# Air Abrasive Disinfection of Implant Surfaces in a Simulated Model of Peri-Implantitis

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CERTIFICATE OF APPROVAL

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MASTER'S THESIS

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This is to certify that the Master's thesis of

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has been approved by the Examining Committee for the thesis requirement for the Master of Science degree in Oral Biology at the June 2016 graduation.

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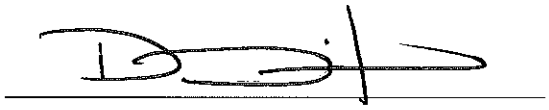

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ABSTRACT

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Simulated Model of Peri-Implantitis

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**Introduction**: Dental implant technology has evolved into a predictable treatment option for the restoration of edentulous sites. However, peri-implantitis is an emerging complication leading to increased morbidity or mortality of osseointegrated implants. The prevalence of peri-implant diseases has been reported to range from 12% to 80% of patients. Treatment of peri-implantitis requires thorough debridement and decontamination of the implant surface. The aim of this *in vitro* study was to evaluate the ability of air-powder abrasion to mechanically decontaminate dental implants in a unique peri-implantitis defect model. A secondary aim was to compare the efficacy of two air-powder abrasion units used for implant decontamination. **Materials and Methods**: 26 OSSEOTITE® implants were inoculated with biofilm media containing *Streptococccus sanguinis* in a peri-implantitis defect model. Six implants served as positive (n=3) and negative controls (n=3) while ten implants were disinfected with Cavitron® Jet Plus™ and ten with AIR-FLOW® PERIO. Residual bacteria were removed from the implants and plated on Brain Heart Infusion (BHI) agar plates. Colony Forming Units (CFUs) were counted after 24 hours. **Results:** Negative control implants were free of bacteria following decontamination. Bacteria were observed on each positive control culture and on 15% of the

experimental cultures. The mean CFUs per plate were 104 for the positive control, 0.85 for the Cavitron® Jet Plus™ and 0.23 for the AIR-FLOW® PERIO. **Discussion:** While complete decontamination of an implant might be unobtainable, the significant reduction in bacteria may allow the host response to better combat remaining periodontal pathogens and initiate healing. When compared to conventional scaling with a curette, air powder abrasion has the advantage of increased ability to contact more of the implant surface potentially resulting in greater elimination of viable bacteria. Evaluating the two instruments, both showed effective results even though there was no statistically significant difference between them. The AIR-FLOW® PERIO did, however, exhibit several characteristics such as the smaller tip design and lateral and vertical spray patterns that could be more beneficial in a clinical setting over the Cavitron® Jet Plus™. **Conclusion**: Air-powder abrasion is a beneficial technique to decontaminate dental implants. In this controlled *in vitro* study, the Cavitron® Jet Plus™ and the AIR-FLOW® PERIO were equally successful at eliminating viable bacteria from dental implant surfaces.

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# CHAPTER I: INTRODUCTION

In 1965 Dr. Brånemark placed the first dental implant. Over fifty years later, implant technology has evolved into a predictable treatment option for the restoration of edentulous sites. Implant rehabilitation is advantageous over conventional prosthetic treatments because implants reduce or eliminate the requirement for partial or complete removable dental prostheses, eliminating the need for removal of tooth structure on adjacent teeth.[1] Research has demonstrated a 96.1% survival rate of osseointegrated implants.[2] Despite this high success rate, implants can manifest diseases of the supporting tissues. Peri-implant mucositis and peri-implantitis result in loss of tissue and possible failure of the implant.

Peri-implantitis is defined as an inflammatory process around an implant. It includes soft tissue inflammation and progressive loss of supporting bone beyond biological bone remodeling whereas peri-implant mucositis encompasses an inflammatory process without bone loss.[3] The prevalence of peri-implant diseases has been reported to range from 28% to 80% of patients.[4-7] It has been estimated that 500,000 implants were placed within the United States in 2014.[8] Therefore, it can be conservatively estimated that at least 150,000 of these implants will manifest disease. Given this high prevalence of disease, there is a crucial need for effective treatment therapies of peri-implant mucositis and peri-implantitis.

