Comparison of an Nd:YAG Laser to Air-Powder Abrasion in Decontaminating Titanium Implants Utilizing a Peri-Implantitis Defect Model

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A Thesis submitted to the Faculty of the
Periodontics Graduate Program
Naval Postgraduate Dental School
Uniformed Services University of the Health Sciences
in partial fulfillment of the requirements for the degree of
Master of Science in Oral Biology

June 2019

Naval Postgraduate Dental School
Uniformed Services University of the Health Sciences
Bethesda, Maryland

CERTIFICATE OF APPROVAL

MASTER'S THESIS

This is to certify that the Master's thesis of

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June 2019

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ABSTRACT

Comparison of an Nd:YAG Laser to Air-Powder Abrasion in Decontaminating Titanium Implants Utilizing a Peri-Implantitis Defect Model

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Introduction:

Implants eliminate the need to prepare natural teeth for fixed prostheses and serve as an alternative treatment option for partially and fully edentulous patients. Implants are equally as susceptible to inflammatory disease processes as the natural dentition. There is currently no universally accepted protocol for the treatment of peri-implantitis. The aim of this study was to compare an air-powder abrasion system (Cavitron JET Plus™, York, PA, USA) to an neodymium-doped yttrium aluminum garnet (Nd:YAG) laser (Millenium Dental Technologies Periolase® MVP-7™, Cerritos, CA, USA) in the ability to eradicate viable bacteria from dental implant surfaces in a peri-implantitis defect model.

Methods:

Fifty-six external hex OSSEOTITE® Biomet 3i™(Biomet 3i, Palm Beach Gardens, FL, USA) dental implants were placed in the defect model and contaminated with *Streptococcus sanguinis*. A total of 6 implants served as positive and negative controls. Ten implants were decontaminated using the air-powder abrasion method, and forty implants were

decontaminated using the Nd:YAG laser. Quantification of viable bacteria was done by counting bacterial colony forming units (CFU) after 48 hours of incubation.

Results:

All positive and negative control groups validated the experimental methodology. Laser treatment was more effective in total elimination of bacteria from contaminated implant surfaces. 78% of Nd:YAG laser plates displayed zero bacterial growth, whereas only 27% of air-powder abrasion plates had zero growth. A Mann-Whitney U Test indicated that the Nd:YAG laser significantly reduced viable bacteria from contaminated dental implant surfaces.

Conclusion:

Utilization of Nd:YAG laser showed a significantly greater ability to decontaminate implant surfaces when compared to the Cavitron JET Plus™ air-powder abrasion system.

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CHAPTER 1: INTRODUCTION

Treatment options for partially and fully edentulous patients have improved remarkably since the development of the titanium dental implant by Dr. Branemark[1]. The prevalence of dental implants has increased eight-fold, from 0.7% to 5.7%, in the period from 1999 to 2016[2]. Recent data indicate that more than 3 million people in the United States have received dental implants, with this number growing by 500,000 a year[3]. Dental implants are positioned in alveolar bone and designed for replacement of teeth lost to disease and trauma, among other causes. Implants eliminate the need to prepare natural teeth adjacent to edentulous areas for fixed prostheses, serve as bridge abutments, reduce the need for removable partial dentures and provide comfort and retention for complete dentures[4,5]. Following treatment with dental implants, patients need regular maintenance of the implant-supported prosthesis to promote long-term health, stability, and function[6]. Dental implants and natural teeth have similar susceptibility to disease, with peri-implant disease exhibiting soft tissue inflammation, loss of bony support, and potential loss of osseointegration. With demand for dental implants growing, greater emphasis must be placed on the diagnosis and treatment of implant diseases such as peri-implant mucositis and peri-implantitis.

CHAPTER II: REVIEW OF LITERATURE

The American Academy of Periodontology defines peri-implantitis as an inflammatory reaction in the soft tissues surrounding a dental implant with evidence of progressive bone loss. Peri-implant mucositis involves an inflammatory process confined to the soft tissues without evidence of bone loss[7]. During the progression of peri-implant mucositis to peri-implantitis, an implant demonstrates a progressive loss of bone support, possibly leading to mobility and ultimate failure[8].

