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The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review

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Abstract

Objective: The objective of the present paper was to review factors and conditions that are associated with hard and soft-tissue deficiencies at implant sites.

Importance: Hard- and soft-tissue deficiencies at dental implants are common clinical findings. They can lead to complications and compromise implant survival and, hence, may require therapeutic interventions. It is, therefore, important to understand the etiology of hard and soft-tissue deficiencies. Based on this understanding, strategies should be developed to correct hard and soft-tissue deficiencies with the aim of improving clinical outcomes of implant therapy.

Findings: A large number of etiological factors have been identified that may lead to hard and soft-tissue deficiencies. These factors include: 1) systemic diseases and conditions of the patients; 2) systemic medications; 3) processes of tissue healing; 4) tissue turnover and tissue response to clinical interventions; 5) trauma to orofacial structures; 6) local diseases affecting the teeth, the periodontium, the bone and the mucosa; 7) biomechanical factors; 8) tissue morphology and tissue phenotype; and 9) iatrogenic factors. These factors may appear as an isolated cause of hard and soft-tissue defects or may appear in conjunction with other factors.

Conclusions: Hard- and soft-tissue deficiencies at implant sites may result from a multitude of factors. They encompass natural resorption processes following tooth extraction, trauma, infectious diseases such as periodontitis, peri-implantitis, endodontic infections, growth and development, expansion of the sinus floor, anatomical preconditions, mechanical overload, thin soft tissues, lack of keratinized mucosa, malpositioning of implants, migration of teeth, lifelong growth, and systemic diseases. When more than one factor leading to hard and/or soft-tissue deficiencies appear together, the severity of the resulting condition may increase. Efforts should be made to better identify the relative importance of these etiological factors, and to develop strategies to counteract their negative effects on our patient's wellbeing.

KEYWORDS
gingival thickness, implantology, osseointegration, osseous defects

INTRODUCTION

The use of dental implants is considered a predictable therapeutic option for the rehabilitation of partially or fully edentulous patients providing long-term function and esthetics.[1–4] Tissue deficiencies at implant sites are common clinical findings.[5,6] Their presence may lead to an increase in marginal bone loss, soft-tissue inflammation, and soft-tissue recession.[7,8] These complications are difficult to treat and may threaten the survival of the implant. Hard-tissue defects at implant sites encompass intra-alveolar, dehiscence, fenestration, horizontal ridge, and vertical ridge defects.[9] Soft-tissue defects include volume and quality deficiencies, i.e. lack of keratinized tissue.[10] These tissue deficiencies may result from a large number of reasons. The aim of the present paper is to describe the factors associated with and/or causing soft- and hard-tissue deficiencies of dental implants.

Some factors need to be considered related to implant therapy within the context of this review. The aim of implant therapy is to provide patients with teeth for function and esthetics in good health. To use implants as anchoring elements for artificial teeth, the implants need to be placed in a position amenable to prosthetic reconstruction. This position may not be within the available bony envelope even in situations, where the bone volume is sufficient for placing implants. The prosthetically ideal position is determined by several factors: 1) the treatment plan, which takes into consideration the aim of prosthetic therapy; 2) the volume and the morphology of the host bone in the area; 3) the morbidity associated with the overall treatment; 4) the costs of the treatment; and 5) the desires of the patient. Hence, although avoidable, bone defects are often the consequence of placing the implant in the prosthetically driven position in ridges with sufficient bone and soft tissue.

Moreover, implants are available in different forms and shapes. For the purpose of this review treatment with rotational symmetric, screw-type implants with diameters of 3.5 to 4.5 mm and lengths of 8 to 14 mm is considered.

Due to ethical reasons, many of the factors described in the present review cannot be studied in randomized controlled clinical trials. Hence, evidence of lower levels like cohort, prospective or cross-sectional study designs or observational studies need to be included in the analysis of the available data. Furthermore, cause and effect are difficult to establish for most of the factors, which only allows describing associations between the factors and the hard and soft tissue defects.

