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Dent Clin N Am 50 (2006) 339–360

THE DENTAL
CLINICS
OF NORTH AMERICA

Consequences of Implant Design

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The use of dental implants to replace missing teeth is becoming a preferred alternative for restorative dentists and their patients. Patients who previously did not seek dental replacements now present to dental practitioners and request information and replacement care. Furthermore, patients have gained such awareness of these new options that they increasingly request modification or replacement of existing dental restorations (eg, dentures, fixed partial dentures, and removable partial dentures). Quality of life analyses indicate that patients perceive their oral health status as improved by their experience with dental implants [1]. Root-form dental implants now comprise the most widely used form of treatment and often have success rates of 90% to 100%. Success and survival rates continue to improve as the physical design, surface technology, and clinician experience evolve.

Currently, two basic types of root-form implants are used. The first category of implants was introduced and developed by Branemark and colleagues [2] and the implants are referred to as two-piece implants. The two pieces consist of an implant body and a separate abutment. The implant is placed during a surgical procedure; the top of the implant is at the level of the bone crest or some distance apical to it (Fig. 1). The gingival tissues are re-approximated for primary closure over the top of the implant, which is then left undisturbed for a period of time, usually 3 to 6 months, for osseointegration. This surgical placement technique is referred to as submerged placement.

After successful integration in the bone, a second surgery is performed and a healing or restorative abutment is connected to the implant (Fig. 2). This is referred to as second-stage surgery. The gingival tissues are re-approximated around the abutment as they would be around a tooth.

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0011-8532/06/$ - see front matter
doi:10.1016/j.cden.2006.03.008 *dental.theclinics.com*

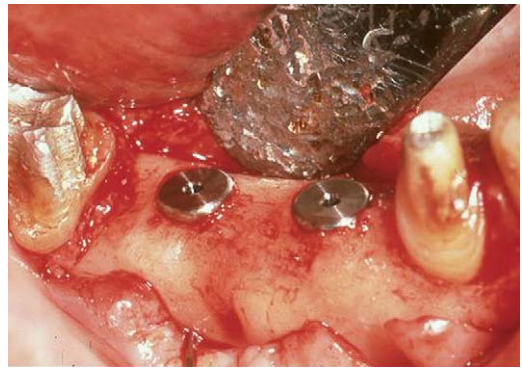

Fig. 1. Clinical photograph of two submerged (two-piece) dental implants in the posterior mandible after flaps were reflected at second-stage surgery. Note that the tops of the implants are placed slightly apical to the alveolar crest and only the thin cover screws can be seen.

A second healing period is allowed for the gingival tissues before restorative procedures are continued.

The second category of implants is referred to as one-piece implants. This concept was introduced and developed by Schroeder [3–5]. A one-piece implant comprises the implant body and the soft tissue healing abutment manufactured as one piece. The implant is surgically placed; the top is positioned coronal to the crest of the alveolar bone and the gingival tissues are reapproximated around the now transgingival implant, rather than over the top of the implant, at the time of implant placement surgery (Figs. 3 and 4). This surgical approach is referred to as non-submerged placement. Another term used to describe this implant category is single-stage implants because no

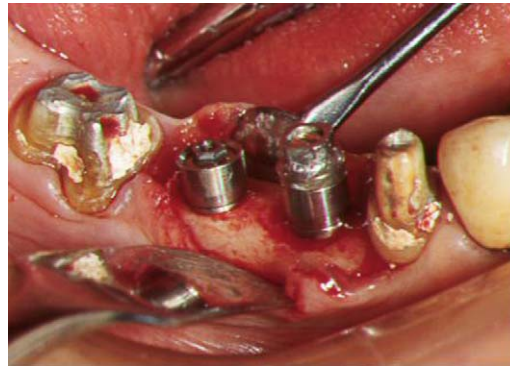

Fig. 2. Clinical photograph of two submerged (two-piece) dental implants in the posterior mandible at second-stage surgery. The thin cover screws are replaced with transgingival abutments. An interface or microgap now exists at the bone crest level where a butt-joint connection exists between the top of the implants and the apical ends of the abutments.

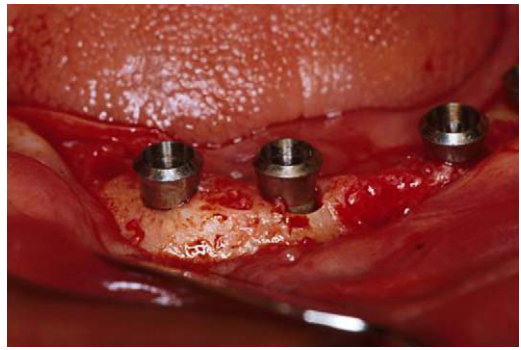

Fig. 3. Clinical photograph of non-submerged (one-piece) dental implants placed in the anterior mandible of an edentulous patient. The surgical flaps reveal the alveolar crest and the implants in the osteotomy preparations. In the middle implant a defect exists and the rough-smooth border of the implant can be seen slightly apical to the bone crest.

second-stage surgery is required. Restorative procedures may commence as soon as healing has occurred.

The discussion in this paper will reflect the terminology of one-piece implants placed using a non-submerged technique, and two-piece implants placed using a submerged technique. These techniques reflect the development and descriptive analysis of implant therapy in the literature. Currently used clinical techniques, however, also include placement of the two-piece implant and abutment components simultaneously in one surgical procedure, during which the gingival tissues are re-approximated around the abutment (ie, two-piece implants placed in a non-submerged approach). Additionally, one-piece implants may be placed subjacent to the buccal portion of the surgical flap for esthetic advantage. This is referred to as semi-submerged placement. Or, the one-piece implant can be completely submerged

Fig. 4. Clinical photograph of two non-submerged (one-piece) dental implants placed in the posterior mandible. This is a 1-week postoperative view after the sutures have been removed. The healing caps placed in the tops of the implants have been removed to reveal the internal aspect of the implants. Note the healthy condition of the peri-implant soft tissues.

at the time of surgical placement (ie, a one-piece implant placed in a submerged or two-stage approach). This might be preferred if bone augmentation procedures are to be combined with implant placement surgery.

