IAOMT ACCREDITATION--
Checklist for Completing Unit 8:
Biological Periodontal Therapy

INTRODUCTION TO UNIT 8

☐ Take the Unit 8 Pre-test. Click here to go to pages 3-4.

☐ Read the “Periodontal Diseases” fact sheet from the National Institutes of Health. Click here to go to pages 5-6.

REQUIRED (MANDATORY) CONTENT OF UNIT 8

☐ Read the “The Biologic Concept for the Management and Treatment of Periodontal and Soft Tissues of the Oral Cavity” article by Mollica and Just. Click here to go to pages 7-17.

☐ Read the “Periodontal Disease and Overall Health: An Update” review by Balakesavan, Gokhale, Deshmukh, and Williams. Click here to go to pages 18-24.

☐ View the IAOMT online learning module “Biological Periodontics” at https://iaomt.org/online-learning/biological_periodontics/
Click here to go to page 25.

☐ Read the “Five-year Retrospective Study of Laser-Assisted Periodontal Therapy” from Kusek, Kusek, and Kusek. Click here to go to pages 26-29.

☐ Read the “Ozone and its Use in Periodontal Treatment” study from Iliadis and Millar. Click here to go to pages 30-35.

Continued on next page...
TEST FOR UNIT 8

☐ Take the Post-Test for Unit 8 at https://www.cvent.com/d/yvq547. Click here to go to page 36.

☐ If you are interested in learning more about any of the topics in this unit, explore the readings in the OPTIONAL Unit 8 PDF file. Note that these are not required materials.

☐ Continue on to Unit 9! Click here to go to https://iaomt.org/accreditation-materials/.
PRE-TEST FOR UNIT 8 TO BE TAKEN BEFORE STUDYING BIOLOGICAL PERIODONTAL THERAPY

*This is a pre-test, and the results are for your records only. You are not expected to know the answers since you have not studied this material yet. The pre-test is simply designed to assist you in recognizing some of the important information that will be presented in this unit. There is no time limit for this test. Choose the option that BEST answers each question.

1. __________ are frequently found in severe cases of gum disease.
   A. one cell animals
   B. animals with more than one cell
   C. A & B
   D. none of the above

2. Research has linked periodontitis to __________.
   A. cardiovascular disease
   B. rheumatoid arthritis
   C. low birth weight and premature births
   D. A & B
   E. all of the above

3. The goal of biocompatible periodontal therapy is to eliminate __________.
   A. tooth structure
   B. infection
   C. antibiotics
   D. surgery
   E. all of the above

4. __________ is the only practical chairside method of determining the presence of a wide range of putative periodontal pathogens.
   A. diagnostic testing for systemic diseases
   B. surgically removing pieces of the infected gum
   C. phase contrast microscopy
   D. administering antibiotics
   E. none of the above
5. The following measure is NOT generally recommended as part of subsequent appointments for biocompatible periodontal therapy:
   A. Pre-scaling rinse with an antimicrobial agent to reduce contaminated aerosols & general microbial loads.
   B. Definitive quadrant scaling.
   C. Subgingival irrigation with antimicrobial agents of all quadrants during each quadrant appointment.
   D. Re-evaluation of the effectiveness and compliance with home care measures via phase microscopy.
   E. Surgical procedures to remove infected areas.

Periodontal diseases are disorders of the gums, or gingiva, and other tissues around the teeth. Periodontal diseases vary in severity, from the reversible, recurring mild inflammation called gingivitis that affects many people, to the sometimes irreversible, severe, chronic periodontitis that badly erodes the bone and other supporting structures of the tooth, possibly leading to tooth loss. An estimated 8 to 10 percent of American adults have some form of periodontal disease. Smoking contributes significantly to the risk of having periodontitis. The risk is also higher in individuals with diabetes.

Yesterday

- In the 1950s, tooth loss was extremely common, largely because of rampant tooth decay and untreated periodontal diseases.

- The primary research focus was on oral bacteria. Periodontal diseases were thought to begin when chalky white deposits called calculus accumulated near the gingiva, along the base of the tooth. Many believed it served as an irritant and wedge that opened a small pocket between the tooth and gingiva, allowing bacteria to freely enter and progressively erode the bone and the other supporting structures of the tooth.

- Periodontal disease was viewed as a linear process that started with gingivitis progressing to loosening of teeth and loss of bone holding the teeth. Without proper treatment, people were told their gingivitis would inevitably progress to periodontitis, advanced disease, and ultimately tooth loss. All people were thought to be susceptible to severe periodontitis, especially as they aged.

Today

- The most recent survey of the nation’s oral health, released in 2005 (National Health and Nutrition Examination Survey) (www.cdc.gov/nchs/nhanes), showed a continued decline in periodontal disease among American adults and an associated reduction in tooth loss. According to the survey, Americans age 20 and older have on average about 24 of their natural teeth. Without research on the causes and treatment of periodontal disease, that number would be much lower.

- The fundamental role of the immune system in causing periodontal diseases was largely overlooked just a generation ago. Research has established that periodontal diseases arise when specific oral bacteria infect gum tissue, triggering a complex immune response and progressive inflammation that play a major role in causing periodontitis.

- Periodontal diseases are no longer viewed as an inevitable result of aging. Even though moderate disease affects a majority of adults, severe periodontitis affects only five to 15 percent of adults.

- Some individuals appear to be more susceptible to severe periodontitis than other people. Scientists are making progress to understand how a person’s genes and environment make him or her more likely to have advanced forms of periodontal disease. (For example, a study entitled “Molecular and population genetics of periodontal pathogens” is in progress in 2010.)

- Gingivitis and more severe periodontal diseases are recognized as distinct conditions. Researchers know that gingivitis does not necessarily lead to severe disease and tooth loss.

- This greater understanding has helped dentists provide better treatment for their patients. In addition to improved consumer dental products to help prevent periodontal disease, increased awareness exists about those at greatest risk who might benefit from more regular periodontal care. These include smokers, people with diabetes, and those taking certain types of anti-seizure medications, cancer drugs, oral contraceptives, and some calcium channel blockers. For example, a study entitled “Neutrophils and periodontitis in diabetes” is currently underway.
Tomorrow

- Scientists now know that the bacteria in our mouths exist as a complex, multi-layered community termed oral biofilm. Scientists already are in the process of dissecting the dynamics of these bacterial communities. This research may give dentists the tools to target their treatment specifically to the bacteria that trigger periodontal disease. At the same time, because biofilms form throughout the body and nature, research advances may have broad applications in medicine and environmental studies.

- For those who develop advanced periodontal disease, researchers are working to regenerate the damaged or lost bone and restore the tooth support to its natural state.

- Oral bacteria shed from chronic periodontal infections enter the circulatory system and may contribute to diseases of the heart and other organs. The role of periodontal diseases in causing or contributing to other serious conditions is the subject of ongoing laboratory and clinical research. As this research unfolds in the coming years, it may be that a trip to the dentist not only could have benefits for your oral health but also help reduce your chances of developing related systemic conditions.

For additional information contact: NIDCR Office of Communications and Health Education at (301) 496-4261.

National Institute of Dental and Craniofacial Research (NIDCR):

www.nidcr.nih.gov
Understanding the biologic concept for the management and treatment of periodontal and associated soft tissue of the oral cavity is one of the cornerstones to overall health and wellness. When we study the human body, we tend to see bits and pieces so we may better understand ourselves. The reality is that the human body is but one complete system intricately woven into the web of life. Understanding this biologic concept gives us a better appreciation for the influences issues like periodontal disease can have on our unique ecosystem.

**THINKING IN A BIOLOGIC MANNER:**

The concept of the cell is, strictly speaking, only a morphological abstraction. Seen from a biologic viewpoint, a cell cannot be considered by itself without taking its environment into account.¹

Linear thinking in medicine, as well as dentistry, goes back to the time of Galileo and Virchow. The school of thought includes the premise that organisms are analogs to technical machines as cellular functional units with defects which must be repaired. In this fundamental concept, acute illness is viewed as cause and effect, drug to cell, lock and key, receptor, then a reaction. This can be clinically objectified as a drug kills a bug (cause) with a then immediate repair (effect). While such a concept might apply to acute illness, it does not apply to the ever-increasing rise in chronic disease and tumor.