# CHAPTER II: REVIEW OF THE LITERATURE

## PERI-IMPLANTITIS

Peri-implantitis has been defined as an inflammatory process around an implant, which includes soft tissue inflammation and progressive loss of supporting bone beyond biological bone remodeling. Peri-implant mucositis, in contrast, encompasses an inflammatory process of these tissues without bone loss.[3] In a cross-sectional study, peri-implant mucositis occurred in 80% of subjects and 50% of implant sites while the prevalence of peri-implantitis was observed in 28% to 56% of subjects and in 12% to 43% of implant sites.[4-7] The true prevalence of peri-implant disease is difficult to ascertain and may be underreported due to varied evaluation techniques among providers, implant systems, definitions of peri-implant disease, subject selection and the length of observation.[7] Furthermore, evidence suggests that peri-implant disease may be higher in patients diagnosed with periodontal disease, systemic chronic disease such as diabetes or patients who smoke tobacco.[9,10]

One of the main etiologies of peri-implant diseases is bacterial plaque. Poor oral hygiene, calculus, history of periodontal disease, residual cement, tobacco use, diabetes, excessive alcohol use, genetics and type of implant surface have all been identified as contributing factors.[7,9-13] The bacterial flora associated with peri-implantitis is similar to that found in chronic periodontitis.[14] Gram-positive, facultative bacteria are normally associated with a healthy tooth or implant sulcus. However, as the supporting tissues of the tooth of implant become diseased, bone and attachment loss occurs and a deep pocket develops. The microbiota also changes to gram negative anaerobic pathogens such as *Porphymonas gingivalis, Tannerella*

*forsythia*, *Fusobacterium nucleatum*, *Peptostreptococcus micros*, *Prevotella nigrescens*, and *Aggregatibacter actinomycetemcomitans* serotype b.[15-19]

If the inflammatory process occurring around implants is not recognized and treated at an early stage, the resulting peri-implant bone loss can be severe and characterized by a deep, narrow, circumferential defect (Figure 1).[20] The nature of this defect perpetuates the disease process by limiting accessibility to thoroughly decontaminate the implant surface during therapy. Access is severely restricted in the apical region. i.e at the bottom of the defect. Without effective debridement, bacteria are not eliminated and the therapeutic goal cannot be achieved. Therefore, treatment techniques must effectively remove bacteria and their products from the diseased implant surface. They should also be able to navigate the deep and tortuous bone defect.

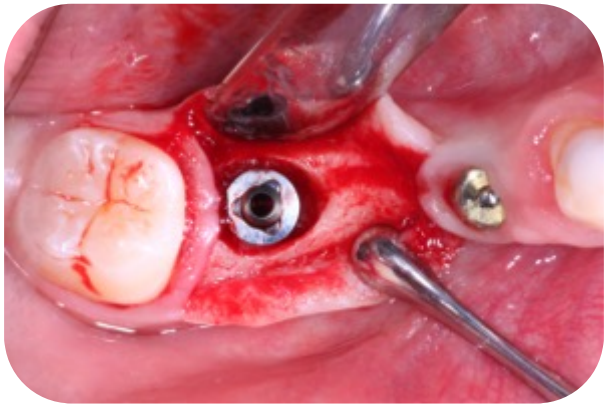

Figure 1. Clinical Peri-Implantitis Circumferential Defect
(Courtesy of Dr. C. Barth)

TREATMENT

There is no universally accepted regimen to treat peri-implantitis. However, the rationale for treatment is similar to treating periodontitis and stems from the commonality of bacterial

plaque as the etiologic agent. Surface debridement and elimination of pathogenic plaque and bacterial products constitute the basic elements for treating both diseases.[9]

There are two main goals for peri-implantitis surgical therapy. The first is to decontaminate and debride the implant surface in order to remove bacteria and local factors such as endotoxin which contribute to the disease process. The second is to resolve pathologic pockets, correct the bony defect, restore osseous topography to achieve re-osseointegration of the implant.[21]

Various techniques have been recommended to debride and decontaminate the implant surface. These include the use of curettes, titanium brushes, sterile saline, air-powder abrasion, pharmacotherapeutics, chemotherapeutics, implantoplasty, biologic materials and lasers.[22] There is evidence in the literature indicating the beneficial effects of each therapy; however, there is no consensus among periodontists for the superiority of one treatment modality over another.[9] The literature also reports successful treatment of peri-implantitis with bone growth and re-osseointegration along the previously diseased surface when mechanical and chemical decontamination techniques were used together with regenerative procedures such as bone grafts and membranes.[23]

