Oral Health and Systemic Health

The Heart
Periodontal disease may increase the risk of cardiovascular disease and fatal heart attacks. In fact, those with gum disease are almost twice as likely to suffer from heart disease as those with healthy gums.¹ ²

The Brain
Periodontal disease may increase the risk of stroke. Research has shown that harmful bacteria in the mouth can make a person more susceptible to developing blood clots and can eventually increase the chance of a stroke.³

The Kidneys
Bacteria often enter the body through the mouth. With poor oral care, infections progress faster. This increases the diseases the kidneys must fight off.⁴

The Lungs
Poor oral health may worsen respiratory illnesses by promoting growth of harmful bacteria that can be transported to the lungs. When the germs reach the lungs, they can breed and multiply to cause pneumonia and bronchitis.⁴

The Pancreas
Diabetics are at greater risk for periodontal disease. Periodontal disease may disrupt the control of blood sugars, which can increase the likelihood of serious complications, such as heart and lung diseases.⁵
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Introduction

It is a common misconception that oral health and general physiological health are separate entities, when in fact oral health involves cells that contribute to the very existence of an individual, thus making it a part of physiology. The mouth is atypically full of natural bacteria, biofilms and various types of plaque, which makes it just as vulnerable to infection and disease as any part of the body that is at risk from bacteria and inflammation. Oral health is an integral part of general health, and should not be isolated as it contributes to determining the overall health condition of an individual.

The American Dental Association (ADA) warns, however, that establishing an association between the two does not automatically mean that one causes the other. Periodontal disease associations to bodily diseases, such as diabetes or cardiovascular diseases, are not necessarily causal in nature since bodily diseases can exist without periodontal complications, thus allowing the theories to stay elusive. Dr. Howard Tenenbaum of the University of Toronto also notes that a perception trap has been created recently, which puts emphasis on dental treatment due to its association to bodily diseases, and not as a standalone entity of health in itself. Dr. Tenebaum rejects this approach since it implies that oral health is a mere health component that should be given secondary attention to because of the new findings that connect systemic diseases and oral diseases. However, he disclaims that it does not mean that he is putting less emphasis on existing oral and systemic health associations.

Oral health has been associated with systemic diseases such as diabetes, cardiovascular diseases, osteoarthritis and respiratory diseases, among others, but none has yet to be completely proven. The 1990’s have seen a number of articles, some with overly dramatic headlines while others a bit more skeptical or inducing of critical thinking. Establishing a causal relationship is difficult in its complexities, as a myriad of variables convolute theories. To this day, the medical industry has only
seen poor oral health as a contributing factor to systemic health complexities rather than a direct cause.

Figure 2. An overview of the different systemic health complications in relation to oral health by Delta Dental
Recent progress in identifying oral microorganisms and clustering them into specific classes and the previous knowledge that certain strains of bacteria are naturally present in the mouth have paved a path towards a more realistic and accurate assessment of focal infection of the oral area. Previous attempts to link oral health and systemic health have gotten far enough as to show that the theories cannot be disproved entirely as the mouth may indeed act as a gateway to disseminating pathogenic organisms to other parts of the body, especially hosts who are immunocompromised such as those suffering from diabetes, malignancies, rheumatoid arthritis, or those receiving immunosuppressive treatments.

Previous epidemiological studies suggested that infection in the oral cavity, most especially apical and marginal periodontitis, is a possible risk factor in the development of systemic diseases because complex microfloras (which have about 200 specie types in apical periodontitis and over 500 specie types for marginal periodontitis) are in existence. Said infections are mostly anaerobic, the most common of isolates being Gram-negative rods. These microfloras have an anatomic proximity to the bloodstream that can blossom into bacteremia, eventually spreading bacterial products, immunocomplexes and components.

Specific types of pathogenic bacteria that colonize the subgingival area have been found to be associated with periodontal infections. Two of these strains, *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis*, have been found present in virulent organisms and periodontal tissue. The type of bacteria in a periodontal disease and the rate of progression of a periodontal infection are also partly determined by systemic and local risk factors, which include pre-existing diseases as seen by areas of plaque retention and deep-probing depths that are associated with cases such as defective restorations.

Other risk factors include the use of tobacco, peptic ulcers, cardiovascular diseases, diabetes mellitus, respiratory diseases or chronic obstructive pulmonary disease (COPD), cancer, allergies, asthma, certain medications, obesity, rheumatoid arthritis, and the most recent addition of Alzheimer’s disease. Studies have also determined other notably important possible risk indicators for periodontal diseases that include coping behaviors, stress and osteopenia as associated with deficiency in estrogen. Other determinants also linked to periodontal diseases include age, hereditary factors and gender. Inflammation also triggers a host to respond to an infection, and this response can contribute to situations that deal with systemic conditions.
INFLAMMATION

The body responds to cellular injury by inflammation. Previous materials have doted on the negative effects of inflammation, failing to focus on the fact that it is one of the body’s coping mechanisms against injury, and is thus a crucial process in order for the human body to survive. Inflammation is a protective response to a cell injury’s initial cause as well as the consequences brought by it. Cell injury usually occurs due to genetic defects, trauma, tissue necrosis, chemical and physical agents, and immune infections and reactions.