In an article written for *The Physician Executive*, the authors stated, “Traditional medical training may actually impede a physician’s ability to solve complex problems because it is based on vertical, linear and traditional thinking.”²
The authors continue to explain that linear thinking is developed through an existing pattern that is methodical, stepwise, intolerant of uncertainty, restricted by relevant information, and with little interest in novel approaches.

So, what is the outcome or reward for depth of knowledge? Non-linear thinking allows for restructuring of an existing pattern, is multidirectional and creative, tolerant of uncertainty, unrestricted by relevant information, open, and welcoming of novel approaches. The resultant outcome and reward is depth and breadth of knowledge.

The significance of these particular thinking concepts lies in the essence of biologic systems. Biologic systems do not show linearity; they are highly interlinked and subject to a balance of biologic flow. Biologic systems are free to exchange energy and matter with their surroundings. The most important energy to give structure and organization to a biologic system is input and information. This information allows a multitude of local and far-ranging reactions and interactions within the system itself. Manifestations of chronic disease have many masks; therefore, symptomatology can be misleading. Thinking biologically, thereby understanding the biologic underpinnings and processes regardless of the “disease,” can give us new insight on treatment options and planning.

**UNIFICATION OF CELLS, TISSUES, ORGANS, AND BODY:**

Whether on the micro or macro level, the connectivity or oneness of a biologic system is critical to ultimately understanding the human body. We have been taught human anatomy in a rather linear way. We learn this way to understand the parts we are made of, such as the femur, temporalis muscle, mandible, and so forth. What is lost at times is how the pieces come together and better yet, actually work together. The human body as a biologic system does not understand itself as millions of little pieces but rather as one completely unified system.

What could be so pervasive, so unifying, as to have the ability to link the entire human body into one? The unifying system in the human body is
what Alfred Pischinger called the “Extracellular Matrix and Ground Regulation.” Every function and every process in the living body involves the matrix in one-way or the other. This matrix is the fabric that binds us together. The matrix allows nutrients, cytokines, hormones, vitamins, and oxygen, to name a few substances, to flow into the cell. At the same time, it allows waste products of cellular metabolism flowing out to this matrix milieu to be presented to the circulatory and lymphatic system for elimination.

All immune responses and tissue repair takes place through the matrix. The extracellular matrix permeates the extracellular spaces of the entire organism, reaching each cell and always acting as one unit. This autoregulating matrix is a complex, ordered aggregate composed of a number of different macromolecules whose structural integrity and functional compositions are important in maintaining normal tissue architecture, as well as in development and tissue specific function.

WE ARE NOT ALONE:

When we consider our biology and our ecology, we do not stand-alone. We are a huge petri dish, and on us, in us, and all over us are bacterial forms. This is not the time to run to the bathroom and wash with anti-bacterial soap or do a deep colonic. The bugs that we grow are mostly good, a symbiotic relationship that keeps us healthy and happy. The most common term today is what is called the human microbiome. The American Academy of Microbiology has defined it in basically two ways. The two definitions are based upon genetic and ecologic conditions:

**Genetic Definition:** Just as the entire collection of human genes is called the human genome, this definition of microbiome means the entire collection of genes found in all of the microbes is associated with a particular host. A broader term, “metagenome,” refers to the entire collection of microbial genes found in a particular environment. A “metagenome” may or may not be host-associated.

**Ecologic Definition:** Ecologists use the term “biome” to describe the collection of plants and animals that live in a particular environment. Thus, there are various terrestrial and aquatic biomes. In the case of humans, when microbiome is used in a ecologic sense, it refers to the
ecosystem made up of microbes within and on the human body – that is, the collection of microbes that live in the human “habitat.”

The microbiome of the oral cavity is a reflection of the entire human organism and has direct implications locally, as well as far reaching implications. It is estimated that about 1000 microbial species have been identified in the human mouth. Typically, an individual will host 100 to 200 species at any one time. This is an ecologic dynamic and will change at any time based upon such things as nutrition, hydration, and general health status.

Like the rest of the body, the oral cavity contains many microhabitats such as tooth surface, tongue, buccal mucosa, and periodontal pockets with its associated soft tissue. The oral cavity is an area that can host both anaerobic (oxygen hating) and aerobic (oxygen loving) bacteria. It is becoming clear that, dependent on the individual’s oral microbiome, this situation could directly promote a healthy or a disease-inducing environment, which can result in, for example, caries of dentition and/or periodontal disease. When a harmonic, well-functioning community of microbes exists that supports a balanced ecology, oral health is maintained. When an imbalance occurs due to a multitude of reasons, pathogenic forms can dominate the ecology with the resultant negative effects we see clinically as disease.

**Traditional View of Periodontal Disease**

75% of adults in the United States have gum disease, but only 60% know about the problem. Meanwhile, many dental and medical professionals are not aware of the latest research about gum problems. However, at least health care practitioners agree on the overall definition of what comprises the gum system itself. The periodontium refers to the gums and the structures that support them, which are connected to the nerve and blood supply and include the following:

- **Gingiva** - gums
- **Sulcus** - the space between the gum and the tooth
- **Cementum** - the root surface of the tooth that connects the tooth to the ligament and bone
- **Periodontal ligament** - the tissues that connect the tooth to the alveolar bone
- **Alveolar bone** - the bone that contains the tooth sockets

Mouths are known to harbor bacteria, and bacteria combines with mucus and other substances to form **plaque**. In regard to bacteria, there can be more than 100 billion microorganisms per mg of dental plaque.\(^9\) Harmless bacteria are mainly recognized as **gram positive aerobic bacteria**, and harmful bacteria are mainly recognized as **gram negative anaerobic bacteria**. Some plaque can be removed by oral hygiene such as brushing and flossing, but plaque that is not removed or is resistant to removal can accumulate to form **tartar**.

**Gingivitis** is a build-up of plaque and tartar which results in gums that sometimes exhibit no symptoms but in other cases can cause gums to bleed easily and make them red and swollen.

**Periodontitis** is more severe and can cause gums to recede, creating gaps or **pockets** that can become infected, especially due to food and other particles collecting in the pockets. It can also lead to destruction of tissue and bone. Symptoms of periodontal disease can include:\(^{10}\)

- Halitosis (bad breath)
- Uncomfortable bite
- Red, purple and/or swollen gums
- Sensitive teeth and gums, sometimes bleeding easily
- Foul taste in mouth
- Newly occurring spaces between teeth
- Formation of abscesses
- Pus between teeth and in gums
- Pain, especially during chewing
- Receding gum line
- Loose teeth

Risk factors for gum disease often accepted and acknowledged by most mainstream dental and medical communities consist of the following:\(^{11}\)

- Genetic susceptibility/heredity
- Gender (males are more likely to have gum disease)
- Hormonal changes in females, especially during pregnancy, just before menstruation, when using oral contraceptives, or after menopause
- Poor immunity and illnesses such as diabetes, cancer, and AIDS
- Certain medications, especially Fosamax for osteoporosis and bisphosphonate drugs for cancer patients
- Drug abuse
- Alcohol abuse
- Smoking
- Issues with dental restorations or bite
- Poor oral hygiene
- Unsatisfactory diet, especially consumption of sugar and acids
- Mouth breathing
- Older age
- Oral infection
- Oral injury
- Oral inflammation

Generally speaking, complications recognized as a result of gum disease are\textsuperscript{12}
- Loss of teeth
- Bad breath
- Cardiovascular issues including coronary heart disease and stroke
- Low birth weight and premature births to mothers with gum disease
- Problems for patients with diabetes
- Respiratory issues, including asthma
- Rheumatoid arthritis

**PERIODONTAL DISEASE AND THE ORAL SYSTEMIC LINK:**

Research has shown that periodontal disease is associated with several other diseases. For a long time, it was thought that bacteria was the factor that linked periodontal disease to other disease in the body; however, more recent research demonstrates that inflammation may be responsible for the association. Therefore, treating inflammation may not only help manage periodontal diseases but may also help with the management of other chronic inflammatory conditions.\textsuperscript{13}
Beyond the scope of what has already been generally accepted about periodontitis (such as its connection to cardiovascular disease\textsuperscript{14} and rheumatoid arthritis\textsuperscript{15}), recent studies continue to offer new, additional information that suggests disease in the gums can spread to other parts of the body and/or disease in other parts of the body can impact the gums.