Mechanical decontamination employing air-powder abrasion has not been extensively studied. In an *in vitro* study by Zablotsky et al. (1992), it was shown that surface detoxification by grit-blasting of implants was superior to topical application of citric acid, stannous fluoride, tetracycline HCl, chlorhexidine gluconate, hydrogen peroxide, chloramine T, sterile water or conventional debridement techniques using plastic sonic scaler tips.[24] Another *in vitro* study using air-powder abrasion found bacterial endotoxin was removed from 98.5% of machined

titanium surfaces, 84.2% of plasma sprayed surfaces and from 88.8% of HA-coated surfaces.[25] Several i*n vitro* studies have shown beneficial results using air-powder abrasion therapy for the treatment of peri-implantitis.[26] Maximo et al. (2009), in an *in vivo* report, demonstrated that treatment consisting of surgical access and implant debridement using Teflon curettes and air abrasion resulted in improved clinical parameters and decreased periodontal pathogens.[27] Schou et al. (2003) compared air-powder abrasion, citric acid, saline and chlorhexidine for the treatment of experimentally induced peri-implantitis in monkeys and found all treatments effective.[28] In a case series, Froum et al. (2012) described a clinical protocol using a combination of modalities to treat 51 implants diagnosed with peri-implantitis. This stepwise decontamination approach utilized air abrasion for 2 minutes, tetracycline (50mg/mL) application for 30 seconds, 0.12% chlorhexidine gluconate application for 30 seconds, copious sterile saline irrigation and bone grafting around the site. This procedure resulted in successful bone fill and resolving the signs and symptoms of peri-implantitis such as reduction of probing depths, decrease in inflammation, added bone fill and a decrease in bleeding on probing over a 3.5 to 7 year follow-up.[21] However, it is not clear which of the steps of this protocol was more efficacious than the others.

Complete access to the implant surface is imperative for successful treatment with air-powder abrasion units. Bacteria located on the inferior boarder of the implant threads are protected and often remain untouched by the abrasive spray. This can be the case especially in deep and narrow peri-implant defects. However, innovative designs of air abrasion unit tips can improve operator access to enhance surface decontamination.

The Cavitron® JET Plus™ and AIR-FLOW® PERIO air abrasive units are indicated for the treatment of peri-implantitis.[29,30] The Cavitron® JET Plus™ tips are inflexible and approximately 1.34mm in diameter at the narrowest point and 1.85mm in diameter at the widest

point. The abrasive spray is ejected unidirectionally from the tip. This arrangement does not allow for precise delivery of abrasive particles to the implant surface within the osseous defect. The AIR-FLOW® PERIO tip is flexible, plastic, and trapezoid shaped with dimensions of 0.47mm x 0.85mm increasing to 0.84mm x 2.15mm. This tip allows for simultaneously delivery of a water and abrasive spray in multiple directions. It is designed to deliver the spray at 90° to the long axis of the instrument and at the apex of the tip.[30] The smaller tip and unique delivery design might allow for easier placement into the deep, narrow circumferential defects associated with peri-implantitis and more effectively clean the diseased implant surface.[31]

## AIM

This *in vitro* study evaluated the efficacy of air-powder abrasion to decontaminate dental implants in a model mimicking a deep, narrow, circumferential, peri-implantitis defect. A secondary goal was to determine if the AIR-FLOW® PERIO, with its unique tip design, was more efficacious than Cavitron® Jet Plus™ at removing viable bacterial from the implant surface.

# CHAPTER III: MATERIALS AND METHODS

## PREPARATION PHASE

This *in vitro* study followed the methodology of a feasibility study (IRBNet #387270) performed by Merchant (2013) and Pasieta (2014). Peri-implant circumferential bony defect models were designed and fabricated at the 3D Medical Modeling Service, Walter Reed National Military Medical Center (WRNMMC), with the following dimensions; 6.5mm deep by 10.5mm wide with a volume of 350µL (Figure 2). The models were mass-produced, using an additive manufacturing (Objet500 Connex 3D printer, Stratsys, Eden Prairie, MN, USA) technique, with a resin material (Renshape Huntsman 7810). The accuracy ranged from 20-85 microns. OSSEOTITE® (Biomet 3i™) dental implants were placed into individual resin models with the implant platform positioned at the base of the model and the apex protruding from the top (Figure 3). The implants were secured in place by the implant specific impression coping and screw. The simulated defect model served as both the reservoir for the contaminant and site where decontamination procedures were performed.

