Diagnostic Parameters for Monitoring Peri-implant Conditions

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Purpose: *To review the literature on clinical, radiographic, and biochemical parameters used for monitoring peri-implant conditions.* ***Materials and Methods:*** *A MEDLINE search was conducted that included articles published in English until the end of August 2003. Results from human and experimental animal studies are presented.* ***Results:*** *The parameters that may be used to assess the presence of peri-implant health and the severity of peri-implant disease include plaque assessment, mucosal conditions, peri-implant probing depth, width of the peri-implant keratinized mucosa, peri-implant sulcus fluid analysis, suppuration, implant mobility and discomfort, resonance frequency analysis, and radiographic evaluation.* ***Discussion:*** *Based on the analysis of the available evidence, it appears reasonable to use a number of clinical and radiographic parameters to discriminate between peri-implant health and disease.* ***Conclusions:*** *Systematic and continuous monitoring of peri-implant tissues during maintenance care is recommended for the early diagnosis of peri-implant disease.* INT J ORAL MAXILLOFAC IMPLANTS 2004;19(SUPPL):116–127

Key words: *clinical parameters, dental implants, dental radiography, diagnosis, long-term evaluation, peri-implant disease*

Oral endosseous implant systems with 2 different healing modalities (submerged and nonsubmerged) have been developed and used successfully for the rehabilitation of partially or completely edentulous patients.[1–20] Knowledge of the biology of osseointegration and peri-implant soft tissue healing has expanded rapidly.[21–27] A comparative study in the beagle dog[28] has provided histologic evidence that the peri-implant hard and soft tissues around 1-stage and 2-stage implant systems do not significantly differ with respect to morphology and composition. Furthermore, studies have provided clinical and radiographic evidence that 2-part implant systems can successfully osseointegrate in the mandible when a nonsubmerged surgical protocol is applied for implant placement.[29–32]

At the population level, longitudinal evaluation of oral implant systems is of primary importance for the assessment of long-term survival and complication rates of each system, for the determination of factors affecting the success of therapy, and for the identification of specific problems. At the individual level, clinical peri-implant evaluation is necessary for the detection of early signs of disease and for the planning of therapeutic interventions. An unbiased comparison of different implant systems is only meaningful if the stages of peri-implant disease are defined and if appropriate clinical parameters and indices are available.

As established in 1993 at the First European Workshop on Periodontology in Ittingen, Switzerland, *peri-implant disease* is a collective term for

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Table 1 Indices Used to Assess Plaque Accumulation Around Oral Implants

Score	Mombelli et al[40] (mPI)	Lindquist et al[47]
0	No detection of plaque	No visible plaque
1	Plaque only recognized by running a probe accross the smooth marginal surface of the implant	Local plaque accumulation
2	Plaque can be seen by the naked eye	General plaque accumulation greater than 25%
3	Abundance of soft matter	

inflammatory processes in the tissues surrounding an implant.[33] *Peri-implant mucositis* was defined as a reversible inflammatory process in the soft tissues surrounding a functioning implant, whereas *peri-implantitis* is an inflammatory process additionally characterized by loss of peri-implant bone. A subgingival biofilm formation has been shown in animal experiments and clinical studies to be an important etiologic factor for the initiation of peri-implant inflammation and subsequent loss of marginal bone.[34–37] In contrast to early implant losses, implant loss occurring during function may be the result of biologic processes characterized by clinical signs (eg, implant mobility) that emerge only when an advanced and possibly irreversible state of the disease has been reached. Therefore, the clinical and radiographic parameters routinely used to monitor oral implants during maintenance care should be of high sensitivity and/or specificity, should be easy to measure, and should yield reproducible data. The aim of this review article is to summarize current scientific evidence on the available diagnostic parameters for the longitudinal monitoring of oral implants.

MATERIALS AND METHODS

A search of Medline/PubMed was performed up to and including August 2003. The search was limited to human and experimental animal studies published in English. The following search terms were used: dental implants, peri-implant health, peri-implant disease, peri-implant mucositis, peri-implantitis, probing depth, bleeding on probing, dental plaque, peri-implant sulcus fluid, peri-implant keratinized mucosa, implant mobility, suppuration, long-term evaluation, and dental radiography. The journals *Clinical Oral Implants Research*, *Journal of Clinical Periodontology*, *Journal of Periodontology*, and *The International Journal of Oral and Maxillofacial Implant*s were searched by hand up to July 2003. The selection criteria included all levels of available evidence: systematic reviews, randomized controlled clinical trials, controlled clinical trials, prospective and retrospective cohort studies, and case reports of human and experimental animal studies. One reviewer (GES) screened titles and abstracts of the search results. The full text of publications of potential relevance was then obtained.