A 2000 study by Li, Kolltveit, Tronstad, and Olsen published in *Clinical Microbiology Reviews* entitled “Systemic Diseases Caused by Oral Infection” examined the links between periodontal disease and cardiovascular disease, infective endocarditis, bacterial pneumonia, low birth weight, and diabetes mellitus.\textsuperscript{16} The researchers of this study specified that bacteria from the mouth could be passed to the bloodstream:

With normal oral health and dental care, only small numbers of mostly facultative bacterial species gain access to the bloodstream. However, with poor oral hygiene, the numbers of bacteria colonizing the teeth, especially supragingivally, could increase 2- to 10-fold\textsuperscript{17} and thus possibly introduce more bacteria into tissue and the bloodstream, leading to an increase in the prevalence and magnitude of bacteremia.\textsuperscript{18}

Other researchers have explored the association between oral infection and illness as well. Michaud, Joshipura, Giovannucci, and Fuchs\textsuperscript{19} concluded that men with gum disease, particularly those who lost teeth within the past four years, were more likely to develop pancreatic cancer.\textsuperscript{20} Slots identified that herpes viruses, especially human cytomegalovirus (HCMV) and Epstein Barr virus (EBV), appear to have roles in gum disease.\textsuperscript{21}

Also of note is that researchers Shimauchi and Ogawa demonstrated in a 2007 study that *Porphyromonas gingivalis* (Pg), a gram-negative anaerobic bacteria recognized for its role in chronic periodontitis, “may be able to adapt to the local immune defense, contributing to the connection between systemic and periodontal disease.”\textsuperscript{22}
Knowing that periodontitis can impact corrosion of amalgam fillings,\textsuperscript{23} gum disease could also be associated with a number of mercury-related illnesses. Additionally, researchers have warned about harmful consequences of infections from root canals that in turn impact the periodontium.\textsuperscript{24}

Furthermore, ten species of bacteria have been found to serve as pathogens for periodontitis, including \textit{Actinobacillus actinomycetemcomitans}, \textit{Porphyromonas gingivalis}, and \textit{Bacteroides forsythus}.\textsuperscript{25} Some researchers have established that pathogen and host-response biomarkers can be correlated with periodontal disease.\textsuperscript{26} Periodontal pathogens \textit{Treponemas} and \textit{Borrelia burgdorferi} were identified in Alzheimer’s disease cases by Miklossy in 2011.\textsuperscript{27} However, this concept is far from new. For nearly a century, light microscopy has been applied to locating the parasite \textit{Entamoeba gingivalis} in diseased gums.\textsuperscript{28}

Treatments commonly practiced in popular, modern dentistry such as root planing and surgery do not seem to address the current science or even the past practices from one hundred years ago that recognize the role of pathogens and inflammation in periodontal disease. In fact, by removing parts of the body and/or making a number of surgical incisions, the disease could potentially be worsened and spread to other parts of the body, and different parts of the periodontium could be damaged as well.

The goal of biologic based periodontal therapy is for the Biologic Dentist to integrate safe and effective therapies to support positive ecologic changes in the oral cavity. This is done by elimination of pathogenic microbial forms and by supporting the patient’s own inherent healing process, not just eliminating the tooth structure.\textsuperscript{29}

The biological dentist uses the least invasive treatment possible for periodontal disease, and s/he does this by removing the smallest amount of tissue required, applying non-toxic antimicrobial agents to reduce pathogens, utilizing precise and definitive scaling, irrigating subgingivally, and cooperating with the patient to evaluate lifestyle choices.
These more health-focused measures offer the patient’s immune system a better opportunity to fight off the infection in the gums. Antibiotics are used only if necessary, and surgery is used only as a last resort. Healing, of the entire body, is the first priority.

5 Mollica P. Pischinger Memorial Lecture, American College of Integrative Medicine and Dentistry, School of Integrative Biologic Dental Medicine. Saddle Brook, New Jersey. 2010.
8 This list was compiled from various sources including
10 This list was compiled from various sources including


• Mayo Clinic. Diseases and conditions: periodontitis. Mayo Clinic website. 


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Periodontal disease and overall health: An update

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ABSTRACT

Periodontitis is a chronic inflammatory disease caused by a mixed microbial infection. The disease is a result of a complex interaction between the bacteria and the susceptible host. The host reaction to the microbial flora leads to a release of pro-inflammatory cytokines and a low grade inflammatory response that has an effect on the overall health status of the patient. Periodontitis has been associated with several systemic conditions such as cardiovascular disease, diabetes, adverse pregnancy outcomes and respiratory infections. In recent years, the relationship of periodontitis to overall health has been explored by numerous researchers who have expanded our understanding of periodontal disease as it affects the overall health of human subjects. This article further examines the relationship of periodontitis to overall health and throws a light on recent associations.

Key words
Anemia, infertility, neoplasms, periodontitis, periodontitis-systemic disease interactions

INTRODUCTION

Periodontal disease is one of the most common diseases of humans and is one of the main causes of tooth loss in adults. Well conducted studies have shown that periodontal disease is significantly associated with certain systemic diseases such as cardiovascular disease,[1,2] diabetes,[3‑5] adverse pregnancy outcomes[6] and respiratory infections.[7] The periodontal disease - systemic disease relationship is supported by over 20 years of studies. The association of periodontal disease with several systemic conditions such as diabetes and atherosclerotic disease is reported to be related to the inflammatory response of periodontal disease. C-reactive protein is an important marker of the inflammatory response and is elevated in subjects with periodontal disease; its levels in peripheral blood are reduced when periodontal disease is treated. Another indication of the systemic inflammatory response associated with periodontal disease is the presence of cytokines, including tumor necrosis factor alpha and interleukins 1 and 6, often found in the circulation of patients with periodontal disease.[8] There are other conditions that also contribute to a systemic inflammatory response including obesity, psoriasis and rheumatoid arthritis along with periodontal disease.[9‑11]

This two way relationship of periodontitis and chronic systemic diseases in turn increase the systemic inflammatory load. This report further examines evidence for the relationship between periodontal disease and overall health.

PERIODONTITIS AND INFERTILITY

Infertility, which affects 15% of all couples worldwide, is defined as the failure of a couple to conceive after one year of unprotected intercourse (WHO 1999). In about half of all cases, infertility is attributed to the male partner (male factor) and results from poor sperm quality. Male factor includes one or more of the following: Low sperm production (oligozoospermia), poor sperm motility (asthenozoospermia) or abnormal sperm morphology (teratozoospermia) Guzick et al.[12] In many cases, infertility of the male partner can be traced to overt infections, immunologic problems, hormonal imbalance, anatomic defects, ejaculatory failures and environmental exposure. In about half of the cases, however, the aetiology of male infertility remains unexplained.

Bieniek and Riedel (1993) published a case series suggesting a direct causal relationship between dental bacterial infections (foci) and therapy-resistant
bacteriospermia, perhaps leading to sub-fertility.\textsuperscript{[13]} They implied that bacteriospermia might be initiated from oral foci of infection spreading through the circulation. Presence of various cytokines, namely tumour necrosis factor alpha (TNF-α), interferon gamma (IFN-γ) and interleukin 1 beta (IL-1β) were shown, in seminal plasma of fertile, infertile and immunoinfertile men using specific immunoradiometric assays. Presence of IL-6 may be associated with infertility and may be of importance in the specific diagnosis and treatment of male\textsuperscript{[14-17]} or female\textsuperscript{[18]} infertility.