The literature has reported a wide range of peri-implant disease prevalence. The consensus report of the Sixth European Workshop on Periodontology determined that peri-implant mucositis occurred in 80% of subjects and 50% of implant sites, while the prevalence of peri-implantitis ranged from 28% to 56% of subjects and 12% to 40% of implant sites[9]. In 2010, Koldsland assessed 109 subjects with a functional loading time of 8.4 years, and categorized the amount of detectable radiographic peri-implant bone loss, inflammation, and presence of bleeding on probing. The investigation reported a severity-dependent range of peri-implantitis from 11.3% to 47.1%[10]. A more recent meta-analysis by Atieh determined that peri-implant mucositis occurred in 63.4% of participants and 30.7% of implants, while peri-implantitis prevalence was determined to be 18.8% of participants and 9.6% of implants[11].

The etiology of peri-implantitis is varied and multi-factorial, yet centers on the presence of a pathogenic biofilm in a susceptible host. Secondary contributing factors include the presence of residual sub-gingival cement associated with cement-retained dental implant restorations and cigarette smoking[12]. Bacteria-induced inflammation is

usually associated with the patient's oral biofilm, and the growth of the bacteria is exacerbated in individuals with poor oral hygiene. When combined with host susceptibility, an increase in bacterial plaque around dental implants may contribute to the onset of peri-implant disease. Analysis of the microflora associated with failing implants reveals an increased number of orange and red-complex, Gram-negative, anaerobic pathogens, including *Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium* and *s*pirochetes[13-15]. Highlighting a possible link between the presence of residual periodontal pathogens and the onset of peri-implant disease, Mombelli reported that the pre-existing oral microflora before implant placement contributes to the composition of the newly established microflora on dental implants after placement[16, 17]. Just as smoking has been proven to increase the risk of periodontitis, evidence supports a similar detrimental effect of smoking on dental implant health. Patients who smoke have a higher rate of implant loss at 15.3% versus only 2% in non-smokers. Furthermore, peri-implant disease is 6.5 times more prevalent in patients with chronic periodontitis than those without a history of periodontal disease[18]. With such evidence in mind, patients with a history of periodontitis should be advised that implant success may be negatively impacted[9, 19].

Along with clinical signs of inflammation and bleeding on probing, peri-implantitis presents as a circumferential, moat-like bony defect around a dental implant[20]. Though peri-implantitis shares a similar pathophysiology with periodontitis, there is no universally accepted treatment protocol for peri-implantitis[21, 22]. A successful outcome measure for treatment continues to be long-term survivability and

re-osseointegration of the implant after regenerative therapy[23]. While it is known that early detection and intervention are mandatory for the preservation of the implant and supporting tissues, expert consensus is lacking for a standardized treatment approach[24]. Although many treatment protocols have been proposed in the literature, most approaches stem from the broad categories of non-surgical, surgical and laser therapy. Louropoulou reported on non-surgical, mechanical decontamination of dental implants utilizing traditional and ultrasonic scalers, reporting damage to the implant surface[25]. The use of non-metal instruments, rubber polishing cups and abrasive pastes has been successful in treating peri-implantitis when used in conjunction with local antimicrobial therapy[26-28]. However, a systematic review by Claffey noted no single method of surface decontamination, including chemical agents, air-powder abrasives or lasers, to be superior[22]. Resective and regenerative techniques, coupled with surface decontamination strategies, have also proven to be successful. Leonardt surgically accessed failing implants, removed granulomatous inflammatory tissues, applied hydrogen peroxide to clean the implant surfaces, and reported a 58% success rate over five years with a corresponding decrease in disease progression[29]. A more recent case series employed a comprehensive surface decontamination protocol with chemical agents, air-powder abrasion, and enamel matrix derivative, followed by regeneration using a bone graft, recombinant platelet-derived growth factor, and a membrane. The investigators reported successful regeneration in all 51 implants with no bone loss over 3 to 7.5 years[21]. While such complex surgical modalities have proven to be effective, success comes with a high financial cost and may increase the risk of patient morbidity[30,

[31].