EVALUATION OF THE ORAL HYGIENE STANDARD

Plaque Assessment

Microbial biofilms have been shown to form on inert biomaterial surfaces in an aqueous environment.[38] Implants placed in the oral cavity represent artificial surfaces colonized by bacteria from saliva and ecologic niches such as periodontal pockets, tonsils, and crypts of the tongue. Experimental and human studies have provided evidence that formation and development of a microbial biofilm represents an important etiologic factor in the pathogenesis of peri-implant disease.[34,37,39–42] Several microbiologic features of the subgingival biofilm around implants have been correlated with the presence of clinically detectable plaque.[43] Furthermore, periodontal pathogens from residual pockets of remaining teeth in patients treated for periodontal disease have been documented to colonize oral implants.[44,45] Mombelli and coworkers[40] modified the original Plaque Index introduced by Silness and Löe[46] to assess biofilm formation in the marginal area around ITI implants (mPI) (Institut Straumann, Waldenburg, Switzerland). Lindquist and associates[47] assessed oral hygiene levels according to a 3-point scale and reported a significant relationship between oral hygiene and peri-implant bone resorption over an observation period of 6 years. Therefore, it appears meaningful to monitor oral hygiene habits by quantifying plaque accumulation. Indices used to assess plaque accumulation around oral implants are presented in Table 1.

Table 2 Indices Used to Assess Marginal Mucosal Conditions Around Oral Implants

Score	Mombelli et al[40] (mGI)	Apse et al[50]
0	No bleeding when a periodontal probe is passed along the mucosal margin adjacent to the implant	Normal mucosa
1	Isolated bleeding spots visible	Minimal inflammation with color change and minor edema
2	Blood forms a confluent red line on mucosal margin	Moderate inflammation with redness, edema, and glazing
3	Heavy or profuse bleeding	Severe inflammation with redness, edema, ulceration, and spontaneous bleeding without probing

EVALUATION OF THE PERI-IMPLANT MARGINAL TISSUES

Mucosal Conditions

In addition to redness and swelling of the marginal tissues, bleeding on probing (BOP), pocket formation, and suppuration have been reported to result from peri-implant infections.[40,48] Assessment of these clinical signs has been considered important in the diagnosis of periodontal diseases. Therefore, the definition of peri-implant parameters based on periodontal indices such as the Gingival Index System[49] (GI) seems indicated. The GI[49] has been modified and adapted (mGI) for application around oral implants,[40] while a simplified GI has been proposed by Apse and associates.[50] Indices used to assess marginal mucosal conditions around oral implants are presented in Table 2.

Around implants, however, soft tissue texture and color depend on the normal appearance of the recipient tissues before implant placement, and may be influenced by the material characteristics of the implant surface.[51] Furthermore, difficulties in recording mucosal inflammation have been reported, such as nonkeratinized peri-implant mucosa normally appearing redder than keratinized tissue.[52] In a longitudinal study, only a weak correlation between GI scores and changes in peri-implant crestal bone level was reported.[52]

Presence or Absence of Bleeding

BOP (notated in clinical records as BOP+) elicited after the insertion of a probe into the sulcus with light pressure (ie, 0.25 N) has been shown to detect the presence of an inflammatory lesion in the gingiva around teeth with a normal[53] and a healthy but reduced periodontium.[54] On the other hand, absence of bleeding on probing (BOP–) has been reported to represent periodontal health with a negative predictive value of 98.5%.[55,56] BOP has been used to assess peri-implant tissue conditions around implants. Lekholm and colleagues[57] found no correlation between BOP and histologic, microbiologic, or radiographic changes around implants. These authors hypothesized that bleeding could have been caused by inappropriate force transmission from the periodontal probe tip to the peri-implant soft tissues. These preliminary findings were confirmed in an animal study.[58] Conversely, findings from animal experiments using ITI implants yielded completely different results.[59] Healthy sites were characterized by absence of bleeding (0%), whereas both peri-implant mucositis and peri-implantitis sites showed substantially increased BOP (67% and 91%, respectively). The reason for these results might be attributed to the different probing forces applied by the various investigators. These findings were confirmed in a prospective study where absence of BOP had a high negative predictive value, thus serving as a predictor for stable peri-implant conditions.[60] Luterbacher and coworkers[61] evaluated the diagnostic characteristics of different BOP prevalences alone or in combination with a microbiologic test for monitoring periodontal and peri-implant soft tissue conditions during maintenance therapy. The authors reported that BOP alone yielded higher diagnostic accuracy at implant sites compared with tooth sites. Furthermore, when combining positive microbiologic test and BOP of 75% or more, the positive predictive values were greater for implants than for teeth. With predictive values of 100% at BOP frequencies of 50% or more at implant sites, this parameter appears to play a central role in monitoring changes in peri-implant tissue conditions.

