Treatment of Peri-implantitis Defects with Autogenous Bone Grafts: Six-Month to 3-Year Results of a Prospective Study in 17 Patients

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As part of an ongoing prospective study, the treatment of peri-implantitis defects using autogenous bone grafts was evaluated. This present report is based on data from 25 ITI screw implants in 17 patients with progressive peri-implant tissue destruction during the maintenance phase. Treatment of these lesions included raising flaps, removal of the surrounding granulation tissue, and air-polishing of the implant surface. Subsequently, corticocancellous bone grafts or particulate bone were placed into the peri-implant osseous defects, and the flaps were sutured around the cervical segment of the implants, allowing for transmucosal healing. Two of the 25 cases resulted in a negative outcome of the procedure. One of the transplants had to be removed 40 days after augmentation because of flap dehiscence and graft mobility. In another patient, the healing period was uneventful until the re-entry surgery, but when the site was reopened, the total graft volume was resorbed. The primary therapeutic success at re-entry surgery evaluated by intraoperative measurements resulted in a median defect depth reduction of 6.9 to 0.7 mm (P = .001), corresponding to a bone repair of 90%. The change in defect width was 1.9 mm (P = .002, repair 100%). A positive result of the reconstructive therapy has been observed during a re-evaluation time of up to 3 years. Median marginal bone loss was reduced from 6.2 to 2.3 mm after 2 and 3 years, respectively. The median vertical bone resorption of 4.5 mm was completely repaired. The crevicular fluid volume, a parameter of the level of marginal inflammation, along with probing depths and attachment levels, were reduced to a physiologic rate. The implant observation period until the first appearance of the lesion seems to be crucial to the effectiveness of the therapy. Early failures appearing within the first 2 years after implant placement showed a more stable therapeutic result over time. (INT J ORAL MAXILLOFAC IMPLANTS 2000;15:125–138)

Key words: *autogenous bone grafts, dental implants/complications, DNA probe, implant failure, ITI screw implant, peri-implantitis*

Clinical studies indicate a high probability of successful soft tissue integration and minimal bone resorption with osseointegrated implants.[1–7]

Moderate loss of bone tissue in the healing and early functional phase is interpreted either as the result of microbial colonization on the implant surfaces following exposure to the oral cavity or as a remodeling process from initial functional load. However, single implants show bone destruction of a progressive character and higher clinical inflammation parameters. The etiopathogenesis of these changes, labeled as peri-implantitis, is multifactorial. The risk of formation of peri-implantitis is determined by factors specific to the patient, such as the presence of a pathogenic microflora, occlusal overload by parafunction or bruxism, lack of passivity of fit, and individual immunologic response.

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Further local factors are of importance concerning the implant site. These include quality and quantity of the bony implant bed and also geometric and occlusal risk factors. The significance of these single influential factors as etiologic covariables for the development of progressive peri-implant bone destruction has been described in the literature by the analysis of failing implants. In reference to the microbiota associated with diseased implants, a dominance of gram-negative anaerobic bacteria and spirochetes corresponds with chronic progressive periodontopathy.[8,9] The periodontopathogens commonly identified in the majority of studies are *Porphyromonas gingivalis* and *Prevotella intermedia*; furthermore, *Actinobacillus actinomycetemcomitans*, *Fusobacterium nucleatum*, *Campylobacter rectus*, *Peptostreptococcus micros*, *Eikenella corrodens*, *Prevotella nigrescens*, and *Capnocytophaga* species were traceable.[8–16] Indication of the influence of overload by parafunctional activity and nonaxial occlusal contacts, respectively, was found by Sanz et al,[17] Quirynen et al,[18] and Rangert et al.[19] Few studies deal with the host immune response to peri-implantitis by determination of the inflammatory mediators prostaglandin E_2 or interleukin-1β in the sulcus fluid. Both mediators are able to induce osteoclastic bone resorption and can disturb the physiologic equilibrium of bone turnover. Jovanovic et al,[20] Kao et al,[21] and Salcetti et al[16] found significantly higher levels of prostaglandin E_2 and interleukin-1β at diseased implants in comparison to healthy sites. In contrast to the findings of the above-mentioned studies, Wilson and Nunn[22] failed to demonstrate a significant relationship between a positive interleukin-1 genotype and implant survival.

For the treatment of peri-implantitis, a variety of therapeutic strategies can be applied (Table 1). As a nonsurgical approach to therapy, systemic or topical antimicrobial regimens offer the possibility of selectively influencing the anaerobic microbiota. Possibilities of surgical intervention include resective techniques for eliminating bone pockets, as in periodontics, which results in control of inflammation but not in defect regeneration. The optimal goal of therapy should be the regeneration of lost alveolar support and thereby reconstruction of the original bone structure. Literature reports dealing with surgical repair of peri-implant defects include, in addition to guided bone regeneration (GBR), defect filling with autogenous bone grafts or bone graft substitutes. Table 1 shows the present state of literature concerning the mentioned therapeutic considerations.[9,13,23–33] By antimicrobial therapy alone the studies of Mombelli and Lang[9] and Buchmann et al[13] have shown an improvement of the soft tissue

parameters and elimination of pathogenic microbiota, but no mentionable bone repair was achieved. For the membrane technique, good results have been demonstrated only by case reports. The success prospective of this treatment with an adequate number of patients has not yet been shown.

Augthun et al[27] observed 15 failing implants with no osseous defect-filling after GBR. Jovanovic et al[29] studied the reparative potential of e-PTFE membrane coverage in peri-implant bone defects and found a soft fibrous filling in 7 of 10 implant sites. Three cases failed to show any improvement at the time of membrane removal. Buchmann et al[32] treated deep peri-implant bone pockets in 5 implants with nonresorbable membranes and autogenous bone. Three implant sites showed an average repair of 58%; in the other 2 cases, the therapy failed and the implants were removed. A high rate of complication (infections, premature membrane exposure) as cause for compromised bone fill was a frequently reported sequela. Results of a pilot study[25] on 14 diseased implants showed no postoperative complications and a reduction of defect depth from 5.8 to 2.1 mm after augmentation with autogenous particulate or block-shaped bone grafts without membrane coverage.

The aim of this study was to report the results of a prospective study on autogenous bone grafts within peri-implantitis therapy. The extent of bone repair, evaluated by intrasurgical measurements, microbiota, and complications, was registered to demonstrate the predictability of the treatment approach. Hard and soft tissue reactions were evaluated up to 3 years after defect revision to assess the maintenance of the newly gained supporting tissue.

MATERIALS AND METHODS

Patient Selection and Reconstructive Surgical Procedure

Within a 4-year period, from April 1994 to March 1998, 17 patients with 25 failing ITI screw implants were consecutively admitted for study. Criteria were progressive crater-like or saucer-shaped peri-implant bone defects and, as a soft tissue parameter, probing depths of more than 5 mm. The implants were to show no mobility and no peri-implant radiolucency. The extent of the defect was not to exceed 90% of the originally osseously anchored part of the implant. Patients without sufficient compliance to the therapy and patients with systemic medical conditions or complications that would preclude any minor oral surgical procedure were excluded from the study.