The healed bone and gingival tissue-to-implant and gingival tissue-to-abutment relationships are analogous to, but different from the dentogingival interface of natural teeth. These relationships depend on the physical design of the implant, the location of the implant components relative to the bone, the surface technology of the implant, and the soft and hard tissue dimensions existent at the time of placement. The long-term stability of these relationships depends on the restorative and occlusal demands placed on the implant, as well as the bacterial colonization of the components and spaces created (Fig. 5).

The connection of restorative components (abutments and crowns) to the restorative interface of the implant creates a space, which can be colonized by oral bacteria. This space is sometimes referred to as the microgap. Research has shown that the creation of the microgap can have a direct influence on bacterial colonization, recruitment and localization of inflammatory cells, and the soft and hard tissue anatomical relationships around the implant complex. Long-term stability depends on the healthy attachment of epithelium, connective tissue, and bone to titanium as well as the subsequent maintenance of bone levels.

Both one- and two-piece implants are surgically placed with similar drill sizes, sequences, and methods. Although there are some variations in manufacturers' recommendations based on design features and materials, the

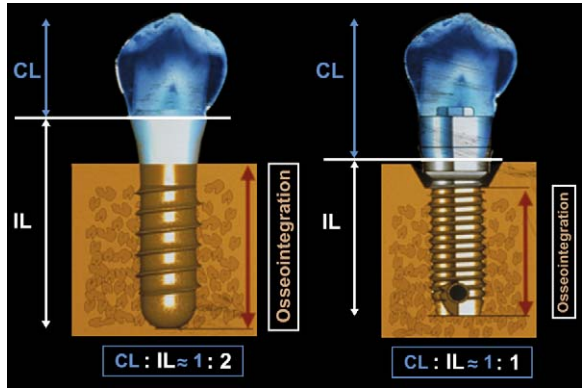

Fig. 5. Schematic diagram of a non-submerged (one-piece) dental implant on the left, and a submerged (two-piece) implant on the right. The one-piece implant has its interface above the bone level; the two-piece implant has its interface at the original bone crest level. After this interface is created at the bone crest, bone resorption occurs mesial and distal (in the schematic) but actually all around the implant, down to the first or second thread level. The crown length (CL) to implant length (IL) is less in the non-submerged (one-piece) design compared with the submerged (two-piece) design. (Courtesy of Institut Straumann AG, Basel, Switzerland, with permission.)

various protocols have become progressively more similar. In most implant systems, a screw-shaped implant macrostructure is used. The implant is screwed into the prepared bony walls of the osteotomy and, in some cases, after the osteotomy has been prepared ("tapped") for the screw threads. A cover screw is attached to the implant and then the flaps are re-approximated.

Marginal bone levels

Successful dental implant therapy requires long-term maintenance of the soft and hard tissues that surround the implant. This is particularly true for the bone-to-implant contact because osseointegration provides resistance to the forces exerted on the implant restoration. Osseointegration is a histological outcome and cannot be clinically ascertained in patients. Therefore, surrogate clinical variables must be used to determine tissue stability around the implant over time. One such surrogate variable that has been used is the level of the osseous tissue mesial and distal of the implant as determined by radiographic evaluation. One convenient aspect of the radiographic evaluation is the level of the bone adjacent to the implant as measured from a predetermined location on the implant restoration. This location is usually at the top of the implant and can also be used before implant restoration to assess the bone level around the implant. This is commonly referred to as the marginal bone level (Figs. 6 and 7). The implant macro-structure is relatively fixed and so provides a constant point from which measurements can be made, in a manner similar to the use of a stent to determine relative attachment levels in periodontal trials.

The predictability of dental implants has been established through longitudinal studies of implant survival or success (the latter being a function of some pre-specified criteria). Parameters that have been followed include detection of mobility, pain, infection, inflammation, and marginal level of bone (also referred to as crestal bone). Particular emphasis was placed on monitoring the marginal bone level over time, because some implants lost a significant amount of marginal bone and the implants failed after becoming mobile. Implant mobility turned out not to be a very sensitive indicator for implant failure because large amounts of bone loss could occur, yet the remaining bone prevented movement of the implant. Thus, when mobility of a previously osseointegrated implant is clinically detected, implant failure invariably occurs. Therefore, evaluating the marginal bone level over time allowed the clinician to better assess the status of the peri-implant tissues and facilitated earlier therapeutic intervention.

Early reports on implants in patients indicated that marginal bone loss occurred in the 1–2 mm range in the first year after restoration and after the first year generally very small amounts of bone loss occurred or the level stabilized. In these studies, the baseline radiograph was made at the time the prosthesis was placed on the implant and the studies generally included a submerged implant that had a machined surface and a butt joint

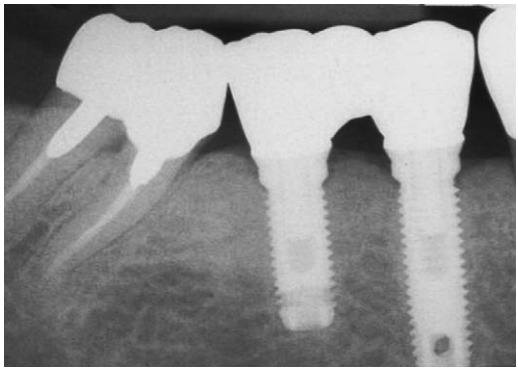

Fig. 6. Periapical radiograph of two submerged (two-piece) dental implants placed in the posterior mandible. The final crowns are connected together. Note the angular bone loss mesial and distal of each implant down to the level of the first thread of the implants. This bone loss is characteristic for this type of two-piece implant.

connection in which an external hex with a screw joint was used to connect the abutment to the implant. The bone levels were not evaluated before the prosthesis was connected because in early studies, the technique prohibited taking radiographs at the time of implant placement. At that time there was a fear of critically damaging the cells that lined the implant preparation which contribute to making the bone-to-implant contact. Therefore, in the early studies, the baseline or first radiograph was taken at the time the prosthesis was inserted and was used to evaluate changes in the marginal bone level over time.