Peri-implant Probing Depth

In contrast to natural teeth, for which average periodontal probing depth (PD) has been reported, the physiologic depth of the peri-implant sulcus of successfully osseointegrated implants has been a matter

of debate. Increasing periodontal PD and loss of clinical attachment are pathognomonic for periodontal diseases. Pocket probing is therefore an important diagnostic process for the assessment of periodontal status and for the evaluation of periodontal therapy. The extent of probe penetration is influenced by factors such as probing force and angulation, probe tip diameter, roughness of the implant or root surface, inflammatory state of the periodontium, and firmness of the marginal tissues. Furthermore, it has to be realized that PD measurements may be affected by compromised access. Data demonstrate that the periodontal probe often fails to locate the histologic level of the connective tissue attachment around teeth.[62,63]

The differences with respect to soft tissue composition, organization, and attachment between the gingiva and the root surface on the one hand and between the peri-implant mucosa and the implant surface on the other make the conditions for PD measurements around teeth and implants not fully comparable.[37,64,65] One potential explanation influencing the differences in probe penetration is that most collagen fibers in the supracrestal connective tissue compartment have been demonstrated to run mostly in a parallel direction to the implant axis.[51,66]

Ericsson and Lindhe[58] used a beagle dog model to compare the extent of probe penetration around teeth and implants under healthy soft tissue conditions. Compared with natural teeth, probe tip penetration around 2-stage submerged implants ended closer to the alveolar crest. The extent of peri-implant probe penetration has also been investigated around 1-stage oral implants in beagle dogs.[59] It was reported that density of the peri-implant tissues influences probe penetration. In the presence of inflamed peri-implant tissues, periodontal probes penetrate close to the alveolar bone, exceeding the connective tissue level by a mean of 0.52 mm. However, if healthy peri-implant conditions or mucositis are present, the probe tips may identify the histologic level of the supracrestal connective tissue attachment. One potential explanation for the different outcomes between the 2 above-mentioned studies may be attributed to the different probing forces employed (0.5 N versus 0.2 N). A recent experimental study in monkeys[65] has documented that PD measurements around teeth and implants are different. While no difference was observed with respect to probe penetration under healthy peri-implant/periodontal conditions, mild and severe marginal inflammation around implants was associated with a significantly deeper probe penetration into the supracrestal connective tissue when compared to that around teeth.

The magnitude of probe penetration into a periodontal pocket depends on the force application.[67,68] Furthermore, simultaneous recordings of probing PD and probing force before and after periodontal therapy have revealed that the force range chosen for repeated probing influences the amount of attachment level change determined.[69,70] The tissue resistance to probing and the accuracy of depth measurement at different force levels (eg, 0.25, 0.5, 0.75, 1.0, and 1.25 N) were compared around nonsubmerged ITI dental implants and teeth in 11 subjects.[71] It was concluded that peri-implant PD measurements are more sensitive to force variation than the corresponding measurements around teeth.

Correlations between bone levels recorded on radiographs and the extent of peri-implant probe penetration have been observed. In the case of screw-type implants, the probe tip appeared to stop 1.4 mm coronally to the bone level.[72] The mean discrepancy between probe penetration and the location of the bone margin in radiographs was 1.17 mm in 100 nonsubmerged hollow-screw and hollow-cylinder implants measured 1 year after implantation.[73] In general, studies have indicated that successful implants allow probe penetration of approximately 3 mm.[40,50,73–78]

Implant shape and surface texture influence penetration of the probe tip. Peri-implant probing is impossible around some implant systems because of characteristics of the design (eg, concavities, shoulders, suprastructure, or steps). Lack of surface smoothness, as with plasma-coated, sandblasted, acid-etched, or threaded implants, might interfere with probe penetration when bone resorption has reached this level and may lead to underestimation of pocket depth.[79] Although convincing evidence is lacking, some authors have expressed concerns about the possibility of introducing bacteria into the peri-implant tissues and about damaging the implant surface with a metallic periodontal probe.[58,65,77,79] Other authors concluded that increased pocket depth could be correlated with a higher degree of inflammation of the peri-implant mucosa[41,57,72,80] but not necessarily with peri-implant bone loss.[81,82] However, absolute values of PD have to be interpreted in the context of surgical implant positioning, eg, submucosal implant placement in esthetic anterior sites versus conventional implant placement in posterior nonesthetic sites. Progressive increases in PD may be an alarming sign. Therefore, the establishment of baseline PD values at the time of delivery of the prosthetic suprastructure is of critical importance in allowing comparison with future PD measurements.

