**. To improve the visualization of the alveolar crest profile, it may be advisable to use various projections and include bitewings in the posterior sections *(Figures 2-15)*.

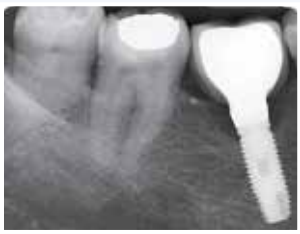


Figure 2-15 a

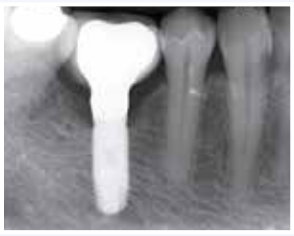


Figure 2-15 b

Figure 2-15 c

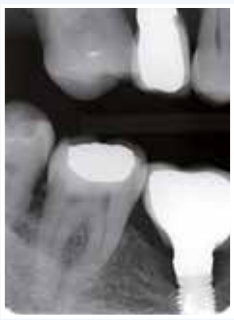


Figure 2-15 d

Figures 2-15. **Radiographic reference taking (baseline section).** The taking of periapical radiographs must be standardized, by seeking a radiographic projection that enables the implant morphology to be clearly identified. Images b) and c) do not enable different parts of the implant to be identified because of lack of perpendicularity of the x-ray beams to the implant axis.

• The thickness of the alveolar process, the superposition of anatomical structures (pyramidal apophysis of the upper jaw or mandibular oblique lines) and the use of bone regeneration with bone particulates have an impact on the image and should be taken into account when monitoring such images *(Figures 2-16)*.

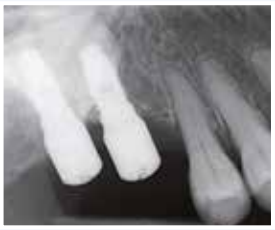


Figure 2-16 a

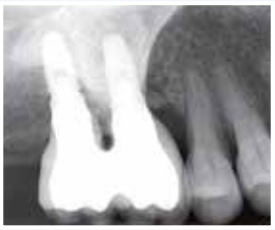


Figure 2-16 b

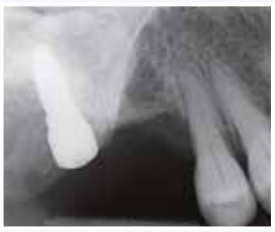


Figure 2-16 c

Figures 2-16. **Limitations of radiographic examination.** a) High radiographic density resulting from lifting the sinus floor and coincidence with the pyramidal process of the maxillary arch match the location of the implants. b) High radiographic density masked the course of the mesial implant peri-implantitis, detected by probing. c) Bone loss cannot be observed, even after the implant has been removed.

Finally, the use of cone beam tomography overcomes some of the diagnostic problems encountered with conventional radiographs. This technique provides a very accurate three-dimensional image of hard peri-implant tissues, although it does not confirm the existence of osseointegration. Due to its high dose of radiation, compared with intraoral radiographs, any prescription should be carefully evaluated.

2.3 OTHER TESTS

1. MICROBIOLOGICAL DIAGNOSIS

Due to the infectious origin of peri-implantitis and its similarity with periodontitis, microbiological tests have been suggested as a possible diagnostic approach to detect more aggressive periodontopathogens.

However, studies have not supported this hypothesis, and microbiological tests are not useful for diagnosing peri-implant diseases. To explain these findings that appear to contradict the infectious etiology of such diseases, experts have pointed out that bacterial species specifically related to peri-implantitis have not yet been identified. In addition, by solely seeking periodontopathogens, the potential role of other pathogens that trigger extraoral infections, or whose culture is difficult, may be overlooked.[26]

A possible alternative use of these tests would not be for diagnosis purposes but as a guide for treatment. Using microbiological cultures to establish an antimicrobial susceptibility profile enables antimicrobial therapy to be tailored to peri-implantitis, which does not respond to broad-spectrum antibiotics.

Finally, we must not forget that **the patient's level of oral hygiene and history of periodontitis are identifiable risk factors** for peri-implantitis. For this reason, although they cannot be used to confirm the diagnosis of peri-implantitis, microbiological tests on the remaining dentition could offer a useful test to determine the risk of peri-implantitis. Interestingly, at present, there is only a single study that reveals the usefulness of these microbiological tests to improve the prognostic capacity of bleeding on probing to detect the clinical course of peri-implantitis.[28]

2. GENETIC DIAGNOSIS

In common with microbiological studies, the similarities between periodontitis and peri-implantitis tend to suggest that genetic tests identifying polymorphism in Interleukin 1 could point to an increased susceptibility to peri-implantitis (consequently, this could be used as a prognostic test to establish the level of risk but not for diagnosis). However, reports from clinical studies that have explored this possible relationship have been inconclusive.

Interestingly, this association does appear to be observed in smokers. That is, **a greater destruction of bone due to peri-implantitis is observed in patients who are smokers and who have polymorphism in their Interleukin 1 gene.** Therefore, in such patients (peri-implantitis+tobacco) it would be interesting to perform this genetic study to gain a better understanding of their level of risk of bone destruction.[29, 30, 31]

3. TREATMENT

3.1 GENERAL PRINCIPLES

How should we treat this condition? The 5 principles

The strategy used to treat peri-implant diseases is simple: firstly, eliminate the cause (etiological treatment) and then attempt to correct the consequences of infection (corrective treatment of sequela).

After accepting the infectious etiology, and because of the clear similarities with periodontal diseases, several protocols have been proposed based on the treatment of periodontitis. The basic aim is to reduce bacterial load in the peri-implant sulcus and over the surface of the implant to a level that does not cause an inflammatory reaction.

This primary aim can be broken down into a few general principles proposed by Mombelli in 1999,[32] namely:

1. **Removal of the biofilm from the peri-implant pocket.**
2. **Decontamination/conditioning of the surface.**
3. **Reduction or elimination of locations that are difficult to clean.**
4. **Establishment of an effective regimen for the patient to monitor plaque to prevent re-infection.**
5. **Bone regeneration/tissue recovery.**

However, there are clear differences between teeth and implants that will have an impact on treatment: metal, design with threads and surface treated to enhance its roughness. These characteristics can favor the formation of a bacterial *biofilm* when exposed to an oral environment. Furthermore, the superstructure design can hinder effective mechanical treatment of the infected implant.

Although less obvious, the main differences between teeth and implants probably lie at the tissue level. In addition to the absence of periodontal ligaments and connective insertions, there are also obvious structural differences in the soft tissues. Experimental studies have revealed that peri-implant mucosa should be considered as a scar that repairs the aggression following placement of the implant.[33]

Peri-implant masticatory mucosa is denser than collagen but less vascular and, more specifically, has fewer fibroblasts in comparison to gingiva. Although there is no clinical evidence that translates the consequences of these findings, it can be inferred that, like all scar tissue, it will have a delayed clinical response, decreased capacity for tissue repair, and even an abnormal immune response.

Taking into account all of these circumstances and by analogy with the systematic periodontal treatment approach proposed by Ramjford,[33] the following treatment sequence is proposed:

1. Systemic phase

2. Etiological phase
2.1 Non-surgical treatment
2.2 Surgical treatment

3. Corrective phase
3.1 Bone regeneration
3.2 Mucosal correction

4. Follow-up or maintenance phase

TREATMENT 1
SYSTEMIC PHASE - Antibiotics

In common with the treatment of periodontitis, this phase should control the systemic conditions that favor local infection, such as congenital or acquired diabetes or immune disorders, and the use of some medications.

During this phase, we should consider the option of administering antibiotics. To date, evidence on the need to administer systemic antibiotics is limited. However, the use of antibiotics has been shown to reduce bleeding on probing and the peri-implant probing depth. Moreover, as it involves infection of a foreign body and possible involvement of extraoral bacteria, such as *Staphylococcus* and *Peptostreptococcus*, the use of systemic antibiotics may be more justified than in periodontitis.

When making the decision, we need to examine the degree, extent, and depth of the inflammation. The following situations support the prescription of systemic antibiotics:

a. The inflammation of peri-implant masticatory mucosa has reached the mucogingival line.

b. Abundant suppuration through the peri-implant sulcus.

c. Existence of an abscess or fistula.

As the flora associated with peri-implant diseases is of mixed type, quite variable, and in most cases dominated by anaerobic gram-negative bacteria, we need to use a **broad-spectrum antibiotic** that will also cover the possibility of Staphylococcus or Peptostreptococcus being present. A first-line antibiotic could be **amoxicillin with clavulanic acid**. In the case of penicillin allergy, other broad-spectrum drugs, such as doxycycline (tetracycline) or ciprofloxacin (quinolone), could be used.

TREATMENT 2
ETIOLOGICAL PHASE - Reducing bacterial load

ETIOLOGICAL NON-SURGICAL TREATMENT: MECHANICAL THERAPY

The aim is to reduce bacterial load to a level that stops inflammation of the peri-implant tissue. This is called "mechanical therapy" and is equivalent to the scaling and root planing used in periodontal therapy. However, the characteristics of implants in terms of their design and surface mean that a different approach is required.

An **essential preliminary phase involves the periodontal treatment of the remaining dentition**. Periodontal health must be achieved by scaling and root planing, as well as training patients in oral hygiene techniques.