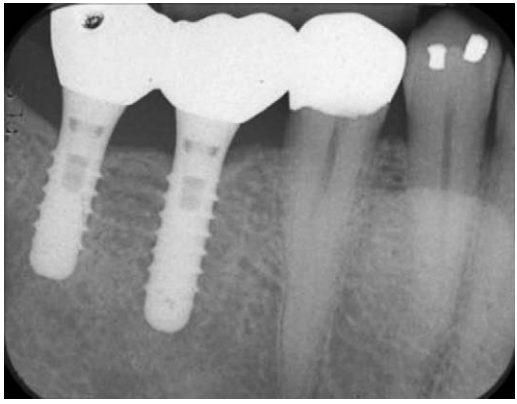

Fig. 7. Periapical radiograph of two non-submerged (one-piece) dental implants placed in the posterior mandible. The final crowns are connected together. The crowns contact the tops of the implants approximately three millimeters above the alveolar crest. This means that the interface or microgap is located coronal to the bone level.

Later studies and experiences clinically indicated that baseline radiographs could be taken at implant placement and could then be used to evaluate the changes in the marginal bone levels beginning with implant surgery. Hermann and colleagues [6,7] performed a series of studies that evaluated radiographic marginal bone changes over time around both submerged and non-submerged implants. A radiograph taken at the time of placement was used to establish a baseline from which changes would be measured. This facilitated evaluation of the marginal bone changes before prosthesis insertion and the biological events that occurred during the soft tissue remodeling as the implant or implant component parts passed through the gingival, and a peri-implant mucosal seal was created. It became evident from these studies that differences occurred in the marginal bone area if the top of the implant stopped at the bone crest level and the implant was first submerged and a second surgery was used to connect a secondary implant component. This was in contrast to a one-piece implant that was made to extend beyond the crest through the soft tissues initially (non-submerged) at the first surgery.

In one set of studies, two-piece implants (originally referred to as submerged-type implants) were placed either at the bone crest level (recommended position), 1 mm above the crest of bone, or 1 mm below the crest of bone and then closure screws were attached and the tissues closed over the top to submerge the implants [7]. In addition, one implant, placed at the crest of bone, had the abutment connected to the implant at the time of implant placement and was, therefore, a two-piece implant placed in a non-submerged surgical approach. These implant configurations were compared with a one-piece implant (originally referred to as a non-submerged type implant) placed with the border of the roughened endosseous portion of the implant at the crest of the bone and the smooth transgingival portion of the implant in the soft tissues. One last configuration was examined that used the one-piece implant with its rough-smooth border placed 1 mm below the alveolar crest. The results demonstrated that minimal amounts of bone loss occurred around the one-piece, non-submerged implant when it is placed as recommended, with its rough-smooth border at the crestal bone level. If this non-submerged implant was placed 1 mm apically, so that 1 mm of smooth collar was within osseous tissue, a small amount of bone loss occurred. If an abutment was connected at the time of first-stage surgery to a typically submerged implant and placed as a non-submerged but two-piece implant, approximately 1.5 mm of bone loss occurred after 1 month in the canine model. After that, minimal bone loss was observed.

No crestal bone changes were observed around the three submerged implants for the 3 months that they were covered with the alveolar mucosa. However, once the second- stage surgery was performed and an abutment was connected to the implant, bone loss was observed within a month around all three designs. Approximately 1.5 mm of bone loss occurred around the implant that was placed with the top of the implant at the

alveolar crest. This was identical to the same design (two-piece) that was placed in a non-submerged approach as described above. In other words, a two-piece design implant that had an interface (called a microgap) between the top of the implant and the abutment, located at the alveolar crest, was associated with about 1.5 mm of bone loss once the connection of the components took place. If these components were connected at the time of first-stage surgery, the bone loss occurred within the first month after implant placement. If however, the implant was first submerged for 3 months and then the abutment was connected, the same amount of bone loss occurred within the first month after the connection was made at second-stage surgery. The investigators suggested the bone loss observed was associated with the microgap (a two-piece implant configuration). Submerging the implant (ie, no microgap) was not associated with bone loss; however, once the abutment was connected and a microgap was created, bone loss occurred identical to the bone loss that occurred if the abutment was connected at the time of first-stage surgery (ie, a two-piece implant configuration placed in a non-submerged approach). Thus, the actual surgical technique of submerging or not submerging the implant does not have marginal bone consequences. However, once the abutment is connected to a submerged implant, bone loss occurs. The bone loss is simply delayed until the abutment is connected and the microgap is created. This association was confirmed by the fact that a one-piece, non-submerged implant was not associated with this bone loss. Thus, marginal bone loss was strongly correlated with microgap creation.