Peri-implant probing should also include the location of the soft tissue margin relative to a fixed landmark point on the implant (eg, implant shoulder for

1-stage nonsubmerged implant systems) or its suprastructure. The combined full-mouth PI and the BOP scores, as well as the mean attachment level of all teeth in a group of partially edentulous subjects, were shown to significantly influence mean PD and connective tissue level around implants.[83] If peri-implantitis is associated with marginal recession, then PD alone may not accurately reflect peri-implant bone loss, whereas increasing loss of connective tissue level is a definite sign of peri-implant pathology. In a longitudinal study, Brägger and coworkers[84] found that the connective tissue level in combination with the radiographic parameters obtained 2 years after implant loading were good predictors of the peri-implant tissue condition. Repeated peri-implant PD measurements may be performed with higher reproducibility by means of an automated controlled-force periodontal probe.[85] Furthermore, animal experiments have shown that, as is the case around teeth,[86] peri-implant probing disrupts the epithelial attachment but does not cause permanent damage to the transmucosal soft tissue seal. In one study, complete attachment of the junctional epithelium was re-established after 5 days following probing using a conventional periodontal probe.[87]

Width of Peri-implant Keratinized Mucosa

Clinical and experimental studies[88,89] have failed to support the concept of an "adequate width" of keratinized tissue adjacent to natural teeth for the maintenance of periodontal health. Implant research has also focused on the necessity of the presence of keratinized mucosa around oral implants. No differences in peri-implant soft tissue recession or bone loss have been found between sites with or without keratinized mucosa following plaque-induced breakdown at implants placed in dogs.[90] On the other hand, ligated titanium or hydroxyapatite-coated implants in monkeys with minimal or no keratinized mucosa demonstrated significantly more recession and connective tissue loss than those surrounded by keratinized tissue.[91,92] This suggests that the absence of keratinized mucosa around implants seems to increase the susceptibility of plaque-induced peri-implant tissue destruction. These findings have been confirmed in other studies,[83,93] suggesting that the presence of keratinized mucosa around implants is strongly correlated with optimal soft and hard tissue health. However, longitudinal clinical studies have failed to reveal major differences in the progression of lesions around implants placed in sites with or without keratinized mucosa, or that the lack of an attached portion of masticatory mucosa may jeopardize the maintenance of soft tissue health.[74,94–97] Furthermore, in the presence of good oral hygiene, the nature of the mucosa may have little influence on the long-term survival of implants. However, suboptimal oral hygiene may lead to greater tissue damage around implants within alveolar mucosa than around implants within keratinized tissue. Proper oral hygiene procedures may also be facilitated in the presence of an adequate band of keratinized mucosa. Prospective longitudinal controlled clinical trials will have to be performed to further elucidate the potential role of a sealing effect of keratinized mucosa on long-term peri-implant health.

Peri-implant Sulcus Fluid Analysis

Several biochemical mediators in the gingival crevicular fluid (GCF) around natural teeth have been identified as potential host markers for periodontal disease activity and progression.[98] To date, only a few studies have reported on the association between signs of peri-implant inflammation and increased levels of inflammatory mediators in the peri-implant sulcus fluid (PISF). A pilot study[99] investigated whether the crevicular fluid volume around osseointegrated implants shows a relationship to peri-implant soft tissue condition. The results indicated a close relationship between PISF volume and plaque accumulation as well as degree of peri-implant soft tissue inflammation. Numerous investigations of potential diagnostic markers of stable and diseased peri-implant conditions have focused on the sulcus fluid analysis of several mediators, including protease activity[60,100,101]; collagenase, gelatinase, and elastase activity[102–107]; aspartate aminotransferase[108]; glycosaminoglycans[109,110]; and proinflammatory mediators such as interleukin-1beta (IL-1β) and prostaglandin E_2 (PGE_2).[80,111] Kao and coworkers[111] reported that PISF-IL-1β levels around diseased implants were approximately 3 times higher than those around stable implants, thus providing evidence for the involvement of this catabolic cytokine in peri-implant bone destruction.

