COMPARISON OF MARGINAL BONE LOSS IN CONVENTIONAL VERSUS OSSEODENSIFICATION IMPLANT SITE PREPARATION: A RETROSPECTIVE STUDY

A Thesis

by

WADE MATTHEW KNIGHT

Submitted to the Office of Graduate and Professional Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

Chair of Committee,	Carlos Parra
Committee Members,	Pooria Fallah Abed
	Paras Patel
Head of Department,	Madhu Nair

May 2021

Major Subject: Oral Biology

ABSTRACT

Introduction: Marginal bone loss is a multifactorial problem affecting the health and longevity of dental implants placed throughout the world. A novel drilling system has been developed that allows for the densification of bone around dental implants through a non-subtractive drilling protocol which allows for higher initial insertion torques.

Materials and Methods: Patients with implants that were placed with either conventional or osseodensification drilling protocols that had been restored for at least 1 year were radiographically and clinically re-evaluated. Radiographic marginal bone levels were measured on the mesial and distal of implants and compared from the time of restoration to ≥12 months post-loading. Pocket probing depth (PD), presence/absence of plaque, presence/absence of bleeding on probing (BoP), and keratinized mucosa (KM) were recorded.

Results: Twenty-two patients with 39 implants were evaluated. The groups were not significantly different in the amount of marginal bone loss present at baseline ($p > 0.05$). The conventional drilling group had a mean marginal bone loss of 0.17 mm ± 0.65 between when the implant was restored and the follow-up appointment, while the osseodensification group had a mean marginal bone loss of 0.096 mm ± 0.66 during the same period. The difference between the groups was not significantly different ($p > 0.05$). The clinical parameters assessed were not significantly different between the groups.

Conclusion: Osseodensification drilling protocols do not lead to an increase in marginal bone loss around dental implants that have been in function for at least 1-year compared to conventional drilling protocols.

ACKNOWLEDGEMENTS

I would like to thank my committee members, Drs. Parra, Fallah, and Patel for their guidance and dedication in seeing this project through. I would like to give a special thanks to some of the members of the Department of Periodontics for assisting me in seeing the research participants, Vanessa, Christina, Angela, Vea, and Karen.

Finally, I would like to thank my wife, Rose Knight, for her love and support during this adventure.

CONTRIBUTORS AND FUNDING SOURCES

Contributors

This work was supported by a thesis committee consisting of Dr. Parra and Dr. Fallah of the Department of Periodontics and Dr. Patel of the Department of Diagnostic Sciences. All work for this thesis was completed by the student independently.

Funding Sources

There are no outside funding contributions to acknowledge related to the research and compilation of this document.

NOMENCLATURE

MBL	Marginal bone loss
GI	Gingival index
PI	Plaque index
PICF	Peri-implant crevicular fluid
PCR	Polymerase chain reaction
BoP	Bleeding on probing
IL	Interleukin
TNF-α	Tumor necrosis factor-alpha
mm	Millimeters
Ncm	Newton centimeters
TAMUCOD	Texas A&M University College of Dentistry
HIPAA	Health Insurance Portability and Accountability Act
KMW	Keratinized mucosa width
PD	Probing depth
SD	Standard deviation
SLA	Sandblasted, large grit, acid etched
CBCT	Cone beam computer tomography

TABLE OF CONTENTS

LIST OF TABLES

1. INTRODUCTION AND LITERATURE REVIEW

Background and Significance

Dental implants are used extensively throughout modern dentistry for the replacement of missing teeth since they were first introduced to the United States. According to Elani et al. (2018), the prevalence of dental implants to replace missing teeth is dramatically increasing and is predicted to reach 23% by 2026.[1] During this time, dental implants have shown high long-term success and survival rates. Survival rates range in the low to mid 90% for many studies.[2-4] Survival rates only give a quantitative evaluation of dental implants, either present or absent, and some argue that survival is an inadequate term because implants can be kept in the mouth even when they have obviously failed.[5] A more stringent method for evaluating dental implants is the term success. Success has been defined by several authors and allows for a qualitative evaluation of dental implants. Albrektsson et al. (1986) defined success as the absence of mobility, peri-implant radiolucency, persistent pain, infection, neuropathy, paresthesia and less than 0.2 mm of vertical bone loss per year after the first year of loading.[6] The criteria for success has been updated several times and is commonly defined as described by Misch et al. (2008) to include the absence of mobility, pain or tenderness on function, exudate history and less than 2.0 mm of vertical bone loss.[5]

The failure of dental implants can further be broken down into early and late failure. Early failure is defined as a failure of the dental implant before the prosthetic restoration has been placed. According to a systematic review by Berglundh et al. 2002), early implant failure ranged

from 0.76 - 7.5%.[7] Late implant failure, also known as failure after loading, ranged from 2.06 – 11.29%.[7]

In order for dental implants to have long-term success, they first need to osseointegrate. Osseointegration occurs when the dental implant and surrounding bone form an intimate union. There are many definitions for osseointegration.[8-10] Zarb (1991) defined osseointegration as "a process whereby a clinically asymptomatic rigid fixation of alloplastic materials is achieved and maintained in bone during functional loading."[10] Primary stability occurs immediately after a dental implant is placed and is crucial in achieving osseointegration. According to Meredith (1998), primary stability is a mechanical phenomenon related to the local bone quality, quantity, implant type and placement method.[11] Primary stability is vital in preventing micromovement during implant healing and is the highest immediately after implant insertion.[12] An increase in stability, after an initial decrease following implant insertion, is noted as remodeling and regeneration of the bone occurs along the implant-bone interface.[11] This increase in stability is referred to as secondary stability.