Another confirmation that marginal bone loss is associated with the presence of the microgap was that as the microgap was moved apically, more bone loss was observed. When the microgap was located 1 mm above the bone crest (ie, the top of the implant was placed 1 mm above the bone crest at the time of first-stage surgery), only a small amount of bone loss was observed. If however, the microgap was located at the bone crest level (ie, the top of the implant was placed at the bone crest level at first-stage surgery), more bone loss was observed. Finally, if the microgap was located 1 mm apical to the bone crest (ie, the top of the implant was placed 1 mm apical to the bone crest level at first-stage surgery), the greatest amount of bone loss was observed in these two-piece configurations. Thus, marginal bone loss strongly correlated with microgap location.

In the experiments described, the bone loss observed in all cases occurred within the first month after microgap creation. After that, no further significant loss of marginal bone occurred. This again suggests that the observed loss is associated with the creation of the microgap and that afterwards the driving force for further bone loss is no longer present. Therefore, the etiology of the marginal bone loss associated with the creation and location of the microgap appears limited to this structure.

In summary, radiographic marginal bone levels have been used as a clinical outcome to determine the status of the implant restoration. Depending

on implant design, marginal bone loss is observed after implant placement and the abutment is connected. Thus, the creation and location of the microgap is associated with marginal bone loss. This bone loss occurs relatively rapidly and then stabilizes. The presence of ongoing bone loss is a clinical sign of instability and likely, pathology. Based on the loading conditions, some bone loss may be observed, but equilibrium tends to be reached in the bone level. Progressive bone loss suggests that a problem exists and the clinician needs to take therapeutic action.

Biologic width around dental implants

Natural teeth are surrounded by gingival soft tissues that provide a biologic seal between the oral cavity and the inside of the body. This unique structure is composed of epithelium and soft connective tissues that are continually bathed in a transudate called gingival fluid. The linear dimensions of this structure have been described and the epithelial and connective tissue dimensions were referred to as the biologic width by Gargiulo and coworkers [8]. Cadaver specimens were measured and mean values determined for the space occupied by the sulcus depth, the junctional epithelium, and the gingival connective tissues. Questions arose about whether the soft tissues around implants had similar structures. A pioneer in endosseous dental implants, Andre Schroeder [3], used histologic specimens that showed both the titanium implant and the surrounding tissues to describe the epithelium and connective tissues around the implant. Buser and colleagues [9] further explored these tissues and described the existence of a junctional epithelium similar to that found around teeth and a surrounding connective tissue, which appeared to encircle the implant. This connective tissue was a 50 to 100 μm avascular zone that ran perpendicular to the implant long axis. Peripheral to this scar-like tissue was a vascular zone and large connective tissue fiber bundles that ran parallel to the long axis of the implant. Although the epithelial attachment to an implant surface was similar to the natural dentition, the connective tissue contact was completely different.

The marginal bone tissue around an implant is directly influenced by the presence or absence of a microgap and its location. Bone loss is associated with the two-piece implant design and is generally not observed with one-piece dental implant designs. Based on these observations, investigators questioned whether a biologic width existed around implants analogous to that seen around teeth. Additional questions concerned the influence of implant design on the biologic width. Weber and coworkers [10] had described histological differences in the location of the apical extension of the junctional epithelium between one-piece and two-piece implant designs. These investigators had observed that around two-piece implant designs the epithelium was always located apical to the microgap, and that the epithelium around two-piece implants was always located more apically than around one-piece implants. Cochran and colleagues [11] measured the linear

soft tissue dimensions around implants and demonstrated that a biologic width existed around endosseous dental implants. In addition, these dimensions were different between one- and two-piece implant designs. The biologic width dimension around one-piece dental implants was similar to the biologic width dimension described by Gargiulo and coworkers [8] for natural teeth. This finding was significant for esthetic reasons and has implications for the surgical placement of the implant. The biologic width dimension for two-piece implants was different (larger) compared with one-piece implants and natural teeth. These findings suggested that the concept of biologic width is valid in both teeth and implants, despite the obvious differences between these two structures. In addition, the presence of the microgap and its location influences the epithelial dimension and location. Thus, a microgap in two-piece dental implant designs influences marginal bone levels and also influences the biologic width of the surrounding soft tissues. The epithelial structure around teeth and implants is similar but the soft connective tissue structure is completely different. In spite of these differences, the biologic width around one-piece implants and natural teeth is similar. These physiologic similarities make it possible for a clinician to create esthetic tooth replacement with implant restorations.

It is not known why the biologic width dimension is similar between one-piece implants and natural teeth in spite of different gingival connective tissue structures. Because the epithelial structure is similar between teeth and implants, it is not surprising that these linear dimensions are similar. What is remarkable is that a junctional epithelium forms around the implant from the existing keratinized oral epithelium similar to what happens around the natural tooth after periodontal surgery. This suggests that the physiologic conditions that govern junctional epithelium formation are independent of the adjacent non-vascular hard structure (an implant or tooth root). In fact, it may be that anytime a nonvascular solid structure is placed into oral epithelium, the host reaction is a physiologic structure (ie, the nonkeratinized junctional epithelium). This likely relates to an acquired pellicle formation, microbial plaque accumulation, and corresponding oxygen tension changes. A remarkable finding is that a hemidesmosomal attachment is formed on the titanium oxide surface similar to that which is formed on the tooth root surface. This again suggests a physiologic structure that forms regardless of the nature of the substrate and again may reflect a host reaction to a nonvascular solid structure in the oral cavity. In regard to the gingival connective tissues, the linear biologic width dimension is similar between the one-piece implant and the natural tooth in spite of completely different structures. This counter-intuitive finding suggests that in spite of dramatic structural differences (in the case of the connective tissues) an overall physiologic phenomenon drives the apico-coronal dimension of the connective tissues and epithelium, which finds expression in the literature as the concept of biologic width. This physiology is unknown but may be related to the location and functional demands of the tissues within

the oral cavity. Thus, the oral cavity demands determine the oral soft tissue dimensions in spite of actual structural differences in the connective tissue contact between teeth and one-piece implants.