In a 3-year longitudinal investigation, Behneke and associates[82] were able to show a positive correlation between PISF volume and the amount of bone resorption. In a subsequent report of the 5-year data,[2] the percentage of sites exhibiting elevated PISF rates increased significantly in the second half of the observation period. Using a cross-sectional study design, Salcetti and coworkers[80] investigated the production of IL-1β, PGE_2, interleukin-6, platelet-derived growth factor, and transforming growth factor beta in the PISF of patients with 1 or more failing titanium implants. Several of these patients had at least 1 other stable implant that did not present with clinical signs of

inflammation or radiographic evidence of peri-implant bone loss. Significant elevations in PISF levels of IL-1β, PGE_2, and platelet-derived growth factor in subjects with failing implant sites were detected when compared with patients with healthy control implants. The significant elevations of IL-1β and PGE_2 at both failing implant sites and at stable implant sites in the same subject indicate that an increased local host response is detectable at the patient level as well as at local sites of peri-implant inflammation.

The finding that enzymes from polymorphonuclear granulocytes (PMN) are detected in high concentrations at sites with peri-implantitis may indicate enhanced PMN cell activity.[105,107] Hultin and colleagues[105] analyzed the composition of PISF at implants in patients with "stable marginal tissue conditions" and peri-implantitis. Implant sites with peri-implantitis had higher concentrations of lactoferrin and elastase activity than control sites. Similarly, Plagnat and associates[107] collected and analyzed PISF from sites with and without clinical and radiographic signs of peri-implantitis. The authors reported that PISF levels of elastase, alpha2-macroglobulin, and alkaline phosphatase were significantly higher at diseased sites than at healthy sites, and that the levels of these markers correlated with clinical symptoms. On the other hand, similar low levels of the above-mentioned markers were found both at baseline and at the 3-year examination in the sulcus fluid around successful implants placed in esthetic anterior sites, indicating stable biochemical peri-implant conditions.[104] Collectively, these data document an important implication of catabolic inflammatory mediators in peri-implant tissue breakdown and indicate a potential value of biochemical markers for monitoring the host response during the supportive phase of implant therapy.

Suppuration

High numbers of PMN cells have been detected around implants that are associated with severe signs of mucosal inflammation.[48] Several histopathologic and immunohistochemical analyses of tissues surrounding implants with signs of peri-implantitis, ie, clinical signs of inflammation and advanced bone loss, have revealed the presence of large inflammatory cell infiltrates.[35,112–115] Sanz and associates[115] analyzed soft tissue biopsies from patients with peri-implantitis and reported that a considerable portion of the connective tissue (ie, 65%) was occupied by an inflammatory infiltrate. Esposito and coworkers[112] analyzed the characteristics of soft tissues surrounding failing implants immunohistochemically. They reported that the marginal portion of the specimens was characterized by an "intense inflammatory and immunologic response." Piattelli and colleagues[114] described histopathologic characteristics of 230 retrieved implants. The authors reported that around implants removed because of peri-implantitis, "an inflammatory infiltrate composed of macrophages, lymphocytes, and plasma cells was observed in the connective tissue." Gualini and Berglundh[113] reported that peri-implantitis lesions contained significantly greater proportions of B-lymphocytes and PMN cells than mucositis lesions. Collectively, the observation that large numbers of inflammatory cells, including PMN cells, occupy the connective tissue infiltrate may explain the presence of suppuration at sites with advanced peri-implant disease.

EVALUATION OF THE BONE-IMPLANT INTERFACE

Implant Mobility and Discomfort

Primary stability at the time of implant placement has been recognized as an important prerequisite for the achievement of osseointegration.[74,94,116] The establishment and maintenance of direct contact at the bone-implant interface are requirements for long-term implant success. Implant mobility is an indication of lack of osseointegration. Even if peri-implant disease has progressed relatively far, implants may still appear immobile because of some residual direct bone-to-implant contact. The recording of implant mobility may be a very specific—but not at all sensitive—clinical parameter in detecting loss of osseointegration. This parameter more likely detects the final stage of osseo-disintegration and, therefore, represents a late implant loss. Furthermore, pain or discomfort may be associated with increased implant mobility and could be one of the first signs indicating a failing implant.[117,118] Persistent discomfort may be evident long before any radiographic change is detectable.[119]