In terms of implants, in order to avoid damaging the titanium surface, the tactic has been to use materials that are softer than titanium itself: rubber polishers, polishing brushes, low-abrasion, fluoride-free and pumice-free prophylaxis paste (Hawe Implant Paste™ by Kerr™), curettes of various materials (plastic Teflon®, carbon, gold-coated, and titanium curettes), ultrasonic tips covered with plastic PEEK (polyetheretherketone; Instrument PI for Piezon® by EMS™; SONICflex® implant tip by KAVO™) or high-pressure jets of glycine particles (Air-FLOW® Soft by EMS™) (Figures 3-1).

Figure 3-1 a

Figure 3-1 b

Figures 3-1. a) Carbon curettes for metal surfaces. b) Details of the active part.

Mechanical therapy under this approach is suitable for removing materia alba and supragingival calculus, in addition to floating bacterial plaque, from the peri-implant sulcus. However, its effectiveness is limited in terms of removing subgingival calculus, as well as the plaque fixed to the surface of the implant, especially in the case of rough surfaces. Moreover, depending on the technique used, it is documented that these soft materials can break down and settle on the surface of the implant, thereby altering cellular adhesion. Therefore, to improve the non-surgical options for the treatment of peri-implant diseases, **the use of adjunctive therapies**, such as antimicrobial mouthwashes, submucosal irrigation with antiseptics and disinfectants, antibiotics for topical application, photodynamic therapy, and lasers have all been proposed. The results of available studies indicate:

- The association of antimicrobial mouthwashes with chlorhexidine and essential oils reduces the number of locations with bleeding on probing.
- Subgingival irrigation with antiseptics improves the probing depth and reduces the number of locations with bleeding on probing.
- The adjuvant use of local antibiotics (tetracycline fibers, sustained release forms of doxycycline, lincomycin, or minocycline) also reduces the number of locations with bleeding on probing and their probing depth.
- The use of laser or photodynamic therapy has not revealed any benefits in comparison to mechanical therapy.

The general conclusion appears to indicate that mechanical therapy is suitable for the treatment of mucositis. However, the results are limited by the probing depth. In terms of peri-implantitis, non-surgical treatment is quite unpredictable.[35]

Based on all the above, the following protocol is proposed:

a. Review the general periodontal status of the patient and carry out any treatment required to achieve periodontal health, which includes adequately controlling plaque. Pay special attention to teaching and verifying hygiene techniques appropriate to the implant-supported prosthesis.

b. Remove the prosthesis or superstructure. If the access to the peri-implant sulcus is inadequate or there is a lack of adjustment or previous loosening, removal of the prosthesis or superstructure should be considered. Not only will this facilitate access to the entire perimeter of the sulcus, but it will facilitate decontamination of the implant's interior, which can act as a bacterial reservoir.

c. Mechanical therapy. Use curettes (carbon, Teflon®, titanium) and special ultrasonic tips designed for implants (PEEK), avoiding damage to the metal areas. The use of infiltrative anesthesia is recommended to prevent discomfort for patients and to ensure that the inferior part of the peri-implant sulcus is reached. Special care should be taken to avoid damaging soft tissues in fine phenotypes.

d. If bicarbonate or glycine jets are used, the risk of emphysema should be evaluated carefully, depending on the degree and extent of the peri-implant soft-tissue inflammation.

e. Use low-abrasion, fluoride-free, and pumice-free prophylaxis paste (Hawe Implant Paste™ by Kerr™).

f. Submucosal irrigation of the peri-implant sulcus with disinfectants and oral antiseptics. It is recommended that a normal saline solution be used initially for washing to remove any floating bacterial plaque, in addition to any calculus remnants, blood, and other organic matter that decreases the effectiveness of disinfectants. Chlorhexidine or 10% povidone-iodine can be used (warning: iodine products cannot be used during breastfeeding, they can interfere with thyroid function tests, and prolonged use in patients under simultaneous lithium therapy should be avoided) *(Figure 3-2 and Figure 3-3).*

Figure 3-2

Syringes prepared with blunt needles for submucosal irrigation of saline solution, chlorhexidine and 10% povidone iodine.

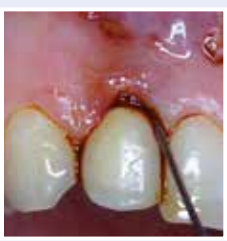


Figure 3-3

Submucosal irrigation of povidone iodine.

g. As an alternative, evaluate the use of topical antibiotics in the peri-implant sulcus. This is recommended for deep probing cases in aesthetic areas, in locations with vertical (infra-bone) bone defects, or in areas difficult to access with mechanical instruments *(Figure 3-4)*.

Figure 3-4

Submucosal application of topical antibiotics.

h. Prescribe a chlorhexidine mouthwash with or without chloride cetylpyridinium every 12 hours for 2 to 4 weeks.

i. See the patient again in 2 to 4 weeks to evaluate the results of the mechanical therapy and to determine the need for surgical access.

ETIOLOGICAL SURGICAL TREATMENT:

All the treatments proposed for the management of peri-implant diseases are based on our knowledge of the treatment of periodontal diseases.

The removal of biofilm on the surface of the implant is the main aim of peri-implantitis therapy. Thus, in some cases, we have to resort to surgical treatments since non-surgical treatments, despite being effective for the treatment of mucositis, are not effective in peri-implantitis.[36]

Non-surgical treatment of peri-implantitis is not predictable: it resolves the inflammatory lesion but does not obtain significant re-osseointegration on the surface of the previously affected implant. The associated use of chlorhexidine only has limited effects on clinical and microbiological parameters. The use of lasers has revealed minimum benefits and requires further evaluation. The use of topical or systemic antibiotics has been proven to reduce bleeding and probing depths. **Surgical treatment is currently the suitable approach used to treat peri-implantitis as it offers greater predictability in stopping peri-implant bone destruction**.[37]

The primary objective of the surgical treatment of peri-implantitis is to access the surface of the implant to **debride, decontaminate,** and resolve the inflammatory lesion.[38] However, even when surgery is the treatment of choice, non-surgical treatment is required first because this enables us to verify the ability of the patient to adopt proper oral hygiene and may even resolve some peri-implant lesions.

DECONTAMINATION

One of the objectives of surgery is to access the surface of the implants in order to decontaminate it. Here, we face perhaps one of the greatest differences compared with the tooth: namely the surface of the implant as opposed to the radicular cementum. The macro-design of implants, along with various modifications to the surface areas, can favor the formation of a bacterial *biofilm* when exposed to the oral environment. In addition, the superstructure design can hinder an effective mechanical treatment of the infected implant.

Animal studies have found that to achieve re-osseointegration, an open debridement and decontamination of the surface of the implant are required. Consequently, **decontamination of the surface of the implant is a mandatory step in the surgical treatment of peri-implantitis.** This implies that, regardless of the surgical technique used, we will always be able to access the problem, debride the biofilm and infected tissue, and decontaminate the implant surface. During the surgical procedure, we can treat the peri-implant soft and hard tissues.

The objectives of this type of cleaning and decontamination must be to:

1. Remove bacterial deposits.

2. Facilitate the rearrangement of soft tissue.

3. Limit and minimize any future bacterial *biofilm.*

Biological contamination is difficult to remove from the implant surface. A number of tools are available for the removal of the subgingival *biofilm* such as plastic, carbon, Teflon, or titanium curettes, modified ultrasonic tips and blast-abrasion systems. All these tools have proved to be inadequate in fully removing the biofilm from the rough surface of the implant,[39,40] and none of the mechanical or chemical decontamination methods have proven to be superior over the others *(Table 3-1)*.

Schwarz[41] compares closed-sky and open-sky treated areas, using the same treatments: laser, vector, and curettage, along with tetracycline. In all three cases, the best results were obtained with

Treatment	Observation Period	Results	Comments
Test: Cleaning with **delmopinol**. Monitoring: None. Antibiotics for 3 weeks.	4 months	Test: Resolution of peri-implantitis but without re-osseointegration. Significant recession of the marginal peri-implant mucosa. Monitoring: Peri-implantitis is not resolved.	Non-submerged model. The results may reflect this.
Group 1: Abrasive pumice with rotating brush. Group 2: Cotton pellets soaked in saline solution.	7 months	Radiological bone height increase: Group 1: 0.65 mm, Group 2: 0.73 mm. Re-osseointegration in both groups: 0.4 mm Bone regeneration: Group 1: 59%, Group 2: 64%.	Submerged model.
Cleaning with chlorhexidine. Monitoring: without GBR. Test: GBR with ePTFE membrane.	6 months	Histology: **Bone regeneration**: M: 31% (0.82 mm), SLA: 15.1% (0.41 mm), TPS: 13.9% (0.33 mm). **Re-osseointegration**: M: 7.05% (0.19 mm), SLA: 11.1% (0.3 mm), TPS: 13.9% (0.33 mm), M+GBR: 61.7% (2.2 mm), SLA+GBR: 83.4%, (2.6 mm), TPS+GBR: 72.6% (2.3 mm), M+GBR: 2% (0.07 mm), SLA+GBR: 19.7%, (0.6 mm), TPS+GBR: 13.6% (0.5 mm).	No statistically significant differences in terms of re-osseointegration, although **significantly more bone filling in groups with GBR**.
Group 1: **Air powder abrasive**. Group 2: **Carbon dioxide laser**. Group 3: ***Prophy jet*** + carbon dioxide laser.	4 months	**No statistically significant difference between groups in terms of bone gain.** The groups treated with laser revealed more bone-implant apposition and group 2 was better than group 3.	The carbon dioxide laser gave somewhat better results.
Surface rinsed with physiologic saline, photosensitization, and ePTFE membrane. Surfaces: Hydroxyapatite (HA), Titanium plasma spray (TPS), Acid etching (AE), Commercially pure titanium (CPTi).	5 months	Bone regeneration: HA: 48.28%, TPS: 39.54%, AE: 26.88%, CPTi: 26.7% Re-osseointegration: HA: 15.83%, TPS: 25.25%, AE: 17.3%, PTi: 24.94%	
Group 1: **Air powder abrasive unit** + citric acid. Group 2: **Air powder abrasive unit**. Group 3: Gauze in saline + citric acid. Group 4: Gauze soaked in saline solution and chlorhexidine alternately. All groups: Autologous bone and ePTFE membrane.	6 months	Bone regeneration: Bone filling **almost complete regardless of treatment**. Re-osseointegration: Bone-implant average contact of **39% to 46% regardless of treatment**.	Conclusion: The simplest method should be the treatment of choice. For example: gauze in saline solution and chlorhexidine.