Bone quality and quantity are essential for initial implant stability and ultimately for long term success.[11] Bone quantity can be easier to determine with modern technology such as cone beam computed tomography, but bone quality can be more difficult to determine. Lekholm and Zarb (1985) classified bone into 4 groups based on the relationship of cortical to trabecular bone. When the bone was thick and cortical with minimal to no trabecular bone, this was labeled type I bone; thick cortical with minimal trabecular bone was labeled type II bone; bone with a thin cortical layer and dense trabecular bone was labeled type III bone; and bone with a thin cortical layer with low density trabecular bone was labeled type IV bone.[13] This classification has limited use for the clinician because it is difficult to accurately determine the bone quality

presurgically. Another classification system was developed by Misch (1990) that classified bone density according to clinical hardness determined while drilling for implant placement. Tactile feel of D1 bone was described as feeling like drilling into oak or maple wood; D2 as drilling into white pine or spruce; D3 as drilling into compressed balsa wood; and D4 as drilling into compressed Styrofoam.[14] While this classification scheme is geared toward the clinician placing dental implants, it still be difficult to determine the bone quality. Trisi and Rao (1999) conducted a study that compared the surgeon's clinical assessment of bone quality as described by Misch (1990)[14] to the histologic structure of the bone qualified by histomorphometric analysis. The study found that clinicians were able to distinguish D1 and D4 bone ($p < 0.05$), but could not significantly assess D2 or D3 bone ($p > 0.05$).[15] Studies have shown that bone density is directly related to bone strength with regards to microfracture.[16, 17] Misch et al. (1999), using the bone quality classification developed by Misch (1990),[14] showed that a tenfold difference in bone strength may exist between D1 and D4; D2 bone had a 47% to 68% greater compression strength than D3 bone.[18] As previously described by Trisi and Rao (1999),[15] clinicians are able to adequately distinguish between hard bone (D1) and soft bone (D4), but they are not able to adequately distinguish between intermediate bone (D2 and D3), which has vastly different mechanical properties.

Studies have looked at the intraoral location of the different bone densities.[15, 19-24] The findings of several groups have been summarized by Resnik and Misch (2021). D1 bone is not seen in the maxilla and rarely seen in the mandible; D2 bone is the most common bone density seen in the mandible with D2 seen in the anterior mandible two-thirds of the time and half of the time in the posterior mandible. D2 bone is seen in the maxilla in one-fourth of patients and is more likely in the anterior and premolar region; D3 bone is present in the maxilla more than half

of the time. 75% of the time the edentulous anterior maxilla has D3 bone. D3 bone is common in the posterior maxilla and mandible; D4 bone is found in the posterior maxilla in 40% of patients and it is seen in the anterior maxilla 10% of the time. D4 bone is not common in the mandible.[25]

Esposito et al. (1998) conducted a systematic review exploring biological factors in implant failure.[19, 26] This group found an implant failure rate 3x higher in the maxilla than the mandible with both early and late failures. Bone quality was one of the main reasons cited for the late failures.

As previously discussed, initial implant stability is imperative for osseointegration. The stability achieved at implant insertion is largely dependent on the bone quality and quantity at the site.[24, 27, 28] Several techniques have been developed to preserve bone and increase insertion torque, such as the osteotome approach or the undersized preparation of the osteotomy.[29-31] A novel technique, using proprietary burs has recently come onto the market. The drilling protocol and system, Densah®, were designed by the Versah Corportation, Jackson, MI. The burs are designed for non-subtractive osteotomy preparation, osseous densification, by compacting bone into the walls of the osteotomy.[32] The drilling system can operate in the standard subtractive method by controlling the direction of rotation for the system, counter-clockwise for osseous densification and clockwise for standard osteotomy preparation. The method of bone compaction is "through the application of controlled deformation due to rolling and sliding contact along the inner surface of the osteotomy with the rotating lands of the densifying bur. The bone deformation occurs through viscoelastic and plastic mechanisms when the load is controlled beneath the ultimate strength of bone."[32] The authors state the system can be used

safely because the drills provide a haptic feedback to the surgeon that allows for the control of forces applied to the bone based on its density.[32]

The osseodensification drilling protocol has been demonstrated in several studies to increase the insertion torque of dental implants. Huwais and Meyer (2017) compared the insertion and removal torques of dental implants using conventional drills, extraction drilling using the osseous densification system (Densah®, Versah), and the osseous densification system by placing dental implants in porcine tibia. Progressive enlargement of the osteotomy was carried out using 2.2, 3.2, 4.2, and 5.2 mm twist drills in the conventional group and with 2.8, 3.8, 4.8, and 5.8 mm Densah burs in the osseous densification group. After the third diameter step of the osteotomy was completed, a 4.1- mm diameter implant was inserted and removed. The drilling sequences were then completed and a 6.0- mm diameter implant was inserted, and biomechanical stability measurements were completed. Results showed that the maximum osseous densification insertion torque for the 4.1 mm implant was 49 $\pm$ 24 Ncm and 108 $\pm$ 56 Ncm for the 6 mm implant which were approximately double the other two methods; the removal torques were 31 $\pm$ 17 Ncm and 85 $\pm$ 49 Ncm for the 4.1- and 6.0-mm implants, respectively. The removal torque for the osseous densification was more than double the other two drilling methods.[32]

Wound healing following osteotomy preparation using osseous densification drilling protocols was explored by Witek et al. (2019). The authors compared the healing of osteotomies in the hips of sheep using conventional twist drills and protocols, osseous densification clockwise and counterclockwise. They report, qualitatively, that the histomorphometric analysis of the 3 treatment modalities all showed bone remodeling and growth with no signs of impaired healing. The presence of bone chips (autograft) was evident in the osseous densification

counterclockwise group and was not seen in the other groups. This serves to show, histomorphometrically, that bone, which is removed during conventional drilling, is packed into the walls of the osteotomy during osseodensification drilling.[33]

Survival rates of dental implants placed with this system seem to be at least as good as other drilling systems. Huwais et al. (2018) performed a multi-center retrospective study looking at survival rates of dental implants placed with simultaneous transcrestal sinus augmentation using the Densah® system. The cumulative 5 year survival rate was 97%.[34]

Excessive insertion torque has led some authors to describe a phenomenon known as "compression necrosis." This term has been defined as "compression of bone beyond its physiologic tolerance may result in ischemia with subsequent necrosis or sequestrum formation, which could ultimately lead to implant failure."[35] Compression necrosis is thought only to occur in cortical bone if it does truly exist.[35, 36] Several studies report no adverse effects on peri-implant bone with high insertion torques. Khayat et al. (2013) performed a study comparing two groups of patients based on the initial insertion torque. The control group consisted of 6 patients and 9 implants with maximal insertion torques of 30-50 Ncm and the test group consisted of 32 patients and 42 implants with maximal initial insertion torques greater than 70 Ncm. The average initial insertion torque of the control group was 37.1 Ncm and 110.6 Ncm for the test group with a maximum insertion torque of 176 Ncm. The mean marginal bone loss for the control group was 1.03 mm at loading, and 1.09 mm at 1-year. The results for the test group were 0.72 mm at loading and 1.24 mm at 1-year. The results were not statistically significantly different between groups.[37]

Risk Factors for Implant Disease

As previously discussed, vertical bone loss, also known as marginal bone loss, is a key descriptor with regards to the success of a dental implant. MBL around a dental implant can threaten its longevity and is a multifactorial process that have been explained with differing reasons, such as peri-implantitis, excessive occlusal forces, iatrogenic dentistry, and restorative related issues to name a few.[38]

Dental implants are susceptible to marginal bone loss caused by chronic inflammatory processes much like the natural dentition. Peri-Implantitis has been defined as "a pathological condition occurring in tissues around dental implants, characterized by inflammation in peri-implant mucosa and progressive loss of supporting bone."[39] Peri-implantitis is preceded by peri-implant mucositis, which has been defined as a reversible inflammatory lesion of the mucosa surrounding a dental implant without the loss of supporting bone,[40-42] much like gingivitis precedes periodontitis in the natural dentition.