METHODS

Electronic searches of the Medline (PubMed) database were performed and complimented by manual searches of relevant recent articles representing original research or review papers. The following basic search terms were applied: hard tissue, bone, soft tissue, mucosa, soft-tissue thickness, keratinized mucosa, tooth extraction, tooth loss, tooth fracture, trauma, periodontitis, peri-implantitis, endodontic lesion, periapical lesion, sinus floor, sinus floor expansion, growth, development, tooth migration, malpositioning, mechanical overload, systemic disease and combined with defect, deficiencies. Data from both clinical and preclinical studies were considered. Papers taken into account had to report evidence on the etiology of hard- and soft-tissue deficiencies of dental implants. No further restrictions were applied. The criteria regarding the methodology of the studies included were broad thus allowing information originating from experimental pre-clinical and clinical trials to case series to be used for this review. Since this review is of narrative nature no formal evidence-based quality assessment was performed of the studies included. The search was limited to the English language. Owing to the heterogeneity of the data no statistical analysis was performed.

OBSERVATIONS AND DISCUSSION

Hard-tissue deficiencies prior to implant placement

Hard-tissue deficiencies prior to implant placement encompass situations, where the available amount of bone does not allow placing a standard implant fully embedded in the local host bone (Table 1).

Tooth loss

Resorbed edentulous ridges may show various forms, whereas certain overall patterns have been identified in 24 maxillary and 99 mandibular completely edentulous dry skulls.[11] Generally speaking the resorption pattern of the mandible is centrifugal and that of the maxilla is centripetal. This resorption process may reach a degree, where the circumference of the mandible is further buccal than that of the maxilla. The investigators surmised that implant placement in such situations is not possible without bone augmentation to correct the bone deficiencies.[11] Many studies have investigated ridge resorption on a longitudinal basis between tooth extraction and up to 12 months thereafter.[12] Changes of the alveolar ridge were studied in 24 patients between tooth extraction and implant placement demonstrating loss of ridge profile.[13] Still another study with 16 extraction sites with spontaneous healing demonstrated vertical and horizontal loss of bone dimensions after full flaps.[14] Multiple additional studies have been published assessing the changes in alveolar bone dimensions between tooth extraction and 3 to 12 months thereafter.[15,16]

TABLE 1 Factors affecting hard- and soft-tissue deficiencies at dental implants

Hard-tissue deficiencies prior to implant placement
Tooth loss
Trauma from tooth extraction
Periodontitis
Endodontic infections
Longitudinal root fractures
General trauma
Bone height in the posterior maxilla (area of the sinus floor)
Systemic diseases
Hard-tissue deficiencies after implant placement
Defects in healthy situations
Malpositioning of implants
Peri-implantitis
Mechanical overload
Soft-tissue thickness
Systemic diseases
Soft-tissue deficiencies prior to implant placement
Tooth loss
Periodontal disease
Systemic diseases
Soft-tissue deficiencies after implant placement
Lack of buccal bone
Papilla height
Keratinized tissue
Migration of teeth and life-long skeletal changes

These resorption processes have been examined longitudinally in animal experiments and have been summarized.[17,18] It has been shown, however that the bone profile of people wearing removable dentures is continuously reduced over time under the denture bases.[19,20]

Evidence: There is a high level of evidence from well-performed prospective clinical studies by various groups of investigators describing the process of loss of alveolar bone occurring following tooth extraction. Some cross-sectional observational studies describe a pronounced loss of alveolar bone and overall ridge profile over long periods of edentulous individuals. Very scarce data is available comparatively studying the prevalence and the severity of hard tissue defects at different time points following tooth extraction.

Trauma from tooth extraction

Trauma during tooth extraction may affect bone healing at the extraction site. In a recent study in five beagle dogs raising of flaps lead to higher resorption rates and hence to smaller dimensions of alveolar process compared to flapless extraction.[21] In a clinical study, 21 patients were either treated with a widely mobilized flap design or a limited papilla sparing flap design.[22] One year after crown placement, the loss of crestal bone on the adjacent teeth had amounted to 1.1 mm in the widely mobilized flap design and to 0.3 mm in the limited flap design. The clinical and preclinical data of these two studies agree. The status of the buccal bone was assessed in 53 sites in 30 patients.[23] Bone dehiscence, plate fracture and complete plate loss occurred in 28%, 9%, and 4% of sites, respectively. In 73 out of 301 tooth extractions a traumatic event (fracture of crowns, roots, or alveolar bone) occurred during the extraction procedure.[24] Of these 73 sockets 18 developed a healing complication. A previous study compared 36 histologic samples of disturbed wound healing with 185 of undisturbed healing.[25] The results showed decreased connective tissue formation in the sites with disturbed wound healing. The investigators concluded that this disturbed wound healing will eventually lead to lower amounts of bone volume in the area of the previous extraction socket. In an experimental study in eight rabbits the buccal wall of the alveolus was deliberately removed in half or the sites (experimental group) and left intact in the control group.[26] Micro CT analysis showed decreased amounts of bone width in the experimental group in the previous socket area.