Table 1 Summary of Data Available in the Literature on the Clinical Outcomes of Various Peri-implantitis Procedures

Peri-implantitis procedures	Authors	Type of study	No. of patients/ implants	% of clinical bone fill	Complications	Evaluation period
Antimicrobial therapy						
Ornidazol (systemic for 10 days)	Mombelli and Lang (1992)[9]	Controlled clinical trial	9/9		None	1 year
Metronidazol or amoxycillin (systemic for 7 days)	Buchmann et al (1996)[13]	Controlled clinical trial	20/20	11%	None	6 months
Resective surgical therapy						
No clinical trials or case reports						
Regenerative surgical therapy						
Bone grafts and bone graft substitutes						
HA or DFDB	Gammage et al (1989)[23]	Case report	2/5		Graft loss: 20%	1 year
DFDB	Lozada et al (1990)[24]	Case report	1/2		None	1 year
AB	Behneke et al (1997)[25]	Controlled clinical trial	10/14	62%	None	2 years
Guided bone regeneration						
e-PTFE + HA	Kraut and Judy (1991)[26]	Case report	1/3		Membrane exposure and removal after 7 weeks	10 months
e-PTFE	Augthun et al (1992)[27]	Controlled clinical trial	12/15	0%	Premature membrane exposure: 87%	6 months
e-PTFE	Lehmann et al (1992)[28]	Case report	1/1	80%	Suppuration and membrane removal after 5 months	6 months
e-PTFE	Jovanovic et al (1992)[29]	Controlled clinical trial	7/10		Premature membrane removal: 30%	6 months
e-PTFE	Hämmerle et al (1995)[30]	Case report	2/2	51%	Suppuration and membrane removal after 4 months	1 year
e-PTFE + HA, DFDB, AB	Mellonig et al (1995)[31]	Case report	3/3		Membrane exposure and removal after 6 to 9 weeks	8 to 12 months
Non resorbable membrane + AB	Buchmann et al (1997)[32]	Controlled clinical trial	5/5	58%	Membrane exposure implant loss: 40%	6 months
Polylactic + AB	Von Arx et al (1997)[33]	Case report	1/1	90%	Membrane exposure and suppuration after 4 weeks	6 months

HA = hydroxyapatite; DFDB = decalcified freeze-dried bone; AB = autogenous bone; e-PTFE = expanded polytetrafluoroethylene.

After diagnosis of peri-implantitis and determination of inclusion or exclusion criteria, a 1-month-long local-disinfecting treatment using weekly submarginal irrigation (Odontoson M, Goof, Hörsholm, Denmark) with iodine solution was provided prior to surgery. Treatment of the defects followed a previously published procedure[25,34] and consisted of elevation of a mucoperiosteal flap and removal of the peri-implant inflammatory granulation tissue using hand curettes without touching the implant surface. Implant surfaces were then treated using an air-powder abrasive instrument (Air Flow SI, EMS, Nyon, Switzerland) with sodium carbonate solution for 30 seconds, with the spray vector directed perpendicular to the implant surface. Following abrasive cleaning, the exposed implant and bony surfaces were rinsed with sterile physiologic saline. At this time, clinical measurements of the peri-implant bony defects were recorded. The augmentation was performed with autogenous block-shaped or particulate bone grafts obtained from the mandibular retromolar area or the symphysis. Following intramarrow penetration of the surrounding cortical bone plate of the recipient sites to allow the migration of angiogenic and osteogenic cells, bone grafts were placed in the defect sites to cover the exposed implant surfaces. For stabilization of the corticocancellous bone grafts, supporting screws of the Memfix kit (Straumann, Freiburg, Germany) were used, and bone chips were maintained in position with fibrin glue (Tissucol, Immuno, Heidelberg, Germany). After adaptation of the

Fig 1a The pretreatment radiograph reveals the crater-like bone defect of the implant in the area of the mandibular right second molar.

Fig 1b Intrasurgical status after removal of the granulation tissue and treatment of the implant surface with an air-powder abrasive.

Fig 1c Application of an autogenous bone graft from the retromolar area and stabilization with a supporting screw.

mucoperiosteal flap to the implant cervical segment, transmucosal healing followed for 3 to 4 months. The implant prostheses were reattached, either directly after augmentation, or within a 14-day period afterwards.

A perioperative antibiotic regimen with metronidazol (Clont, 2×400 mg per day) was administered for 7 days. In patients with screw-fixed bone grafts, re-entry followed after 3 to 4 months for removal of the supporting screws and clinical assessment of bone regeneration. Figs 1a to 1g demonstrate the step-by-step procedure in a patient exhibiting a crater-like defect with additional loss of the buccal bone wall. Treatment of the patients was conducted according to a standardized protocol,

where surgical procedures and follow-up examinations were administered by a single examiner.

Patients were informed of the terms for participating in the study, and data were used according to the declaration of Helsinki[35] and guidelines set forth by the University of Mainz for biomedical research in human subjects.

Microbial Sampling

Before the pretreatment was started, the implant sites were investigated for the presence of putative periodontopathic organisms, using specific DNA probes for *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Prevotella intermedia* (DMDx/PathoTek Test, Wybert, Lörrach, Germany). For the sampling
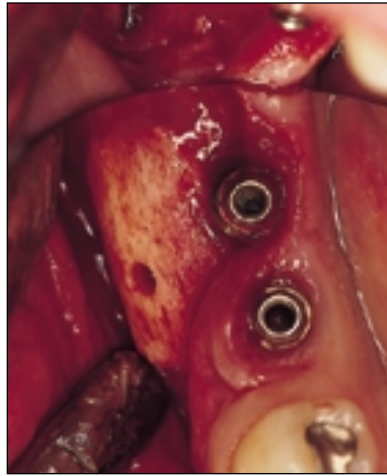
Fig 1d Clinical status at reopening (4 months after reconstructive therapy) and following removal of supporting screws demonstrates osseous healing of the previously existing bone defect.
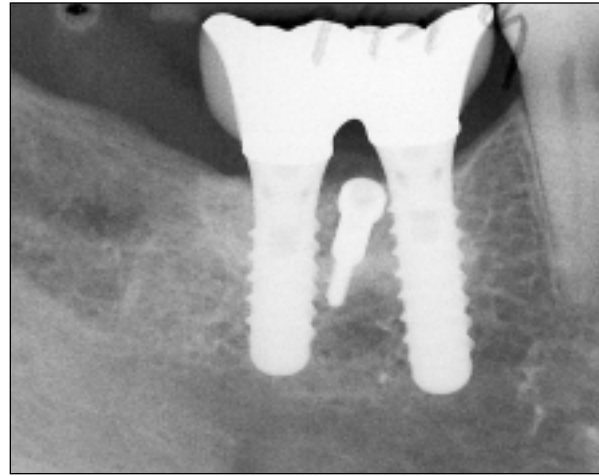
Fig 1e Periapical radiograph taken at the time of re-entry shows the normal trabeculation of the repaired area.