Klinger A \textit{et al.}, studied a possible relationship between infertility and the presence of periodontal disease.\textsuperscript{[19]} They conducted a study to examine the association between fertility parameters and the periodontal status of men attending a fertility and in \textit{vitro} fertilization (IVF) clinic. The investigators found a positive correlation between deep periodontal pockets and sperm sub-motility. It can be postulated that bacteriospermia and cytokine production due to periodontal disease which acts as a foci of infection can increase the risk for male infertility.\textsuperscript{[18]} However, it might also be plausible that periodontal disease is merely a risk indicator for infertility, arising from common exposures such as environmental, nutritional, stress-related, behavior oriented or genetic. This may point to a possible association between male infertility, diminished semen quality and periodontal infection in men attending fertility and IVF clinics. However, due to the small sample size and less than ideal statistical methods used, these findings need to be interpreted with caution within the context of the study.

Hart reported that the negative effect of periodontal disease on conception was of the same order of magnitude as the effect of obesity.\textsuperscript{[20]} This was the first report to suggest that periodontal disease might be one of several factors that could be modified to improve the chances of pregnancy. The researchers followed a group 3737 pregnant women who were taking part in a Western Australian study, the SMILE study,\textsuperscript{[20]} and analyzed information on pregnancy planning and pregnancy outcomes for 3416 of them. The information about time of conception was available for 1,956 women and of these 146 women took longer than 12 months to conceive. Periodontal disease was found to be a modifiable risk factor for impaired fertility in women.

Collectively, the evidence suggests that periodontal disease gives rise to foci of infection which leads to the increase in pro-inflammatory cytokines. This leads to increased risk of infertility in male patients and adverse pregnancy outcomes in female patients.

**ANEMIA AND PERIODONTITIS**

Anemia is one of the most common global public health problems in developed as well as developing countries. Worldwide, anemia affects 1.62 billion people, representing 24.8% of the total population.\textsuperscript{[21]} Anemia of Chronic Disease (ACD) is the most common form of anemia observed in clinical medicine.\textsuperscript{[22,23]} ACD is defined as the anemia occurring in chronic infections, inflammatory conditions or a neoplastic disorder that is not due to narrow deficiencies or other diseases, and occurring despite the presence of adequate iron stores and vitamins.\textsuperscript{[24,25]}

The possible explanation for ACD was postulated by Cartwright in 1966.\textsuperscript{[26]} He reported that at least three pathologic processes are involved in ACD: Shortened erythrocyte survival, failure of the bone marrow to increase red blood cell (RBC) production to compensate for this increased demand, and impaired release of iron from the reticuloendothelial system. The pro-inflammatory cytokines such as interleukin 1 (IL- 1), IL- 6, and tumor necrosis factor–alpha (TNF α) are thought to act as mediators in suppressing erythropoiesis from the bone marrow leading to anemia.

The association of anemia and periodontitis has been explored since the early 20\textsuperscript{th} century. Early reports suggested that anemia was a cause of destructive periodontitis and not a consequence of it. Lainson \textit{et al.}, implicated anemia as a systemic cause of periodontitis.\textsuperscript{[27]} Chawla \textit{et al.}, suggested that anemia is an important factor in the etiology or pathogenesis of periodontal disease.\textsuperscript{[28]} On the other side, Siegel \textit{et al.}, reported a depression in the number of erythrocytes apparently secondary to the presence of periodontal disease.\textsuperscript{[29]} Hutter \textit{et al.}, evaluated the blood parameters in patients with chronic periodontitis and concluded that these patients show signs of anemia.\textsuperscript{[30]} Gokhale \textit{et al.}, performed a cross sectional study which included a total of 60 systemically healthy male patients.\textsuperscript{[31]} Red blood cell parameters were evaluated from the peripheral blood samples. They concluded that patients suffering from chronic periodontitis have a statistically significant lower number of erythrocytes and lower hemoglobin as compared to healthy controls.

A recent interventional trial by Pradeep \textit{et al.} supports the earlier findings regarding signs of anemia in patients with chronic periodontitis.\textsuperscript{[32]} They treated 60 chronic periodontitis patients, who had lower hemoglobin levels, with non-surgical periodontal therapy. After a follow up of 6 m, they found a statistically significant improvement in the red blood cell parameters. This finding presents early clues that treating periodontal disease may be beneficial in the management of anemia and suggest a role for periodontal disease in ACD.

**PERIODONTITIS AND CHRONIC KIDNEY DISEASE**

The number of patients with chronic kidney disease (CKD) is growing and is projected to rise in the future. With this...
in mind, it is important to understand the relationship of CKD and periodontal disease. CKD is associated with many physiologic changes that might contribute to the development of periodontal disease [Figure 1]. These include xerostomia, decreased salivary pH levels and decreased mineralization of enamel.[33] Additionally, some of the medications commonly prescribed to CKD patients may increase the risk of developing periodontal disease. Periodontal disease may represent a modifiable contributor to the already high inflammatory burden in patients with CKD, especially in those with diabetes. Treatment of periodontal disease in these patients could decrease the overall chronic inflammatory burden and its sequel.

Borawski et al. compared the periodontal status of three groups of adult CKD patients: (i) undergoing maintenance hemodialysis (HD), (ii) treated with continuous ambulatory peritoneal dialysis (CAPD) and (iii) pre-dialysis CKD patients.[34] The results were related to those obtained in generally healthy individuals with advanced periodontitis requiring specialized treatment and in general population subjects. This study showed a marked level of periodontitis in adult CKD patients compared with the general population. The disease is particularly severe in maintenance HD patients and comparable to its full-symptomatic form in subjects requiring specialized treatment. The results also indicate that periodontal disease is less severe in CAPD patients and moderate in pre-dialysis CKD subjects. In summary, periodontal disease is prevalent, severe and under recognized in CKD patients.

Fischer et al., assessed the potential bidirectional relationship between CKD and periodontal disease and potential mediators of this relationship using structural equation models.[35] The direct effect of one factor (periodontal disease) on the outcome (CKD) while simultaneously controlling for direct effects of many other factors (diabetes, hypertension, socio-economic status etc.) were studied.

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**Figure 1:** Hypothetical structural equation models outlining 4 possible relationships between CKD and periodontal disease. Not shown are relevant potential risk factors of diabetes e.g., obesity, hypertension and race/ethnicity.
Models A, C and D appear to be the most plausible models. Each model suggests that a bidirectional relationship may exist between CKD and periodontal disease. Periodontal disease impacts CKD directly; CKD impacts periodontal disease directly; and periodontal disease indirectly affects CKD through diabetes duration and hypertension.

Results from models A, C and D also suggest a bidirectional relationship between periodontal disease and diabetes duration. These findings support direct relationship of diabetes and CKD (Model D) and that diabetes impacts CKD indirectly through periodontal disease (Model B) and hypertension.

Fisher et al. investigated the association between chronic kidney disease and clinical measures and serologic markers of periodontal infection in 4,053 patients. Nine percent of the study population had chronic kidney disease, 22% had high A. actinomycetemcomitans antibody titer, 24% had high P. gingivalis antibody titer, 9% had periodontal disease and 17% were edentulous. After simultaneously adjusting for recognized risk factors, adults with a high A. actinomycetemcomitans titer were less likely to have chronic kidney disease, suggesting a composite systemic antibody response to clusters of periodontal pathogens which may be important. The authors concluded that the results support considering edentulism and low serum titer to A. actinomycetemcomitans as risk indicators for chronic kidney disease. The rationale for including edentulism in the analysis was based on the observation that periodontal disease is the major cause of edentulism among 40-to 69-year-olds, with 60.5% of teeth extracted due to periodontal disease, tooth loss was a marker of past periodontal disease among adults over 55 years of age, and non-surgical periodontal therapy was associated with a reduction in the rate of tooth loss.

In a cross sectional study Fisher et al. identified 12,947 adults 18 years or older for information for kidney function and at least one risk factor in the Third National Health and Nutrition Examination Survey (NHANES III). The main predictor was periodontal status. The prevalence of Chronic kidney disease and periodontal disease was 3.6% and 6.0% respectively; and the prevalence of edentulism was 10.5%. Adults with periodontal disease and edentulous adults were twice as likely to have chronic kidney disease. In summary, the findings support the conclusion that periodontal disease is a potential nontraditional risk factor associated with CKD.