Treatment	Observation Period	Results	Comments
Laser therapy with application of hydrogen peroxide solution. Group 1: Machined surface + cotton pellets in saline solution. Group 2: SLA + cotton pellets in saline solution. Group 3: Machined surface. Group 4: SLA.	6 months	**The filling of the bone defect ranged from 72% to 82%.** Re-osseointegration as a percentage of the defect: Machined/laser 21% (0.46mm). Machined/saline solution 22% (0.42mm). SLA/laser 74% (1.13mm). SLA/saline solution 84% (1.22mm).	**The characteristics of the implant's surface are more important than the decontamination method.**
Gauze soaked in chlorhexidine and saline solution alternately. Group 1: Debridement. Group 2: Autologous bone. Group 3: Autologous bone and platelet-enriched plasma.	6 months	**Connective tissue encapsulation area** separating the bone from the implant surface in all groups. Re-osseointegration (within the three most coronal threads): Group 1: 6.5%, Group 2: 19.3% Group 3: 50.1%	

Table 3-1: Modified from Claffey N et al.[35] **Decontamination.** A main aim of periodontal therapy is to remove supra and subgingival soft and hard deposits from the root surface; this task is difficult to accomplish only by mechanical means. There are several tools available for it, such as brushes and rubber cups, plastic curettes, Teflon or titanium carbon, modified tips for ultrasound, and jet abrasion systems. No method for chemical or mechanical surface decontamination has proven to be better than another.

(GBR: Guided bone regeneration; ePTFE: Expanded polytetrafluoroethylene; SLA: Sand blasted large grit acid etched).

surgical treatment. Laser achieves the highest level of re-osseointegration. However, this approach requires further evaluation. We do not yet know whether adjunctive use of a systemic antibiotic could be helpful.

Implantoplasty (correction of the rough surface of the implant and elimination of threads to achieve a smooth and polished surface) has proven to be the most effective method to stop bone loss. By combining implantoplasty and surgical treatment, we increase the survival of implants and observe a greater decrease of probing depths and bone loss.[42] For this reason, we recommend implantoplasty for the decontamination of surfaces *(Figures 3-5)*.

TYPES OF SURGERY

Three approaches can be used for the surgical treatment of peri-implantitis:

1. Using **access surgery** we lift a full-thickness flap to access the surface of the implant and can therefore decontaminate the surface and debride the bone defect *(Figures 3-6)*.

2. Using **resection techniques**, we also perform apical repositioning techniques, with the removal of soft and hard tissue to reduce the pocket. One section of the implant surface will also be exposed to facilitate patient hygiene *(Figures 3-7)*.

Figure 3-5 a

Figure 3-5 b

Figure 3-5 c

Figure 3-5 d

Figure 3-5 e

Figures 3-5. **Implantoplasty.** Sequence of burs used: a) diamond bur; b) and c) ceramic polishers (Arkansas stone and silica); d) and e) metal polishers (rubber tips for polishing amalgam).

Figure 3-6 d

Figure 3-6 e

Figure 3-6 f

Figure 3-6 g

Figures 3-6. d) preoperative clinical image; e) full thickness flap: intraoperative view of implants, where plaque deposits attached to the implant surfaces can be seen; f) debridement of contaminated peri-implant tissues, where we observe that the bone defect is horizontal and with no intraosseous component that enables us to consider a regenerative treatment; g) implantoplasty by means of rotating instruments.

Figure 3-6 a

Figure 3-6 b

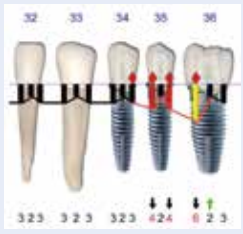


Figure 3-6 c

Figures 3-6. **Access surgery.** a) Clinical image of a patient in whom we detected bleeding and increased probing depths; b) pre-surgical radiographic view in which peri-implant bone loss can be observed; c) data from the periodontal examination prior to treatment.

Figure 3-6 h

Figure 3-6 i

Figure 3-6 j

Figures 3-6 images 2 years after surgery. h) Remains with no bleeding and with no pockets with a stable bone level in the radiographic image. As a result of the treatment there was a recession of the peri-implant mucosa. i) radiograph in which bone stability is confirmed and where the area of the implant in which we remove the threads by means of implantoplasty can be observed; j) periodontal records at 24 months after treatment.

In both access and resection surgery we can use antimicrobials and antiseptics as adjunctive treatments.

3. Finally, using **regenerative techniques** we endeavor to recover the bone tissue lost through the use of biomaterials, grafts, bone substitutes *(Figures 3-8)* or barrier membranes *(Figures 3-9)*.

As we will see, these three types of surgeries are not mutually exclusive and can be combined to adapt to each case *(see Combined Therapies)*.

CRITERIA USED TO SELECT THE TYPE OF SURGERY

Different surgical techniques can yield different results, depending on the situation. Although long-term studies are still needed, we can provide a number of recommendations:

- A recent review[43] by Chan et al. *(Table 3-2)* established that we can expect a 2-3mm reduction in probing depths (PD) as a result of surgical treatment, regardless of the type used. The results of regenerative treatments, with or without membrane, show a maximum PD reduction of 5.4mm[44, 45] and 2mm bone filling. However, although bone filling is observed in regenerative treatments, this type of treatment is the least predictable and offers the greatest variability in terms of results.

Figure 3-7 a

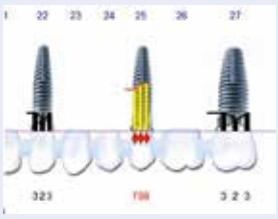


Figure 3-7 c

Figure 3-7 b

Figures 3-7. **Resection surgery.** a) Clinical image of a patient with a maxillary hybrid prosthesis in which bleeding and increased probing depths are retained in one of the implants (position 2.3) following the non-surgical treatment phase; b) pre-surgical radiographic view in which peri-implant bone loss can be observed; c) data from periodontal examination.

Figure 3-7 d

Figure 3-7 e

Figure 3-7 f

Figure 3-7 g

Figure 3-7 h

Figure 3-7 i

Figures 3-7. d) supracrestal horizontal incision and distal vertical discharge; e) full thickness flap that enables us to perform surgical debridement of soft and hard tissue; f) implantoplasty of the contaminated surface of the implant; g) placement of a connective tissue graft to compensate for postoperative retraction; h) discontinuous suture; i) immediate postoperative period (2 months).

Figure 3-7 j

Figure 3-7 l

Figure 3-7 k

Figures 3-7. Postsurgical images. j) Bleeding and peri-implant pockets disappear; k) x-ray in which bone stability is confirmed and where the area of the implants in which we remove threads by implantoplasty of the surface can be observed; l) post-treatment periodontal records.

Figure 3-8a

Figure 3-8 b

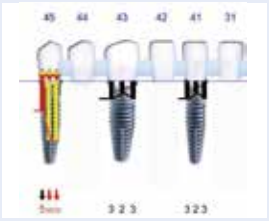


Figure 3-8 c

Figures 3-8. **Regenerative surgery.** a) Buccal and lingual incisions at the level of a distal abutment implant of a hybrid lower prosthesis in which bleeding and increased probing depths are maintained following the non-surgical treatment phase; b) pre-surgical radiographic view in which we can see peri-implant bone loss with the possibility of performing regenerative treatment; c) data from periodontal examination.

Figure 3-8 h

Figure 3-8 i

Figure 3-8 j

Figures 3-8. h) clinical condition of soft tissue at 12 months; i) x-ray in which the bone filling of the intraosseous component and bone stability of the suprabony area in which we perform the implantoplasty is verified; j) post-treatment periodontal records.

Figure 3-8 d

Figure 3-8 e

Figure 3-8 f

Figure 3-8 g

Figures 3-8. d) flaps in position; e) clinical situation after performing implantoplasty of the suprabony area of the implant; f) decontamination of the area of the intracrestal implant and filling of the defect with a bovine xenograft; g) discontinuous suture. Non-submerged healing.