With no universal consensus for the treatment of peri-implantitis, a continued interest in evaluating alternative methodologies persists. One possible option that has been explored in the literature is the air-powder abrasive system. An *in vitro* study by Quintero compared two air-powder abrasion devices, the Cavitron® Jet Plus (Dentsply Sirona, Charlotte, NC, USA) and the Air-Flow Handy Perio® (Hu-Friedy, Chicago, IL, USA), in the ability to eliminate viable bacteria from implant surfaces in a simulated model of peri-implantitis. The author reported that both devices significantly reduced bacterial colony forming units from baseline, and the two systems were equally effective[32]. In a literature review of 27 articles evaluating air-powder abrasive treatment for surface decontamination, Tastepe concluded that *in vivo* data on air abrasives was insufficient, but *in vitro* studies showed promising results[33]. While clinical data is limited, the overall body of evidence supports air-powder abrasion as a safe and effective method for implant surface decontamination.

Another alternative for implant surface decontamination is laser therapy. The ability of lasers to perform photoantisepsis, or selective killing, is a unique capability that has the potential to target well-known periodontal pathogens such as *Porphyromonas gingivalis* and *Prevotella intermedia*[34]. The energy of the Nd:YAG laser passes through water and hydroxyapatite, but is absorbed by darker pigments found in melanin, hemoglobin, and certain periodontal pathogens[35]. The use of the Nd:YAG laser in dental applications ranges from minor soft tissue surgery to the proprietary LANAP© and LAPIP© protocols for the treatment of periodontal and peri-implant disease. While

there are a limited number of controlled clinical trials reporting on the efficacy of the Nd:YAG laser in treating peri-implantitis, outcomes from case reports and *in vitro* studies have been promising. In a 2016 review of lasers in periodontal and peri-implant therapy, Mizutani suggested that lasers might reduce the need for established, invasive surgical procedures, and that evidence supports the use of lasers for the treatment of peri-implant mucositis and peri-implantitis[36]. A recent study compared Nd:YAG laser-assisted mechanical debridement of implants versus mechanical debridement alone, revealing that laser treatment was more effective in reducing inflammation in the first 3 months, but the added benefit of the laser dissipated at the 6-month evaluation[37].

Given the wide variety of commercially available lasers in dentistry and the unique impact of each specific laser wavelength on soft and hard tissue, an understanding of the interplay between laser energy and dental implants is paramount[38]. Implant surface temperature changes during laser decontamination have been investigated with controversial outcomes. The variations in laser wavelength, power output, time, operation mode, and provider skill make comparison of the available data from published investigations difficult. The inherent variability is concerning as temperatures in excess of 44-47^{o}C for more than 1 minute can cause an increase in implant failure[39]. Nevins' study on the use of Er:YAG laser to decontaminate dental implant surfaces noted that there was a slight rise in temperature, but with no visible damage to the adjacent bone[24]. An investigation by Oyster used a CO_2 laser for decontaminating the surface of implants. Using pig mandibles, Oyster operated the laser at various power levels ranging from 2-4 Watts in continuous mode with times of 2, 4, 6,

and 8 seconds. He showed that using the lowest power setting and time produced a minimal temperature change of 1.2°C. Conversely, using the highest wattage setting and longest time resulted in a temperature change of 11.7°C, producing an overall temperature of 48.7 °C that was potentially harmful to osseointegration[40]. Kreisler looked at implant-bone temperature changes during a simulated surface decontamination of implants using an Er:YAG laser. Titanium plasma-sprayed, sandblasted acid-etched, and hydroxyapatite- coated dental implants were placed into porcine bone blocks, and immersed in 37°C water to simulate *in vivo* thermal conductivity. The 2,940 nanometer wavelength Er:YAG employed a 540 micrometer tip with the option for simultaneous water cooling. Results showed that at various depths, with or without water cooling, the Er:YAG did not exceed 47°C after 120 seconds of use[41]. Providing additional support for the safety of laser therapy, Monzavi examined different cooling systems associated with an Er:YAG laser. Implants were placed in resected sheep mandibles, irradiated with the laser, with either water and air spray, air spray alone, or no water and air spray. He found that water and air spray and air spray alone significantly limited temperature changes as compared to no cooling method at all, but even in the absence of cooling, the maximum temperature change was 10 °C[42]. A secondary area of concern in laser therapy involves the possibility of implant surface alterations during treatment. A study by Kilinc evaluated the interaction of laser energy with eight different dental materials, including titanium. Using a CO_2, Nd:YAG and diode laser with equivalent power, laser energy was directed towards the various materials at a 45° angle for 30 seconds. After high-power scanning electron microscope (SEM)