Thus, the evidence suggests that periodontitis and CKD share a bidirectional relationship with direct and indirect effects on each other. Both the diseases lead to an increase in the systemic inflammatory load.

### PERIODONTITIS AND CANCER

With increasing attention being focused on oral/systemic interactions, studies have suggested that periodontal disease may be associated with increased cancer risk. Current understanding would indicate that tooth loss in older individuals may often be a result of periodontal disease.

In the first study in which the periodontium was assessed, Tezal et al. used a cross-sectional analysis of data obtained from the (NHANES III; National Center for Health Statistics 1994). Following adjustment for age, gender, race, ethnicity, education, tobacco use, alcohol consumption, and occupational hazard, clinical attachment loss was significantly associated with the presence of oral tumors (OR: 4.6; CI: 2.3-9.3). Additional analyses considering the interactions between clinical attachment levels (CAL) and smoking indicated that CAL was a significant risk for tumor (OR: 21.76; CI: 3.6-131.63) in current smokers, suggesting that it is a risk modifier. This concept is strengthened by the observation that CAL had no effect on tumor risk for former smokers or people who never smoked and hence, is probably not an independent risk factor.

Rosenquist et al., in a case control study of a Swedish population, alcohol consumption, tobacco use, oral hygiene, dental status and dental radiographic status were evaluated for increasing risk for oral cancer. Upon radiographic assessment, a high level of marginal bone was noted to have an increased risk for oral cancer in unadjusted analyses (OR: 3.00; CI: 1.0-8.7); however, this failed to reach significance in adjusted analyses. Regular dental check-ups were noted to be associated with a decreased risk of oral cancer in adjusted analyses (OR: 0.4; CI: 0.2-0.6).

In a subsequent study, Tezal et al. carried out a case-control study of pre-existing data for patients. Analyses following adjustments for the confounders of age, smoking habit, and number of missing teeth indicated that for every millimeter of alveolar bone loss, there was a 5.2-fold increase in the risk of tongue cancer (OR: 5.2; CI: 2.6-10.4). Other variables studied, including caries, restorations, and root canal treatment, failed to show any significant association with tongue cancer.

The most recent published study assessing the association between oral hygiene, periodontal disease, and oropharyngeal and oral cancer was a cross-sectional prospective case-control study. In this study Rezende et al. reported that advanced periodontal disease was greater in the subjects with oral and oropharyngeal cancer. Up to 76% of the cancer subjects had periodontal probing pockets greater than 6 mm compared to 20% of the patients without cancer. No statistically
significant differences could be found for caries, missing teeth, restorations, or prostheses.

In a well conducted study, Michaud et al. analyzed periodontal disease, tooth loss and cancer risk in a male health professional cohort.\(^{[45]}\) From this study, the five main cancers experienced by this cohort were colorectal, melanoma of the skin, lung, bladder, and prostate. Following adjustment for known cancer risk factors such as smoking history and diet, compared to individuals with no reported history of periodontal disease, individuals with a self reported history of periodontal disease demonstrated an increased risk for total cancer (HR: 1.14; CI: 1.07–1.22). For specific cancers, a past history of Periodontal disease was associated with increased risk for lung (HR: 1.36; CI: 1.15–1.60), kidney (HR: 1.49; CI: 1.12–1.97), pancreas (HR: 1.54; CI: 1.16–2.04) and hematological cancers (HR: 1.30; CI: 1.11–1.53). These findings for lung and pancreas were in agreement with previously published studies. The findings for kidney and hematological cancers were new and have not been reported previously. In contrast to previous studies, the association for esophageal cancer, while increased, was not significant after adjusting for smoking status.

Overall, the authors concluded that periodontal disease appeared to be associated with a small but nonetheless significant risk for cancer in general. Some influence of smoking was noted in smokers but the associations persisted in people who had never smoked. Whether some of these associations were due to direct effects of periodontal disease on cancer or the result of being more a surrogate marker requires further investigation.

The most widely quoted study is population-based and derived from data obtained from the NHANES I Epidemiologic Follow-up Study.\(^{[46]}\) The authors reported that an association between periodontitis and lung cancer, after adjustment for known risk factors, could be demonstrated. However, they cautioned that this periodontitis-cancer association could be spurious. Another published report in which periodontitis and lung cancer was studied does not support a link. In this study,\(^{[47]}\) associations between tooth loss and mortality patterns in a cohort from Glasgow were studied. The authors concluded there was no association between external causes of death and tooth loss as a continuous (HR: 0.97; CI: 0.92–1.03) or categorical variable for missing five to eight teeth (HR: 0.74; CI: 0.45–1.21) or missing nine or more teeth (HR: 0.89; CI: 0.42–1.88). In addition, no evidence of an association between lung cancer and tooth loss was found, with or without adjustment for smoking.

While the literature is scant on this topic, to date it does not seem to support any association between periodontal condition and lung cancer.

Pancreatic cancer is the fourth leading cause of cancer death in the U.S. It is an extremely difficult cancer to treat and little is known about what causes it. One established risk factor in pancreatic cancer is cigarette smoking; other links have been made to obesity, diabetes type 2 and insulin resistance. The oral cavity provides a gateway between the external environment and the gastrointestinal tract, and it facilitates both food ingestion and digestion. Oral hygiene and tooth loss can potentially affect gastrointestinal flora and nutritional status, and thus they have implications for the development of chronic diseases. The consequences of poor oral hygiene are dental plaque, periodontal disease, and tooth loss.\(^{[48]}\)

Stolzenberg-Solomon et al., hypothesized that tooth loss may be associated with pancreatic cancer.\(^{[49]}\) This study suggests a positive association between tooth loss and the development of pancreatic cancer but further studies were needed to fully evaluate the association between tooth loss and pancreatic cancer. Tooth loss that occurs through poor dental hygiene may be a marker for more deleterious gastrointestinal flora and, consequently, greater endogenous nitrosation.\(^{[50]}\) It has been estimated that 45%-75% of nitrosamine formation comes from endogenous formation by salivary and gastrointestinal bacteria converting nitrate to nitrite and nitrosamines,\(^{[51‑55]}\) with the rest coming from immunostimulation and macrophage response via intermediate production of nitric oxide.\(^{[56]}\) Endogenous formation of nitrosamines in the oral cavity in persons with poor oral hygiene is 8-fold that in persons with good oral hygiene.\(^{[50]}\)

Hujoel et al., in their study utilizing the NHANES I data to investigate the association between periodontitis and various cancers found no association for pancreatic cancer.\(^{[56]}\)

A subsequent study by Michaud et al., investigated the association of periodontitis in 216 males diagnosed with pancreatic cancer from a larger cohort of 48,375 men.\(^{[57]}\) This study provides the first strong evidence that periodontal disease may increase the risk of pancreatic cancer. The results showed that, after adjusting for age, smoking, diabetes, body mass index and a number of other factors, men with periodontal disease had a 63% higher risk of developing pancreatic cancer compared to those reporting no periodontal disease. The most convincing finding was that never-smokers (in subjects with periodontitis) had a two-fold increase in risk of pancreatic cancer.

A possible explanation for the results is that inflammation from periodontal disease may promote cancer of the pancreas.

Another explanation, according to Michaud, is that periodontal disease could lead to increased pancreatic...
carcinogenesis because individuals with periodontal disease have higher levels of oral bacteria and higher levels of nitrosamines, which are carcinogens, in their oral cavity. More research is needed to confirm and explore the role of inflammation in cancer.

PERIODONTITIS AND MORTALITY

Further prospects for a relationship between oral health and increased risk of total death and death from cancer have been made from a cohort study on rural Chinese. It was concluded that tooth loss (which occurs through poor dental hygiene) was significantly associated with increased risk for total death from cancer and from upper GI cancer.