Fig 1f Occlusal view of the implant-supported fixed prosthesis.
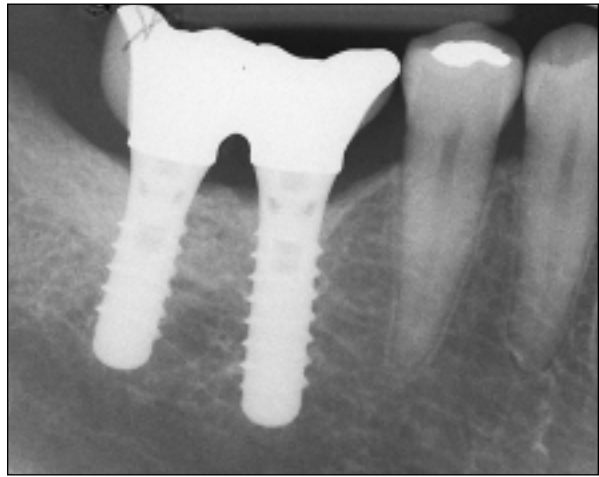
Fig 1g Periapical radiograph 3 years after reconstructive therapy, demonstrating normal bone structures.

of subgingival plaque, the implant sites were isolated with cotton rolls and gently air dried. Sterile paper points were placed into the sulcus and left in situ for 10 seconds.

Intrasurgery Recordings

During reconstructive surgery and re-entry, the measurements of the peri-implant defect morphology, recorded to the nearest 0.5 mm at the buccal, lingual-palatal, mesial, and distal aspects of each implant using a calibrated periodontal probe, included the following parameters:

- Defect depth: distance from the implant top surface to the fundus of the defect

- Bone level: distance from the implant top surface to the most coronal point of the alveolar crest
- Defect width: distance between the most coronal point of the alveolar crest and the implant surface

Radiographic Evaluations

Periapical radiographs were obtained with the long-cone technique using Rinn film holders (Dentsply, Konstanz, Germany). After calculating the magnifying factor using the known implant length, the radiographs were analyzed for changes in the alveolar bone levels with respect to the baseline data (implant placement). The distance between the implant top surface and the first visible bone contact was defined as marginal bone loss and measured to

Table 2 Schedule for Data Collection

Time point	Clinical recordings	Radiographic evaluation	Microbial sampling	Intrasurgery recordings
Beginning of initial treatment	•	•	•	
End of initial treatment	•			
Reconstructive surgery				•
Re-entry surgery	•	•		•
6 mo after reconstructive surgery	•			
12 mo after reconstructive surgery	•	•		
24 mo after reconstructive surgery	•	•		
36 mo after reconstructive surgery	•	•		

the closest 0.1 mm at the mesial and distal aspect of each implant. In addition to the determination of marginal bone loss, bone resorption was morphologically differentiated in horizontal and vertical components. All measurements were made independently by 2 of the authors. If there was a discrepancy of 0.5 mm or less, the mean value of the 2 measurements was used. In situations with greater discrepancies, the radiographs were analyzed again and discussed until consensus was reached.

Clinical Data

The clinical examinations were performed after removal of the prostheses and focused on mucosal peri-implant conditions and implant mobility:

- Probing depth, measured to the nearest 0.5 mm with a Plast-o-Probe (Maillefer, Stuttgart, Germany) at the buccal, lingual-palatal, mesial, and distal surfaces of the implants
- Distance between implant shoulder and mucosal margin, measured to the nearest 0.5 mm with the same probe at the same 4 locations
- Attachment level, calculated by adding probing depth and distance for each site
- Crevicular fluid volume, collected with indicator strips (Merck, Darmstadt, Germany) inserted buccally and lingual-palatally in the peri-implant sulcus for 30 seconds
- Periotest value, measured buccally at a distance of 3 mm from the implant shoulder (Siemens, Bensheim, Germany)

All patients were enrolled in a strict maintenance program, with 4 appointments in the first year and then annual check-ups. During these visits the evaluated parameters were assessed by 1 qualified investigator and, if necessary, the patients were instructed in oral hygiene procedures. Table 2 shows the schedule for data collection.

Statistical Analyses

The graphic presentation of research parameters for the descriptive statistics was done by notched box-and-whisker plots. The mean values and the number of implants are expressed in the footnotes to the graphs. Wilcoxon's signed rank test was used to evaluate differences in the defect size recorded at the time of reconstructive surgery and re-entry. For unpaired observations, the Mann-Whitney U test was used. P values of less than .05 were considered statistically significant and thus clinically meaningful.

RESULTS

Patient Pool and Complications

Over the inclusion period, a total of 17 patients (11 females, 6 males, mean age 51.7 years) with 25 diseased implants was consecutively treated and documented in this study. Of the 25 implants, 13 had been placed in edentulous mandibles, 8 in partially edentulous mandibles, and 4 in partially edentulous maxillae. In 8 situations, the peri-implant defects became apparent within the first 2 years of the functional phase, and the other 17 implants were late failures. With 18 implants, defect-filling was achieved with block-shaped bone grafts. In these cases, a re-entry followed to remove the supporting screws, so that the regeneration of bone could be clinically evaluated by renewed intraoperative measurements. Seven sites could be refilled with particulate graft material, as the morphology of the defect under consideration to the circular preserved bone walls reduced the risk of graft displacement. In 18 sites the grafts were harvested from the retromolar area, and in 7 sites the mandibular symphysis was used as a donor site for bone transfer. After augmentation, all implants could be followed for 6 months, 18 implants were followed for 1 year, 13 implants were followed for 2 years, and 10 implants were followed for 3

Table 3 Type of Complication, Therapy, and Treatment Outcome of the 6 Patients with a Compromised Healing Phase

Patient no.	Type of complication	Therapy	Amount of clinical bone fill
1	Flap dehiscence (1 × 1 mm) at 18 days	Irrigation with chlorhexidine rinse	86%
2	Flap dehiscence (2 × 3 mm) at 9 days	Osteoplasty	100%
3	Flap dehiscence (2 × 3 mm) at 7 days	Osteoplasty	94%
4	Flap dehiscence (3 × 4 mm) at 21 days	Osteoplasty	78%
5	Flap dehiscence (8 × 5 mm) and graft mobility at 30 days	Graft removal, treatment failure	0%
6	Graft resorption	Explantation, treatment failure	9%
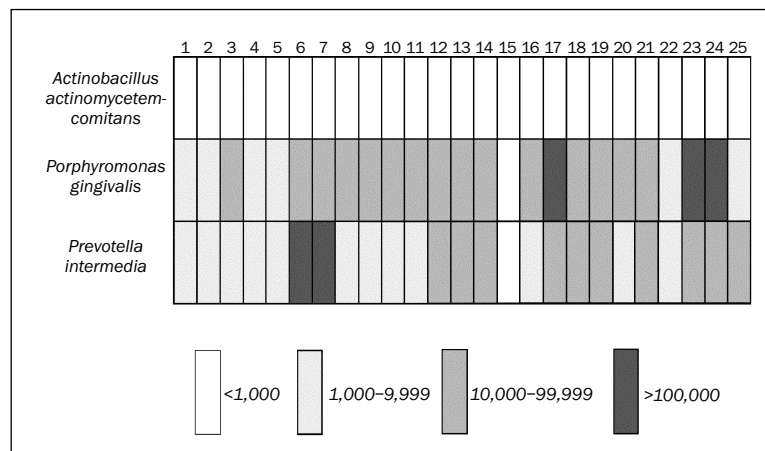
Fig 2 Microbial levels of *A actinomycetemcomitans, P gingivalis,* and *P intermedia* in the 25 diseased implant sites using DNA probes.

years. One patient with 1 implant was considered to be a drop-out, since he could not continue to attend further follow-up appointments after the 1-year observation period, because of a change of residence.