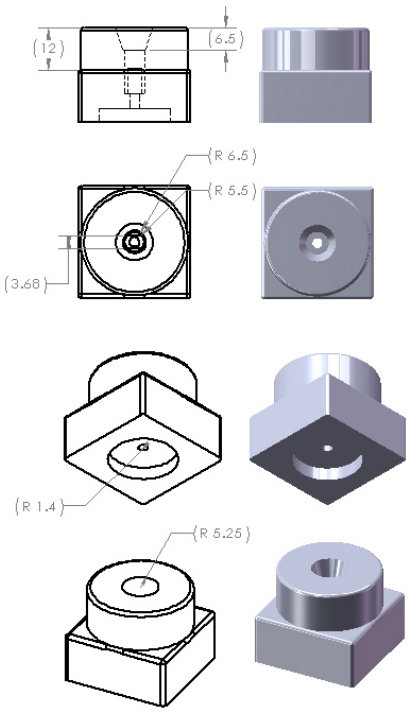


Figure 2. Peri-Implantitis Model Design (Courtesy of Dr. K. Merchant)

Figure 3. Fabricated Peri-Implantitis Model (Courtesy of Dr. K. Merchant)

*Streptococcus sanguinis* was sub-cultured in Brain Heart Infusion (BHI) broth and incubated (37°C, 5% $CO_2$, 95% humidity) for 16 hours. The contamination media was prepared by transferring 1mL of the BHI-bacterial culture into 9mL of fresh, pre-warmed Biofilm Media (BM) and incubated under the same conditions for an additional 5 hours. A spectrophotometer (Genesys 10S UV-VIS, Thermo Fisher, Waltham, MA, USA) was used to monitor bacterial growth until an optical density of greater than 0.5 at 600nm was achieved. A bacterial culture was plated in duplicate, using BHI agar plates, at $10^{-3}$, $10^{-4}$, $10^{-5}$ dilutions to validate the actual bacterial concentration surrounding the implants (Appendix 1).

All experimental units were assembled from autoclaved or pre-sterilized components in a biological safety hood using sterile technique. All components were sterilized in an autoclave at 270° Fahrenheit at 42 psi with an exposure time of 15 minutes and drying time of 30 minutes. Twenty-six tapered, external hex Biomet 3i™ OSSEOTITE® implants with polished collars (5mm platform) were placed into the following groups; treatment A (n=10), treatment B, (n=10), positive control (n=4) and negative control (n=2). Using sterile technique, individual units were assembled by first placing an impression coping into a resin model previously numbered with an indelible marker (1-26). The implant was removed from its packaging and placed onto the impression coping. The coping screw was then hand-tightened to create a hermetic seal between the implant platform and the base of the model.

CONTAMINATION PHASE

Experimental (n=20) and positive control (n=4) implants were inoculated with 350µL of suspended contamination media pipetted into the simulated bony defect of the resin model. Negative controls (n=2) had 350µL of sterile Biofilm Media pipetted into the simulated bony

defect. The media suspension surrounded equal portions of the polished collar and roughened surface of the implants. Following media placement into the defect, at least 5 apical implant threads remained exposed above the inoculum. These threads were used to handle the implant after the decontamination procedure was performed. To prevent the media from evaporating, a sterile test tube lid was packed with sterile 2x2 gauze and saturated with 1.5mL of BHI solution. Sterile aluminum foil was used to wrap both the defect model and test tube lid securely together. After the implants were inoculated, all samples were incubated at 37°C, 5% $CO_2$ and 95% humidity for 24 hours.

DECONTAMINATION PHASE

Following the incubation period, the excess media was removed from the defect space using a thin tipped pipette without touching the implant surface. The experimental implants were then immediately decontaminated using either the AIR-FLOW® PERIO (treatment A) or the Cavitron® JET Plus™ (treatment B). The air-powder abrasion instruments were used to deliver an abrasive powder abrasive (AIR-FLOW® PERIO - Glycine, Amorphous Silica; Cavitron® JET Plus™ - Sodium Bicarbonate, Amorphous Silica, Mint Flavoring with Sodium Saccharin) and water circumferentially around the implant threads and the implant collar for 60 seconds. A new instrument tip was used for each implant undergoing decontamination.