Tramini et al., investigated tooth loss and associated factors in elderly patients in France who had been institutionalized long term. This was a cross-sectional study. The authors concluded that oral health can affect the general health of the patient which in turn can lead to increased risk of premature death.

Söder et al., published the results from a 16-year longitudinal study investigating periodontitis and premature death. It was concluded that young periodontitis patients with missing molars were at higher risk for premature death by life threatening diseases such as neoplasms than were their more healthy counterparts.

Ajwani et al., conducted a prospective study in 364 individuals in the age group above 75 years. They followed the patients for 5 years and found that after controlling for the common risk factors, periodontitis doubled the risk of cardiovascular related mortality.

SUMMARY

In recent years periodontitis has been linked to a number of systemic conditions. Substantial research indicates that oral health is an important component of general health, and individuals with periodontitis may be at risk for other diseases as well. The search for molecular mechanisms linking periodontitis to changes in systemic health has resulted in the evolution of a new era of multidisciplinary biomedical literature. However, an understanding about the effect of treatment of periodontal disease on systemic health is unclear. Well conducted intervention studies are required to substantiate the benefit of periodontal therapy in preventing or in reducing the risk of systemic disease. There is a need for a coherent approach by the dentists and the physicians to substantiate the two way relationship between oral and systemic disease. In this era of evidence based approach, further studies are required to close the gap between medicine and dentistry.

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YOU NOW NEED TO VISIT IAOMT’S “ONLINE LEARNING CENTER” TO WATCH THE BIOLOGICAL PERIODONTICS VIDEO AT https://iaomt.org/online-learning/biological_periodontics/.

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UPON COMPLETION OF THE “BIOLOGICAL PERIODONTICS” VIDEO, YOU WILL NEED TO CONTINUE WITH THE ADDITIONAL REQUIREMENTS FOR UNIT 8, WHICH INCLUDE MORE READINGS AND COMPLETING THE UNIT 8 TEST.
The use of dental lasers for the treatment of periodontal disease is accepted in some areas of dentistry, while in others it is thought to be antidotal therapy. This article seeks to show that laser-assisted periodontal therapy is a viable, noninvasive method for treating periodontal disease.

Periodontal disease is a chronic inflammatory disease caused by a bacterial infection. For this reason, the bactericidal and detoxifying effects of laser treatment are advantageous in periodontal therapy. The effectiveness of this therapy involves suppressing certain bacteria such as *Aggregatibacter actinomycetemcomitans*, an invasive bacterium associated with aggressive forms of periodontal disease that cannot be treated readily with conventional scaling and root planing (SRP). This bacterium is present on diseased root surfaces; as a result, it can invade the adjacent soft tissues as well, making removal by mechanical instrumentation difficult.

It is impossible to achieve success with traditional periodontal methods of treatment due to the great difficulty in terms of completely removing bacterial deposits and their endotoxins from deep areas of periodontal pockets. In addition, antibiotics that are used to prevent bacterial colonization after periodontal treatment help to increase the resistance of the microorganism.

According to the literature, using diode lasers in conjunction with SRP accelerates and enhances wound healing, making it more comfortable, while decreasing gingival bleeding, inflammation, and pocket depths. A 2002 position paper from the American Academy of Periodontology stated that gingival curettage consistently fails to provide any advantage in treating chronic periodontitis compared to SRP alone. The current article challenges this assertion by describing a five-year retrospective study that shows how laser technology made a consistent difference in the health of chronic periodontal patients. It is the authors’ opinion that the biofilm attaches to the inner lining of the epithelium and bony walls exposed to the bacteria. That biofilm will continue to destroy sulcular and junctional epithelium if it is not eliminated.

**Protocol**

Patients with pockets of 5 mm or more and those with bleeding and/or suppuration were considered candidates for laser-assisted periodontal therapy. SRP was performed three months before the start of laser-assisted periodontal therapy, due to the maximum utilization of insurance for most patients.

Optimally, patients would return for a series of subsequent appointments to address every pocket that exceeded the healthy 3 mm. For example, a patient whose deepest pocket measured 7 mm would come in for a total of four appointments every 7–10 days. Each time the laser was used to treat that pocket, 1 mm of it would heal from the apical to the coronal. Using this example, all 7-mm pockets would be treated first, while the 6-mm pockets would be treated at the second appointment (7–10 days later). This process would continue for each appointment until all pockets had a healthy depth of 3 mm.

Each appointment began with ultrasonic scaling at a low setting, applied to all pockets in a slow, sweeping motion. This technique is used to smooth the root surface, in addition to the regular cleaning or SRP that the patient had undergone previously. After ultrasonic scaling was completed, a strong topical anesthetic (*Cetacaine, Cetylite Industries*) was applied. Although the laser is virtually pain-free, some patients might feel an uncomfortable amount of heat. Dentists might also opt to use local anesthetics for patients who have lower pain tolerances.
**Laser setup**

To prevent it from stripping the epithelial attachment, the laser tip should be measured 1 mm less than the deepest pocket being treated. Before placing the tip into the pocket, the laser must be “initiated” with black articulating tape (Accu/ilm II, Parkell). This initiation pinpoints the laser energy to the end portion of the fiber-optic tip (Fig. 1), making it possible to emit laser energy only to the intended areas rather than laterally.

The diode laser should be set at the lowest possible setting. The laser’s energy is directed to the margin of the infected pocket without actually entering the pocket. The margin will start to turn white, which indicates that the laser setting is correct; it might be necessary to adjust the laser in 0.1 W increments to achieve this result (Fig. 2). Once the correct wattage has been achieved, the clinician should move the laser into the pocket for 5–10 seconds at a time. Each time the laser is removed from the pocket, it might carry a small amount of debris; wet gauze will remove this debris from the fiber-optic tip (Fig. 3 and 4). The clinician should inspect the fiber-optic tip to make sure it is still initiated each time before entering the pocket. The clinician should continue until no more debris can be removed or fresh bleeding occurs (Fig. 5).

After treating the infected pocket, the margins of the pocket must coagulate to help the healing process, as going into the pocket repeatedly with the laser can leave the borders jagged. The clinician needs to return to the lowest possible setting with the initiated fiber-optic tip. The laser energy should be traced along the margins of the pocket (at a distance of 1–2 mm) for approximately 20 seconds (Fig. 6).

Finally, the diode laser is used for biostimulation to aid in healing the damaged cells that line the wall of the inner epithelium.\(^8\)–\(^{11}\) In the authors’ experience, 6 J is the optimum setting for the diode laser.

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Fig. 1. An example of an initiated fiber-optic tip.

Fig. 2. The laser at the gingival margin.

Fig. 3. Debris removed from the diseased pocket.

Fig. 4. The laser is used to coagulate the pocket.
when it is approximately 4–5 mm from the treated area (Fig. 7). (This is the minimum distance to ensure that the laser energy is diffused sufficiently so that it provides biostimulation only and does no cutting.) Using a microbrush, the clinician should apply liquid vitamin E to the treated pockets (Fig. 8). (The efficacy of vitamin E has not been validated in the literature, but the authors have experienced positive tissue response with its use.)

Patients were instructed to avoid certain foods for at least 24 hours, including crunchy or spicy foods, foods with tiny seeds, and foods that might become lodged in the space created by the procedure. Patients were asked to avoid smoking for at least 24 hours, rinse with warm salt water at least twice daily for three days post-treatment, and avoid flossing and hard brushing for 48 hours post-treatment. If the patient experienced discomfort after the procedure was complete, ibuprofen or a similar pain reliever could be used, but typically this was not necessary. After the initial 48 hours, patients could brush and floss according to their normal routine.

Patients should return three months after the last appointment so that the dentist can determine the progress of pocket healing. Since periodontal disease is a lifelong struggle for most patients, this procedure might need to be repeated every 3–24 months, depending on the patient’s home care regimen.

**Materials and methods**

This study examined the use of diode lasers on periodontal pockets to determine their bactericidal attributes and their ability to improve periodontal conditions.