Since peri-implantitis and periodontitis are preceded by soft tissue inflammation without the loss of supporting hard tissues, does the soft tissue inflammation occur at the same rate and to the same degree? Salvi et al. (2012) performed an investigation to compare the rates of formation of peri-implant mucositis and gingivitis in the mandible following cessation of oral hygiene practices for 21 days followed by 21 days of optimal oral hygiene to see how quickly the lesions resolved. The subjects in the study received a prophylaxis procedure followed by a period of 3-4 weeks where the subjects were asked to perform optimal home care. At the end of this period, subject's home care was evaluated to ensure full-mouth plaque scores and full-mouth bleeding scores were 15% or less. Subjects were then instructed to withhold oral hygiene practices on

mandibular teeth and implants for 21 days. Subjects then reinstituted optimal oral hygiene practices. Clinical parameters for the teeth and implants were measured at 6 locations per site at several time points during both phases to include gingival index (GI) and plaque index (PI).[43-45] The median PI at baseline for teeth was 0 and 0.17 for implants, which was a statistically significant difference ($p < 0.05$). PI increased each of the 3 weeks and by week 3 the median PI was 1.67 for teeth and 1.33 for implants. The difference between the median PI at tooth and implant sites at the 3 observation periods were statistically significant ($p < 0.02$). At baseline, the median GI was 0 for teeth and 0.17 for implants ($p = 0.12$). The median GI increased significantly during the 3 measurement periods: 0.83, 1, 1 and 1.33, 1.33, 1.5 for teeth and implants, respectively. After reinstitution of oral hygiene practices, the median GI decreased over the next 3 weeks; 0.83, 0.33, 0.33 and 0.75, 0.5, 0.5 for teeth and implants, respectively. At the end of the 3 weeks of reinstituted oral hygiene, the median GI was significantly different between implants and teeth ($p < 0.04$).[46] This study showed a stronger inflammatory response in the soft tissue around dental implants as compared to the natural dentition. This agreed with other studies with regards to the increase in gingival inflammation following cessation of oral hygiene practices.[47, 48] Salvi et al. (2012) differed from these studies in that they found no difference between the inflammatory response of the natural tooth and dental implant.[46]

Histologically, the lesions of peri-implantitis and periodontitis are similar, but important differences are apparent. Burglundh et al. (2011) conducted a systematic review and found that plasma cells and lymphocytes make-up the largest portions of inflammatory cells in both groups. In the peri-implantitis lesion, a larger portion of neutrophils and macrophages were evident as compared to the periodontitis lesion.[49]

The microbiome of the peri-implant tissues has been analyzed by different groups.[50, 51] Casado et al. (2011) conducted a study in which 30 patients were divided evenly into 3 groups based on clinical and radiographic analysis: peri-implant health, peri-implant mucositis, and peri-implantitis. The peri-implant crevicular fluid was collected around each implant and analyzed by polymerase chain reaction. The results showed that the periodontal pathogens *Actinobacillus actinomycetemcomitans* (Aa), *Porphyromonas gigivalis* (Pg), *Prevotella intermdia* (Pi), *Tannerella forythensis* (Tf), and *Treponema denticola* (Td) were present at all sites: peri-implant health, peri-implant mucositis, and peri-implantitis.[50] A study by Renvert et al. (2007) found similar results. This group looked at the microbiota around healthy implants, and those with peri-implant mucositis and peri-implantitis using DNA-DNA hybridization. This group defined peri-implant mucositis as a probing depth of 4 mm or greater and the presence of bleeding on probing and with less than 3 threads of bone loss; they defined peri-implantitis as 3 or more threads of bone loss between the 1-year and the final radiographic examination at 5-years with BoP. The only difference this group found was an elevated level of *E. corrodens* ($p < 0.05$) in the peri-implant mucositis group as compared to the healthy group. No difference in microbiota was found between peri-implant mucositis or peri-implantitis, or between healthy implants or peri-implantitis. This group also looked for differences in the microbiota of dental implants of the three groups, health, peri-implant mucositis, peri-implantitis compared to the natural dentition. They found "no differences in the microbiota between implant and tooth samples…" They did find a difference in dentate individuals with and without a history of periodontitis. The history of periodontitis group had a significant difference in *F. nucleatum sp. Vincentii* ($p < 0.02$) and *N.* mucosa ($p < 0.05$) compared to individuals without a history of periodontitis. The authors concluded, "The prevalence of *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema*

denticola, and *A. actinomycetemcomitans* considered as key pathogens in periodontitis are low and do not seem to differ by implant status."[51]

The expression of proinflammatory mediators is an important consideration when evaluating marginal bone loss around dental implants. Faot et al. (2015) conducted a systematic review with the goal of comparing the inflammatory profile of peri-implant crevicular fluid of healthy dental implants to those with peri-implant disease. This systematic review included studies conducted between 1996 and 2013. The authors ended with 19 studies in the systematic review and 7 studies in the meta-analysis. The meta-analysis revealed a statistically higher level of IL-1β in peri-implantitis ($p = 0.001$) and peri-implant mucositis ($p = 0.002$) as compared to healthy controls. There was no significant difference in the expression of IL-1β between peri-implantitis and peri-implant mucositis ($p = 0.80$). A significant difference was found between the amount of TNF-α in peri-implantitis sites compared to healthy implants ($p = 0.02$).[52] Increased levels of IL-1β has been associated with deeper probing depths ($p < 0.0001$), higher plaque index ($p < 0.0001$), higher gingival index ($p < 0.0001$), and more bone loss ($p < 0.0001$) around dental implants.[53] IL-1β and TNF-α have been shown to stimulate bone resorption and inhibit formation of bone.[54, 55] The systematic review by Faot et al. (2015) also showed increases in the proinflammatory cytokines IL-4, IL-6, IL-8, IL-12, IL-17 in sites with peri-implant disease as compared to healthy implants.[52] Other studies have shown a reduction in the levels of the anti-inflammatory cytokine, IL-10, as implants progress from health to peri-implantitis.[56, 57] Summarily, pro-inflammatory cytokines can be used to diagnose peri-implant disease, but levels of these markers are not able to discern between peri-implant mucositis and the conversion to peri-implantitis.[52]