Longitudinal assessment of individual implant mobility may be performed for screw-retained suprastructures. For obvious reasons, this cannot be applied to all cemented and tooth/implant-supported restorations. An electronic device (Periotest; Siemens, Bensheim, Germany), originally designed to measure the damping characteristics of the periodontium around natural teeth,[120,121] has been recommended to monitor initial degrees of implant mobility or horizontal displacement. However, differences in Periotest values (PTVs) have been reported for implants in the mandible and in the

maxilla,[73] with implants in the maxilla showing significantly higher PTVs. In patients treated with Brånemark System implants (Nobel Biocare, Göteborg, Sweden), this procedure was found to be related to the type of jaw treated, implant and abutment length, condition of the peri-implant tissues, and bone density.[122] Despite some positive claims for this method,[2,82,123,124] the prognostic accuracy of PTVs for the diagnosis of peri-implantitis and early signs of implant failure has been criticized because of the lack of resolution, poor sensitivity, and susceptibility to operator variables.[125]

Resonance Frequency Analysis

A new, noninvasive device based on the principles of resonance frequency analysis (RFA) has been developed to measure primary implant stability and to monitor implant stability over time.[126] This method evaluates the stiffness of the bone-implant interface by means of a signal transducer connected to a frequency response analyzer (Osstell; Integration Diagnostics, Göteborg, Sweden). The resonance frequency of the transducer-implant unit is calculated from the peak amplitude of the signal and is graphically illustrated on the Osstell display as the peak of a frequency-amplitude plot. In addition, an implant stability quotient (ISQ) is displayed as a number between 1 and 100. This ISQ value has been introduced to quantify the frequency measurements of oral implants with a range between 3,500 and 8,500 Hz. Several investigations[127–132] have shown that the ISQ value of a stable osseointegrated implant increases with time, suggesting an increase in the bone-implant contact area. On the other hand, crestal bone loss around implants has been correlated with loss of implant stability.[124,129] This may allow detection of an increase in implant mobility before clinical signs are recorded.[133] However, conclusive data on the bone-implant interface and RFA values are still lacking.[134]

RADIOGRAPHIC EVALUATION

Long-term preservation of crestal bone height around osseointegrated implants is often used as a primary success criterion for different implant systems. Originally, a mean crestal bone loss ≥ 1.5 mm during the first year after loading and ≥ 0.2 mm/year thereafter had been proposed as one of the major success criteria.[94,135] This success criterion, however, has recently been questioned, because longitudinal studies have provided evidence that crestal bone loss around osseointegrated implants in well-maintained patients may be minimal.[2,18,136]

Conventional radiography represents a widely accepted technique for the long-term evaluation of marginal bone changes at interproximal sites of osseointegrated implants. In general, the long-cone paralleling technique, supported by positioning devices, is used. It should be noted that conventional radiography yields a high proportion of false negative findings, ie, it has low sensitivity in the detection of early pathologic and/or bone remodeling changes.[137] Therefore, radiographic methods are confirmatory rather than exploratory and should only be considered in conjunction with assessment of the clinical parameters.[138] Nevertheless, the distance from a landmark on the implant (eg, implant shoulder for 1-stage transmucosal implant systems or apical termination of the cylindric portion of the implant for 2-stage submerged implant systems) to the alveolar bone crest represents a reliable parameter for long-term monitoring in clinical practice.

It should be pointed out that radiographic evidence of bone-to-implant contact does not imply osseointegration on a histologic level.[139] More importantly, if clinical parameters indicate peri-implant disease (eg, increased PD, BOP+, suppuration), additional radiographs should be obtained to evaluate the extent of peri-implant crestal bone loss. For longitudinal clinical research purposes, radiographs should be obtained at baseline and at 1-, 3-, and 5-year intervals. Thereafter, they should be obtained every 5 years if marginal peri-implant bone stability has been demonstrated.[140]

Computer-assisted image analysis has been shown to improve the diagnostic accuracy (ie, increased sensitivity) of detecting minimal periodontal tissue changes.[137,141] Consequently, the use of digital image analysis has expanded into implant dentistry to monitor peri-implant bone healing and gain or loss of alveolar bone density.[142]

CONCLUSION

Evidence from the presented literature indicates that the use of a number of clinical, biochemical, and radiographic parameters is meaningful in the evaluation of peri-implant tissue status. Research efforts are currently under way to relate biologic parameters to morphologic changes in peri-implant structures. However, reliable prognostic indicators for peri-implant hard and soft tissue changes are still lacking. This is not surprising, because the same phenomenon (ie, development of diagnostic tests for the assessment of active periodontal tissue destruction) has challenged periodontal research in recent decades.

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