It has been shown that around natural teeth, the epithelial component is more variable than is the connective tissue component. That is, the connective tissue dimension remains more stable over time. Hermann and colleagues [12] evaluated the changes over time in the biologic width dimensions around one-piece implants and determined that the connective tissue dimension around implants was more stable than the epithelial dimension, a phenomenon also observed in the natural dentogingival interface. Interestingly, this study included implants that were not loaded (ie, the implants did not have restorations) and implants that were loaded (with restorations) for 3 months and for 1 year. The biologic width dimension did not vary significantly regardless of whether the implant was unloaded, loaded for a short time, or loaded for a long time. This suggests again that the formation of a biologic width is a physiologic response in the oral cavity and is not dependent on the presence or absence of loading, or the length of loading time. This is reinforced by analogy with the natural dentition where a biologic width is formed around teeth that may not be in occlusion, such as around third molar teeth or teeth that have lost the antagonist tooth in the opposing arch. The fact that the connective tissue dimension is more stable over time than the epithelium dimension, both around teeth and one-piece implants, may be related to the fact that the connective tissues once formed are predominated by the protein collagen, and as collagen matures, more cross linkages occur which stabilizes this tissue. This highly cross-linked connective tissue structure would then be more resistant to dimensional change over time. In the case of the junctional epithelium however, this structure is constantly being challenged by microbial growth and pathologic microbial products. The host reacts by changes in the inflammatory immune system including widening of the intercellular epithelial spaces and the recruitment of polymorphonuclear leukocytes. The host response would be expected to fluctuate greatly over time depending on the challenge, which varies daily based on host stress, home oral hygiene, professional hygiene etc. Thus, it would not be unexpected to see more changes in the epithelial dimension compared with the connective tissue dimension around both teeth and implants. Another point regarding the biologic width is that the epithelium is always found below the microgap on a histological basis.

Bacterial challenges around implants

The natural dentition is continuously challenged by microbial plaque, which consists of hundreds of species of bacteria. These bacteria and their products elicit a host inflammatory–immune reaction. Dental implant restorations face the same microbial challenge but, unlike the natural tooth,

consist of multiple component parts. The connection of these multiple component parts has changed over the years but screw connections are most common. This often results in the creation of an interface between components located within the tissues surrounding the implant. For example, two-piece implants by design have an interface at the crestal bone level where the top of the implant contacts the abutment that fits on top of the implant. Further coronally, an interface is found where the crown meets the abutment. One-piece implant designs result in only one interface between the top of the implant (which by design extends coronal to the crestal bone) and the crown. As noted above, these interfaces or microgaps are associated with marginal bone loss when they are located close to or within the bone tissue. The question then becomes why bone loss is associated with the interfaces (microgap).

Experimental research and investigation of implant components from patients indicates that the interfaces become contaminated with bacteria and their products. Experimental studies have connected the components together on the bench top under ideal conditions of asepsis, not likely to be attained in the mouth because of the presence of bacteria. These implants were then incubated in solutions that contained various bacterial species. Under these scenarios (which are much more favorable than would actually exist in the mouth), bacterial contamination is found in all the interfaces examined. In addition, implant components taken from patients also reveal bacterial contamination of the internal aspects of the components. Thus, bacteria are able to penetrate the interface and create microbial niches in the interfaces [13–19].

Studies examining the soft tissues that surround the implant have demonstrated that inflammatory cells are present in variable amounts adjacent to the implant depending on the implant configuration [19]. These studies have examined the inflammatory cells in the soft tissues adjacent to both one- and two-piece implant designs with varying relations between the microgap and the alveolar crest. In addition, a two-piece implant placed in a non-submerged approach (the abutment was connected to the implant at the time of first-stage surgery) was also examined for the presence of inflammatory cells in the soft tissues around the implant. The analysis was performed 6 months after implant surgery and two-piece implants had abutments connected at a second-stage surgery 3 months after implant placement. The results revealed that two-piece implants placed at the alveolar crest resulted in identical inflammatory cell accumulation patterns regardless if the abutment was connected at first or second-stage surgery. In these cases, the most inflammatory cells were located at the level of the interface (where the original bone crest was located) and the number of cells decreased as one moved away from the interface both in an apical and in a coronal direction (Fig. 8). The predominant inflammatory cell was the polymorphonuclear leukocyte, which is normally associated with a more acute reaction. This peak of inflammatory cells correlated with the interface whether the

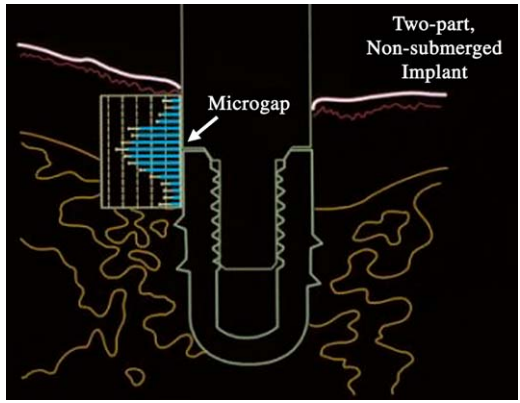

Fig. 8. Schematic drawing of a submerged (two-piece) dental implant design with abutment attached at the time of first-stage surgery (implant placement surgery), which results in a two-piece implant placed in a non-submerged surgical approach. This shows the result after 6 months of healing where the alveolar bone crest has moved from the abutment or implant interface down the implant after bone loss has occurred. If the inflammatory cells are counted along the side of the implant soft tissues, the greatest number of cells (represented in the inset graph as the longest bars) is located at the microgap or interface between the implant and abutment. The literature demonstrates that bacteria are found in the microgap and likely cause recruitment of the inflammatory cells.

interface was moved apically or coronally. Furthermore, this accumulation of inflammatory cells (and hence inflammatory reaction) was not observed when no interface was present (ie, adjacent to a one-piece implant). The one-piece implant design had many fewer inflammatory cells around the implant: the majority of the cells were located coronally near the junctional epithelium. The predominant cell type around this implant configuration was the mononuclear cell, the number of which diminished in an apical direction. These studies also demonstrated that the amount of bone loss was positively correlated with the accumulation of inflammatory cells apical to the microgap around the two-piece implants. Such bone loss (or inflammatory cells) was not observed around the one-piece implant design.