While the terms ‘peri-implantitis’ and ‘periodontitis’ conveys the similarities of the disease processes around the dental implant and natural tooth, the processes do vary in their course due to the physiologic relationship of the dental implant and the natural tooth to their individual environments in health. The dental implant lacks cementum, a periodontal ligament, bundle bone, as well as the dento-alveolar and dento-gingival fibers, and in health, the dental implant is ankylosed to the host bone, whereas the natural tooth is mobile in the socket.[58] Studies have shown that peri-implantitis may begin sooner and progress at a faster rate than periodontitis.[59-61] Derks et al. (2016) conducted a retrospective study of a cohort of Swedish patients, n=53 with 105 implants. This cohort had moderate/severe peri-implantitis, which was defined as BoP and/or suppuration and greater than 2 mm of peri-implant bone loss as identified by radiographic examination at their 9-year implant follow-up. The group found that 51% of implants had greater than 3 mm of bone loss and 29% had greater than 4 mm. It was estimated that 52% and 66% of implants had > 0.5 mm of marginal bone loss at years 2 and 3, respectively. This group found a non-linear progression of bone loss around the implants.[62] The non-linear progression of peri-implant bone loss was also found by Fransson et al. (2010).[63] This group showed that the rate of peri-implant bone loss increased over time.

The space between the implant and abutment connection is termed the microgap. When this area is exposed to the oral environment, marginal bone loss usually results. Barboza et al. (2002) performed a split-mouth human study in which subjects received implants on both sides of the mandible. One-half of the implants were submerged, and the other half were left exposed to the oral cavity to heal. After 4 months of healing, the non-submerged implants had more bone loss than the submerged implants. This group hypothesized that the crevice (microgap) serves as a nidus for dental plaque resulting in marginal bone loss.[64] When the microgap is placed at the

bone crest, at least 0.5 mm of marginal bone loss is expected.[64, 65] Hermann et al. (2001) showed that movement at the microgap between implant components, implant body and abutment, cause crestal bone loss even when the microgap is placed coronal to the bone crest. In this study, the researchers placed implants with the microgap 1 mm coronal to the bone crest. They found significantly less bone loss ($p < 0.02$) occurred when the abutment was welded to the implant, preventing movement between the components.[66]

The microgap can be moved horizontally away from the bone crest when the abutment has a smaller diameter than the implant at the connection. This is termed platform switching. Platform switching has been reported to preserve marginal bone when compared to platform matched implants.[67, 68] Atieh et al. (2010) conducted a systematic review and meta-analysis and found that platform switched implants had significantly less marginal bone loss compared to platform matched implants. The advantages of platform switching on bone levels is most favorable when the implant – abutment difference is 0.4 mm or greater.[69]

A 'biologic width' of tissue thickness is important in maintaining marginal bone levels around implants. Tomasi et al. (2014) conducted a human study that examined the morphogenesis of the mucosal attachment to dental implants. This group found that at 8 weeks the soft tissue had an average thickness of 3.6 mm, which was 1.9 mm of barrier epithelium and 1.7 mm of connective tissue.[70] Linkevicius et al. (2015) conducted a study in which the soft-tissue thickness was measured at the time of implant placement. The groups were stratified based on tissue thickness of 2 mm or less (n=40) and greater than 2 mm (n=40). The implants were placed in a single stage and platform switching was performed for all implants. The results at 1-year of function showed that the thin tissue group had 1.17 mm of crestal bone loss and the thicker tissue group had 0.21 mm of crestal bone loss. The difference between groups was

significant ($p < 0.001$). This study showed that a minimal amount of tissue is needed above the shoulder of the implant to reduce marginal bone loss and the effects of platform switching are not appreciated when the soft tissue thickness is 2 mm or less.[71] This result agreed with the work of Vandeweghe and De Bruyn (2012), which found a protective effect of platform switching, about 30% less marginal bone loss, only when the mucosal thickness allowed the establishment of a biologic width.[72]

Surgical factors at the time of implant placement have been shown to lead to marginal bone loss. Spray et al. (2000) showed that significantly less bone loss occurred when 1.8 mm or more of buccal bone was present after the osteotomy was completed compared to sites with less buccal bone at uncovery.[73] Thermal damage can occur during implant osteotomy preparation, which can lead to necrosis of the hard.[74] Bone temperature increases with the number of times a drill has been used and in guided implant placement, possibly due to the guide blocking proper irrigation.[75]

Presently, literature supports the survival of implants placed in humans and supports increased insertion torque in animal models with osseosdensification drilling protocols. A gap in the literature exists comparing osseosdensification drilling protocols to conventional drilling protocols with regards to marginal bone loss in a sample of human participants.

This retrospective study was undertaken with the aims to evaluate if implants placed using an osseodensification implant site preparation have more marginal bone loss compared to implants placed with conventional site preparation with at least 1-year of loading and secondary aims of comparing clinical measurements between the two different protocols.

2. MATERIALS AND METHODS

Patient Enrollment

The Institutional Review Board of Texas A&M University College of Dentistry reviewed and approved the protocol for this retrospective study (IRB2019-1494D). A records search was completed at TAMUCOD for dental implants that were placed in the Department of Periodontics using conventional and osseodensification drilling protocols. The records search encompassed a period between February 2017 and May 2019. Individual records were then screened to verify that inclusion and exclusion criteria were met in patients that had dental implants placed in the Department of Periodontics.

Inclusion criteria consisted of single-unit dental implants that had been loaded for at least 12 months and that had radiographs at the time of implant loading. Participants were excluded if they had uncontrolled systemic conditions, such as diabetes mellitus, pregnant women, and smokers. Age, gender, implant size, brand, and location were recorded for each patient.