The postoperative course was uneventful for 19 of the 25 implants. Table 3 provides detailed information about patients with compromised healing, including the type of complication, the method of treatment, and the amount of clinical bone fill. Four patients developed small soft tissue dehiscences, resulting in partial exposure of the grafts, but they healed completely following osteoplasty or chlorhexidine rinses. In these patients the healing complications did not have a negative influence on the amount of repair; at reopening, the bone fill ranged from 78% to 100%. One of the transplants had to be removed 40 days after augmentation because of major flap dehiscence and graft mobility. The graft was replaced 3 months later, after healing of the mucosal wound. This second augmentation was successful, but the case was considered a failure of therapy. In another patient, the healing period was uneventful until re-entry surgery, but when the site was reopened, the total graft volume was resorbed and the implant had to be removed. Intraoperative complications or postoperative donor site complications, such as wound dehiscences, neurosensory disturbances, chin ptosis, or labiomental fold irregularities, did not occur with any of the patients.

Microbial Analyses

In Fig 2 the results of the DNA probes for each of the 25 implants are depicted. *Actinomyces* species could not be found in any of the sites respectively or were below detection level. *P gingivalis* and *P intermedia* were, with the exception of only 1 patient, identified
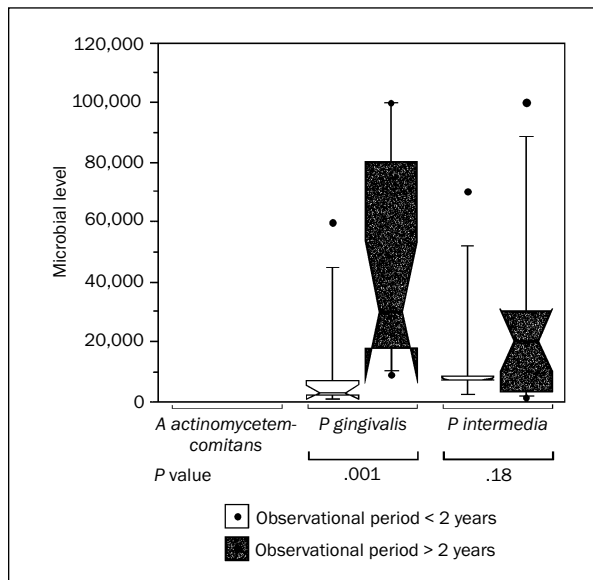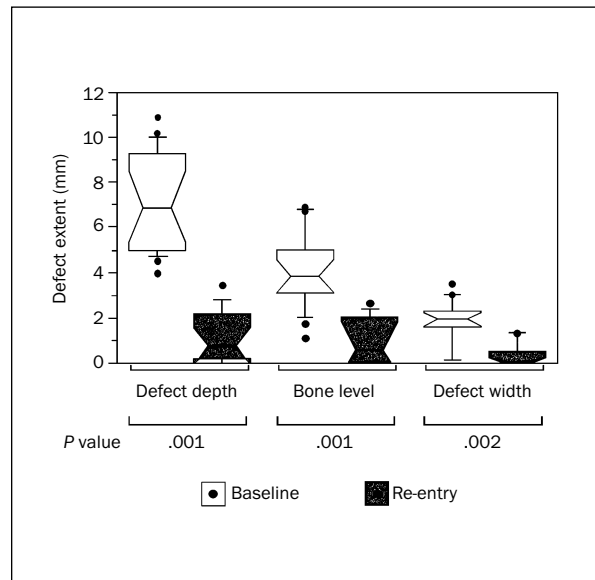
Fig 3 Box plots of the prevalence of *A actinomycetemcomitans, P gingivalis,* and *P intermedia,* depending on the implant observation period. The differences among the observation period categories for *P gingivalis* were significant.

Fig 4 Diagram showing the changes in defect extent in patients who received block-shaped autogenous bone grafts between initial and final measurements. Significant improvement was seen for all parameters.

in all samples. In regard to *P gingivalis*, 18, and in regard to *P intermedia*, 12 of the 25 probes showed a prevalence of greater than 10^4 organisms. Concerning each site, in most cases both species could be detected either in low proportions (6 samples) or in a high incidence (11 samples). Figure 3 represents the prevalence for *P gingivalis* and *P intermedia* dependent on the time after implant placement up to the appearance of peri-implant breakdown. The median amount of *P gingivalis* and *P intermedia* was at a level of 2,000 and 7,000 organisms, respectively, in sites with an observation period of less than 2 years. Implants that remained functional for a period longer than 2 years showed, for both putative pathogens, with a median of 30,000 and 20,000 organisms, respectively, elevated levels in peri-implant sulcus, which was significant for *P gingivalis* ($P = .001$).

Intrasurgery Observations

For 18 of 25 implants, the augmentation was done with mini-screw stabilized block grafts. To remove the supporting screws at re-entry, bone repair could be clinically measured and related to the initial peri-implant destruction. Figure 4 shows the initial as well as the final values for defect depth, bone level, and defect width. The 4 records of each implant were joined by calculating the mean value. For all 3 parameters, the change in defect extent between initial and final measurements was significant. The median defect depth decreased from 6.9 mm to 0.7 mm ($P = .001$), corresponding to bone repair of

90%. A repair of 84% of the bone level was a result of a median reduction from 3.8 mm to 0.6 mm ($P = .001$). The median defect width decreased from 1.9 mm to 0 mm ($P = .002$). For this parameter, filling of the initial bone defect was complete with 100%.

Radiographic Evaluation

The course of marginal bone loss in total, as well as refined in its horizontal and vertical components, is expressed in Fig 5. The diagrams depict the changes with respect to the postoperative radiograph. Between implant placement and diagnosis of peri-implantitis, median marginal bone loss of 6.2 mm was observed. This consisted of a horizontal component of 1.7 mm and a vertical component of 4.5 mm. Radiographic examination 3 to 4 months after bone grafting showed a reduction of marginal bone loss to 1.3 mm. For the 1-year examination the calculated median was 2.3 mm; after 2 and 3 years, the value stabilized at 2.4 mm. The morphologic differentiated evaluation of bone resorption revealed for the horizontal component (measured remote to the implant, as the distance between implant shoulder and osseous crest) that the initial value of 1.7 mm dropped to a median of 0.9 mm 3 months posttreatment. In the sequel, again an increase became evident, which was most distinct in the first year, resulting in a final value of 2.3 mm after 3 years. In contrast, vertical bone resorption, ie, the angular defect, showed a total leveling of its initial value from in median 4.5 mm to 0 mm after 3 years.
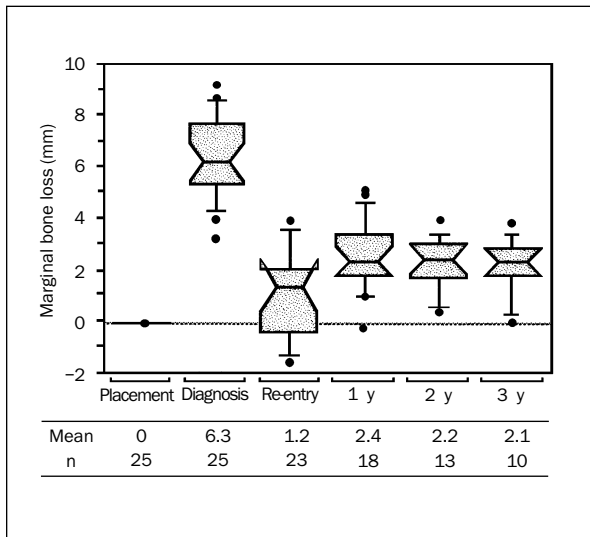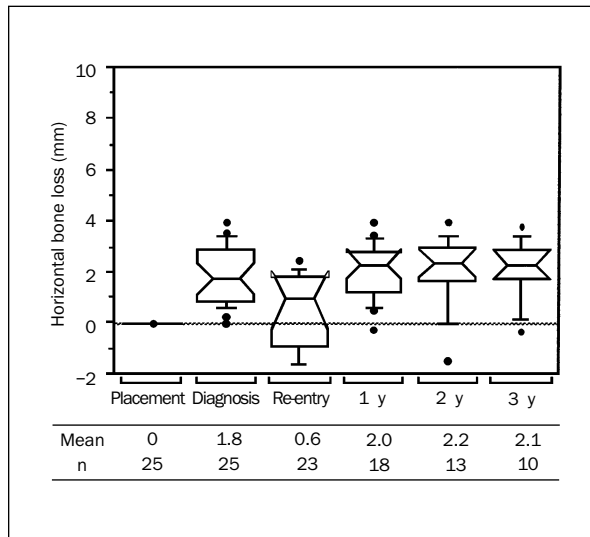
Fig 5a