Evidence: Some data from preclinical studies exist assessing the effect of trauma to the healing process of the alveolar process. Clinical investigations reporting on hard- and soft- tissue defects resulting from traumatic tooth extraction are scarce.

Periodontitis

Chronic periodontitis has been defined as "an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment, and bone loss. It is characterized by pocket formation and/or gingival recession".[27] As periodontitis progresses the tooth supporting bone of the alveolar process is continuously resorbed adjacent to the teeth.[28] In a group of 20 patients, who had lost teeth due to periodontal disease, implant placement was not possible due to a lack of bone volume at the sites.[29] In a control group of 10 patients implants could be placed without bone augmentation in sites, where teeth had been lost due to aplasia, endodontic infections, or trauma.

Evidence: Controlled clinical studies are largely lacking comparing the need for bone regeneration, when teeth are lost due to periodontal disease or to other reasons. Many studies reporting bone regeneration procedures describe the reasons for tooth extraction, which also include periodontal disease.

Endodontic infections

Loss of supporting periodontal and surrounding bone at teeth may also result from infectious processes other than marginal periodontal disease namely by apical periodontitis.[30]

Endodontic infections are a common clinical finding leading to resorption of periapical bone.[31–35] Whereas the marginal bone may still be intact, the bone resorption around the apex of the tooth may reach a degree clinically affecting the feasibility of implant placement using standard procedures. The bone deficiencies may render implant placement more difficult. Moreover, depending on the degree of bone resorption implant placement may not be possible at all.[36] Few controlled studies with small patient samples have compared the outcome of implants immediately placed into extraction sockets of teeth exhibiting apical periodontitis to implants replacing teeth without apical periodontitis.[16,37,38]

Evidence: Scarce evidence from controlled clinical studies (three studies, 1- to 5-year observation rates, < 50 patients) indicates that at sites with periapical infections survival (96% cumulative survival rate > 5 years) and complication rates of implant are similar to implants placed in non-infected sites.

Longitudinal root fractures

Furthermore, longitudinal root fractures may lead to bone resorption and thus cause hard-tissue deficiencies at implants.[39] Pattern and amount of bone resorption are depending on factors like the type of the fracture, the extent and the duration until a therapeutic intervention.[39–41] Evidence based data is largely missing for early diagnosis of vertical root fractures.[42] Epidemiologic studies have reported vertical root fractures to account for around 10% of reasons for extractions of endodontically treated teeth.[43] At the time of tooth extraction and implant placement varying extents of bone deficiencies may be present.[44]

Evidence: Information is very scarce assessing the extent of bone destruction caused by vertical root fractures and the bone defects resulting, when implants are placed. Available data are limited to describing the occurrence of bone destruction associated with longitudinal root fractures. In addition, some prevalence data exist for longitudinal root fractures of endodontically treated teeth.

General trauma

A frequent clinical reason making it necessary to place implants is trauma. Trauma may affect teeth alone or may affect teeth, mucosa, bone along with intraoral and perioral tissues.[45] When the alveolar process and/or the body of the mandible and the maxilla are involved, a reduced volume of bone available for implant anchorage will result.[46]

Evidence: Trauma as a cause of loss of tissue is obvious. Analysis regarding frequency and extent of soft- and hard-tissue defects in such situations compared to normal ones is missing. There are no data on survival and complications of implants in prosthetically optimal position versus implants in suboptimal position following surgical reconstruction of the lost tissues.

Bone height in the posterior maxilla (area of the sinus floor)

The height of the bone in the posterior maxilla is bordered by the floor of the sinus and by the crest of the alveolar bone. Often times the height of this bone is insufficient for the placement of implants of standard length and consequently bone defects will result.[6,47–50] With the progressing age of patients the floor of the maxillary sinus expands in the caudal direction thus decreasing the bone height.[51] This process is more pronounced when teeth are extracted (average loss of height 2.2 mm) as compared to dentate sites (average 1.8 mm).[48] Additional findings support these data reporting lower height in edentulous regions (average height 7.1 mm) as compared to dentate regions (average height 9.7 mm).[50] As a consequence, oral surgical interventions will become necessary[52,53] thus allowing implant placement.[54]

Evidence: There is a high level of evidence describing the frequent presence of bone defects at implant sites in the posterior maxilla.