Potential participants were then contacted by phone to see if they were interested in participating in this research. A voicemail was left if the call was not answered. The potential participants were called a week later and another voicemail was left if the call went unanswered. Individuals that agreed to participate in the research were scheduled.

Clinical Protocol

Informed consent and HIPAA authorization were acquired from each participant. Clinical and radiographic examinations were conducted for each implant. The radiographic examination consisted of bitewing and peri-apical radiographs of the dental implants. Periapical radiographs were obtained using a parallel long-cone technique.[76] The clinical examination included the implant of interest and the natural teeth on either side of the implant. Plaque index which consisted of a yes/no questionnaire if plaque was detected by running a UNC-15 periodontal probe through the gingival sulcus at 4 surfaces per tooth. Keratinized mucosa width (KMW) values were recorded to the nearest millimeter on buccal of maxillary teeth/implants and on the buccal and lingual of mandibular teeth/implants. Tissue thickness was defined as thick if the periodontal probe was inserted into the sulcus and could not be seen and thin if the probe could be visualized through the sulcus.[77] This was measured on the buccal and lingual/palatal. Probing depth and bleeding on probing were recorded. The implants and adjacent teeth were photographed on the buccal and lingual/palatal. Participants were notified if their implants were diseased as indicated by BoP, deeper PDs, and signs of extensive marginal bone loss and were referred for treatment as necessary.

Radiographic Examination

The examiners underwent a calibration exercise to measure inter- and intra-rater reliability prior to measuring marginal bone loss on the study participants. The mesial and distal marginal

bone loss of 15 implants, not included in this study, were measured independently by both examiners at two separate time points. Inter- and intra-rater reliability were both above 80%.

The digital radiographs collected at time of restoration and follow-up were calibrated and evaluated using the software ImageJ (National Institutes of Health, Bethesda, MD, USA). Images were calibrated by setting the known width of the dental implant as the scale. Marginal bone loss measurements were performed by two separate examiners working independently. The measurements from each clinician were averaged for each surface of each implant. If any measurement varied by more than 1 mm, a third calibrated examiner was available to take measurements. In this case, all three measurements would be averaged.

Power Analysis

A power analysis was performed with an effects size of 0.75, a power of 80% and a significance level at 5%. A sample size of twenty-nine per group was determined to be adequate.Statistical Analysis

Marginal bone loss measurements were calculated as a mean on the mesial and distal of each implant and expressed in mm $\pm$ standard deviation (SD). Inter- and intra-rater reliability tests was performed using Cronbach's Alpha test. Normality of the distribution of the data was analyzed by measuring skewness, kurtosis, and performing a Kolmogorov-Smirnov test. Differences between groups were compared using an Independent T test. Probing depths (PD) and keratinized mucosa width were expressed in mm $\pm$ standard deviation per implant surface, while bleeding on probing (BoP) and plaque scores were expressed as a mean percentage (%) of

all sites. All data was entered in statistical software for analysis (SPSS software, IBM, Chicago, IL, USA)

3. RESULTS

Patient Enrollment

A total of 94 implants in 58 patients were identified that underwent implant placement using osseodensification implant drilling protocols. 12 patients with 19 implants agreed to participate in the study and underwent the clinical and radiographic examination. 46 patients with 75 implants did not participate in the study (Table 1).

Table 1 Reasons for Not Participating in the Study (Test Group)

Implant not restored with 1-year of loading	18 patients (30 implants)
Voicemails not returned	18 patients (21 implants)
Not a single-unit implant supported prosthesis	5 patients (17 implants)
Patient not interested in participating	3 patients (3 implants)
Missing radiographs at loading	1 patient (3 implants)
Implant failed	1 patient (1 implant)

The test group was matched to a control group that consisted of 10 patients with 20 implants that were placed with conventional drilling protocols in the same time frame as the test group.

The mean age of the test group was 60. 9 $\pm$ 9.9 years (mean $\pm$ SD) and ranged between 47-76 at the time of surgery. This group was 75% female and 25% male. The mean age of the

control group was 58.1 ± 9.5 years (mean ± SD) and ranged between 46-70 at the time of surgery. This group was 60% female and 40% male (Table 2).

Table 2 Demographics of Participants

	Control group	Test group
Age in years (mean ± SD)	58.1 ± 9.5	60.9 ± 9.9
Gender – n(%)		
Male	4(40)	3(25)
Female	6(60)	9(75)

Characteristics of Implants

All 39 implants evaluated in this study were bone-level Straumann SLA implants placed in posterior sites. 89.5% of implants in the test group were placed in the maxilla compared to 40% of the control group implants (Table 3).

Table 3 Characteristics of Implants

	Control group n(%)	Test group n(%)
Location of implant		
Maxilla	8(40)	17(89.5)
Mandible	12(60)	2(10.5)

Table 3 Continued

Molar	12(60)	9(47.3)
Premolar	8(40)	10(52.6)

Clinical and Radiographic Measurements

Clinical measurements were not significantly different between the groups ($p > 0.05$) (Table 4). The groups did not significantly differ in the amount of marginal bone loss at the time of loading ($p > 0.05$), 0.039 mm $\pm$ 0.650 and 0.234 mm $\pm$ 0.706 for control and test groups, respectively. There was no significant difference in marginal bone loss between loading and follow-up between the groups, ($p > 0.05$). The control group had a mean marginal bone loss of 0.17 mm $\pm$ 0.65 between loading at follow-up, while the test group had a mean marginal bone loss of 0.096 mm $\pm$ 0.66 between loading and follow-up. Table 5 presents a breakdown of radiographic measurements based on the mesial and distal of each implant. Furthermore, there was no significant difference between measurements when breaking the results down into the mesial and distal for PD or MBL and the buccal and lingual for KMW.