	Placement	Diagnosis	Re-entry	1 y	2 y	3 y
Mean	0	6.3	1.2	2.4	2.2	2.1
n	25	25	23	18	13	10

Fig 5b

	Placement	Diagnosis	Re-entry	1 y	2 y	3 y
Mean	0	1.8	0.6	2.0	2.2	2.1
n	25	25	23	18	13	10

Figs 5a to 5c Box plots illustrating alterations in total marginal bone loss and in horizontal and vertical components from the examination immediately after implant placement, to the radiographic examination at the time of diagnosis of peri-implantitis prior to reconstructive surgery, and to the re-examinations at 3 months (re-entry) and 1, 2, and 3 years posttreatment. The radiographic examination carried out 3 months after therapy demonstrated refill of the bone amounting to a median of 4.9 mm. During the following 3 years, the radiographic data revealed a constant course. The morphologic differentiation in horizontal and vertical components showed that remodeling of the peri-implant osseous defects was the combined result of a slight horizontal resorption of the marginal alveolar crest (1.4 mm after 3 years) and a complete refill of the angular portions of the defects.
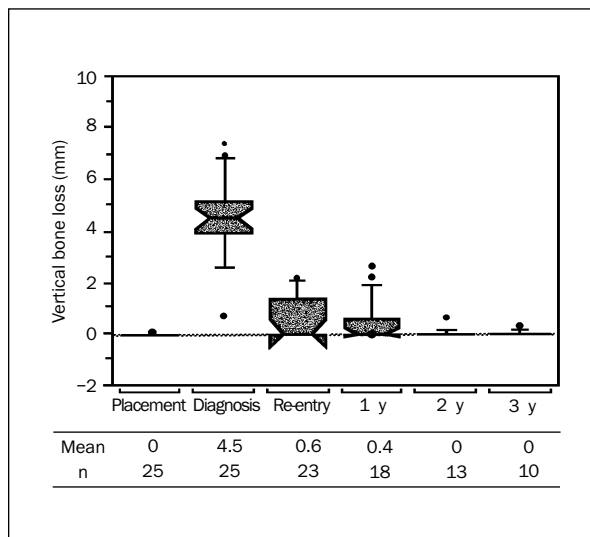

Fig 5c

	Placement	Diagnosis	Re-entry	1 y	2 y	3 y
Mean	0	4.5	0.6	0.4	0	0
n	25	25	23	18	13	10

Based on radiographic examination, Fig 6 represents the success of the reconstructive therapy related to the observation period until the appearance of peri-implantitis. If the peri-implant breakdown appeared as an early failure within the first 2 years after implant placement, the median radiographically evaluated bone repair during the first 3 months was 3.9 mm. The longitudinal monitoring of these patients indicated no recurring incidence of loss of bone support. In the group of late failures (observation period > 2 years), the primary repair after 3 months was 7 mm. This change represented a more relevant improvement when compared to the early failure group; however, during the following period there was a trend toward moderate loss

of repaired bone, so that, in the late failure group, after 3 years, remaining bone fill reached 3.7 mm.

Clinical Observations

During the destructive peri-implant processes, a significant increase in sulcus fluid flow rate (Fig 7) to 5.5 mm could be observed, when compared with the initial value of 0.6 mm at the time of prosthesis placement. The local-disinfecting pretreatment already reduced the crevicular fluid volume to 2.0 mm, and in the 3-year recall phase, stabilization at a level of 1 mm was found. Probing depths and attachment levels (Fig 8) rose between the time of prosthesis placement and the diagnosis of peri-implantitis, from medians of 2.0 mm and 1.9 mm to 5.0 mm
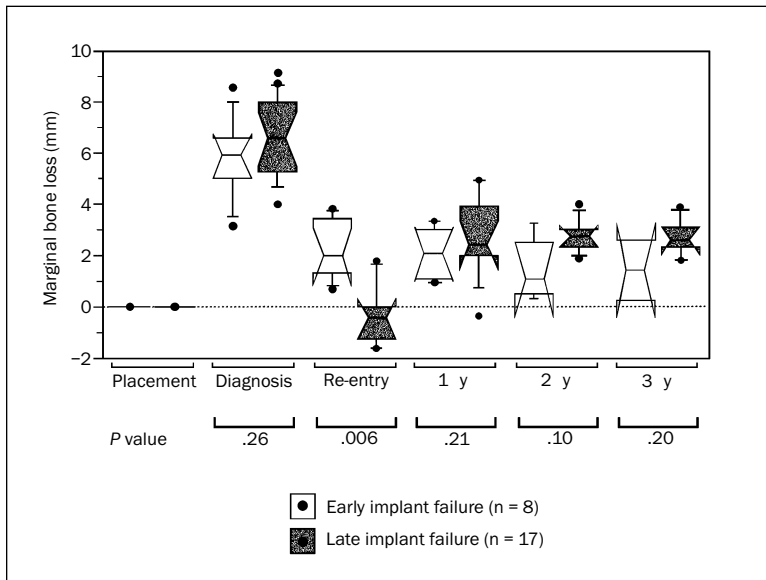
Fig 6 Longitudinal evaluation of marginal bone loss in 2 observation period categories: 1 ≤ 2 years (early failure) and > 2 years (late failure). Compromised implants with a short observation period showed a more stable result of reconstructive therapy over time.
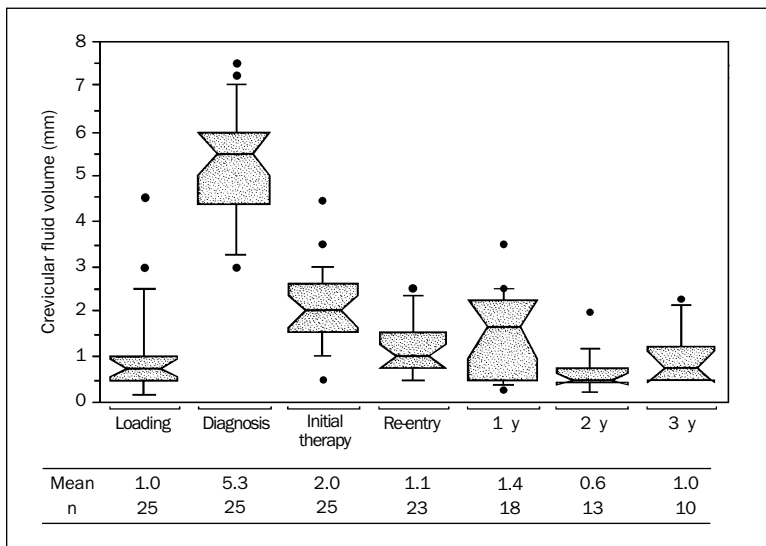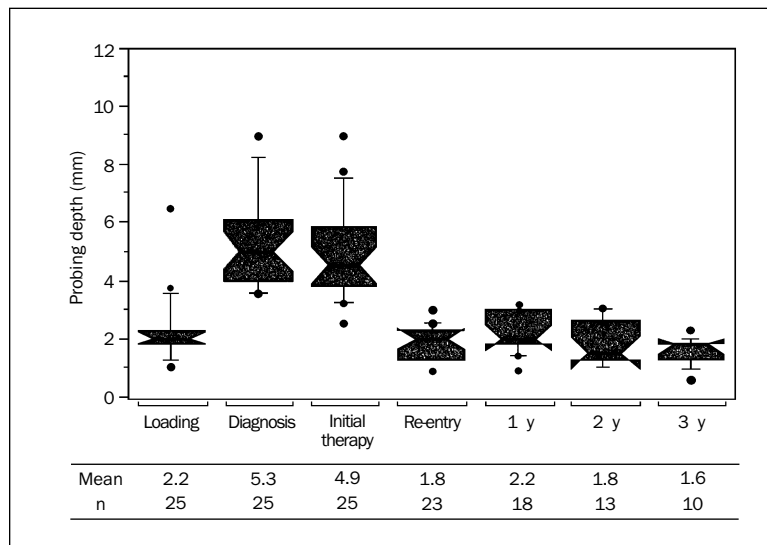