Systemic diseases

Some systemic diseases are associated with abnormal and incomplete tooth and bone formation during growth and development like ectodermal dysplasia.[55] When tooth development does not take place, the alveolar process is not formed at all or is reduced in its volume.[56] The resulting bone deficits may reach different degrees of magnitude. With increasing amounts of lacking bone, implant treatment becomes more and more difficult and bone grafts harvesting with associated patient morbidity becomes necessary.[57,58] Twenty-four patients received 88 implants after tumor resection in the maxilla.[59] All patients needed to be reconstructed with bone transplants prior to implant placement. At a median of 99 months of follow-up time, the cumulative survival rate amounted to 89%. As a treatment option short implants were tested in a recent study.[60] At the 5-year examination, the survival rate ranged from 74% to 95%.

Evidence: As stated above for trauma lack of bone formation as a cause of lack of tissue is obvious. Again, analysis regarding frequency and extent of soft- and hard-tissue defects in patients suffering from malformation or substantial loss of bone is missing. Similarly, there are no data on survival and complications of implants in prosthetically optimal position versus implants in suboptimal position following surgical reconstruction of the lost tissues.

Hard-tissue deficiencies after implant placement

Hard-tissue deficiencies after implant placement may generally be placed into two categories: bone deficiencies

associated with healthy situations, and those associated with diseases and malfunctions.

Defects in healthy situations

Defects of the alveolar process also exist, when teeth are present. The prevalence of dehiscence and fenestrations defects in modern skulls has been described to amount to 4.1% and 9.0%, respectively.[61] After tooth removal and implant placement, bone defects will result. Defects existing in healthy anatomical situations encompass dehiscence defects, fenestration defects, and infrabony defects.[9,62,63]

In addition, at intact ridges the prosthetically correct implant positions may not be within the bony envelope. Lingual undercuts are a frequent finding in the mandibular anterior and the premolar and molar areas. The prevalence of undercuts has been reported in cross-sectional studies to range from 36% to 66% in the posterior area[63–65] and from 2.4% to 8% in the anterior area.[64,66] Recently, a variant of mandibular anatomy has described and termed "hourglass" shape.[67] Ten out of 719 patients in need of full mandibular reconstruction exhibited this variant of mandibular shape.[67]

Evidence: Well-conducted cross-sectional clinical studies exist describing the frequency of bony undercuts in the mandible possibly leading to bone defects at implants in these sites. No valid data are available describing the prevalence of clinical conditions with these defect situations.

Malpositioning of implants

A factor, which has been given increased attention recently, is malpositioning of implants. In a group of 125 implants malpositioning was identified as the most important factor with an odds ratio of 48 associated signs and symptoms of peri-implant tissue breakdown.[68] Malpositioning as the reason for explantation was reported in 22 (14%) out of 151 implants scheduled for removal.[69] Buccal mucosal recession was observed to be significantly associated with more buccal implant positioning in a prospective cohort study including 30 implants placed in esthetic sites.[70] These findings were corroborated in a retrospective study with 42 single implants in the esthetic zone reporting a significant association of buccal mucosal recession with buccal implant positioning.[71] Another retrospective study photographically analyzed the level of the mucosal margin at 85 single tooth implants in the esthetic zone compared with the reference central incisor.[72] Again mucosal recession was associated with buccal implant position. Similarly, a multivariate analysis performed in a group of 93 patients with single implant reconstructions found a correlation between the bucco-oral position of the implant and the height of the buccal crest 4 months after implant placement.[73] Thus, each 1 mm that the implant was placed more buccally from the center of the alveolus resulted in a more apical position of the buccal crest of 0.22 mm.

Evidence: Few prospective cohort studies report in a structured manner on the effect of implant positioning on the hard and soft tissues at the implant site. In addition, several reports of single or multiple cases deal with reconstructive difficulties when dealing with malpositioned implants. These include fabrication of specific prosthetic parts, leaving certain implants unrestored and surgical interventions to remove implants or reposition them in a more favorable prosthetic location.