Following decontamination, the experimental implants were rinsed with 20mL of sterile saline to remove any extraneous abrasive. The experimental, positive control and negative control implants were removed from their respective models using sterile technique under a biological safety hood. A sterilized hemostat was used to hold and transfer each implant from the resin model into a 15mL centrifuge tube containing 2.0mL of phosphate buffered saline

(PBS). The tubes were vortexed in a multi-tube vortex mixer (model number 945093, Fisher Scientific, Waltham, MA, USA) for 60 seconds at 1000 RPM to dislodge remaining bacteria. After vortexing, the centrifuge tubes containing the bacterial suspension were placed in an ice bath to suspend bacterial growth.

## VALIDATION OF THE METHODOLOGY

A series of pilot tests were performed to validate the methodology. The resin defect model, with a secured implant, was tested for leakage and evaporation by placing erythrosine dye into the well and assessing 24 hours later. This test demonstrated a watertight seal was created between the implant platform, the impression coping and the base of the model. In a second test, 350µL of sterile biofilm media was syringed into the wells of three separate defect models. In model #1, no additional coverings were wrapped around the implant and defect model. For model #2, sterile aluminum foil was wrapped around the implant and defect model. And, in model #3, a sterile test tube lid was packed with sterile 2x2 gauze and saturated with 1.5mL of BHI solution. The defect model and test tube lid were secured by wrapping a sterile aluminum foil around both parts. The models with media were placed in the incubator (37°C, 5% $CO_2$, 95% humidity) for 24 hours. The media was then removed from the models with a pipette and measured. Model #1 showed the most evaporation at ~50% while model #2 had ~40% evaporation. Model #3 showed the least amount of evaporation at ~15%. The latter model was chosen for the methodology. Additionally, in the feasibility study performed by Pasieta (2014), it was discovered that streaking of the bacteria on the agar plates using a loop resulted in occasional gouging of the agar medium negating the results for that plate. Therefore, a drip technique was devised where the 10uL of the inoculum was added to the upper part of a BHI

agar plate and the plate tilted at a 45° angle to allow the inoculum to flow downward creating 2 bacterial streaks inoculum.

## DATA COLLECTION AND STATISTICAL ANALYSIS

Standard microbial culture technique was used to determine results for this experiment. Each BHI plate was identified with a pre-assigned number corresponding to a master list of all test and control specimens. All the samples were plated using undiluted inoculum in duplicate using the previously described drip technique and incubated at 37°C, 5% $CO_2$, 95% humidity for 24 hours. After incubation, bacterial colonies on each plate were counted by two blinded evaluators using the Scienceware® Colony Counter (model 378620002, Scienceware® Bel-Art Products, Wayne, NJ, USA) to record the colony forming units (CFUs).

Data was summarized by computing mean CFUs grown from each implant across each of the two streaks from the duplicated plates. A power analysis confirmed statistical significance using 4 positive control, 2 negative control, 10 experimental treatment A and 10 experimental treatment B implants. Due to the suspected non-normality of such data, a nonparametric statistic (the Mann-Whitney U Test) was used to answer the aims of this study: 1) the comparison between mean CFUs from implants treated with the Cavitron® JET Plus™ vs. untreated Positive Control implants; 2) the comparison between mean CFUs from implants treated with the AIR-FLOW® PERIO vs. untreated Positive Control implants; and 3) the difference between the average number of bacterial colonies from implants treated with the Cavitron® JET Plus™ vs. those treated with the AIR-FLOW® PERIO.

## CHAPTER IV: RESULTS

The mean concentration of *Streptococcus sanguinis* in the contaminated media was $1.65x10^7$ CFUs per milliliter at an optical density of 0.62 at 600nm. Appendix 2 displays the number of colonies on each bacterial streak for the experimental, positive control and negative control implants.

| Condition | N | Mean | Standard Deviation | Median | Min | Max |
|---|---|---|---|---|---|---|
| **Positive Control** | 4 | 104.1 | 55.9 | 103.0 | 45.5 | 165.0 |
| **AIR-FLOW® PERIO** | 10 | 0.2 | 0.3 | 0.0 | 0.0 | 1.0 |
| **Cavitron® Jet Plus™** | 10 | 0.9 | 2.3 | 0.0 | 0.0 | 7.3 |
| **Negative Control** | 2 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |

Table 1. Positive Control, Experimental and Negative Control Implant Results

All positive controls demonstrated bacterial growth. Negative controls displayed no growth on any culture. Table 1 and Figure 4 demonstrate representative examples of bacterial growth from each condition. The positive control implants exhibits large amounts of bacteria averaging 104.1 ±55.9 CFU/streak. Figure 5 displays bacterial colonization on the positive control implants while Figure 6 is an example of the negative control representing no bacterial growth.