Fig 7 Box plot illustrating alterations in crevicular fluid volume following reconstructive therapy. Subsequent to the bone grafting, crevicular fluid decreased from 5.5 mm (diagnosis) to 1.0 mm (re-entry) (median values). During the following 3 years, no further changes were noted.
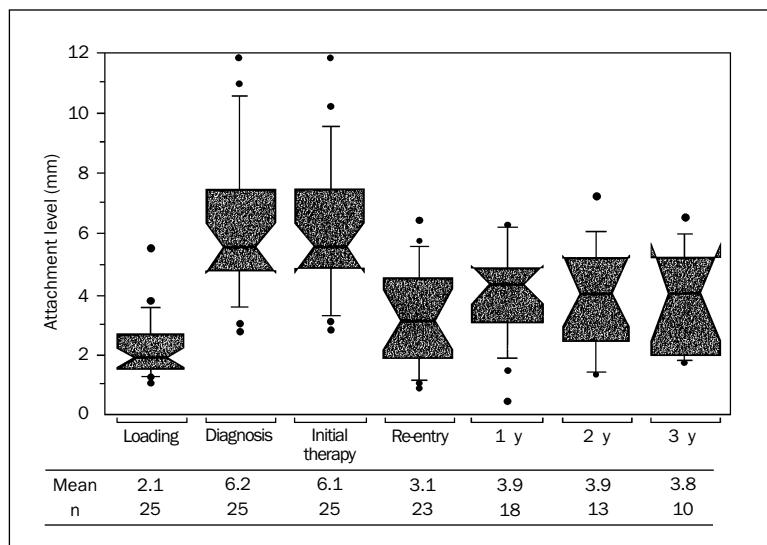
and 5.5 mm, respectively. The first check-up, 3 months after defect revision, resulted in a decrease of median probing depths to 2.0 mm; the values at the 1-, 2- and 3-year examinations also stayed within an acceptable range (1.5 mm to 2.1 mm). Attachment levels tended to rise during the first year after reconstruction, and in the subsequent research period, no further attachment loss occurred. The results of the assessment of implant mobility with

the Periotest device showed that the appearance of progressive peri-implant bone loss was reflected in an increasing Periotest value, from a median of –4 at baseline after completion of the prosthetic treatment, to a median of –1 at the time of diagnosing the peri-implant destructive disease. In the examinations following augmentation, decreasing values of –3 (1 year after augmentation) and –4 (2 and 3 years after augmentation) indicated ongoing remodeling.

Figs 8a and 8b Box plots showing alterations in probing depth and attachment level. A median reduction of 3.3 mm in probing depth was found during the 3-year trial. Because of slight marginal recession, the implants tended to lose attachment between the 3-month (re-entry) and 1-year examinations.

	Loading	Diagnosis	Initial therapy	Re-entry	1 y	2 y	3 y
Mean	2.2	5.3	4.9	1.8	2.2	1.8	1.6
n	25	25	25	23	18	13	10

Fig 8a

	Loading	Diagnosis	Initial therapy	Re-entry	1 y	2 y	3 y
Mean	2.1	6.2	6.1	3.1	3.9	3.9	3.8
n	25	25	25	23	18	13	10

Fig 8b

DISCUSSION

The treatment of progressive peri-implant destruction in the period of functional loading is a serious challenge for the maintenance therapy of endosseous implants. The advantage of regenerative techniques, in comparison to resective methods, lies in the possibility of reconstruction of lost bony support that has been destroyed by the disease process. It can be seen from the results of the present study that this goal of therapy can be reached with sufficient predictability, by using autogenous bone grafts. The bone grafting entailed filling a median of 90% of the defect, and the sites were stable for periods ranging from 6 to 36 months posttreatment.

With a failure of therapy in 2 of 25 patients, the predictability of the procedure was highly acceptable. The amount of defect filling in the present study compared with the results cited in the literature review (Table 1), indicates a similar positive outcome described only in the case reports of Lehmann et al[28] and Von Arx et al.[33] The successful treatment of peri-implantitis presented in this paper may be attributed to the use of autogenous bone as an augmentation material with the possibility of maintenance of cellular viability and rapid revascularization. Furthermore, the pretreatment involving repetitive submarginal irrigation leading to a reduction of the adherent microbial layer on the implant surface[9,36] and to replacement of the inflammatory

tissue by collagen fibers[25] may be a prerequisite for therapeutic success. The intraoperative decontamination of the implant surface with a modified airabrasive device presents another cofactor for enhancing effectiveness. Dennison et al[37] studied various methods of implant surface conditioning and achieved the highest detoxification effect by using air-polishing. However, the application of an air-powder abrasive instrument is controversial because there is a risk of emphysema[38] and possible loss of surface energy which may negatively influence the potential for reosseointegration.[39,40]

In the present study, the application of a membrane was relinquished to reduce the risk of compromised bone fill as a result of membrane exposure. The frequency of premature membrane exposure ranges from 30%[29] to 87%[27] and therefore was the most problematic and common complication described for guided bone regeneration in peri-implantitis therapy. Soft tissue dehiscences subsequent to the application of autogenous block grafts do not necessarily imply treatment failure, as controlled healing is possible and can be achieved by osteoplasty. Another advantage of autogenous bone grafts without barriers lies in the fact that complete coverage to guarantee submerged healing is not necessary. This avoids enlarged flap mobilization and therefore reduces the risk of dehiscence and inflammation. The superstructure can be reconnected 1 to 2 weeks after surgery, which saves the patient from an unnecessarily long period of compromised oral function. A disadvantage of the presented method can be seen in the multistep procedure, which includes 4 weeks of pretreatment, grafting with the removal of host donor tissue, and a professionally supervised maintenance phase.