Figure 3-9 a

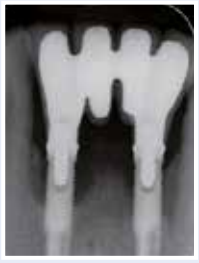


Figure 3-9 b

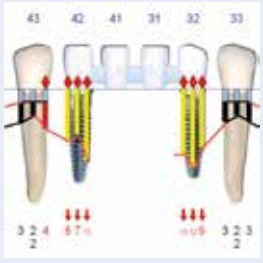


Figure 3-9 c

Figures 3-9. **Regenerative surgery:** a) Clinical diagnosis in which we detect increased probing depths at the level of implants inserted in positions 32 and 42, in addition to fixed prosthetic abutments to replace the mandibular incisors; b) pre-surgical radiographic view in which we can observe peri-implant bone loss with the possibility of performing regenerative treatment; c) data from periodontal examination.

Figure 3-9 d

Figure 3-9 e

Figure 3-9 f

Figure 3-9 g

Figure 3-9 h

Figure 3-9 i

Figure 3-9 j

Figures 3-9. d) intraoperative view of implants after raising full thickness flap where we observe the granulation tissue associated with the implant surface; e) debridement and decontamination of both implants by means of titanium curettes, serum and povidone iodine, with the bone defects affecting both implants remaining clear; f) filling of the defect with a bovine xenograft; g) placement of an ePTFE membrane with titanium reinforcements which we fix using titanium screws; h) discontinuous suture and submerged healing; i) removal of the membrane; j) condition of soft tissue at 6 weeks, after connecting the implants with healing abutments.

Figure 3-9 k

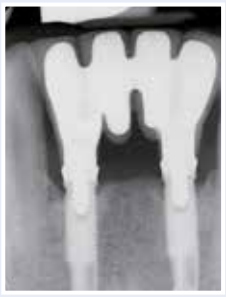


Figure 3-9 l

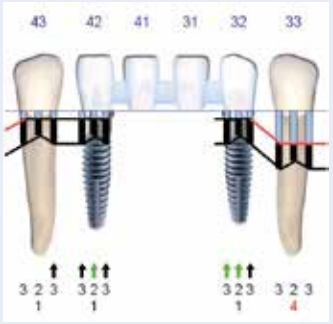


Figure 3-9 m

Figures 3-9. k) clinical view of soft tissue in postoperative review at 2 years. The patient wanted to keep the same prosthesis; soft tissue loss can be observed; l) radiograph in which the bone filling achieved is confirmed; m) data from periodontal examination.

- We now know that better clinical and radiographic results are obtained with non-regenerative surgical techniques (access or resection) compared with non-surgical treatment. We must use these techniques for defects with a low or zero potential for regeneration. With implantoplasty, we achieve more stable results in terms of bone loss.

The characteristics of the peri-implant bone defect, its configuration, and location will indicate the technique of choice in each case (Figures 3-10):

a. **For intra-bony circumferential defects** regenerative techniques should be used.

b. **For fundamentally supra-bony component defects** resection surgery is used when the area involves little or minor aesthetic aspects.

c. **For defects in areas involving an aesthetic aspect or for initial peri-implant defects,** non-surgical treatments will be used. If we do not achieve good results with these treatments, we will opt for access surgery.

COMBINED THERAPIES: FACTORS TO BE TAKEN INTO ACCOUNT

A wide variability of results with the proposed treatments for resolving peri-implantitis can be observed. Therefore, we propose a combination of therapies, associating resection surgery with implantoplasty and bone regeneration *(Figures 3-8)*. In many cases, after implantoplasty in the supracrestal area of the defect and in the dehiscence, we attempt to regenerate the intra-bone component using bone grafts and a resorbable membrane *(Figures 3-9)*. Good short- and medium-term clinical and radiographic outcomes have been achieved with this approach.[47]

Despite the heterogeneity of available studies, everything points to the fact that the surgical treatment of peri-implantitis is a predictable method for controlling the clinical course of the disease, and that patients who receive it obtain at the very least a short-term benefit. When assessing the factors to consider for selecting the type of treatment, it is worth highlighting: the area of the mouth where the problem is located, the amount of bone loss, intrasurgical anatomy of the bone defect, and biomaterials to be used.

Procedure		PD Reduction (mm)	PD Reduction (%)	Rx (mm)	CIL Gain (mm)	CIL Gain (%)	BOP Reduction (%)	Rec (mm)
Access surgery and debridement	No. of Studies	4	4	1	2	1	2	2
	Results	2.38 + 0.53	37.9	0.1 + 1.9	1.20 + 2.11	2.22	41.1	1.31 + 0.61
Resection surgery	No. of Studies	2	2	N/A	1	1	1	1
	Results	2.04 + 0.15	33.4	N/A	0.6	-4.3	21.2	1.44 + 0.39
Bone grafts or substitutes	No. of Studies	5	4	6	1	1	3	2
	Results	2.32 + 1.29	37.1	2.10 + 0.56	0.6 + 0.5	8.2	39.6	0.87 + 0.88
Grafts + Membranes	No. of Studies	11	11	7	7	6	6	6
	Results	3.16 + 0.62	48.2	2.16 + 0.80	1.99 + 0.46	28.1	50.2	0.39 + 0.28

Table 3-2: Modified from Chan HL et al.[42] Summary of the meta-analysis results by Chan et al.
As we see, we can expect a 2 to 3mm reduction in the probing depths as a result of surgical treatments. The results show 2mm of bone filling in regenerative treatments, with or without membrane.
(PD: probing depth; Rx: radiographic bone filling; CIL: clinical insertion level; BOP: bleeding on probing; Rec: mucosal recession).

Schwarz defect Ia: Bone, vestibular or lingual dehiscence-type defect.

Schwarz defect Ib: Bone, vestibular, or lingual dehiscence-type defect. Semi-circular bone reabsorption at the center of the implant body.

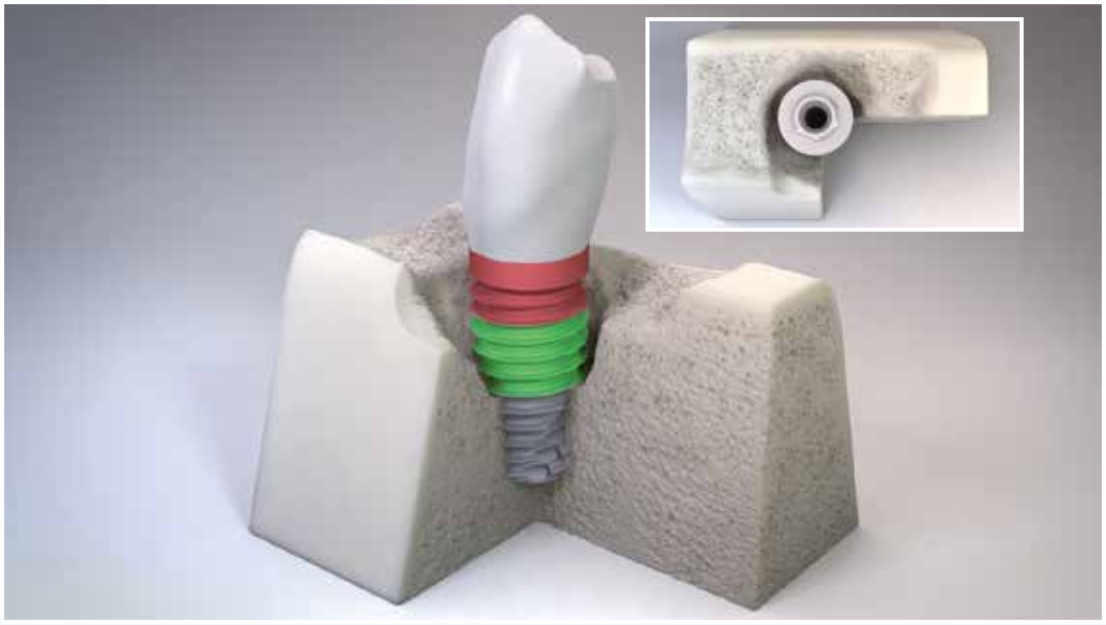
Schwarz defect Ic: Bone, vestibular, or lingual dehiscence-type defect. Circular bone reabsorption on maintenance of the lingual or vestibular cortical.

Schwarz defect Id: Circumferential bone reabsorption. Loss of the buccal and palatal/lingual cortical plates.

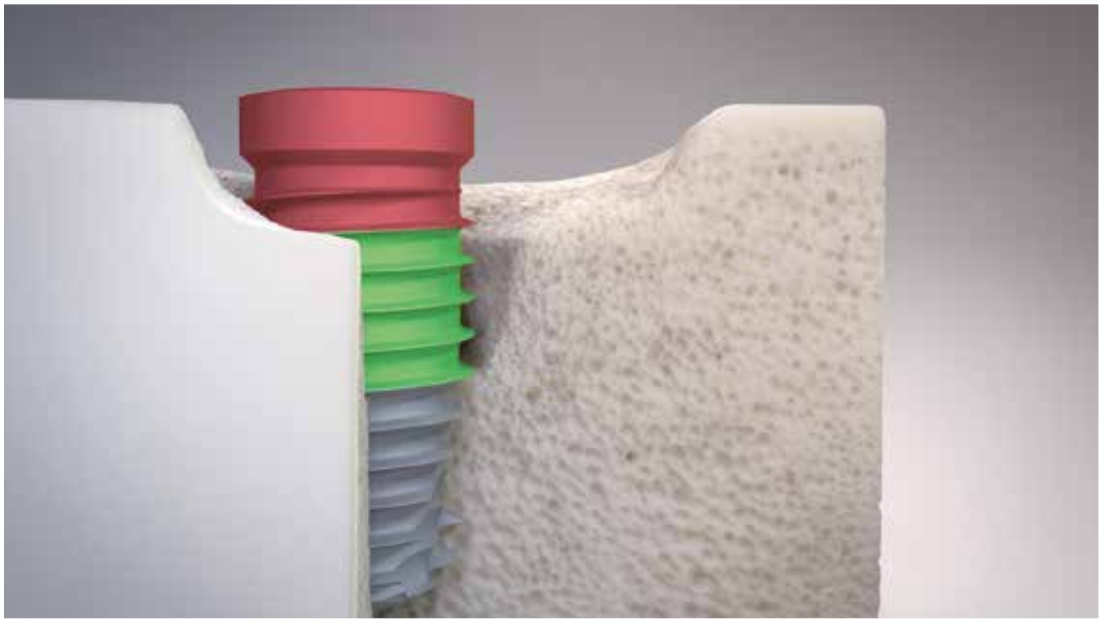
Schwarz defect: Circumferential bone reabsorption. Buccal and palatal/lingual cortical plates maintained.