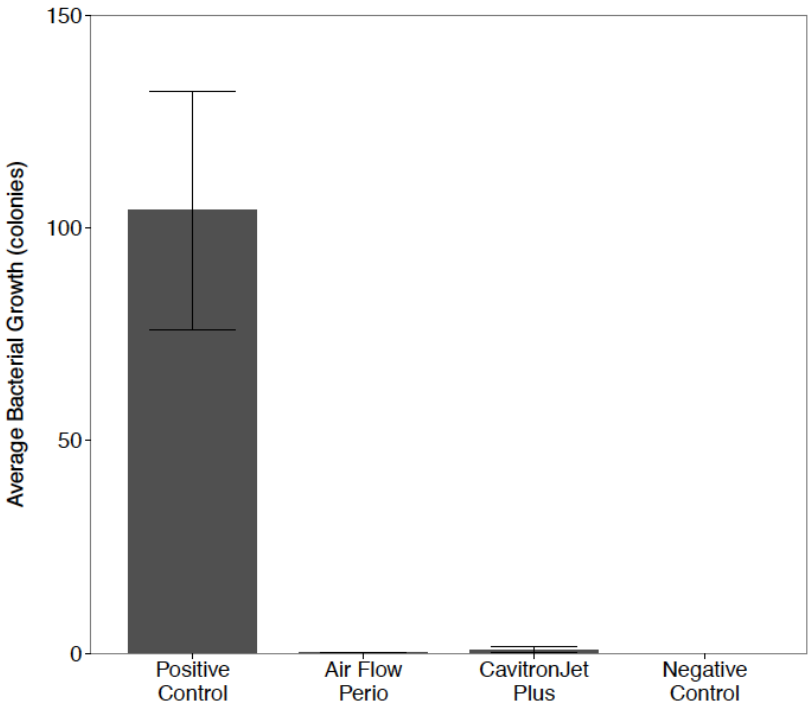


Figure 4. All CFU Growth

Figure 5. Positive Control

Figure 6. Negative Control

Figure 8. Cavitron® Jet Plus™ Agar Plate

Figure 9. AIR-FLOW® PERIO Agar Plate

Both air-powder abrasion devices demonstrated the ability to reduce viable bacteria from the implant surface demonstrating a significant difference in remaining CFUs between the implants decontaminated with air-powder abrasives and the positive controls. The data is summarized in Figure 7 which plots the average number of treated colonies for the Cavitron® Jet Plus™ (0.85 CFU) and AIR-FLOW® PERIO (0.23 CFU). Figures 8 and 9 exemplify the limited number CFUs grown on the agar plates for the treated implants. The comparison of the two treatment conditions yielded no statistically reliable difference in bacterial elimination.