Peri-implantitis

Peri-implantitis includes the following components: "changes in the level of crestal bone, presence of bleeding on probing and/or suppuration; with or without concomitant deepening of peri-implant pockets".[74] Peri-implantitis leads to the loss of hard and soft tissue at implant sites (for details see the review on this topic of this workshop).

Mechanical overload

Mechanical overload has been described as another possible factor leading to hard-tissue deficiencies at implants.[75] Mechanical overload may be categorized into two different entities: loading forces preventing the implant to osseointegrate during the healing phase, and loading forces destroying a previously established osseointegration. The absence of micromotion is not a prerequisite for successful osseointegration. It has been shown that during the phase of bone integration of an implant micromotions of less than 50 μm to 150 μm are still amendable to successful bone integration.[76] Excessive strain can lead to bone resorption, whereas magnitudes below this strain result in bone apposition. The clinically responsible parameters for the pathway of overload of already integrated implants have not been identified thus far.[77–81]

Evidence: The evidence for overload of osseointegrated implants leading to hard and/or soft tissue defects is very scarce. There is a complete lack of well-structured studies testing overload in a clinical environment. The evidence for loss of osseointegration due to overload is limited to anecdotal reports of single or multiple cases.

Soft-tissue thickness

It has recently been investigated whether the thickness of the soft tissues influences the behavior of the crestal bone during tissue integration of implants. Twenty-three implants were placed in 19 patients.[82] The implants were divided into two groups related to soft tissue thickness. At the one-year follow-up examination the marginal bone loss at the implants in the thin group was in the magnitude of 1.5 mm, whereas the thick group only measured around 0.3 mm. Implant abutment connections were evaluated in another study.[83] In addition, the investigators analyzed the effects of the buccal soft tissue thickness on marginal bone level changes in 32 patients.

They found a significant correlation between soft tissue thickness and bone loss with more loss (0.3 mm versus 0.1 mm) at thin soft tissue sites at the 1-year examination. The findings that thin soft tissues lead to increased marginal bone loss were confirmed in a recent study.[84] In addition to the thin and thick tissue-groups the investigators followed a third group with about 30 patients, where they increased the thin soft tissue at implant placement by grafting. The results showed bone loss, which was not different from the thick soft tissue-group.[84] Using a different implant system, patients were also stratified into three groups of about 30 patients each.[85] Groups 1 and 2 exhibited thin soft tissues, whereas group 2 received grafts for increasing the thickness and group 1 did not. Group 3 had thick soft tissues. One year after implant placement group 1 had lost significantly more marginal bone (about 1.2 mm) than groups 2 and 3 (about 0.2 mm), which were no different from each other.[85] Yet another study stratified the patients according to mucosal thickness into two groups of 40 patients each. At the 1-year examination after implant placement, the group with thin tissues showed 1.2 mm and the group with thick tissues 0.2 mm of crestal bone loss.[86] These clinical results are in line with a previous preclinical study, where thinning out of the mucosa at implant sites lead to increased marginal bone loss.[87] It has been hypothesized that one of the reasons for this is the reestablishment of the biological width around implants penetrating the mucosa.[88,89] Since this biologic width usually exceeds 2 mm for titanium and zirconia dental implants[90] a resorption of the crestal bone is postulated to take place to generate space for connective tissue and epithelium adherence to the implant surface. These studies combined suggest that thin soft tissues covering the surgical sites can be a reason for hard-tissue deficiencies at implants.

Evidence: There is a significant amount of controlled prospective studies with medium size patient samples indicating that thin soft tissues lead to increased marginal bone loss compared to thick soft tissues at implants. The majority of the data, however, have been published by one specific group of researchers.

Systemic diseases

Hard-tissue deficiencies after implant placement may also result from systemic diseases, from bone diseases, from the intake of medications, and from certain forms of therapies. Most notably the prolonged medication of high doses of bisphosphonates[91] increases the risk of bone necrosis of the jaws in conjunction with implant therapy.[92,93] In addition, high dose radiotherapy in the jawbone regions may lead to impaired bone turnover and thus to bone loss at implants.[94,95] In addition, increased bone loss as well as soft-tissue recession has been noted in some papers on long-term results, when patients underwent radiotherapy.[96]

Evidence: There is some evidence from case reports and case series demonstrating that implants in patients suffering from certain systemic diseases suffer from increased rates of hard tissue deficiencies.