Schwarz defect II: Supra-alveolar defect.

Figure 3-10a. **Peri-implant defects according to Schwarz et al.** [45]



Combined Schwarz defect Ie + II: Intraosseous circumferential and horizontal.



Lateral view of the combined defect. Defect part above the bone in red and intraosseous part in green.

Figure 3-10b. **Combined defects.** Most bone defects caused by peri-implantitis enable us to use combined treatments. In the area above the bone (in red) we perform resection or access surgery techniques in aesthetic areas; and in the intraosseous area (in green), we perform regenerative surgery techniques. In both cases we need to treat the surface of the implant: at the level above the bone we perform implantoplasty, while at the intraosseous level we use chemical or laser techniques. We know that the characteristics of the surface of implants could have an impact on clinical outcome, such that surfaces with more roughness provide greater re-osseointegration.

The **stage of the peri-implant disease** will also guide the choice of the type of surgery. Initial peri-implantitis (bone loss < 25% of the length of the implant), moderate bone loss (25% to 75%), and advanced bone loss (> 75%) require different treatment protocols *(Figure 3-11: See decision tree)*. For mucositis, non-surgical mechanical treatment and adjunctive use of antiseptic mouthwashes may be effective. For initial peri-implantitis, we recommend using the same treatment, accompanied by local and systemic antibiotics, and then evaluating surgery based on the patient's response. For moderate peri-implantitis, we recommend starting with surgical treatments, while for advanced phases, we also recommend removing the implant[48] *(Figures 3-12)* in cases with mobility, extreme malpositioning, and extensive bone loss (over two-thirds of the length of the implant) and retentive defects that are able to be regenerated.

TREATMENT 3
CORRECTIVE PHASE
Resolve the bone defect and obtain 2mm of masticatory mucosa

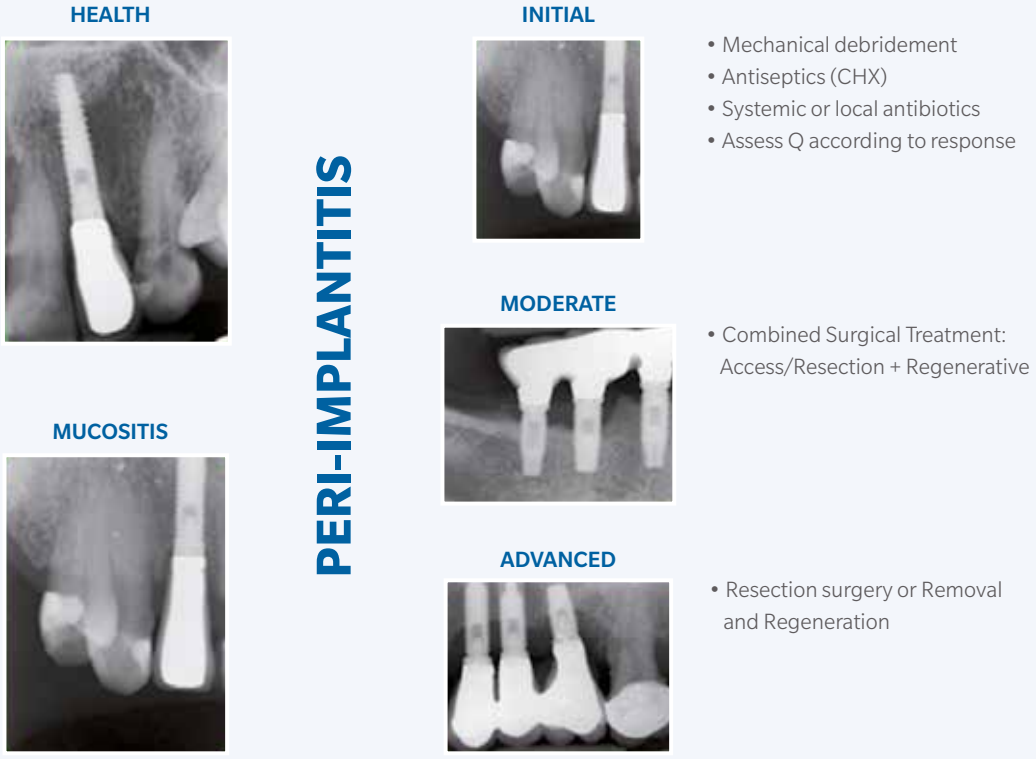


Figure 3-11 **Decision tree.** For mucositis, non-surgical mechanical treatment and adjunctive use of antiseptic mouthwashes may be effective. For initial periodontitis, it is advisable to apply the same treatment, accompanied by local and systemic antibiotics to then evaluate a surgical approach according to the patient's response. For moderate periodontitis, we recommend combined surgical treatments, while for advanced periodontitis we also recommend removal of the implant (Figure 3-12) in cases in which there is mobility, extreme malpositioning, extensive bone loss exceeding 2/3 of the length of the implant and retentive defects that may be regenerated.
Serino G, Turri A. Outcome of surgical treatment of peri-implantitis: results from a 2-year prospective clinical study in humans. Clin Oral Imp Res 2011; 22: 1214–1220.

REGENERATIVE BONE TREATMENT

After halting the course of peri-implantitis and removing the inflammation, one treatment goal is to **fill the defect with bone and achieve re-osseointegration**. As we have already mentioned, although 2mm of bone filling and up to a 5.4mm reduction in probing depth are observed with regenerative treatments, they are less predictable and produce a wider variability of results. However, we must determine the cases for which it is most suited.

Figure 3-12 a

Figure 3-12 b

Figures 3-12. **Removal**. There are cases in which the implant's viability is not possible, and we have to resort to extracting the implant.

- If the lesion around the implant is crater-shaped, especially in more aesthetic areas, regenerative techniques should be used. Regenerative procedures using grafts with or without a membrane are those that obtain the best results. However, they are also the least predictable. So far, there is no evidence to recommend the use of a bone or another type of graft (autogenous, autologous, or xenograft) and the use of a membrane remains controversial, probably because its exposure is the most common complication encountered with this type of technique, meaning that results can be compromised.

Bone filling and re-osseointegration are determined by:

- Anatomy and configuration of the defect
- Various configurations of implant surfaces
- Presence of masticatory mucosa

We must remember that regenerative approaches do not resolve the inflammation, but aim to resolve the bone defects created by the disease. Bearing this in mind, we can recommend these techniques in order to achieve better medium- to long-term results from our treatments. They should also be considered in highly aesthetic areas when the defects allow such techniques.

MUCOSAL CORRECTIVE TREATMENT

The Third European Workshop on Periodontology concluded that, from a clinical perspective, there was no difference in prognosis for implants with high hygiene levels, regardless of whether they were surrounded by masticatory mucosa or alveolar mucosa.[49] Hindsight has now given us another perspective. At present, we know that at least 2mm of masticatory mucosa around the implants offers protection against bone loss[50, 51, 52] and improves aesthetic results.[53]

For this reason, we must take advantage of the placement of implants to increase masticatory mucosa levels. It is also important to use surgical access for the etiological treatment of peri-implantitis to increase masticatory mucosa levels via a submucosal connective tissue graft to compensate for the shrinking of the peri-implant margin that occurs when resolving the inflammation.[54] Finally, we can consider correcting mucogingival peri-implant defects. However, we must take into account the special characteristics of peri-implant tissues, which hinder the already low predictability of such procedures.

TREATMENT 4
FOLLOW-UP OR MAINTENANCE PHASE
Risk factors and prevention

RISK FACTORS

Due to the success of our treatments, both over the short and long term, one of our objectives will be to monitor the risk factors that have been identified as involved in the onset and development of peri-implant diseases. We know that bacterial accumulation in the form of a *biofilm* is the main etiological factor of these diseases. There is strong evidence that poor oral hygiene, a history of periodontitis, and consumption of tobacco are risk factors for peri-implantitis.[55]

However, according to the literature, other factors may also be involved *(Table 3)*. Some depend on the patient (genetics, diabetes, and alcohol intake), others on local conditions (cement, material, and connection of the prosthesis; maintenance of biological space; microbiology; occlusion, and tissue), and, finally, the characteristics of the implant (design, material, 3D positioning, immediate technique, and platform switching) have also been mentioned.

However, as we stated above, the strongest evidence points to poor hygiene, a history of periodontitis, and smoking, while the association with other factors is less well established.