The observation period of the implant until the appearance of destructive peri-implant processes seems to have an influence on the regenerative potential. Long-term failures, which occurred after the second year of implant placement, were, according to radiographic results, associated with higher amounts of repaired bone at the time of re-entry than early failures. However, within the first year after reconstruction, signs of recurrent bone loss were noticed. A possible explanation may be the different etiology of peri-implant bone loss. Early failures might be the result of compromised perioperative conditions (insufficient bone quantity, poor bone quality, heat injury to the implant bed, postsurgical complications). For long-term failures, microbial, immunologic, and biomechanical factors are more likely to be the reason. This statement is supported by the DNA probe findings in this study, which demonstrated elevated levels of *P gingivalis*

and *P intermedia* almost exclusively for those implants with more than 2 years of observation.

The results of the microbiologic examination for the presence of putative periodontopathic organisms are consistent with the reports of Becker et al,[10] Rosenberg et al,[8] Mombelli and Lang,[9] Kohavi et al,[11] Sbordone et al,[12] Buchmann et al,[13] and Gouvoussis et al.[15] The mentioned studies, as well as the presented findings, demonstrated that levels of *P gingivalis* and *P intermedia* account for 10% of the total microbiota. *A actinomycetemcomitans* could not be detected in any of the 25 samples from the failing implants, so that the data of this study reinforce results available from the literature. It can be summarized that this bacterial species may not be an etiologic agent of destructive peri-implant disease. *P gingivalis*, though relatively uncommon in healthy peri-implant sites because of the production of a large array of virulence factors, is of considerable interest as a probable risk factor for peri-implantitis. The species has been shown to have the ability to influence the turnover of bone negatively by producing collagenase, proteolytic factors, cell toxins, and most of all, lipopolysaccharide. Lipopolysaccharide can stimulate peripheral mononuclear cells to produce interleukin-1, which subsequently results in bone loss. The relevance of these inflammatory mediators for progressive loss of peri-implant bone has been described by Kao et al[21] and Salcetti et al.[16] The authors observed interleukin-1 levels in the crevicular fluid of compromised implants that were 3 times higher than in healthy sites.

While the primary amount of bone fill in reconstructive peri-implantitis therapy continued to be important, the maintenance of newly gained supporting tissue over the subsequent loading period is the next obstacle to overcome. In the present report, the sites were followed over periods ranging from 6 to 36 months posttreatment. The longitudinal radiographic analysis resulted in a total leveling of vertical bone loss, which remained persistent over the observation period up to 3 years. As these vertical osseous lesions may involve increased progressive bone destruction, thus jeopardizing long-term success of the implants, their elimination described in this study can be interpreted as a sign of disease reversal. Concerning the horizontal components of bone loss, a moderate decrease in the crestal bone level could be observed, especially in the first year after the reconstructive procedure. This horizontal resorption is possibly the consequence of periosteum disturbance during the reconstruction and re-entry surgery, respectively. The crevicular fluid volume, taken as measure for the degree of inflammation, as well as the probing depth, could be reduced to a

physiologic level following bone grafting. The constant course of these parameters within the 3-year follow-up period has shown no signs of relapse so far. Within the first year after reconstruction, an increase in the attachment level of a median of 1.1 mm was found. Since probing depths were stable, it can be concluded that recession of the marginal soft tissue had occurred. When the recession is associated with an exposure of the implant surface, the treatment approach may interfere with esthetics.

CONCLUSIONS

The results of this study suggest that the use of autogenous bone grafts appears to be an efficacious treatment approach for restoring the hard tissue support of dental implants following progressive bone loss caused by peri-implantitis. The presented method resulted in new clinical bone fill in the previous defects and resolved the peri-implantitis lesions over an observation period of up to 3 years. The degree of "re-osseointegration" in these areas with new bone formation remains unknown, because histologic verification was not available in this clinical study. Several animal studies[39–44] have demonstrated that the bone fill occurring with various methods in the previous peri-implantitis defects ranges from 23% to 77%. The amount of "re-osseointegration" that had taken place, however, was limited to 0% to 36%, and the new bone-to-implant contact appeared most consistently at the base of the angular bony defects. At the coronal part of the experimental implant sites, a thin connective tissue capsule was found to separate the implant surface from the newly formed bone. Further research is needed to provide the maintenance of the achieved hard tissue gain over an extended period of time. From the clinical aspect, a relevant question will be whether it is indispensable to completely reconstitute lost tissues as an ultimate goal of regenerative therapy, or whether an osseous defect fill with incomplete "re-osseointegration" is sufficient to avoid disease recurrence and guarantee favorable long-term prognosis for the rescued implants.

REFERENCES

1. Adell R, Eriksson B, Lekholm U, Brånemark P-I, Jemt T. A long-term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws. Int J Oral Maxillofac Implants 1990;5:347–358.
2. Lekholm U, van Steenberghe D, Herrmann I, Bolender C, Folmer T, Gunne J, et al. Osseointegrated implants in the treatment of partially edentulous jaws: A prospective 5-year multicenter study. Int J Oral Maxillofac Implants 1994;9:627–635.
3. Spiekermann H, Jansen VK, Richter EJ. A 10-year follow-up study of IMZ and TPS implants in the edentulous mandible using bar-retained overdentures. Int J Oral Maxillofac Implants 1995;10:231–243.
4. Lindquist LW, Carlsson GE, Jemt T. A prospective 15-year follow-up study of mandibular fixed prostheses supported by osseointegrated implants. Clin Oral Implants Res 1996;7:329–336.
5. Behneke A, Behneke N, d'Hoedt B, Wagner W. Hard and soft tissue reactions to ITI screw implants: 3-year longitudinal results of a prospective study. Int J Oral Maxillofac Implants 1997;12:749–757.
6. Behneke N, Behneke A, Fuhr K, d'Hoedt B. Langzeitergebnisse mit IMZ- und TSP-Implantaten im zahnlosen Unterkiefer. Dtsch Zahnärztl Z 1997;52:283–290.
7. Buser D, Mericske-Stern R, Bernard JP, Behneke A, Behneke N, Hirt HP, et al. Long-term evaluation of non-submerged ITI implants. Part 1: 8-year life table analysis of a prospective multicenter study with 2359 implants. Clin Oral Implants Res 1997;8:161–172.
8. Rosenberg ES, Torosian JP, Slots J. Microbial differences in 2 clinically distinct types of failures of osseointegrated implants. Clin Oral Implants Res 1991;2:135–144.
9. Mombelli A, Lang NP. Antimicrobial treatment of peri-implant infections. Clin Oral Implants Res 1992;3:162–168.
10. Becker W, Becker BE, Newman MG, Nyman S. Clinical and microbiological findings that may contribute to dental implant failure. Int J Oral Maxillofac Implants 1990;5:31–38.
11. Kohavi D, Greenberg R, Sela MN. Klinische und mikrobiologische Befunde bei osseointegrierten Implantaten. Parodontologie 1995;4:229–305.
12. Sbordone L, Barone A, Ramaglia L, Ciaglia RN, Iacono VJ. Antimicrobial susceptibility of periodontopathic bacteria associated with failing implants. J Periodontol 1995;66:69–74.
13. Buchmann R, Khoury F, Hesse T, Müller RF, Lange DE. Antimikrobielle Therapie der periimplantären Erkrankung. Z Zahnärztl Implantol 1996;12:152–157.
14. Augthun M, Conrads G. Microbial findings of deep peri-implant bone defects. Int J Oral Maxillofac Implants 1997;12:106–112.
15. Gouvoussis J, Sindhusake D, Yeung S. Cross-infection from periodontitis sites to failing implant sites in the same mouth. Int J Oral Maxillofac Implants 1997;12:666–673.
16. Salcetti JM, Moriarty JD, Cooper LF, Smith FW, Collins JG, Socransky SS, Offenbacher S. The clinical, microbial, and host response characteristics of the failing implant. Int J Oral Maxillofac Implants 1997;12:32–42.
17. Sanz M, Alandez J, Lazaro P, Calvo JL, Quirynen M, van Steenberghe D. Histopathologic characteristics of peri-implant soft tissues in Brånemark implants with 2 distinct clinical and radiological patterns. Clin Oral Implants Res 1991;2:128–134.
18. Quirynen M, Naert I, van Steenberghe D. Fixture design and overload influence marginal bone loss and fixture success in the Brånemark system. Clin Oral Implants Res 1992;3:104–111.
19. Rangert B, Krogh PHJ, Langer B, van Roekel N. Bending overload and implant fracture: A retrospective clinical analysis. Int J Oral Maxillofac Implants 1995;10:326–334.
20. Jovanovic SA, James RA, Lessard G. Bacterial morphotypes and PGE_2 levels from the perigingival site of dental implants with intact and compromised bone support [abstract 1395]. J Dent Res 1988;67:287.