Soft-tissue deficiencies prior to implant placement

Soft-tissue deficiencies prior to implant placement encompass the following situations: the available amount of soft tissue does not 1) easily allow soft-tissue coverage of bone volume augmentations; 2) allow tension free primary coverage of the site of implant placement; or 3) allow tension free adaptation of the keratinized soft-tissue flap around the neck of the placed implant (Table 1).

Tooth loss

As stated above with respect to hard-tissue deficiencies, the changes to the ridge occurring after tooth loss are the most common reason leading to soft-tissue deficiencies prior to implant placement. At the same time as the bony profile of the alveolar ridge is reduced in size following tooth loss, the covering soft tissue is also reduced. When implants are to be placed after bone and soft-tissue healing are completed, a diminished amount of soft tissue to cover the site of implantation and concomitant bone regeneration can be an important clinical problem.[90]

Extraction sockets left for spontaneous healing exhibited vertical and horizontal loss of ridge volume as assessed on study casts. Significant vertical but not horizontal resorption was confirmed in a study with 10 extraction sockets in five patients.[97] Silicone impressions at 101 sites taken before and 3 months after tooth extraction for combined assessment of ridge dimensions including both hard and soft tissues revealed only small changes to the ridge.[98] When assessing study cast in 44 patients immediately after tooth extraction of posterior teeth with full thickness flaps and 12 months later, the magnitude of change to the outer contour of the alveolar process has been estimated to amount to 50% in bucco-lingual direction with the resorption being clearly more pronounced at the buccal compared to the lingual surfaces.[99] The crestal resorption during this same time frame was in the magnitude of 1 to 2 mm. The patterns of resorption more than 12 months after tooth extraction have not been studied in detail.

Evidence: There is a high level of evidence from well-performed prospective clinical studies by various groups of investigators describing the process of loss of covering soft tissues occurring following tooth extraction.

Periodontal disease

When left untreated, periodontitis will lead to loss of periodontal support including recession of the soft tissues and

resorption of the tooth-supporting bone.[28] Chronic periodontitis has been defined as "an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment and bone loss. It is characterized by pocket formation and/or gingival recession".[27] In cases of recession the available soft tissue is reduced compared to a healthy situation.

Evidence: Controlled clinical studies are largely lacking comparing the effect of the soft tissue available, when teeth are lost due to periodontal disease or to other reasons. Few studies reporting regenerative procedures after tooth extraction also assess the amount of soft tissue present in a comparative manner between sites with and without periodontal disease.

Systemic diseases

Some systemic diseases are associated with abnormal and incomplete bone formation, e.g. osteogenesis imperfecta.[100,101] The reduced bone formation may result in a bone volume too small to place implants. The soft tissues cover the bone volume present. When more bone volume is needed for implant placement, bone augmentation will be necessary. The available soft tissue may then be insufficient to cover the new bone volume during the regeneration surgery. This lack of soft tissue may render implant treatment more challenging.

Evidence: to date there is scarce data looking into means to increase the amount of soft tissue to facilitate the coverage of bone augmentation sites.

Soft-tissue deficiencies after implant placement

Lack of buccal bone

The lack of buccal bone at implants has been reported to be associated with decreased height of facial soft tissues.[102,103] Twenty-four patients received dental implants immediately placed into extraction sockets.[102] Guided bone regeneration (GBR) was performed and single crowns were inserted. Seven years later, cone-beam computed tomography (CBCTs), were taken to assess the labial bone. Of the 14 patients attending the follow-up examination five exhibited no buccal bone, whereas nine showed intact buccal bone plates. In the sites with intact radiographic buccal bone height, the facial mucosa was at clinically normal levels, i.e. the bone fully covered the implant surface intended for bone contact. In the situations with a lack of buccal bone at the implant, the investigators reported an average facial recession of 1 mm.[102] A large variability of the height of the buccal bone was observed in 17 of 20 patients attending a 10-year examination following immediate implant placement concomitant with GBR.[103] The mean distance from the buccal implant shoulder as assessed on CBCTs amounted to 1.6 mm, whereas the range reached from 0.1 mm to 14.9 mm. In a recent study, 18 implants completely surrounded by native bone were compared with 10 implant exhibiting bone defects treated by GBR.[104] Assessments of buccal soft tissue contours were done prior to implant placement and 3 years thereafter. During this time, the buccal contour increased to a significantly higher degree (mean 1.2 mm) in the GBR sites compared to the native bone sites (0.6 mm). In 20 patients presence of the buccal bone plate was observed 6 years following implant placement and concomitant bone augmentation.[105] The soft tissues esthetics reached high scores using the pink esthetic score (mean 8.25, range 5 to 10). In a group of 22 patients with buccal bone defects smaller than 6 mm, 11 were randomly assigned to no bone augmentation treatment.[7] Although, the bone height slightly decreased, the soft tissue levels remained stable over the 18-month period with no difference compared to the 11 sites with initial GBR to correct the bone defects. In another study 24 bone defects at implant sites were treated with GBR.[8] Four months later the remaining defect sizes were assessed and classified as absent, minimal up to 1 mm, or advanced $>$ 1 mm. Four years later a follow-up examination was performed. Whereas the probing pocket depths were similar in all three groups the values for mucosal recession and for bleeding on probing were higher in the defect groups compared to the group with complete bone coverage of the implant.[8]