Table 4 Clinical Measurements

	Control group	Test group
Mesial probing depth (mm) $\pm$ SD	2.75 $\pm$ 0.81	3.13 $\pm$ 0.77
Distal probing depth (mm) $\pm$ SD	2.75 $\pm$ 0.81	2.92 $\pm$ 0.71

Table 4 Continued

Buccal keratinized mucosa width (mm) ± SD	2.90 ± 1.33	2.76 ± 0.97
Lingual keratinized mucosa width (mm) ± SD	4.27 ± 0.90	3.50 ± 0.71
Plaque score (% of sites)	23.75	27.63
Bleeding on Probing (% of sites)	11.67	14.91

Table 5 Radiographic Measurements

	Control group	Test group
Mesial bone loss – Restoration (mm) ± SD	-0.13 ± 0.58	0.27 ± 0.71
Distal bone loss – Restoration (mm) ± SD	0.21 ± 0.68	0.20 ± 0.59
Mesial bone loss – Follow up (mm) ± SD	0.15 ± 0.66	0.25 ± 0.59
Distal bone loss – Follow up (mm) ± SD	0.27 ± 0.66	0.41 ± 0.54

4. DISCUSSION AND CONCLUSIONS

Bone quality is the lowest in the posterior maxilla, and this can lead to significantly increased implant failure rates.[19, 26] Osseodensisfication drilling protocols have been shown to increase initial insertion torque[32] by compacting autogenous bone, that is typically removed during conventional drilling protocols, into the walls of the osteotomy.[33] This local improvement in bone quality can help to explain the distribution of test patients having implants placed in the maxilla. 89.5% of test group implants were placed in the maxilla compared to 40% for the control group. Aside from the ability to increase insertion torque, osseodensification drilling protocols can be used to perform indirect sinus lifts, which can also help to explain the skewed rate of use in the posterior maxilla.

The goal of this research project was to see if a measurable difference existed between the control and test protocols with regards to marginal bone loss. No difference could be found in terms of marginal bone loss between loading and follow-up. Also, there was no difference in terms of preloading marginal bone loss between the groups. This is important because bone loss following osseodensification drilling protocols could present prior to loading. Due to the retrospective nature of our study, standardization of implant placement could not be accounted for.

A low early implant failure rate was observed for the test group. 95 implants were identified that underwent the test procedure, and of these, only one implant failed for an early survival rate of 98.4%. The implant was found to be mobile at the 2nd stage surgery and was removed. The survival rate was 100% for the 19 implants in the test group that were evaluated. None of the

implants, control or test, required surgical therapy for any reason. Four implants, 2 test and 2 control, were referred for non-surgical therapy due to the amount of BoP and erythema of the peri-implant soft tissue. These implants were diagnosed with peri-implant mucositis.

Probing depth measurements in both groups were relatively shallow. A probing depth of 5 mm or less is generally considered healthy.[58] Only one surface of the 39 implants had a probing depth greater than 5 mm.

The level of BoP in both groups may be considered high, ranging between 11.67 – 14.91%. This may be explained by the fact that dental implants are surrounded by a soft tissue cuff that offers less resistance to dental probe penetration than the gingiva of the natural dentition.[78, 79] Localized "dot bleeding" can be a sign of trauma from probing rather than bleeding that results from plaque induced inflammation.

An adequate band of keratinized mucosa was seen around the majority of the implants in this study. Generally, the natural tooth compared to the contralateral implant will have more keratinized mucosa.[80-82] We did make a comparison between implants and contralateral teeth, but we were able to compare implants to the adjacent natural dentition and there was no significant different in the amount of keratinized mucosa between the adjacent natural dentition compared to either group of the study.

This study demonstrates comparable radiographic and clinical outcomes between conventional and osseodensification drilling protocols, which serves to lend credibility to the efficacy of these drilling procedures. Future studies are needed to evaluate long-term success in a sufficiently large group. A randomized control trial with standardized implant placement would serve to further elucidate the efficacy of this novel technique.

Conclusion

Osseodensification drilling protocols do not adversely affect clinical or radiographic parameters 1-year post-loading. Early survival rates above 98% are favorable. This procedure may serve to locally improve bone quality characteristics in unfavorable areas of the mouth and should be considered when questions about bone quality exists.

REFERENCES

1. Elani HW, Starr JR, Da Silva JD, Gallucci GO. Trends in Dental Implant Use in the U.S., 1999-2016, and Projections to 2026. J Dent Res 2018;97(13):1424-30.
2. Derks J, Hakansson J, Wennstrom JL, et al. Effectiveness of implant therapy analyzed in a Swedish population: early and late implant loss. J Dent Res 2015;94(3 Suppl):44S-51S.
3. Roos J, Sennerby L, Lekholm U, et al. A qualitative and quantitative method for evaluating implant success: a 5-year retrospective analysis of the Branemark implant. Int J Oral Maxillofac Implants 1997;12(4):504-14.
4. Gjelvold B, Kisch J, Chrcanovic BR, Albrektsson T, Wennerberg A. Clinical and radiographic outcome following immediate loading and delayed loading of single-tooth implants: Randomized clinical trial. Clin Implant Dent Relat Res 2017;19(3):549-58.
5. Misch CE, Perel ML, Wang HL, et al. Implant success, survival, and failure: the International Congress of Oral Implantologists (ICOI) Pisa Consensus Conference. Implant Dent 2008;17(1):5-15.
6. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. The International journal of oral & maxillofacial implants 1986;1(1):11.
7. Berglundh T, Persson L, Klinge B. A systematic review of the incidence of biological and technical complications in implant dentistry reported in prospective longitudinal studies of at least 5 years. J Clin Periodontol 2002;29 Suppl 3:197-212; discussion 32-3.
8. Branemark PI, Adell R, Breine U, et al. Intra-osseous anchorage of dental prostheses. I. Experimental studies. Scand J Plast Reconstr Surg 1969;3(2):81-100.

9. Albrektsson T, Sennerby L. State of the art in oral implants. J Clin Periodontol 1991;18(6):474-81.

10. Zarb G AT. Osseointegration—A requiem for the periondontal ligament?—An editorial. Int J Periodontics Restorative Dent 1991;11:88-91.

11. Meredith N. Assessment of implant stability as a prognostic determinant. Int J Prosthodont 1998;11(5):491-501.

12. Gomes JB, Campos FE, Marin C, et al. Implant biomechanical stability variation at early implantation times in vivo: an experimental study in dogs. Int J Oral Maxillofac Implants 2013;28(3):e128-34.

13. Lekholm U, Zarb, GA. Patient selection and preparation. In: Branemark PI, Zarb, GA, Albrektsson, T., editor. Tissue-Integrated Prostheses: Osseointegration in Clinical Dentistry. Chicago: Quintessence; 1985. p. 199-209.

14. Misch CE. Density of bone: effect on treatment plans, surgical approach, healing, and progressive loading. Int J Oral Implantol 1990;6(2):23-31.