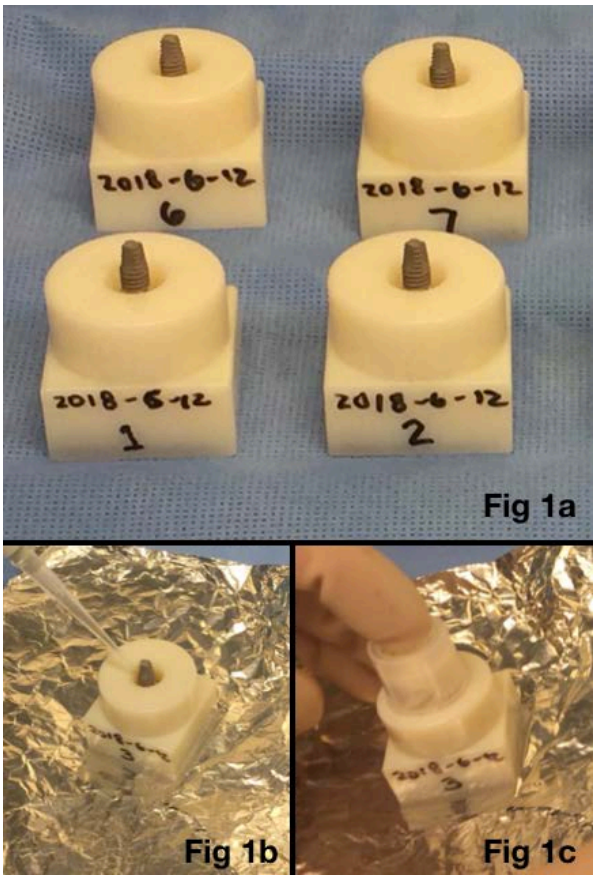
evaluation, the author determined that the Nd:YAG laser adversely affected titanium, all metallic materials, and tooth-like structures[43]. Similarly, Romanos showed that the Nd:YAG laser caused significant damage to titanium disk surfaces when analyzed by SEM[44]. However, despite the findings of Kilinc and Romanos, it is important to note the reported angle of 45^{o} to the long axis of the surface. Such an exaggerated angle is in direct conflict with the recommended parallel orientation of the laser energy.

Given the established success of air-powder abrasion in the decontamination of implant surfaces, and the potential for the Nd:YAG laser to perform in a similar capacity, the aim of this *in vitro* study is to compare the air-powder abrasion system to the Nd:YAG laser in the ability to eradicate *Streptococcus sanguinis* from roughened dental implant surfaces.

CHAPTER III: MATERIALS AND METHODS

Mirroring the methodology of Quintero, additive manufacturing technology (Objet500 Connex, Stratasys Inc, Eden Prairie, MN) was used to fabricate resin (RenShape Huntsman 7810) peri-implantitis models[32]. The 3-dimensional defect models (6.5mm depth x 10.5mm width) contained a centrally placed cylindrical well (350mL volume) to hold one dental implant. Fifty-six external hex OSSEOTITE® Biomet 3i™ (Biomet 3i, Palm Beach Gardens, FL, USA) implants (5mm width) were randomly divided into the following groups: positive control (n=4), negative control (n=2), air-abrasion (n=10), and Nd:YAG laser (n=40). All components were sterilized in advance. Impression copings were inserted into the base of each defect model, individually packaged for sterilization, and exposed to ethylene oxide (50°C, 16 hrs). Screws and drivers were also packaged and sterilized in an autoclave (270°F, 42psi, 15 mins). *Streptococcus sanguinis* (American Type Culture Collection, #10556, Manassas, VA, USA) was sub-cultured in Brain Heart Infusion (BHI) (Hardy Dynamics, VWR Scientific, Radnor, PA, USA) broth and incubated (37°C, 5% CO_2, 95% humidity) for 24 hours. A contamination media suspension was prepared by transferring 0.9mL of the BHI culture into 44.1mL of fresh, pre-warmed Biofilm Media and incubated under the same conditions. Bacterial growth was monitored using spectrophotometry (Genesys 10S UV-VIS, Thermo Fisher, Waltham, MA, USA). Upon reaching an optical density of 0.5 at 600nm, growth was inhibited by placing the suspension on ice, yielding a concentration of $1.5x10^8$ colony forming units (CFU) per milliliter.