Evidence: There are conflicting results from controlled prospective clinical studies and from cohort studies reporting whether or not the buccal bone plate will remain stable over time and will support the soft tissue buccal to the implant.

Papilla height

Another major soft-tissue deficiency is the reduced papilla height between two adjacent implants.[106,107] This situation can cause significant esthetic problems in the visible area. In 33 patients, 136 measurements of papilla height between two implants were performed. The mean papilla height from the bone crest to the top of the papilla amounted to 3.4 mm with a large variability reaching from 1 to 7 mm.[108] This is considerably less than the previously reported value of the normal papilla height of 5 to 6 mm between two adjacent teeth.[109] The papillae at single tooth implants were assessed in 27 implants in 26 patients. The mean papilla height at the 52 sites available for measurement amounted to 3.9 mm between a single implant and an adjacent tooth.[110]

Evidence: Clinical cross-sectional and some longitudinal studies indicate that the papilla height between implants and teeth is affected by the level of the periodontal tissues at the teeth. The height of the papilla between implants is determined by the bone crest between the implants. These processes, however, are not well understood due to the lack of well-controlled studies.

Keratinized tissue

The need for an adequate band of keratinized tissue at implant sites has been discussed controversially in the past.[111] The possible association between the width of the keratinized mucosa at implant was studied in a group of 39 patients.[112] Patients had been treated 5 to 10 years before this examination. In addition to the width of the keratinized mucosa mobility of the mucosal margin was assessed. The statistical analysis failed to reveal an association between the width of the keratinized mucosa or the mobility of the marginal mucosa at the implant sites regarding plaque accumulation, gingivitis, bleeding on probing, or probing pocket.[112] Over a period of at least 3 years, 339 implants were longitudinally followed in 69 patients.[113] Subgroups were made according to the amount of keratinized mucosa present. Results revealed no difference regarding changes in marginal bone levels. The gingival index (0.9 vs 0.8) and the modified plaque index (1.5 vs 1.3) were, however, higher in the subgroup with keratinized mucosa of < 2 mm compared with the subgroup with > 2 mm.[113] In another clinical study thirty patients were identified with < 1 mm of keratinized mucosa at implant sites.[114] Half of the patients underwent surgery for widening of the band of keratinized mucosa and half did not. After an observation period of 10 years a significant difference in gain of keratinized mucosa was present (intervention group 3.1 mm, non-intervention group 0 mm). None of the clinical parameters studied (Quigley-Hein plaque index, bleeding on probing, probing pocket depth, presence of peri-implantitis) were different between the two groups.[114] In contrast, 58 patients with 307 implants completed the 5-year examination of a study assessing the relationship between the width of the keratinized mucosa at implants and some clinical parameters in edentulous mandibles with fixed reconstructions.[115] At sites with < 2 mm compared with > 2 mm of keratinized mucosa the investigators reported higher plaque scores (0.7 vs 0.4) and bleeding tendencies (0.2 vs 0.1) at lingual sites and more recession (0.7 vs 0.1) at buccal sites. No additional differences were reported.[115] Fifteen edentulous patients with mandibular overdentures on four implants were stratified according to the presence or absence of keratinized mucosa at the buccal aspects of the implants.[116] The 19 implants in 15 patients with at least 2 mm of keratinized mucosa had significantly lower plaque (0.3 vs 0.6) and gingival indices (0.1 vs 0.6) than the 17 implants in 15 patients without keratinized mucosa.[116]

When primary coverage of an implant site is aimed at following tooth extraction, a buccal flap is normally raised, advanced and placed in contact with the lingual flap. In 11 patients, ridge preservation was performed and the site was either closed by advancing the buccal flap or not covered to allow for open healing.[117] The 6-month reevaluation revealed the mucogingival junction to be displaced coronally to a significantly greater extent in the group with flap closure (3.8 mm) compared to the control group (1.2 mm). This lack of keratinized tissue is normally more pronounced at the buccal aspect compared to the lingual one.