15. Trisi P, Rao W. Bone classification: clinical-histomorphometric comparison. Clin Oral Implants Res 1999;10(1):1-7.

16. Carter DR, Hayes WC. Bone compressive strength: the influence of density and strain rate. Science 1976;194(4270):1174-6.

17. Rice JC, Cowin SC, Bowman JA. On the dependence of the elasticity and strength of cancellous bone on apparent density. J Biomech 1988;21(2):155-68.

18. Misch CE, Qu Z, Bidez MW. Mechanical properties of trabecular bone in the human mandible: implications for dental implant treatment planning and surgical placement. J Oral Maxillofac Surg 1999;57(6):700-6; discussion 06-8.

19. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (II). Etiopathogenesis. Eur J Oral Sci 1998;106(3):721-64.

20. Friberg B, Jemt T, Lekholm U. Early failures in 4,641 consecutively placed Branemark dental implants: a study from stage 1 surgery to the connection of completed prostheses. Int J Oral Maxillofac Implants 1991;6(2):142-6.

21. van Steenberghe D, Lekholm U, Bolender C, et al. Applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures. Int J Oral Maxillofac Implants 1990;5(3):272-81.

22. Fugazzotto PA, Wheeler SL, Lindsay JA. Success and failure rates of cylinder implants in type IV bone. J Periodontol 1993;64(11):1085-7.

23. Herrmann I, Lekholm U, Holm S, Kultje C. Evaluation of patient and implant characteristics as potential prognostic factors for oral implant failures. Int J Oral Maxillofac Implants 2005;20(2):220-30.

24. Jaffin RA, Berman CL. The excessive loss of Branemark fixtures in type IV bone: a 5-year analysis. J Periodontol 1991;62(1):2-4.

25. Resnik R, Misch. C. E. Bone Density: A Key Determinant for Treatment Planning. In: Resnik R, editor. Misch's Contemporary Implant Dentistry. 4th ed. Canada: Elsevier; 2021.

26. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. Eur J Oral Sci 1998;106(1):527-51.

27. Todisco M, Trisi P. Bone mineral density and bone histomorphometry are statistically related. Int J Oral Maxillofac Implants 2005;20(6):898-904.

28. Ekfeldt A, Christiansson U, Eriksson T, et al. A retrospective analysis of factors associated with multiple implant failures in maxillae. Clin Oral Implants Res 2001;12(5):462-7.

29. Turkyilmaz I, Aksoy U, McGlumphy EA. Two alternative surgical techniques for enhancing primary implant stability in the posterior maxilla: a clinical study including bone density, insertion torque, and resonance frequency analysis data. Clin Implant Dent Relat Res 2008;10(4):231-7.

30. Stavropoulos A, Nyengaard JR, Lang NP, Karring T. Immediate loading of single SLA implants: drilling vs. osteotomes for the preparation of the implant site. Clin Oral Implants Res 2008;19(1):55-65.

31. Summers RB. A new concept in maxillary implant surgery: the osteotome technique. Compendium 1994;15(2):152, 54-6, 58 passim; quiz 62.

32. Huwais S, Meyer EG. A Novel Osseous Densification Approach in Implant Osteotomy Preparation to Increase Biomechanical Primary Stability, Bone Mineral Density, and Bone-to-Implant Contact. Int J Oral Maxillofac Implants 2017;32(1):27-36.

33. Witek L, Neiva R, Alifarag A, et al. Absence of Healing Impairment in Osteotomies Prepared via Osseodensification Drilling. Int J Periodontics Restorative Dent 2019;39(1):65-71.

34. Huwais S, Mazor Z, Ioannou AL, Gluckman H, Neiva R. A Multicenter Retrospective Clinical Study with Up-to-5-Year Follow-up Utilizing a Method that Enhances Bone

Density and Allows for Transcrestal Sinus Augmentation Through Compaction Grafting. Int J Oral Maxillofac Implants 2018;33(6):1305-11.

35. Bashutski JD, D'Silva NJ, Wang HL. Implant compression necrosis: current understanding and case report. J Periodontol 2009;80(4):700-4.

36. Winwood K, Zioupos P, Currey JD, Cotton JR, Taylor M. The importance of the elastic and plastic components of strain in tensile and compressive fatigue of human cortical bone in relation to orthopaedic biomechanics. J Musculoskelet Neuronal Interact 2006;6(2):134-41.

37. Khayat PG, Arnal HM, Tourbah BI, Sennerby L. Clinical outcome of dental implants placed with high insertion torques (up to 176 Ncm). Clin Implant Dent Relat Res 2013;15(2):227-33.

38. Qian J, Wennerberg A, Albrektsson T. Reasons for Marginal Bone Loss around Oral Implants. Clinical Implant Dentistry and Related Research 2012;14(6):792-807.

39. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Periodontol 2018;89 Suppl 1:S267-S90.

40. Zitzmann NU, Berglundh T. Definition and prevalence of peri-implant diseases. J Clin Periodontol 2008;35(8 Suppl):286-91.

41. Lindhe J, Meyle J, Group DoEWoP. Peri-implant diseases: Consensus Report of the Sixth European Workshop on Periodontology. J Clin Periodontol 2008;35(8 Suppl):282-5.

42. Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. J Periodontol 2018;89 Suppl 1:S257-S66.

43. Loe H, Silness J. Periodontal Disease in Pregnancy. I. Prevalence and Severity. Acta Odontol Scand 1963;21:533-51.

44. Mombelli A, van Oosten MA, Schurch E, Jr., Land NP. The microbiota associated with successful or failing osseointegrated titanium implants. Oral Microbiol Immunol 1987;2(4):145-51.

45. Silness J, Loe H. Periodontal Disease in Pregnancy. Ii. Correlation between Oral Hygiene and Periodontal Condtion. Acta Odontol Scand 1964;22:121-35.

46. Salvi GE, Aglietta M, Eick S, et al. Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. Clin Oral Implants Res 2012;23(2):182-90.

47. Pontoriero R, Tonelli MP, Carnevale G, et al. Experimentally induced peri-implant mucositis. A clinical study in humans. Clin Oral Implants Res 1994;5(4):254-9.

48. Zitzmann NU, Berglundh T, Marinello CP, Lindhe J. Experimental peri-implant mucositis in man. J Clin Periodontol 2001;28(6):517-23.

49. Berglundh T, Zitzmann NU, Donati M. Are peri-implantitis lesions different from periodontitis lesions? J Clin Periodontol 2011;38 Suppl 11:188-202.