Just before contamination, the sterile components were assembled under a clean safety hood using aseptic technique. Implants were removed from their industry packaging, positioned in the well onto the inverted coping, and secured with a screw to create a leak-proof seal (Fig. 1a). Fifty sterile, experimental implant/coping/defect (ICD) models were inoculated by transferring 350mL of contamination media into the model well, immersing the coronal two-thirds of the implant (Fig. 1b). The apical one-third remained uncontaminated and later served as a convenient handle to manipulate the implant (Fig. 1b). Test tube caps containing sterile gauze saturated with BHI were positioned over the ICD models to reduce media evaporation (Fig 1c). The fully assembled models were individually wrapped in sterile aluminum foil and incubated (37°C, 5% CO_2, 95% humidity) for 24 hours.



Following the incubation period, any remaining contamination media was carefully suctioned (Fig 2a), and the implants were immediately decontaminated.

Ten experimental ICD models were disinfected using air-powder abrasion (Cavitron JET Plus™, York, PA, USA). The tip of the

air-powder abrasion instrument was maneuvered around the entire implant for 60 seconds, delivering amorphous silica sodium bicarbonate and water at 20-40psi (Fig 2b). Ten experimental ICD models were disinfected using Nd:YAG laser (Millenium Dental Technologies Periolase® MVP-7™, Cerritos, CA, USA). The laser unit was activated (150 µsec pulse duration, 200mJ per pulse, 20Hz repetition rate) and the energy was delivered through a 360-micrometer fiber. The laser beam was directed parallel to the long axis of the implant and moved in a slow, circumferential motion from the apex to implant platform over 20 seconds (Fig 2c).

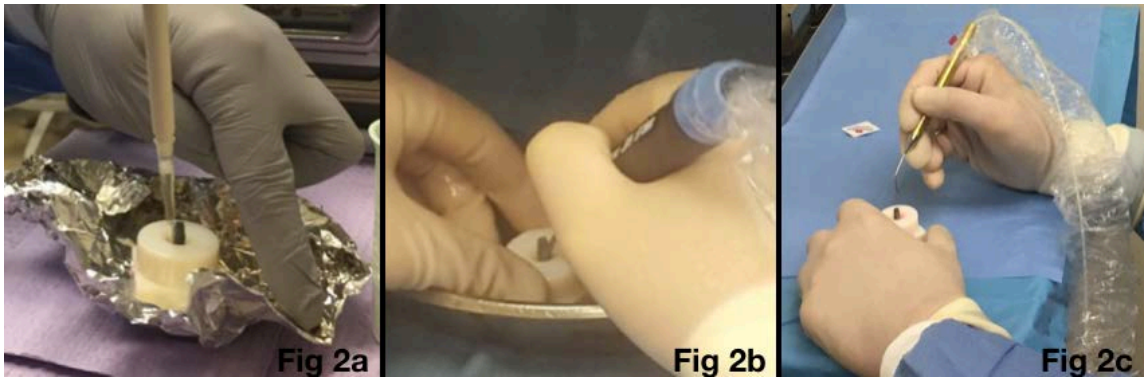


Fig 2a

Fig 2b

Fig 2c

Serving as positive controls, four additional ICD models were prepared, contaminated, but not disinfected. Two ICD models were used as negative controls. They were prepared similarly with two modifications:

1.) Inoculation with 350mL of sterile BHI

2.) Not subjected to a disinfection procedure

Following decontamination, sterilized hemostats were used to grasp the apical third of all experimental and control implants to detach them from the associated resin model. The individual implants were rinsed with sterile saline for 15 seconds to remove any extraneous material, placed into a 15mL centrifuge tube containing 2mL of lactated

Ringer's solution, and vortexed (60 secs, 1000 rpm) in a multi-tube vortex mixer (Fisher Scientific, Waltham, MA, USA). Immediately after vortexing, lactated Ringer's solution was used to prepare 1:10, 1:100, and 1:500 dilutions. For each dilution, three 10uL aliquots of the inoculum were added to the upper part of the BHI agar plate. The plates were then tilted at a 45° angle, allowing downward flow of the inoculum to create three bacterial streaks (Fig 3).

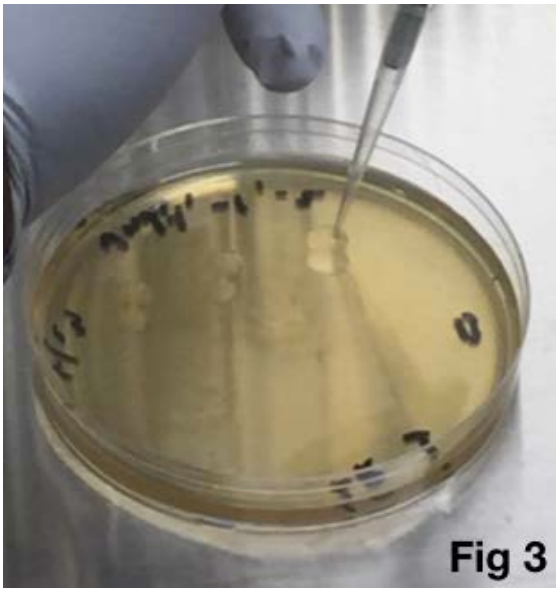


Fig 3

All plates were incubated (37°C, 5% CO_2, 95% humidity) for 24 hours. Following incubation, the plates were randomized, and CFU's were recorded using an automated counter (Scienceware® Colony Counter®, Bel-Art Products, Wayne, NJ, USA).

CHAPTER IV: RESULTS

Data analysis was based upon CFU count. A preliminary review of the data revealed 3 extreme outliers in the Nd:YAG group, prompting a modification to the study in an effort to increase overall power and determine validity of the preliminary data. The modification increased the number of Nd:YAG experimental ICD models to a total of 40, and two additional ICD models were employed as positive controls. In summary, the experimental data was derived from the following groups: 40 Nd:YAG ICD models , 10 air-powder abrasion models, 6 positive and 2 negative control models.

Table 1. Bacterial Growth Relative to Decontamination Method

Modality	Positive Control	Air-Powder Abrasion	Laser	Negative Control
Minimum CFU	1.4×10^5	0	0	0
Maximum CFU	2.9×10^5	3.0×10^2	8.0×10^3	0
Mean CFU	1.9×10^5	1.4×10^2	2.1×10^2	0

Data is presented in Table 1. As expected, none of the negative controls exhibited growth, while all positive controls displayed substantial bacterial growth. Positive controls had a mean of 1.9×10^5 CFU, thus providing validation for the implant contamination methodology. Implant decontamination in both treatment groups resulted in a 1.0×10^3 reduction in bacterial growth versus positive controls.