PREVENTION

As with any disease, prevention is the best form of treatment. Monitoring requires the regular scheduling of appointments to methodically re-evaluate the situation to determine, if necessary, a treatment tailored to the clinical findings.

Therefore, every patient with a dental implant must be:

1. **Trained in oral hygiene techniques.** The patient's ability to maintain good oral hygiene is a prerequisite for the long-term success of our treatments. Even if we manage to treat the peri-implantitis successfully, we will fail if we make the mistake of not providing adequate training and encouraging patients to maintain oral hygiene levels as part of their rehabilitation, as these are crucial elements in the development or reactivation of peri-implant diseases.

2. **Advised about risk factors** *(Table 3)*. As mentioned above, it is very important for the success of our treatments.

3. **Included in a monitoring program**, meaning that patients are evaluated at regular intervals to monitor the condition of their peri-implant tissues, to verify their oral hygiene, to monitor plaque levels, and remove supra- and sub-gingival *biofilm (see in Clinical Examination Figure 2-1)*. Moreover, when relapses are identified, we will need to re-treat the patients. It has been shown[56, 57] that a lack of monitoring results in a higher incidence of peri-implant diseases, so this phase is key to the long-term success of implant therapy.

The follow-up plan includes *Monitoring* and *Actions* or *Treatment*, meaning that when signs occur, we may have to make decisions not initially planned.

a. **Monitoring.** This implies a diagnosis, *(see the Diagnosis section)* to detect the disease at an early stage and the sharing of responsibility between the professional and the patient, who must be an active part of this phase. To this end, we must increase the patients' awareness of the risk factors that may be present and teach them to recognize the signs of potential problems (inflammation, spontaneous bleeding, or when using hygiene devices) to ensure patients seek professional advice when the first warning signs appear and take appropriate actions.

Patient factors:
- Genetics
- Poor oral hygiene
- Smoking
- Periodontal condition
- Systemic diseases: diabetes
- Alcohol intake

Local conditions:
- Prosthesis
  - Cemented
  - Materials
  - Connection/disconnection
- Maintenance of biological space/Platform switching
- Microbiology
- Occlusion
- Tissues

Implant factors:
- Design
- Materials
- Immediate implant
- 3D Placement
- Platform switching

Table 3. **Risk Factors**
There are different factors that can have a negative impact on the clinical course of peri-implant diseases. The existence of these factors is not equally distributed within the population. Therefore, there are patients with a greater likelihood of suffering from peri-implant problems. The long-term success of our treatments largely depends on awareness and monitoring of these factors.

They must cover:

- Analysis of systemic conditions: poorly controlled diabetes, tobacco, etc.
- Evaluation of the patient's peri-implant and periodontal status (residual pockets).
- Evaluation of the prosthesis condition, which must be verified to determine whether the patient's hygiene technique is adequate and correct the situation if this is not the case.

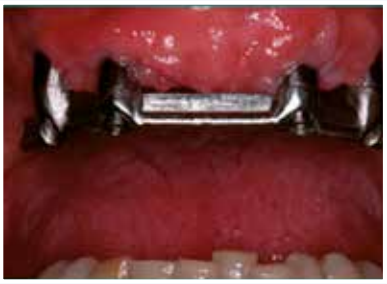
b. Action or Treatment. This phase is preventive and potentially therapeutic. It should be tailored to the diagnosis made during the monitoring phase. During this phase, the causes and risk factors of the case will be monitored. Normally, this merely involves removing soft or calcified deposits with plastic instruments and mechanically cleaning the implants with rubber polishers and prophylaxis paste *(step A of the CIST protocol)*.[58] However, all patients should be subject to two actions: strengthen oral hygiene and assess the need for mechanical treatment *(see PIITN)*. In addition, depending on the type of prosthesis, we conducted a series of actions to verify not only the patient's health, but also the condition of the components *(Figures 4-1)*. If we detect disease, we will start actively treating the peri-implantitis.

SCHEDULING MONITORING SESSIONS

The scheduling of monitoring appointments will vary depending on the patient's risk profile. In view of the risk factors outlined and from a practical point of view, we differentiate from the outset between two types of patients based on their periodontal history: patients with current or previous periodontitis and patients without, or with no history of periodontitis. However, this is not a rigid classification, and it will vary according to the clinical course of each patient, as patients will either remain in their initially assigned group or may change.

a. Patients who have or have had periodontitis. We apply the "Bern spider" model,[59] which evaluates six risk factors and establishes a concrete diagnosis, prognosis, and treatment schedule based on the resulting profile. Patients are evaluated at the end of the active treatment and, after this, at regular intervals. In addition, the graph provides information to patients and encourages them, while also justifying the schedule proposed by the professional. High levels of patient cooperation will highlight the gradual reduction of the overall risk. The hexagon or spider takes into account local and systemic factors, in addition to alterable and unalterable factors, and distinguishes between high, medium and low risk *(Figure 4-2)*.

Overdentures

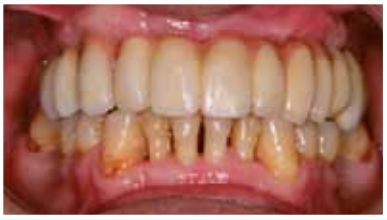


· Removable = easier access for hygiene.
· Review screws and abutments every year.

Dento-alveolar

· Remove and clean annually.
· Review screws and abutments (fracturing and loosening) every year.

Dental



· Remove and clean annually.
· Review screws and abutments (fractures and loosening) every 2 years.

Figure 4-1. The design and type of prosthesis are very important local factors. A patient with a prosthesis that is easy to clean is not the same as one with a prosthesis that is difficult to clean. In addition, there are also relevant factors when determining the frequency of sessions as part of the follow-up program.

b. Patients with no history of periodontitis. While monitoring systematic factors is also important in such patients, we believe that local factors are key in preventing peri-implant problems. This means tobacco habits and systemic pathologies should be evaluated, but we will not concentrate on those local factors which predispose to the onset of these pathologies. The placement of implants and design of the patient's implant prosthesis will impact the maintenance appointment schedule and the procedure to be carried out *(Figure 4-1)*:

- If the placement of implants and design of the prosthesis enables the patient to implement adequate oral hygiene, we will schedule sessions every 6-12 months, taking into account the general risk factors: smoking, systemic diseases, and hygiene, in a similar way to what we do with patients without an implant prosthesis who come to our clinic for conventional prophylaxis.

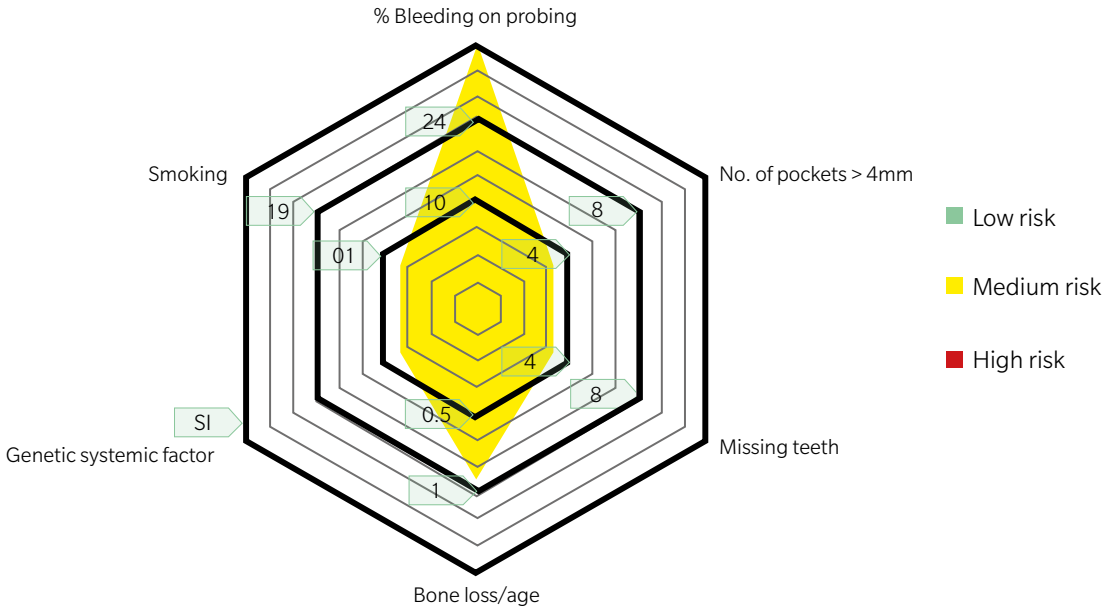


Figure 4-2 **Example of a periodontal risk spider's web based on the Bern model.** The assessments carried out at the level of the patient, tooth and dental surface. At the patient level modifiable factors such as smoking and unmodifiable factors such as genetic abnormalities or systemic diseases are taken into account. At the level of the tooth, tooth loss and bone loss related to age; and, finally, regarding local factors, bleeding and probing depth.

- If, however, the placement of implants or design of the prosthesis makes hygiene difficult (the latter being a situation that we should try to avoid by modifying the design, for example, in the case of a dentoalveolar prosthesis), we include this in Group VIII, subgroup A of the 1999 Armitage classification *(Figures 4-3; see also Figure 1-8 in section 1)* even though the patient has no history of periodontitis, given that this type of implant-supported restoration could be considered as an *Acquired Condition* that modifies or favors plaque-induced gingivitis or periodontitis. It is in these patients that verification of the probing depth and bleeding and the presence/absence of masticatory mucosa (≤1mm) are crucial to determining the frequency of visits.