50. Casado PL, Otazu IB, Balduino A, et al. Identification of periodontal pathogens in healthy periimplant sites. Implant Dent 2011;20(3):226-35.

51. Renvert S, Roos-Jansaker AM, Lindahl C, Renvert H, Rutger Persson G. Infection at titanium implants with or without a clinical diagnosis of inflammation. Clin Oral Implants Res 2007;18(4):509-16.

52. Faot F, Nascimento GG, Bielemann AM, et al. Can peri-implant crevicular fluid assist in the diagnosis of peri-implantitis? A systematic review and meta-analysis. J Periodontol 2015;86(5):631-45.

53. Yaghobee S, Khorsand A, Paknejad M. Comparison of interleukin-1beta levels in gingival crevicular fluid and peri-implant crevicular fluid and its relationship with clinical indexes. J Dent (Tehran) 2013;10(1):1-9.

54. Gowen M, Wood DD, Ihrie EJ, McGuire MK, Russell RG. An interleukin 1 like factor stimulates bone resorption in vitro. Nature 1983;306(5941):378-80.

55. Stashenko P, Dewhirst FE, Rooney ML, Desjardins LA, Heeley JD. Interleukin-1 beta is a potent inhibitor of bone formation in vitro. J Bone Miner Res 1987;2(6):559-65.

56. Fonseca FJ, Moraes Junior M, Lourenco EJ, Teles Dde M, Figueredo CM. Cytokines expression in saliva and peri-implant crevicular fluid of patients with peri-implant disease. Clin Oral Implants Res 2014;25(2):e68-72.

57. Casado PL, Canullo L, de Almeida Filardy A, et al. Interleukins 1beta and 10 expressions in the periimplant crevicular fluid from patients with untreated periimplant disease. Implant Dent 2013;22(2):143-50.

58. Araujo MG, Lindhe J. Peri-implant health. J Periodontol 2018;89 Suppl 1:S249-S56.

59. Axelsson P, Lindhe J. Effect of controlled oral hygiene procedures on caries and periodontal disease in adults. J Clin Periodontol 1978;5(2):133-51.

60. Loe H, Anerud A, Boysen H, Smith M. The natural history of periodontal disease in man. The rate of periodontal destruction before 40 years of age. J Periodontol 1978;49(12):607-20.

61. Schatzle M, Loe H, Lang NP, et al. Clinical course of chronic periodontitis. III. Patterns, variations and risks of attachment loss. J Clin Periodontol 2003;30(10):909-18.

62. Derks J, Schaller D, Hakansson J, et al. Peri-implantitis - onset and pattern of progression. J Clin Periodontol 2016;43(4):383-8.

63. Fransson C, Tomasi C, Pikner SS, et al. Severity and pattern of peri-implantitis-associated bone loss. J Clin Periodontol 2010;37(5):442-8.

64. Barboza EP, Caula AL, Carvalho WR. Crestal bone loss around submerged and exposed unloaded dental implants: a radiographic and microbiological descriptive study. Implant Dent 2002;11(2):162-9.

65. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the canine mandible. J Periodontol 1997;68(2):186-98.

66. Hermann JS, Schoolfield JD, Schenk RK, Buser D, Cochran DL. Influence of the size of the microgap on crestal bone changes around titanium implants. A histometric evaluation of unloaded non-submerged implants in the canine mandible. J Periodontol 2001;72(10):1372-83.

67. Gardner DM. Platform switching as a means to achieving implant esthetics. N Y State Dent J 2005;71(3):34-7.

68. Lazzara RJ, Porter SS. Platform switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. Int J Periodontics Restorative Dent 2006;26(1):9-17.

69. Atieh MA, Ibrahim HM, Atieh AH. Platform switching for marginal bone preservation around dental implants: a systematic review and meta-analysis. J Periodontol 2010;81(10):1350-66.

70. Tomasi C, Tessarolo F, Caola I, et al. Morphogenesis of peri-implant mucosa revisited: an experimental study in humans. Clin Oral Implants Res 2014;25(9):997-1003.

71. Linkevicius T, Puisys A, Steigmann M, Vindasiute E, Linkeviciene L. Influence of Vertical Soft Tissue Thickness on Crestal Bone Changes Around Implants with Platform Switching: A Comparative Clinical Study. Clin Implant Dent Relat Res 2015;17(6):1228-36.

72. Vandeweghe S, De Bruyn H. A within-implant comparison to evaluate the concept of platform switching: a randomised controlled trial. Eur J Oral Implantol 2012;5(3):253-62.

73. Spray JR, Black CG, Morris HF, Ochi S. The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering. Ann Periodontol 2000;5(1):119-28.

74. Eriksson AR, Albrektsson T. Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit. J Prosthet Dent 1983;50(1):101-7.

75. dos Santos PL, Queiroz TP, Margonar R, et al. Evaluation of bone heating, drill deformation, and drill roughness after implant osteotomy: guided surgery and classic drilling procedure. Int J Oral Maxillofac Implants 2014;29(1):51-8.

76. Updegrave WJ. The paralleling extension-cone technique in intraoral dental radiography. Oral Surg Oral Med Oral Pathol 1951;4(10):1250-61.

77. Kan JY, Rungcharassaeng K, Umezu K, Kois JC. Dimensions of peri-implant mucosa: an evaluation of maxillary anterior single implants in humans. J Periodontol 2003;74(4):557-62.

78. Lang NP, Wetzel AC, Stich H, Caffesse RG. Histologic probe penetration in healthy and inflamed peri-implant tissues. Clin Oral Implants Res 1994;5(4):191-201.

79. Abrahamsson I, Soldini C. Probe penetration in periodontal and peri-implant tissues. An experimental study in the beagle dog. Clin Oral Implants Res 2006;17(6):601-5.

80. Chang M, Wennstrom JL, Odman P, Andersson B. Implant supported single-tooth replacements compared to contralateral natural teeth. Crown and soft tissue dimensions. Clin Oral Implants Res 1999;10(3):185-94.

81. Chang M, Wennstrom JL. Soft tissue topography and dimensions lateral to single implant-supported restorations. a cross-sectional study. Clin Oral Implants Res 2013;24(5):556-62.

82. Parpaiola A, Cecchinato D, Toia M, et al. Dimensions of the healthy gingiva and peri-implant mucosa. Clin Oral Implants Res 2015;26(6):657-62.