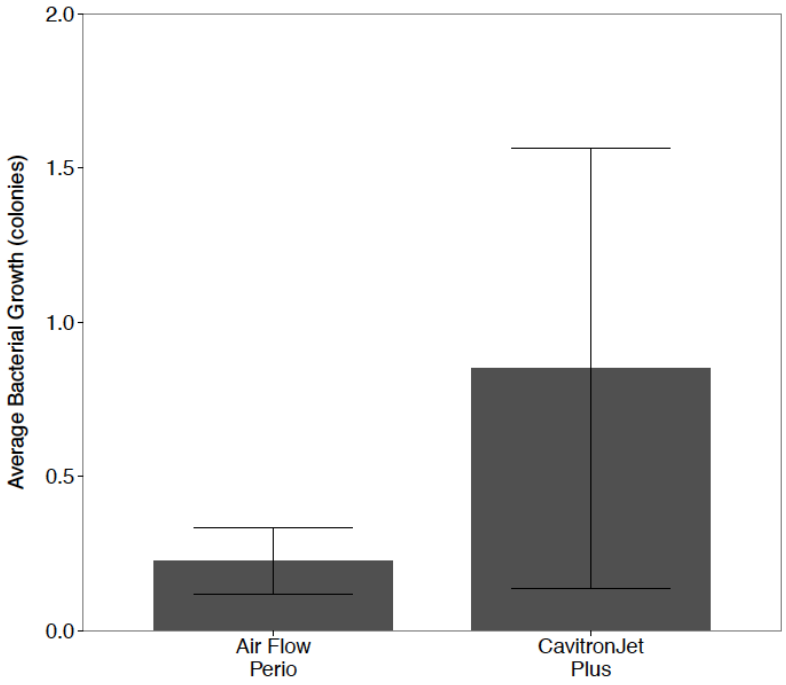


Figure 7. AIR-FLOW® PERIO vs Cavitron® Jet Plus™ CFU Growth

Negative controls verified that sterile technique throughout the experimentation yielded no contamination of the BHI agar plates or implants with any foreign bacteria.

# CHAPTER V: DISCUSSION

This *in vitro* study evaluated the ability to mechanically decontaminate dental implants in an *in vitro* peri-implantitis model using air-powder abrasion. Additionally, the Cavitron® Jet Plus™ and AIR-FLOW® PERIO were compared to examine if one unit exhibited a decontamination advantage over the other. The results demonstrated that both air-powder abrasion devices significantly reduced viable bacteria from the implant surfaces. No significant difference was found between the devices.

Several clinical studies have corroborated the benefits of air-powder abrasion for the treatment of peri-implant lesions. Maximo (2009) evaluated surgical treatment of peri-implantitis using an air-powder abrasion decontamination technique. Post-surgical plaque samples were analyzed using DNA-DNA checkerboard hybridization for 40 bacterial species. There was a 21% reduction in red complex bacterial after mechanical anti-infective therapies.[27] Periodontal parameters and cytokine levels were evaluated in another peri-implantitis study. Diseased implants were surgically accessed and debrided using abrasive sodium carbonate air-powder and resin curettes. A post-surgical decrease in tumor necrosis factor-alpha (TNF-α) and improvement in the ratio of osteoprotegerin (OPG) to receptor activator of nuclear factor- kappa B ligand (RANKL). These values resembled levels associated with healthy implants. Also, plaque index, bleeding on probing and probing depths improved.[32] A retrospective study assessed clinical benefits of surgically treated peri-implant defects using the AIR-FLOW® PERIO device versus curettes and a sterile saline impregnated cotton pellet rub. As found with the previous two studies, clinical parameters significantly improved when compared to the control counterparts.[31] While complete decontamination of the implant appears to be

unobtainable, the reduction in bacterial load can allow the host response to better combat periodontal pathogens and initiate healing through re-osseointegration.

The comparison between the two air-powder abrasion instruments revealed no significant differences in their ability to eliminate viable attached bacteria. One might expect that the design difference of the AIR-FLOW® PERIO instrument tip would have produced a superior result. However, it may be postulated that the similar results stemmed from the increased access to decontaminate the implant surfaces in this *in vitro* model when compared to an *in vivo* situation.

When assessing the attributes of the two air-powder abrasion instruments, the AIR-FLOW® PERIO provided other benefits. It was much easier to insert the tip into the defect. The Cavitron® Jet Plus™ tip was larger making it difficult to place in between the implant and defect wall. The AIR-FLOW® PERIO also projected water and the powder abrasive laterally and from the tip while the Cavitron® Jet Plus™ only discharged water and powder from the tip. Furthermore, the Cavitron® Jet Plus™ discharged a larger spray radius spreading contaminated materials over a greater area. Although no significant differences in eliminating viable bacteria were found between the Cavitron® Jet Plus™ and the AIR-FLOW® PERIO *in vitro*, the AIR-FLOW® PERIO design was superior to use from a clinician's perspective in this model.

A shortcoming of this *in vitro* study was the use of a mono-bacterial environment and lack of a true biofilm to mimic a contaminated implant. *Streptococcus sanguinis* was selected for this study because it is an initial colonizer of dental plaque found in patients with periodontal disease.[18] Also, it is a gram-positive, facultative cocci that was easy to culture and could be reliably identified. However, bacterial ecosystems *in situ* are complex arrays of species. They have associated biofilms and often are associated with calculus. These biofilms contain an extracellular polymeric substance made of glycoproteins, extracellular DNA, proteins, and

polysaccharides, which increases their resistance to displacement by mechanical means. To enhance clinical relevance of *in vitro* models the incorporation of multi-bacteria biofilms and calculus would more accurately represent *in situ* conditions when testing the efficacy of air-powder abrasion therapy.