CONCLUSIONS

1. Monitoring implant patients is necessary and the long-term success of our treatments is largely dependent on such follow-up.

2. All follow-up appointments must diagnose the condition of peri-implant tissues.

3. Treatment (action) will depend on the diagnosis made at any time during the follow-up.

4. The follow-up appointments protocol should be based on the clinical indices, the dependent risk factors of each patient, and the design of the implant prosthesis.

Figure 4-3 a

Figure 4-3 b

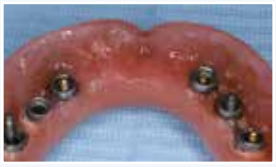


Figure 4-3 c

Figure 4-3 d

Figure 4-3 e

Figures 4-3. In some patients, local factors that predispose them to the onset of peri-implant diseases are more important. Staying with the periodontal analogy, implant-supported restorations could be considered as an Acquired Condition that favors peri-implant inflammatory diseases:
a) the proximity between implants makes patient hygiene difficult.
b) some hybrid-type prosthetic designs, such as the flanges of the prosthesis in the image, make patient hygiene virtually impossible.
c) some materials also promote adhesion of bacterial plaque. The prosthesis in the image was manufactured with a ceromer.

4. PERI-IMPLANT INDEX OF TREATMENT NEEDS (PIITN)

Combining the treatment strategy of the CIST protocol of Lang et al.[60] and the CPITN philosophy proposed by Ainamo,[61] we propose the following *Peri-implant Index of Treatment Needs* (PIITN) to facilitate decision-making when scheduling treatment for these kind of problems.

We intend to provide clinicians with a practical tool that enables them to target their decisions in light of the evidence available but without overlooking the peculiarities that each specific case can present. Table 4-1 defines the PIITN values that we must assign to patients following a clinical and radiographic examination and expresses the degree of involvement and activity of the peri-implant problem. Table 4-2 outlines the therapeutic approaches, from the least to the most aggressive, according to the involvement of the soft and hard tissues. Finally, Table 4-3 lists the recommended therapeutic approach to be performed based on the PIITN value assigned to the patient according to the findings of the examination.

Peri-implant Index of Treatment Needs (PIITN)			
Value 0	• No plaque and/or calculus • No signs of inflammation, bleeding on probing (BOP) and/or suppuration • No increase in probing depth • No radiographic bone loss		
Value 1	• Existence of plaque and/or calculus • No signs of inflammation, bleeding on probing (BOP) and/or suppuration • No increase in probing depth • No radiographic bone loss		
Value 2	• Existence of signs of inflammation and/or bleeding on probing (BOP) and/or suppuration • Increase in probing depth • No radiographic bone loss	**2a:** Probing depth ≤5mm	
		2b: Probing depth >5mm	**2b+:** Aesthetic area
			2b++: Non-aesthetic area
Value 3	• Existence of signs of inflammation and/or bleeding on probing (BOP) and/or suppuration • Increase in probing depth • Existence of radiographic bone loss	**3a:** Bone loss <25% of the length of the implant	**3a+:** Aesthetic area
			3a++: Non-aesthetic area
		3b: Bone loss 25% to 75% of the length of the implant	**3b+:** Horizontal bone defect
			3b++: Vertical bone defect
			3b+++: Combined bone defect
		3c: Loss >75% of the length of the implant	**3c+:** Replaceable defect
			3c++: Non-replaceable defect
Value 4	• Implant mobility • Serious aesthetic defect • Severe malpositioning preventing bone loss stabilization • Peri-implantitis refractory to prior treatment		

Table 2

Table 4-1.

Therapeutic Levels of Peri-implant Diseases		
Approach A	Instructions on oral hygiene	
Approach B	Mechanical treatment and submucosal irrigation	**B1:** Application of local antibiotics
		B2: Prescription of systemic antibiotics
Approach C	Surgical access with surface decontamination (*) Consider connective tissue graft	**C1:** Implantoplasty
		C2: Resection techniques
		C3: Regenerative techniques
Approach D	Removal (*) Consider connective tissue graft	**D1:** Isolated removal
		D2: Removal with regeneration

Table 4-2.

PIITN VALUE			Therapeutic Approach
Value 0: Health			**A**
Value 1: Deposits			**A+B**
Value 2: Inflammation, bleeding (BOP) and/or Suppuration Probing increase	**2a:** PD ≤5mm		**A+B**
	2b: PD >5mm	**2b+:** Aesthetic area	**A+B(1)**
		2b++: Non-aesthetic area	**A+B+C**
Value 3: Radiographic bone loss	**3a:** <25%	**3a+:** Aesthetic area	**A+B1/B2**
		3a++: Non-aesthetic area	**A+B(2)+C1/C2***
	3b: 25% to 75%	**3b+:** Horizontal bone defect	**A+B(2)+C1/C2***
		3b++: Vertical bone defect	**A+B(2)+C3***
		3b+++: Combined bone defect	**A+B(2)+C1/C2+C3***
	3c: >75%	**3c+:** Replaceable bone defect	**D2***
		3c++: Non-replaceable bone defect	**A+B(2)+C1/C2***
Value 4: Therapeutic failure			**D1/D2***

Table 4-3.

REFERENCES

1. Mombelli A, van Oosten MA, Schurch E Jr, Land NP. The microbiota associated with successful or failing osseointegrated titanium implants. Oral Microbiol Immunol 2; 1987;145–151.

2. Zitzmann NU, Berglundh T. Definition and prevalence of peri-implant diseases. J Clin Periodontol 2008;35(Suppl. 8):286–291.

3. Mombelli A, Lang NP. Clinical parameters for the evaluation of dental implants. Periodontology 2000; 1994;4:81–86.

4. Mombelli A, Müller N, Cionca N. The epidemiology of peri-implantitis. Clin Oral Implants Res 2012;23:67–76.

5. Klinge B, Meyle J . EAO Consensus Report: Peri-implant tissue destruction. The Third EAO Consensus Conference 2012. Clin Oral Implants Res 2012;23 (Suppl.6):108–110.

6. Lindhe J, Meyle J. Peri-implant diseases: Consensus report of the Sixth European Workshop on Periodontology. J Clin Periodontol 2008;35 (Suppl.8):282–285.

7. Mombelli A. Prevention and therapy of peri-implant infections. In: Proceedings of the 3rd European Workshop on Periodontology. Lang NP, Karring T, Lindhe J. Eds. Quintenssence Verlags-GmbH: Berlin, 1999.

8. Tarnow D, Cho S, Wallace S. The effect of inter-implant distance on the height of inter-implant bone crest. Int J Periodontics Restorative Dent 2013;33:785–793.

9. Peng M, Fei W, Hosseini M, Gotfredsen K. Influence of implant position on clinical crown length and peri-implant soft tissue dimensions at implant-supported single crowns replacing maxillary central incisors. J Periodontol 2000;71:546-549.

10. Isidor F. Occlusal loading in implant dentistry. In: Proceedings of the 3rd European Workshop on Periodontology. Lang NP, Karring T, Lindhe J, Eds. Quintenssence Verlags-GmbH: Berlin, 1999.

11. Armitage GC. Development of a Classification System for Periodontal Diseases and Conditions. Ann Periodontol 1999;4:1–6.

12. Mombelli A, Lang NP. The diagnosis and treatment of peri-implantitis. Periodontol 2000; 1998;17:63–76.

13. Lang NP, Berglundh T. Working Group 4 of Seventh European Workshop on Periodontology. Peri-implant diseases: Where are we now? Consensus of the Seventh European Workshop on Periodontology. J Clin Periodontol 2011;38 (Suppl. 11):178–181.

14. Roos-Jansåker AM, Renvert H, Lindhahl H, Renvert S. Nine- to fourteen- years follow-up of implant treatment. Part III: Factors associated with peri-implant lesions. J Clin Periodontol 2006;33: 296–301. Workshop on Periodontology. J Clin Periodontol 2011;38 (Suppl.11):178–181.

15. Fransson C, Wennstrom J, Berglundh T. Clinical characteristics of implants with history of progressive bone loss: A prospective study. Clin Oral Implants Res 2008;19:142–147.

16. Schou S, Holmstrup P, Stoltze K, Hjørting-Hansen E, Fiehn NE, Skovgaard LT. Probing around implants and teeth with healthy or inflamed peri-implant mucosa/gingiva. A histologic comparison in cynomolgus monkeys (*Macaca fascicularis*). Clin Oral Implants Res 2002;13:113–126.

17. Mombelli A, Muhle T, Bragger U, Lang NP, Burgin WB. Comparison of periodontal and peri-implant probing by depth-force pattern analysis. Clin Oral Implants Res 1997;8:448–454.

18. Heitz-Mayfield LJA. Peri-implant diseases: diagnosis and risk indicators. J Clin Periodontol 2008;35 (Suppl. 8):292–304.

19. Etter TH, Hakanson I, Lang NP, Trejo P, Caffese R. Healing after standardized clinical probing of the peri-implant soft tissue seal: A histomorphometric study in dogs. Clin Oral Implant Res 2002;13:571–580.

20. Luterbacher S, Mayfield L, Brägger U, Lang NP. Diagnostic characteristics of clinical and microbiological tests for monitoring periodontal and peri-implant mucosal tissue conditions during supportive periodontal therapy (SPT). Clin Oral Implants Res 2000;11:521–529.