21. Kao RT, Curtis DA, Richards DW, Preble J. Increased inter-leukin-1β in the crevicular fluid of diseased implants. Int J Oral Maxillofac Implants 1995;10:696–701.

22. Wilson TG, Nunn M. The relationship between the inter-leukin-1 periodontal genotype and implant loss. Initial data. J Periodontol 1999;70:724–729.

23. Gammage DD, Bowman AE, Meffert RM. Clinical management of failing dental implants. J Oral Implantol 1989;15:124–131.

24. Lozada JL, James RA, Boskovic M, Cordova C, Emanuelli S. Surgical repair of peri-implant defects. J Oral Implantol 1990;16:42–46.

25. Behneke A, Behneke N, d'Hoedt B. Regenerative Behandlung knöcherner Defekte mit autologen Knochentransplantaten im Rahmen der Periimplantitistherapie. Z Zahnärztl Implantol 1997b;13:5–14.

26. Kraut RA, Judy KWM. Implant preservation using guided tissue augmentation membrane and porous hydroxyapatite. J Oral Implantol 1991;8:55–58.

27. Augthun M, Richter EJ, Hauptmann S, Yildirim M. Untersuchungen zur Behandlung von tiefen periimplantären Knochentaschen mit ePTFE-Membranen. Z Zahnärztl Implantol 1992;8:246–250.

28. Lehmann B, Brägger U, Hämmerle CHF, Fourmousis I, Lang NP. Treatment of an early implant failure according to the principles of guided tissue regeneration (GTR). Clin Oral Implants Res 1992;3:42–48.

29. Jovanovic SA, Spiekermann H, Richter EJ, Koseoglu M. Guided tissue regeneration around titanium dental implants. In: Laney WR, Tolman DE (eds). Tissue Integration in Oral, Orthopedic and Maxillofacial Reconstruction. Chicago: Quintessence, 1992:208–215.

30. Hämmerle CHF, Fourmousis I, Winkler JR, Weigel C, Brägger U, Lang NP. Successful bone fill in late peri-implant defects using guided tissue regeneration. A short communication. J Periodontol 1995;66:303–308.

31. Mellonig JT, Griffith G, Mathys E, Spitznagel J. Treatment of failing implants: Case reports. Int J Periodontics Restorative Dent 1995;15:385–395.

32. Buchmann R, Khoury F, Müller RF, Lange DE. Die Therapie der progressiven marginalen Parodontitis und Periimplantitis. Dtsch Zahnärztl Z 1997;52:421–426.

33. Von Arx T, Kurt B, Hardt N. Treatment of severe peri-implant bone loss using autogenous bone and a resorbable membrane. Clin Oral Implants Res 1997;8:517–526.

34. Behneke A, Behneke N. Komplikationen in der Belastungsphase und ihre Therapiemöglichkeiten. In: Koeck B, Wagner W (eds). Implantologie. Praxis der Zahnheilkunde, vol 13. München: Urban & Schwarzenberg, 1996:307–325.

35. Deklaration von Helsinki, beschlossen auf der 18. Generalversammlung in Helsinki, Juni 1964; revidiert von der 29. Generalversammlung in Tokio, Oktober 1975; von der 35. Generalversammlung in Venedig, Oktober 1983; und der 41. Generalversammlung in Hongkong, September 1989. Dtsch Ärztebl 1991;87:4691–4692.

36. Braß M, Anil A. Periimplantäre Therapie mittels subgingivaler Irrigation und intraoperativem Einsatz von Salzstrahlgeräten. Z Zahnärztl Implant 1991;7:239–242.

37. Dennison SA, Hürzeler B, Quinones C, Caffesse RG. Contaminated implant surfaces: An in vitro comparison of implant surface coating and treatment modalities for decontamination. J Periodontol 1994;65:942–948.

38. Bergendal T, Forsgren L, Kvint S, Löwstedt E. The effect of an airabrasive instrument on soft and hard tissues around osseointegrated implants. A case report. Swed Dent J 1990;14:219–223.

39. Grunder U, Hürzeler MB, Schüpbach P, Strub JR. Treatment of ligature-induced peri-implantitis using guided tissue regeneration: A clinical and histologic study in the beagle dog. Int J Oral Maxillofac Implants 1993;8:282–293.

40. Hanisch O, Tatakis DN, Boskovic MM, Rohrer MD, Wikesjö UME. Bone formation and reosseointegration in peri-implantitis defects following surgical implantation of rhBMP-2. Int J Oral Maxillofac Implants 1997;12:604–610.

41. Günay H, Skuballa C, Neukam FW. Experimentelle Untersuchung zur Behandlung von periimplantären Knochendefekten. Z Zahnärztl Implantol 1991;7:16–24.

42. Jovanovic SA, Kenney EB, Carranza FA, Donath K. The regenerative potential of plaque-induced peri-implant bone defects treated by a submerged membrane technique: An experimental study. Int J Oral Maxillofac Implants 1993;8:13–18.

43. Singh G, O'Neal RB, Brennan WA, Strong SL, Horner JA, van Dyke TE. Surgical treatment of induced peri-implantitis in the micro pig: Clinical and histological analysis. J Periodontol 1993;64:984–989.

44. Persson LG, Araújo MG, Berglundh T, Gröndahl K, Lindhe J. Resolution of peri-implantitis following treatment. An experimental study in dog. Clin Oral Implants Res 1999;10:195–203.