These inflammatory cell findings are suggestive of mechanisms that may relate to the tissue changes that occur around the different implant designs. One possibility is that the interfaces become colonized with a biofilm after being exposed to the oral environment (ie, during abutment connection and second-stage surgery). The growth of the bacteria and subsequent release of pathological products provide a continual stimulus to the host, which reacts by sending inflammatory cells to the site (in this case the adjacent interface soft tissues). These cells in turn, if located adjacent to or within a certain dimension to the alveolar crest, stimulate the recruitment and differentiation of osteoclast cells which then start resorption of the bone. This bone resorption continues until there is a certain distance between the site of infection (the interface or microgap) and the alveolar

bone, essentially walling off the source of the infection. This might be considered the effective range of the biofilm and is analogous to a similar plaque–bone distance observed by Waerhaug around periodontally involved teeth [20]. Further significant bone loss would not be observed because the infection is now a set dimension away from the bone crest. This scenario is exactly consistent with the marginal bone changes described above. Recall, for example, that two-piece implants placed in a non-submerged approach (ie, the abutment was connected at the time of implant placement) resulted in bone loss in the first month and then little loss occurred afterwards (ie, the host reacted to the infection and once bone was a set distance away from the infection, no further bone loss occurred). Similarly, submerged two-piece implants did not experience bone loss until the second-stage surgery when an abutment was added and an interface (microgap) was created (ie, an infected interface was created). In all these cases, bone loss occurred again within the first month and little loss occurred after that (ie, the host reacted by resorbing bone to a set dimension away from the infection). Also, the bone loss increased as the interface was moved apically but then after one month, the bone level stabilized (ie, the infection was placed more closely to the bone so more bone loss occurred but once a set distance occurred away from the infection, the bone loss stopped). These findings are all consistent with bacterial contamination of the interface, an inflammatory reaction by the host to that contamination, and bone changes associated when the inflammation approached the bone within a certain dimension. Further support comes from the observation that there were no such bone changes around one-piece implants and no peak of inflammatory cells. These findings are reinforced in clinical studies of two-piece machined implants where bone loss occurred to the level of the first thread when an abutment was connected at second-stage surgery. This was such a consistent finding that a mean 1.5 mm of bone loss was accepted as one of the success criteria in the first year of loading for this design of implant [21].

The quality of the inflammatory reaction adjacent to the interface of two-piece designs after six months in the canine proved to be interesting. Predominantly polymorphonuclear leukocytes and some monocytes were observed. This suggests that the host reacts with a chronic acute type reaction to the interface. It also suggests that this reaction is persistent and that new pathogenic substances are being released over time from the interface. This again is consistent with the scenario described above whereby bacteria occupy the interface, they grow and flourish within the interface, and continually release substances that the host must deal with yet the host cannot eliminate. This is consistent with plaque formation on the tooth root surface that stimulates an inflammatory reaction in the tissues (gingivitis), and if the inflammation approaches the alveolar crest within a certain dimension, bone loss is initiated (periodontitis). This is an effective strategy for the host to try to isolate an infection which it cannot effectively eliminate. The body similarly tries to isolate an endodontic infection at the apex of the tooth by

resorbing the periapical bone and forming an epithelial lined cavity which results in a radiolucent periapical lesion. These implant findings are also consistent with descriptions of the inflammatory reaction around teeth and its correlation to periodontal bone loss. Several investigators have described an extended arm of inflammation or radius of infection. Although the names differ, the concept is the same (ie, when inflammation reaches a certain distance from the alveolar crest, bone loss results around the tooth) [20]. This discussion suggests that the same phenomenon occurs around implants that have contaminated interfaces (ie, when the interface is located at or near bone, bone loss is initiated until a certain distance is reached so that the infection and associated inflammation is no longer within reach of the bone tissue).

These results, taken together, reveal that interfaces between implant components that become contaminated should be avoided near alveolar bone and in the more apical area of the soft tissues around the implant (Fig. 9). Some implant systems have attempted to either eliminate the infection from the interface or move the infected interface away from the bone level. For example, one solution has been to place an anti-infective material at the interface to help with the infection and subsequent inflammatory reaction. However, this approach has not been widely adopted. Another, more elegant solution has been to shift the interface away from the bone by having the abutment fit within the inside of the two-piece implant so that the interface is separated from the bone horizontally by the thickness of the implant outer wall to the inner wall, which mates with the abutment. Another approach would be to effectively seal the interface against bacterial contamination. This latter approach seems unlikely using butt joints on components but may be possible if cold welds (such as can be created

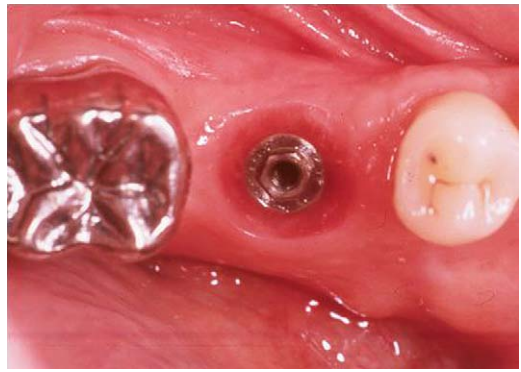

Fig. 9. Clinical photograph of the top of a submerged (two-piece) dental implant after the temporary restoration has been removed. With this implant design, an external hexagonal piece extends coronally and the abutment fits over the top. The abutment (or in some cases, the crown) extends to the top of the implant which was placed at the alveolar crest (thus creating an interface or microgap at the bone level).

with some Morse tapers) could be created between the implant and the abutment (presumably with internal connections of the abutment in the implant). These are attractive possibilities but need to be proven with data and histological evaluations. Without such data, they remain only as attractive possibilities.