21. Jepssen S, Rhüling A, Jepsen K, Ohlenbusch B, Albers H. Progressive peri-implantitis. Incidence and prediction of peri-implant attachment loss. Clin Oral Implants Res 1996;7:133–142.

22. Luterbacher S, Mayfield L, Brägger U, Lang NP. Diagnostic characteristics of clinical and microbiological tests for monitoring periodontal and peri-implant mucosal tissue conditions during supportive periodontal therapy (SPT). Clin Oral Implants Res 2000;11:521–529.

23. Heitz-Mayfield LJA. Peri-implant diseases: Diagnosis and risk indicators. J Clin Periodontol 2008;35 (Suppl. 8):292–304.

24. Lang NP, Berglundh T. On Behalf of Working Group 4 of the Seventh European Workshop on Periodontology: Peri-implant diseases: Where are we now? Consensus of the Seventh European Workshop on Periodontology. J Clin Periodontol 2011;38 (Suppl. 11):178–181.

25. Hollender L, Rockler B. Radiographic evaluation of osseointegrated implants in the jaw. Experimental study of the influence of radiographic techniques on the measurement of the relation between the implant and bone. Dentomaxillofac Radiol 1980;9:91–95.

26. Sewerin IP. Errors in radiographic assessment of marginal bone height around osseointegrated implants. Scand J Dent Res 1990;98:428–433.

27. Lang NP, Berglundh T. On Behalf of Working Group 4 of the Seventh European Workshop on Periodontology: Peri-implant diseases: Where are we now? Consensus of the Seventh European Workshop on Periodontology. J Clin Periodontol 2011;38 (Suppl.11):178–181.

28. Luterbacher S, Mayfield L, Brägger U, Lang NP. Diagnostic characteristics of clinical and microbiological tests for monitoring periodontal and peri-implant mucosal tissue conditions during supportive periodontal therapy (SPT). Clin Oral Implants Res 2000;11:521–529.

29. Feloutzis A, Lang NP, Tonetti MS, Bürgin W, Brägger U, Buser D, Duff GW, Kornman KS. IL-1 gene polymorphism and smoking as risk factors for peri-implant bone loss in a well-maintained population. Clin Oral Implants Res 2003;14:10–17.

30. Gruica B, Wang HY, Lang NP, Buser D. Impact of IL-1 genotype and smoking status on the prognosis of osseointegrated implants. Clin Oral Implants Res 2004;15:393–400.

31. Jansson H, Hamberg K, DeBruyn H, Brattha G. Clinical consequences of IL-l genotype on early implant failures in patients under periodontal maintenance. Clin Implant Dent Relat Res 2005;1:51–59.

32. Mombelli A. Prevention and therapy of peri-implant infections. In: Proceedings of the 3rd European Workshop on Periodontology. Lang NP, Karring T, Lindhe J. Eds. Quintenssence Verlags-GmbH: Berlin, 1999: 281–303.

33. Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thornsen P. The soft tissue barrier at implants and teeth. Clin Oral Implants Res 1991;2:81–90.

34. Ramfjord SP. A rational plan for periodontal therapy. J Periodontol 1953;24:75–87.

35. Lindhe J, Meyle J.Peri-implant diseases: Consensus report of the Sixth European Workshop on Periodontology. J Clin Periodontol 2008;35 (Suppl. 8):282–285.

36. Renvert S, Roos-Jansaker AM, Claffey N. Non-surgical treatment of peri-implant mucositis and peri-implantitis: A literature review. J Clin Periodontol 2008;35 (Suppl. 8):305–315.

37. Claffey N, Clarke E, Polyzois I, Renvert S. Surgical Treatment of Peri-implantitis. J Clin Periodontol 2008;35 (Suppl 8):316–332.

38. Lindhe J, Meyle J. Peri-Implant diseases: Consensus report of the Sixth European Workshop on Periodontology. J Clin Periodontol 2008;35 (Suppl 8):282–285.

39. Schwarz F, Sculean A, Romanos G, Herten M, Horn N, Scherbaum W, Becker J. Influence of different treatment approaches on the removal of early plaque biofilms and the viability of SAOS2 osteoblasts grown on titanium implants. Clin Oral Investig 2005; 9:111–117.

40. Schwarz F, Rothamel D, Sculean A ,Georg T, Scherbaum W, Becker J. Effects of an Er:YAG laser and the Vector ultrasonic system on the biocompatibility of titanium implants in cultures of human osteoblast-like cells. Clin Oral Implants Res 2003;14:784–792.

41. Schwarz F, Jepsen S, Herten M, Sager M, Rothamel D, Becker J. Influence of different treatment approaches on non-submerged and submerged healing of ligature induced peri-implantitis lesions: An experimental study in dogs. Journal of Clin Periodontol 2006;33:584–595.

42. Romeo E, Ghisolfi M, Murgolo N, Chiapasco M, Lops D, Vogel G. Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped implants. Part I: Clinical outcome. Clin Oral Implants Res 2005;16:9–18.

43. Chan HL, Lin GH, Suarez F, MacEachern M, Wang HL. Surgical management of peri-implantitis: A systematic review and meta-analysis of treatment outcomes. J Periodontol 2014;85:1027–1041.

44. Froum SJ, Froum SH, Rosen PS. Successful management of peri-implantitis with a regenerative approach: A consecutive series of 51 treated implants with 3- to 7.5-year follow-up. Int J Periodontics Restorative Dent 2012;32:11–20.

45. Khoury F, Buchmann R. Surgical therapy of peri-implant disease: A 3-year follow-up study of cases treated with 3 different techniques of bone regeneration. J Periodontol 2001;72:1498–1508.

46. Schwarz F, Sahm N, Schwarz K, Becker J. Impact of defect configuration on the clinical outcome following surgical regenerative therapy of peri-implantitis. Journal of Clin Periodontol 2010;37: 449–455.

47. Schwarz F, Sahm N, Becker J. Combined surgical therapy of advanced peri-implantitis lesions with concomitant soft tissue volume. Clin Oral Implants Res 2014;25:132–136.

48. Serino G, Turri A. Outcome of surgical treatment of peri-implantitis: Results from a 2-year prospective clinical study in humans. Clin Oral Implants Res 2011;22:1214–1220.

49. Tonetti M, Sanz M. Consensus report of session B. Tissue integration. In: Proceeding of the 3rd European Workshop on Periodontology. Lang NP, Karring T, Lindhe J. Eds. Quintessence: London, 1999:185–188.

50. Costa FO, Takenaka-Martinez S, Costa OM, Ferreira SD, Silva GLM, Costa JE. Peri-implant disease in subjects with and without preventive maintenance: A 5-year follow-up. J Clin Periodontol 2012;39:173–181.

51. Linkevicius T, Apse P, Grybauskas S, Puisys A. The influence of soft tissue thickness on crestal bone changes around implants: A 1-year prospective controlled clinical trial. Int J Oral Maxillofac Implants 2009;24:712–719.

52. Puisys A, Linkevicius T. The influence of mucosal tissue thickening on crestal bone stability around bone-level implants. A prospective controlled clinical trial. Clin Oral Implants Res 2015;26:123–129.

53. Jung R, Sailer I, Hämmerle C, Attin T, Schmidlim P. In vitro color changes of soft tissues caused by restorative materials. Int J Periodontics Restorative Dent 2007;27:251–257.

54. Schwarz F, Sahm N, Becker J. Combined surgical therapy of advanced peri-implantitis lesions with concomitant soft tissue volume augmentation. A case series. Clin Oral Implants Res 2014;25:132–136.

55. Heitz-Mayfield LJ. Peri-implant diseases: Diagnosis and risk indicators. J Clin Periodontol 2008;35 (Suppl 8):292–304.

56. Matarasso S, Rasperini G, Iorio Siciliano V, Salvi GE, Lang NP, Aglietta M. A 10-year retrospective analysis of radiographic bone-level changes of implants supporting single-unit crowns in periodontally compromised vs. periodontally healthy patients. Clin Oral Implants Res 2010;21:898–903.

57. Pjetursson BE, Helbling C, Weber HP, Matuliene G, Salvi GE, Brägger U, Schmidlin K, Zwahlen M, Lang NP. Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. Clin Oral Implants Res 2012;23:888–894.

58. Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: Their prevention, diagnosis and treatment. Clin Oral Implants Res 2000;11 (Suppl. 1):146–155.

59. Lang NP, Tonetti MS. Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). Oral Health Prev Dent 2003;1:7–16.

60. Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: Their prevention, diagnosis and treatment. Clin Oral Implants Res 2000;11 (Suppl. 1):146–155.

61. Ainamo J, Barmes D, Beagrie G, Cutress T, Martin J, Sardo-Infirri J. Development of the World Health Organization (WHO) Community Periodontal Index of Treatment Needs (CIPTN). International Dental Journal 1982;32:281–291.

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[1] Östman PO†, Wennerberg A, Albrektsson T. Immediate Occlusal Loading Of NanoTite™ PREVAIL® Implants: A Prospective 1-Year Clinical And Radiographic Study. Clin Implant Dent Relat Res. 2010 Mar;12(1):39-47. n = 102.

† Dr. Östman has a financial relationship with Biomet 3i LLC resulting from speaking engagements, consulting engagements and other retained services.

* 0,37 mm bone recession not typical of all cases.

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