Evidence: There are numerous prospective, controlled clinical trials assessing the associations between clinical and radiographic parameters and the presence or absence of a band of keratinized mucosa at implant sites. To date, the results are inconclusive regarding the effect on long-term health and maintenance of dental implants exhibiting these clinical conditions. The effects of clinical manipulations on the position of the mucogingival junction have only scarcely been studied and are, hence, poorly understood.

Migration of teeth and life-long skeletal changes

Discrepancies between implants and teeth may develop due to tooth wear and changes in the anatomy of face and jawbones in adults long after the patient has finished growth and development.[118] This will cause discrepancies of the facial tissue heights between the implant crowns and the natural teeth. Similar to tooth wear these changes occur slowly and take time to manifest clinically. With the increased use of osseointegrated implants over longer periods of time these problems are expected to increase. Changes in the maxillary and mandibular arches occur continuously. From an original sample of 89 boys and 86 girls aged > 3 years, 15 men and 16 women could be reexamined at 45 years of age.[119] Between 13 to 45 years of age the maxillary arch length decreased an average of 5.7 mm in males and 4.6 mm in females. During the time period from 8 to 45 years of age the mandibular arch length decreased on average by 7.4 mm in males and 8.3 mm in females. In another study, 14 females with implants bilaterally in the maxillary molar region and at least one implant in the incisor region were longitudinally followed in the age range from 9 to 25 years.[120] During the observation period the results showed an average eruption of the maxillary incisors of 6 mm downward and 2.5 mm forward. The maxillary first molars experienced an average eruption of 8 mm downward and 3 mm forward underscoring the continuous skeletal changes over time.[120] Wear facets at approximal surfaces of molars and premolars were studied in a sample of 376 skulls.[121] Tooth wear was a common finding and increasing with age. In addition, various patterns of wear were identified. The position of single implant reconstructions was studied in a group of 82 patients, of which 47 were available for examination 18 years after implant reconstruction.[122] In 40% of the patients the implant reconstruction showed signs of infraposition compared to the adjacent teeth. In a recent retrospective study, 174 implants in 128 patients were examined for interproximal contact loss after implant restoration times ranging from 3 months to 11 years.[123] More than half (53%) of the

reconstructions showed interproximal contact loss. Seventy-eight of these open contacts were located mesially and 22% distally. Eight implant reconstructions exhibited mesial and distal interproximal contact loss.[123] Over an observation period of 16 years tooth movements were examined adjacent to 28 single-tooth implants.[124] Tooth movements included vertical and palatal displacements and occurred in some but not all patients. In a sample of 146 implants in 105 patients loss of the interproximal contact was examined prospectively over time.[125] During the observation period, 43% of 186 interproximal contacts were lost with a significantly greater incidence at the mesial (52%) compared to the distal (16%) aspect. Using the pooled data, the investigators calculated that half of the interproximal contacts might be lost in 5.5 years of function.

Evidence: Whereas migration of teeth adjacent to implants is well documented in prospective and in cross-sectional studies, the clinical consequences regarding hard- and soft-tissue defects are poorly examined and understood.

CONCLUSIONS

Hard- and soft-tissue deficiencies at implant sites may result from a multitude of factors. They encompass natural resorption processes following tooth extraction, trauma, infectious diseases such as periodontitis, peri-implantitis, endodontic infections, growth and development, expansion of the sinus floor, anatomical preconditions, mechanical overload, thin soft tissues, lack of keratinized mucosa, malpositioning of implants, migration of teeth, lifelong growth, and systemic diseases. There are varying levels of evidence for the different factors. For some there are well-controlled studies, whereas for others there is little to no scientific evidence. More research is needed to better identify the factors possibly leading to hard- and soft-tissue deficiencies at implant and their clinical impact.

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