A total of 70 non-smoking patients with no implants needed SRP, and had been under care for periodontal disease continuously for at least five years. Using the protocol described above, 810 nm and 940 nm diode lasers (Biolase Technology, Inc.) were used. As described previ-
ously, the cases were rescaled at the time of laser treatment using a light stroke and the lowest setting. Some cases were retreated to maintain healthy pockets. In all cases, 400 μ fiber-optic tips were used. As a starting point for tissue interaction, the lasers were set at 0.5 W in continuous wave mode.

Fiber-optic tips were cleaved (that is, the tip was cut to get a straight fiber) and used for 810 nm lasers and both 810 nm and 940 nm lasers were initiated. Each tooth had six measurable pockets (that is, mesial facial, center facial, distal facial, mesial lingual, center lingual, and distal lingual) and teeth were treated in all four quadrants. Four hygienists performed the treatments and did probing readings, while another hygienist did only probing readings.

Results
A total of 2,103 pockets were treated among the 70 patients. Of the 2,103 pockets, 1,278 were found in molars, 556 in premolars, and 269 in anterior teeth. Of the 1,278 molar pockets, 973 (76%) had been restored to a healthy pocket depth of 3 mm after five years of treatment. Of the 556 premolar pockets, 466 (84%) had been restored to a healthy pocket depth of 3 mm after five years of treatment. Of the 269 pockets in the anterior teeth, 240 (90%) had been restored to a healthy pocket depth of 3 mm after five years of treatment.

Conclusion
In all, 80% of the pockets treated using the diode laser were restored to a healthy pocket depth of 3 mm. These results suggest that this treatment modality should become an adjunct for treating periodontal infections.

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References
Ozone and its use in periodontal treatment

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ABSTRACT

Objectives: To evaluate the effects of ozone in periodontal treatment in dental practice. Methods: An evaluation of the current state of knowledge regarding the application of ozone in periodontal treatment revealed limited available literature. Therefore an audit was conducted in dental practice in order to evaluate the effects of ozone in periodontal treatment. Twenty-five patients were treated with gaseous ozone after having had failed conventional periodontal treatment. BPE scores and the six deepest pockets were measured in each patient before and after the use of ozone. Results: From the initial number of twenty-nine patients selected, twenty-five patients attended both follow up appointments. Based on BPE scores, twenty of the patients have overall improvement while five of the patients continued to have deterioration. Eight patients had an improvement in depths of periodontal pockets by three millimetres, sixteen patients had improvement by one to two millimetres and one patient did not improve. The depth of pockets after the use of ozone decreased significantly (P < 0.001). Conclusion: The audit revealed that gaseous ozone significantly (P < 0.001) reduced the depth of pockets in patients with periodontal disease. The positive results encourage further investigation in the subject.

Keywords: Ozone; Periodontal; Audit

1. INTRODUCTION

Ozone was discovered by Schonbein in 1840 and has been used widely in industry [1]. Schonbein reported that the electrolysis of water produced an odour at the positive electrode [2]. He named that ozone from the ancient Greeks who also observed this strong odour after electric storms and they called it “ozein” (“to have a smell”).

Ozone is a triatomic molecule and exists as colourless gas with a pungent odour at room temperature, detectable at concentrations as low as 0.02 to 0.05 ppm (by volume) [3]. It is a highly corrosive, toxic and a powerful oxidant. Ozone exists in the atmosphere with the highest levels are in stratosphere in the region known as the ozone layer between 10 and 50 km above the sea level. In nature, ozone is also commonly found as a result of lightning strikes during thunderstorms and waterfalls [2]. Ozone absorbs dangerous B and C ultraviolet radiations making it very useful, yet it can be also very toxic for the pulmonary tract especially when it mixes with carbon monoxide (CO), N2O and traces of acids as it occurs in smog [1].

In dentistry ozone has been recognised for its antimicrobial effect and it can be used as a useful disinfectant in clinical applications [4,5]. It is a part of the evolving minimally invasive dentistry theme (MI) and it’s aim of preserving the original tissues where possible. It is claimed that ozone promotes haemostasis, enhances local oxygen supply and inhibits bacterial proliferation [1]. Although there are some promising studies, ozone has not been proven superior to other clinical approaches [6]. There is still need for more scientific data on the subject, as clinical evidence for application of ozone in dentistry is not extensive [4]. Furthermore, there is little evidence for the use of ozone in periodontal treatment and there is a need for more studies in this particular field [7-10].

It is known that ozone can kill bacteria by rupturing their cell membranes within a few seconds. In medicine and dentistry, ozone is used as a powerful sterilizing agent either in the gaseous or aqueous phase, as it successfully kills bacteria, fungi and viruses. Ozone has been found to have a bactericidal effect, particularly in staphylococcal, streptococcal and other infections. Recent research showed that exposure of carious dentine specimens to ozone reduced the levels of pathogenic microorganisms in these samples [4]. Ozone can be used for sterilisation of heat sensitive materials including medical
devices and narrow lumen devices [11]. Some researchers [12] found that even soaking toothbrushes contaminated with oral microorganisms in ozonated water has good disinfectant results. There are currently two ozone generators used in clinical dental applications and both utilise corona discharge (HealOzone by KaVo and Ozotop by TTT, see Figure 1).

Ozone has powerful microbicidal properties, however, this action not only affects micro-organisms but also all the other living systems. Ozone’s concentration in the blood is very important and high levels can be very cytotoxic producing even haemolysis. Baysan and Lynch [13] have listed the potential advantages and adverse reaction of ozone in clinical use.

Periodontitis is chronic gingivitis with associated loss of attachment. The development and course of periodontitis appears to be dependent upon specific inherited, behavioural or environmental conditions—so called risk factors. These risk factors are biologically linked to the disease. There are certain risk determinants (genetics, socio-economic status and gender) that cannot be modified. In periodontitis treatment is based initially on the supragingival calculus removal and possibly root planning combined with oral hygiene instructions. However, the clinical signs of illness (including the presence of microorganisms) can still be found after mechanical debridement, so chemomechanical treatment approach has been suggested as being effective. Ozone may be effective because of its antimicrobial effect.

A literature review revealed little clinical data on the use of gaseous ozone in periodontal disease and conflicting opinions on its benefit [7-10]. Therefore the authors decided to review outcomes from patients under treatment. Recent papers have reported a greater reduction in plaque index, gingival index and bleeding index following the use of ozone irrigation compared to the use of chlorhexidine [14]. Huth et al. [15] similarly showed significant results with gaseous and aqueous ozone and concluded that they merit further investigation. A study by Hauser et al. [16] investigated the use of gaseous ozone on bacteria adhering to implant surfaces and showed a selective reduction in bacteria, concluding that gaseous ozone may have a role in treatment of peri-implantitis.

The aim of the audit was to assess the effect of ozone which is being currently used as an adjunctive treatment in the management of periodontal disease in a UK General Dental Practice.

2. MATERIALS AND METHODS

An audit was been undertaken to answer the clinical question whether the ozone makes a difference in the treatment of periodontal disease.

2.1. Strategy and Subjects

From December 2007 all the patients who presented with periodontal disease in one of the author’s practice (DI), were treated with conventional periodontal treatment. This includes oral hygiene instruction and follow-up, supra and subgingival scaling and a Basic Periodontal Examination, (BPE). The BPE, as defined by the British Society of Periodontology [17], requires that the periodontal tissue should be examined with a standardised periodontal probe using light pressure to examine the tissue for bleeding, plaque retentive factors and pocket depth. A code 0 - 4 is then determined as follows:

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No bleeding or pocketing detected</td>
</tr>
<tr>
<td>1</td>
<td>Bleeding on probing—no pocketing &gt; 3.5 mm</td>
</tr>
<tr>
<td>2</td>
<td>Plaque retentive factors present—no pocketing &gt; 3.5 mm</td>
</tr>
<tr>
<td>3</td>
<td>Pockets &gt; 3.5 mm but &lt; 5.5 mm in depth</td>
</tr>
<tr>
<td>4</td>
<td>Pockets &gt; 5.5 mm in depth</td>
</tr>
</tbody>
</table>

The patients constituted the regular, everyday patients’ list. Twenty-nine patients (16 female, 13 male) aged between thirty-two and fifty-four, who failed to respond well to the conventional periodontal treatment were offered further treatment with ozone. The treatment with ozone, including potential risks and benefits, was fully explained. This was the normal treatment procedure within the practice. An audit was carried out to review the outcomes and assess the benefits of this approach as a guide to designing future treatment strategy in the practice.