Restoration of one-piece implants

When using one-piece implants, the coronal aspect of the implant is visible and more accessible clinically; seating the abutments is straight forward. The practitioner can visually confirm that components are fully seated. Therefore, confirmatory radiographs to verify seating may not be required (Figs. 4 and 10). This can facilitate quick placement as well as replacement of abutments and cover screws. Also, access to the top of the implant facilitates seating and verification of impression components and thus can save time during this process.

Simplified impression techniques have been developed to take advantage of the clinically accessible top of the implant. For example, in some systems, self-retained plastic components are used to record and transfer the exact clinical position of the implant as well as the restorative margin and the position of the abutment to the alveolar crest. These plastic components can be picked up in a closed tray final impression. An appropriate analog is then securely placed in each implant site in the impression and the working cast is poured. The process is similar, if not less complicated, than conventional crown and bridge restoration. This technique is described in some detail elsewhere in this issue (see Haubenreich and Robinson). Cementation of such implant-supported restorations can routinely be accomplished (Fig. 11).

One-piece implants make cementation of restorations practical. Cement-retained restorations are increasingly used because of the overall ease of use, the lower costs involved, and the minimal maintenance required.

Fig. 10. Buccal view of two one-piece implants placed in the posterior mandible before abutment placement. Note the healthy condition of the peri-implant soft tissues.

Fig. 11. Stone model of a non-submerged (one-piece) dental implant and cemental abutment placed in the mandibular posterior sextant. Once this model has been created, crown fabrication can occur using conventional crown-and-bridge techniques.

Furthermore, the similarity to conventional crown and bridge restorations is comfortable for most dentists. At the time of cementation with a one-piece system, residual cement can be eliminated more thoroughly when the top of the implant is exposed rather than situated at the bone level. Apically placed subgingival one- or two-piece implants may, however, make it difficult to remove cement and it may be preferable to retain the screw in those cases.

The use of one-piece solid abutments has simplified the restoration of one-piece implants. One-piece abutments consist of both the abutment and the screw portion for connection to the implant as one manufactured part. The entire abutment is screwed into the implant (Fig. 12). Anti-rotation for this abutment can be ensured by a minimally tapered cone-in-socket fit of the abutment into the implant rather than an external hex connection. This connection design, referred to as a Morse taper configuration, is a reliable, stable, non-loosening attachment mechanism which prevents further rotation of the abutment and eliminates the necessity of an additional abutment

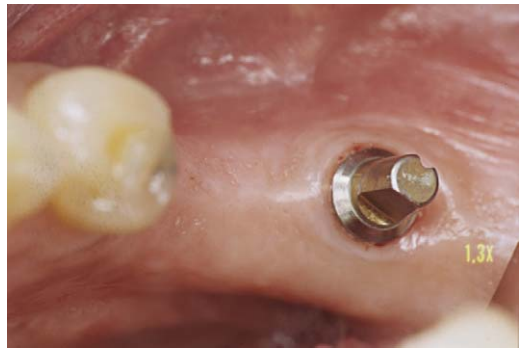

Fig. 12. Solid abutment screwed into a one-piece (non-submerged) implant.

screw. The problem of screw loosening—as seen in two-piece abutments—is eliminated or greatly reduced by this design for one-piece implants. Abutment screw fracture secondary to screw loosening and subsequent metal fatigue is also avoided as the force on the abutment is transmitted to the mating walls of the Morse taper and not the screw threads. One-piece abutments do not have a screw junction at the level of the alveolar crest and the cone arrangement of the connection helps distribute forces favorably [22].

Some implant designs also incorporate flat surfaces within the implant that facilitate exact transfer of the position of the implant to the working model (ie, an implant level impression). An array of abutment designs has been introduced to manage multiple restorative challenges. These abutment options include custom-cast and custom-milled technologies. Abutment connection via screw-retained components is also practical on one-piece implants. This approach is indicated in some circumstances. For example, when restorative space is minimal (the space available between the top of the implant and the opposing occlusion), abutment and crown retention can be accomplished without the abutment surface length necessary for cement retention. Additionally, the Morse taper type of connection stabilizes the screw joint and distributes forces away from the screw. This design element greatly minimizes problems with screw loosening.

Another circumstance that favors screw retained abutments occurs when the top of the implant is located significantly apical to the soft tissue margin. When the margin is placed apically, the removal of excess cement is quite difficult and tissue trauma may result. Residual cement is likely and will prove harmful to all tissue components in its presence. Bone loss and continued inflammation are likely. These potential problems may be avoided with cement-retained restorations which use an internal Morse taper to prevent screw loosening.

As with screwed-in abutments on one-piece implants, the microgap, which is created between the screw-retained abutment and the top of the one piece implant, is located away from the bone crest. This has the same effect of minimizing the inflammatory reaction on the bone and the adjacent soft tissue at the bone level. Screw-retained abutments allow a selection of angled abutments which can be used to overcome angulation problems following implant placement. Custom abutments and computer-designed, computer-generated abutments also are available. The final restoration can be retained by cement or screw to the screw-retained abutment.