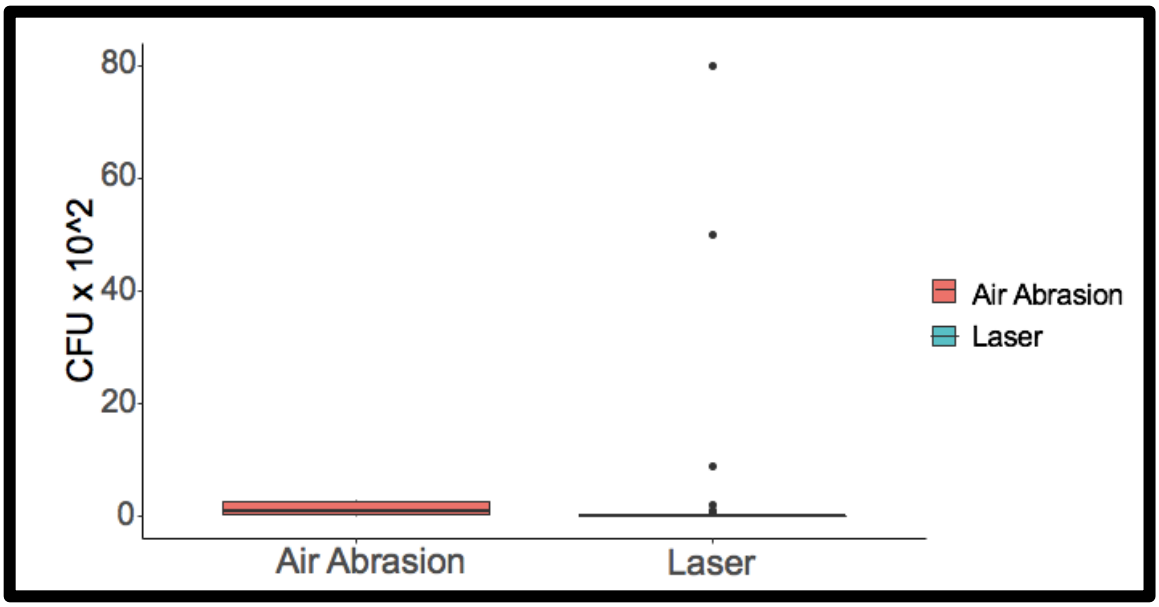


Fig 4. CFU's per disinfection modality including outliers

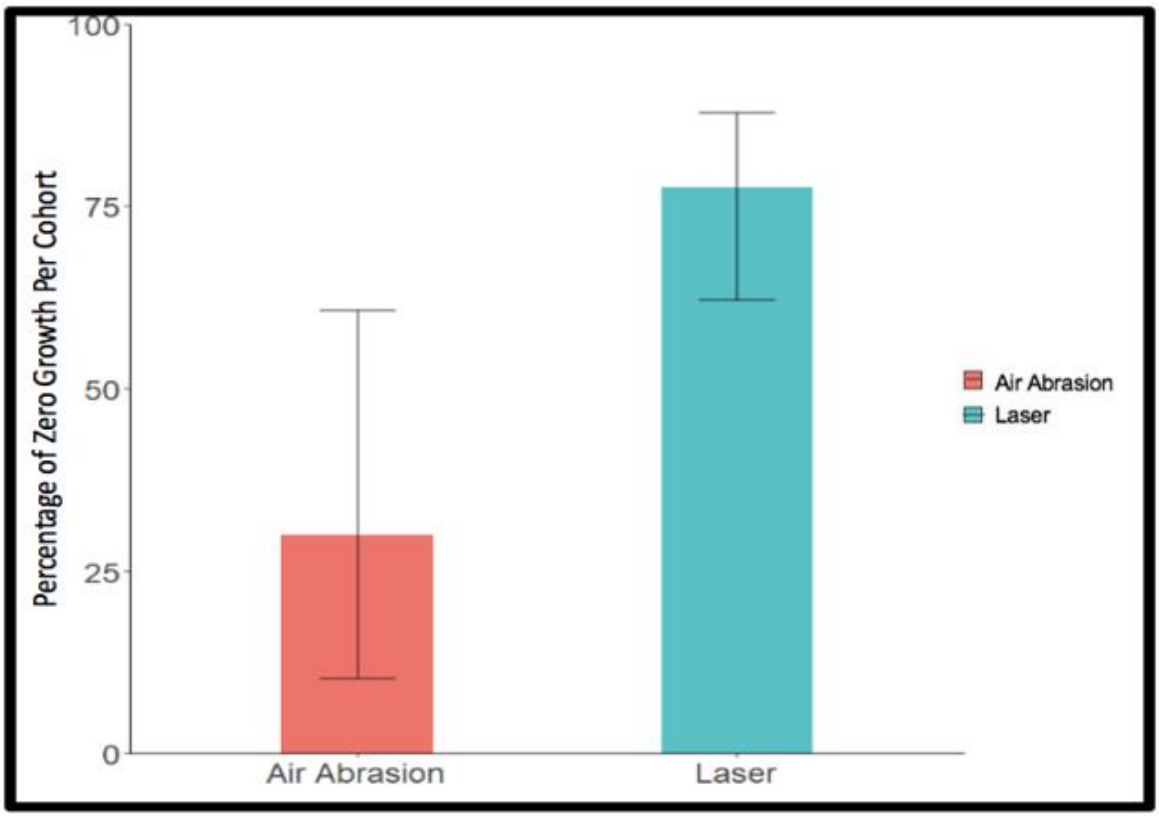


Fig 5. Percentage of zero growth per cohort

A difference in decontamination capabilities between the air-powder abrasion unit and Nd:YAG laser was observed in the number of plates displaying a total absence of bacterial growth. As seen in Figure 5, a Mann-Whitney U Test indicated a significant difference ($p=0.0028$) when comparing the percentage of Nd:YAG laser plates with zero growth, 78% (95% CI:62%-88%), to the percentage of air-powder abrasion plates with zero growth 27% (95% CI: 10%-61%).

CHAPTER V: CONCLUSION

This investigation compared air-powder abrasion to an Nd:YAG laser in the ability to decontaminate roughened dental implant surfaces. Considering that there is no universally accepted regimen for the treatment of peri-implantitis, determining the most conservative, cost-effective method is necessary. The results of this *in vitro* study revealed that, although both the air-powder abrasion unit and the laser significantly reduced bacterial CFU's, the Nd:YAG laser showed a significantly greater ability to eliminate 100% of bacteria from the implant surface. Seventy-eight percent of laser treated plates had zero growth while only 27% of the air-powder abrasion plates displayed zero growth ($p=0.0028$).

Interestingly, early results with the laser-treated ICD models showed a few "extreme" outliers. The authors theorize that technique sensitivity and operator experience with the laser may have played a role. A critical step required for the delivery of consistent laser energy involves cleaving the fiber. While the laser unit has a calibration meter to allow for confirmation of proper cleaving and ultimate power output prior to energy delivery, the inherent variability may have contributed to the unexpected data points. As cleaving efficiency and familiarity with the laser unit increased, total elimination of viable bacteria peaked at 78%, but the authors suspect that the trend would have continued towards 100% with additional Nd:YAG test units. A possible shortcoming of the experimental protocol was the use of a non-pathogenic bacterium for contamination of ICD models. While the use of a known periodontal