Additional concerns when using air-powder abrasion as a treatment modality is the potential to induce an air emphysema or air embolisms, a forceful introduction of gases into soft tissue spaces, fascial planes or the bloodstream. Adverse effects of air emphysema or air embolisms can include swelling, pain, dysphagia, dyspnea, apnea, loss of consciousness, ataxia, convulsions, numbness or dysesthesia, weakness, nausea or vomiting. A review of the literature by Graumann (2013) reported 9 air emphysema and 3 air embolism incidents related to air-powder abrasion occurred between 1977 and 2001. Each occurred during non-surgical therapy when the instrument was left in the sulcus for an extended period of time without movement. When placed in context to the vast number of times air-powder abrasion is employed for non-surgical therapy, the information suggests that there is minimal risk associated with air-powder abrasion therapy.[33] However, care is needed when using these instrument to avoid adverse events.

There is no established standard of care for the treatment of peri-implant diseases. Future studies should investigate decontamination protocols evaluating the efficacy of individual steps in the procedure. Lacking a predictable therapy, risk for severe morbidity or implant loss will continue to increase in the future.

# VI: CONCLUSION

Under the conditions of this study, air-powder abrasion alone effectively decontaminated dental implants. Both the Cavitron® Jet Plus™ and the AIR-FLOW® PERIO appeared equally successful at eliminating viable bacteria from dental implant surfaces. Additional animal and human studies are needed to validate clinical efficacy of air-powder abrasion as a sole treatment modality and to compare air-powder abrasion to other implant decontamination therapies.

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# VIII: APPENDICES

Appendix 1

| | **Dilution** | | |
|---|---|---|---|
| | **$10^{-3}$** | **$10^{-4}$** | **$10^{-5}$** |
| **Plate 1** | 152 | 14 | 1 |
| **Plate 2** | 204 | 24 | 0 |
| **Plate 3** | 153 | 15 | 4 |
| **Plate 4** | 127 | 13 | 0 |

Appendix 1. Concentration of *S. sanguinis* in Contamination Media

Appendix 2

| | | Standard Microbial Culture | | | |
|---|---|---|---|---|---|
| | | Undiluted Plate 1 | | Undiluted Plate 2 | |
| | | Streak 1 | Streak 2 | Streak 1 | Streak 2 |
| Experimental | | | | | |
| Cavitron® JET Plus™ | Implant 1 | 0 | 0 | 0 | 3 |
| | Implant 2 | 0 | 0 | 0 | 0 |
| | Implant 3 | 0 | 0 | 0 | 0 |
| | Implant 4 | 1 | 0 | 0 | 0 |
| | Implant 5 | 0 | 0 | 0 | 0 |
| | Implant 6 | 0 | 0 | 0 | 0 |
| | Implant 7 | 0 | 0 | 0 | 0 |
| | Implant 8 | 4 | 10 | 9 | 6 |
| | Implant 9 | 0 | 0 | 1 | 0 |
| | Implant 10 | 0 | 0 | 0 | 0 |
| AIR-FLOW® PERIO | Implant 11 | 0 | 4 | 0 | 0 |
| | Implant 12 | 0 | 0 | 0 | 0 |
| | Implant 13 | 0 | 0 | 0 | 0 |
| | Implant 14 | 1 | 0 | 0 | 1 |
| | Implant 15 | 0 | 0 | 0 | 0 |
| | Implant 16 | 0 | 0 | 0 | 0 |
| | Implant 17 | 0 | 0 | 2 | 0 |
| | Implant 18 | 1 | 0 | 0 | 0 |
| | Implant 19 | 0 | 0 | 0 | 0 |
| | Implant 20 | 0 | 0 | 0 | 0 |
| Positive Controls | | | | | |
| Implant 21 | | 141 | 151 | 134 | 120 |
| Implant 22 | | 50 | 56 | 35 | 41 |
| Implant 23 | | 183 | 160 | 154 | 163 |
| Implant 24 | | 73 | 43 | 80 | 82 |
| Negative Controls | | | | | |
| Implant 25 | | 0 | 0 | 0 | 0 |
| Implant 26 | | 0 | 0 | 0 | 0 |

Appendix 2. Experimental, Positive Control and Negative Control Implant Colony Forming Units