Restoration of two-piece implants

The restoration of two-piece implant systems can be considered as part of the two-stage surgical placement. After implant healing in the bone, a full-thickness flap is typically used to gain access to the implant with a mid-crestal incision. A soft tissue healing abutment, in most cases, is placed onto the top of the implant. Sometimes, a non-crestal incision is used to remove

the planned healing incision line from directly over the implants to retain keratinized tissue and gain access to the top of the submerged implant. A further healing period is then required for the gingival tissues to mature around the now transgingival, or transmucosal, component on top of the two-piece implant.

After the healing component is placed at the time of the second-stage surgery, a radiograph may be required to ascertain whether the components involved are fully seated, particularly if the top of the implant is obscured by gingival tissue or fluids. Prosthetic components can be difficult to connect to the implant when the components join at a level that is apical to the bone crest, especially if bone has grown adjacent to or over the top of the implant during healing.

In some techniques, the second-stage placement of the healing or prosthetic component is accomplished without a flap by using a punch technique. In this technique, or at anytime an abutment must be placed or replaced, soft tissue interference with full seating can be problematic. Radiographic verification of component seating is usually necessary. Also, radiographic verification is necessary when impression components must be seated subgingivally; the mating components must be completely in contact. Similar problems are encountered when one-piece implants are placed in two-stage techniques and the top of the implant is intentionally positioned subgingivally (eg, in some esthetic situations).

Following impression procedures, the final restoration is attached to the abutment by way of screw retention or—more commonly now—cementation. Depending on the thickness of the tissue, the subgingival location of the abutment again may make complete removal of residual cement problematic. In some instances, the final restoration can be fabricated such that the restoration mates with the top of the two-piece implant located at or below the alveolar crest.

The restorative options for one-piece and two-piece implants continue to evolve. Most options for abutment design and abutment materials are available for both implant types. The two basic modes of treatment are differentiated by the connection design facilitated by one-piece implants, supra-crestal placement of the top of the implant, and the resulting location of the prosthetic connection relative to the bone. An understanding of the prosthetic components available as well as the healing characteristics of the implant system involved is essential for successful implant restorations.

Esthetic implications

The stability of the peri-implant tissues over time is critical to implant success and patient satisfaction. Biologic principles govern the relationships of these tissues and have physiologically established dimensions. Tooth esthetics is dependent on the dimensions of the teeth, and their positions and lengths relative to adjacent soft tissues and teeth. As with natural teeth,

implant restorations should ideally have non-inflamed, keratinized gingival margins coronal to the perceived level of the cemento-enamel junction. The relative positions of the height of the gingival margins are just as important on implant restorations as they are on teeth and tooth restorations. And, just as with natural teeth, inflammation can cause alterations of soft tissues and their anatomic relationships.

Fundamental to the dimensions and anatomy of the soft tissues is the position and anatomy of the underlying bone. Unfavorable alterations of bony anatomy and levels thus have esthetic consequences in the supported soft tissues. The degree and localization of inflammation, and consequent changes in crestal bone are clearly related to the presence, location, and sizes of interfaces (microgaps). The marginal bone levels around one-piece implants with a rough-smooth border have been shown to be stable at that border position [6,23,24]. The interface with one-piece implants is most often placed sufficiently above the crestal bone and apical to the gingival margin so that long-term anatomic relationships of bone and soft tissues, and thus esthetics, are preserved. Two-piece designs often result in bone loss of approximately 2 mm and soft tissue loss of 1 mm [6,25]. These dimensions and changes must therefore be taken into account in the surgical placement of esthetic implant restorations. Together the implant design, as well as the accuracy of the surgical placement, become especially important for implants and supported restorations placed in the esthetic zone.

Summary

There are two general surgical approaches for the placement and restoration of missing teeth using endosseous dental implants. One approach places the top of the implant at the alveolar crest and the mucosa is sutured over the implant, which results in a submerged surgical approach. An alternative approach places the coronal aspect of the implant coronal to the alveolar crest and the mucosa is sutured around the transmucosal aspect of the implant. This results in a non-submerged surgical approach. Different implant designs are generally used for submerged and non-submerged approaches and these designs have biological implications. When a submerged implant design is used, secondary implant components are added that extend through the mucosa to place the implant restoration. The connection of these components requires a second surgical procedure for the patient and results in interfaces in close proximity to the alveolar crest. These connections typically are flat connections maintained by screws within the secondary components. Data demonstrate that such connections become contaminated with bacteria and that the host reacts by creating an inflammatory immune reaction. This host response results in bone loss and soft tissue changes including an enlarged biologic width dimension and recession. With non-submerged implant designs, only one surgical procedure is required and no interfaces are created at the alveolar crest. Consequently, the host inflammatory immune

response is negated and the hard and soft tissue changes are minimized. The restoration of both implant designs can be achieved with either cemented restorations or restorations retained by screws. Screw-retained restorations offer the advantage of retrievability. However, in single teeth and short-span implant restorations, removal is not usually indicated. In these situations, cemented restorations offer simplicity, similarity to conventional crown and bridge techniques, and low maintenance. Regardless of implant design, surgical technique, or final restoration retention, endosseous dental implants have revolutionized restorative dentistry and made a significant impact on improved patient care.

Acknowledgment

The authors thank Madge Cluck for her excellent assistance with the preparation of this article. Thanks also go to Dolores Garza for her help with the manuscript. The following are acknowledged for their contributions to the figures: Dr. Michael Mills, Dr. Nina Broggini, and Institut Straumann AG.

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