Five patients did not receive ozone therapy, so could not be included in the audit, because they suffered from respiratory diseases or experienced other serious health problems.

2.2. Protocol

From the patients who were treated with conventional periodontal treatment and did not respond well, twenty nine were included in the audit. Each patient had a BPE score and the depth of periodontal pocket from all teeth
with pockets measured. All had broadly similar levels of disease. The WHO 621 probe was used to measure the depth of pockets. The probe was placed gently in the pocket and the depth was measured in millimetres according to the scale on the probe. BPE scores were measured according to recommendations of British Society of Periodontology [17].

A complete radiographic survey was performed for every patient so as to help in diagnosis. The recruited patients were treated with a combination of conventional periodontal treatment and gaseous ozone application for 18 sec in every pocket (according to the manufacturer’s instructions). After the treatment oral hygiene instructions were given to the patients and follow up appointments were scheduled in 6 weeks and in three months time.

Patients were asked to keep their oral hygiene daily diary. On follow up appointments all the patients had periodontal pockets measured and they were also asked about any problems after ozone treatment. The questions were about the status of the gingiva (bleeding or any discomfort) in the following days after the treatment with ozone. This was in a form of verbal questionnaire during the follow up appointment. On the three months follow up appointment BPE scores were also measured. Treatment with ozone was not repeated on follow up appointments. All the measurements, treatment and follow up appointments were carried out by one of the authors.

The six deepest pockets were taken into account when applying statistical analysis. Differences in pockets depth before and after application of ozone were analysed using the t-test. Statistical analysis was performed with the SPSS software package.

2.3. Equipment

Ozotop (TTT, Switzerland) an ozone generator that was available in the author’s practice was used (Serial Number: 0307/2006) as shown in Figure 1. This generator uses a corona discharge system and it produces 30 mg ozone per hour. There are three different tips for the oral cavity and the perio-tip was used for the periodontal treatment. The generator has no built-in scavenging system so suction with the special accessory for ozone mouth evacuation (Ozodam, TTT Switzerland) was used, according to manufacturer instructions. Suction with Ozodam was positioned close to the perio-tip in order to minimize the concentration of ozone in the mouth as this approach increases the safety of ozone use [18].

3. RESULTS

From the initial number of twenty-nine patients (16 females, 13 males), twenty-five patients attended both follow up appointments (6 weeks and 3 months) and four of the patients failed to attend either one or both appointments and consequently were excluded from the analysis. All the patients were asked to keep their oral hygiene daily diary but only sixteen complied with this instruction and returned their diaries.

Only BPE scores and depth of pockets measured on second follow up appointment were taken into consideration and were included in the analysis. The purpose of the first follow up appointment was to make sure that all patients complied with the oral hygiene instructions given and to evaluate the initial results as seen by the difference in the depth of pockets.

The depths of pockets before and after ozone treatment were measured as described in protocol. Based on BPE scores it was apparent that twenty (80%) of the patients have improved overall. Fifteen had improvement in more than three regions and five in one or two regions. Five of the patients not only did not improve but had deteriorated. Figure 2 shows the change in BPE scores for each of the 25 patients who attended both appointments.

The six deepest pockets were taken into account when applying statistical analysis (before and after the treatment). Figure 3 illustrates the pocket depth measurements of the six deepest pockets before and after ozone application. Eight patients had an improvement in depths of periodontal pockets by three millimetres, sixteen patients had improvement by one to two millimetres and one patient did not improve and the pocket depth had increased by one millimetre. The depth of pockets after the use of ozone decreased significantly (P < 0.001). The measurements were taken three months after the initial ozone treatment.

4. DISCUSSION

It has been reported by many authors and supported by this literature review that ozone has an antimicrobial effect. Ozone may be effective as it is known to kill microorganisms by rupturing their cell walls and cytoplasmic membranes [19]. This involves chemical modification and fragmentation of mono unsaturated and polyunsaturated fatty acids in the cell wall [20]. When the membrane is damaged, its permeability increases and ozone molecules can readily enter the cells [19].

It is known that specific bacteria are implicated as causative factors that can lead to the development of periodontitis and that oral microbial plaque consists of different types of bacteria that live on host surfaces. Current treatment of periodontitis is based on disinfecting the supragingival and subgingival tissues in order to re-establish a microorganism-free environment [21]. This fact renders ozone application reasonable and justifiable treatment option [8]. It has been shown in-vitro that ozone is bactericidal against periodontopathic microorganisms [9].

In the present audit gaseous ozone was used. The choice of gaseous ozone was based on the ozone generator cur-
Figure 2. The improvement in BPE scores for each of the 25 patients showing changes in score after ozone application, overall showing an improvement (P < 0.001).

Figure 3. Differences in pockets depth before and after application of ozone for 25 patients included in the audit, series 1 - 6 indicated the same six deepest pockets measured in each patient. Overall improvement was significant (P < 0.001).

To make sure that the observed therapeutic effect was that of ozone patients were also instructed not to use any of the available antimicrobials after the treatment with ozone. It is known that agents like chlorhexidine are being used as part of the chemo-mechanical treatment approach of chronic periodontitis and can significantly reduce the depth of deep and medium-deep pockets [22]. Patients were given oral hygiene instructions and were asked to keep their oral hygiene daily diary.

In the present audit, the patients displayed a variety of teeth with periodontal disease including localization, number of diseased teeth and variability of surfaces involved. It was decided that only the six deepest pockets from every patient will be included in the audit. These pockets were only measured before and after the application of gaseous ozone. This approach although practical might have affected the BPE score. In some patients, although all the teeth with pockets including the six deepest improved by few millimetres, their BPE score did not. This may be due to the fact that for various reasons not all the treated pockets improved. It might be that these were the patients who did not keep systematically daily diaries and consequently did not comply with the given oral hygiene instructions.

The present audit showed an overall beneficial effect of ozone application with 80% of the patients showing significantly (P < 0.001) improved BPE scores after gaseous ozone application. Additionally, pocket depth analysis revealed that 32% of the deepest pockets improved by more than three millimetres, 64% by one to two millimetres and 4% did not improve or deteriorated. The results support the use of ozone as an adjunct to mechanical treatment in periodontal patients in agreement with other studies [9,10].

However, there are recognized limitations to the present audit as it was not a research study and so no control group was possible. First, this is not a randomized, controlled and blinded study with all the limitations that this may have and inevitably there may be observational bias introduced, as all the measurements were taken by the author. One another factor that may also affect the validity and reliability of the obtained results is the small number of patients included in the audit. This may introduce type II error. In addition, the statistically significant results found in the audit may not translate necessarily into clinically significant results. One additional factor is the short length of follow-up period in this study. It is important to obtain long lasting therapeutic results in order to justify the use of ozone. In this audit the aspect of cost effectiveness was not addressed although it is an additional and important factor in clinical practice and the positive results in long term should justify the cost of ozone therapy.

The patients included in the audit did not experienced
respiratory or other medical problems and there were no adverse effects observed after the use of ozone. However, they were not screened for smoking although the deleterious effects of smoking on periodontal health have been well-documented [23-25]. This might be responsible for some patients not responding to the treatment. However, the patient population represented subjects who did not respond to conventional periodontal treatment. It is possible that patients who are diagnosed and treated for the first time might have responded more favourably to ozone treatment. Nevertheless, the authors wished to audit the usefulness of ozone in more resistant cases.

5. CLINICAL SIGNIFICANCE

Ozone offers a simple adju nctive therapy for managing periodontal disease alongside conventional methods. Further research is indicated.

6. CONCLUSION

This audit showed beneficial results of gaseous ozone treatment in patients with periodontal disease. These are encouraging results that call for further future randomised controlled studies to obtain high quality evidence for clinical practice.

REFERENCES


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