pathogen may have made the *in vitro* model better simulate clinical circumstances, *Streptococcus sanguinis* has been validated in previous publications[45-47].

Given the historical success of air-powder abrasion and the success of both air-abrasives and laser therapy in this study, it is important to review the advantages and disadvantages from the end-user's perspective. Cost and specialized training are two important considerations for laser therapy. The retail cost for the Nd:YAG laser varies by manufacturer, but in this study, the air-powder abrasion unit was approximately 20 times cheaper than the laser. As with most advances in technology, additional training for the provider and the dental team is essential for patient safety and success. While both modalities utilized for decontamination in this study require baseline training, the inherent risk of laser irradiation in patient care warrants more attention. Furthermore, air-powder abrasion units, such as the Cavitron JET Plus™, are more commonplace in dental practice as part of the dental hygiene armamentarium. Advantages of laser therapy, within the context of this in vitro study, include less treatment time, better visualization of the field that is free of abrasive paste slurry, and potentially, more complete eradication of bacteria. Lastly, and possibly of substantial consideration, is the FDA-approved claim of the Periolase® MVP-7™ manufacturer that advertises true regeneration as a treatment outcome[38, 46, 48-51].

Within the confines of this *in vitro* study, Nd:YAG laser was nearly 3 times more effective in total kill of *Streptococcus sanguinis*, cultured on roughened dental implant surfaces in a simulated model of peri-implantitis. However, both modalities, air-powder abrasion and Nd:YAG laser treatment, significantly reduced viable bacteria counts as

compared to positive controls. Future clinical research is needed to validate the Nd:YAG laser as an independent option for the treatment of peri-implant disease.

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