Inflammation is a change that is local and reactive, usually involving the dissemination of antibacterial agents from neighboring cells that act as defense against infection. It is also responsible for facilitating tissue repair and healing. It contains the injurious or infectious agent and thus serves as the body’s defense mechanism so it can restore itself to a morphological function and form that is of normalcy. A cellular and vascular reaction takes place during an inflammatory response, mediated by factors that are chemical in nature that are usually derived from cells or plasma proteins. Heat, swelling, redness and loss of function are usual symptoms of inflammation. Other symptoms include leukocytosis or a spike in number of white blood cells, fever, the existence of proteins that are in acute-phase such as fibrinogen, serum amyloid A protein and C-reactive proteins, as well as sepsis.

The two types of inflammation are chronic and acute. Chronic inflammation, as the word suggests, takes place in a lengthier duration and histologically manifests by the existence of macrophages and lymphocytes that usually result in tissue necrosis and fibrosis. Lengthier periods of inflammation may be thought of as a healing process gone haywire, which may prompt detrimental changes in localized tissues and possible in other parts of the body. Acute inflammation usually takes place within a shorter and quicker duration. It develops with an exudation of plasma proteins and fluid, as well as a leukocyte emigration, most of which are neutrophils.

Figure 3. Before and after of gingival inflammation
In order to appreciate the process of inflammation, understanding chemical mediators is important since these tend to control the response of an inflammation. Inflammatory mediators stem from plasma proteins as well as cells that include platelets, monocytes or macrophages, mast cells, and neutrophils. These mediators are usually triggered by host proteins or bacterial products. Chemical mediators bind themselves to certain receptors of target cells and may stimulate contraction of the smooth muscle, undergo direct activity of enzymes, increase neutrophil chemotaxis and vascular permeability, and bring about pain or facilitate oxidative damage. Most mediators have a short life span but can cause great damage. Chemical mediator examples include arachadonic acids (such as prostaglandins and leukotrienes), cytokines (such as tumor necrosis factor and interleukin), and vasoactive amines (such as histamine and serotonin).

**INFLAMMATION AND ITS CONNECTION TO ORAL HEALTH**

The periodontium is significantly affected by the process of inflammation. Gram-negative and gram-positive bacteria are released, together with other products that are biologically active, by the plaque biofilm and colonize the surface of the tooth in interproximal areas as well as in the gingival margin. These biologically active products include protein toxins, cytokines and endotoxins. They travel to the gingival epithelium where a host response is initiated, eventually resulting in gingivitis. Clinical changes such as the tissue color turning red from a healthy pink, bleeding while probing, and swelling are some of the evidences. Gingivitis may prevail for years without treatment since it is typically pain-free, or worse, it may be seen by practitioners as a situation that requires less attention as compared to periodontitis. Nonetheless, a persistent chronic gingivitis may actually be a greater basis for systemic health concern as compared to the more readily treatable periodontitis condition.

The sulcular epithelium is penetrated by soluble compounds as the biofilm continues to expand in number. This gives the gingival epithelium the go signal to excrete chemical mediators that include prostaglandins, interleukin 1 beta (IL-1), matrix metalloproteinases, and tumor necrosis factor alpha (TNF-α). These biologically active products direct neutrophils to the inflammation area and proceeds to influence chemotaxis, possibly causing a permeability increase of gingival cells, permitting the emigration of plasma proteins into the tissue from the blood vessels. More
mediators are produced additionally as the process of inflammation progresses, and more cell types are directed to the area of injury including monocytes, T-cells and neutrophils. Consistent inflammation signals fibroblasts and pro-inflammatory cytokine production in the tissues. Oral bacteria with specific antibodies penetrate the peripheral blood. Fibrinogen and its complement, as well as CRP, are formed within the liver and by local cells when there is an activation of an acute-phase response. A close investigation of pregnancy outcomes has been carried out due to the possible exacerbation of the proteins. This biological mechanism basis is starting to emerge, which prompts further study to establish the existence of a causal relationship.

Figure 4. illustration suggesting that inflammation is a common link between oral health and systemic diseases
Diabetes Mellitus

The link between diabetes and oral health is majorly based on the mutual relationship between glycemic control and periodontal disease. Oftentimes, periodontal diseases are viewed as the sixth diabetes complication in oral health. In a Cochrane review, it was stated that though small in amount, there is significant progress in the betterment of blood sugar control due to treatment of pre-existing gum diseases in type two diabetes patients. Even if evidence is continuously being built in order to support this association, it is crucial to understand that the link between oral health and diabetes surpasses this relationship. Other elements that build the association between oral health and diabetes include proper dieting through oral function, development of self-management skills, control of oral disease through positive health behavior, as well as more common risk factors that are modifiable such as poor nutrition and tobacco use, which both have an effect on diabetes and oral health.

Oral diseases are commonly associated with impaired social functioning, anxiety, pain and possible tooth loss that affects one’s ability to maintain a nutritious diet. Studies have interestingly shown that general well-being and quality of life are linked to a person’s chewing ability, since this has an impact on the ability to freely choose preferred types of food and to enjoy meals. However, another study of adults with ages 60 years old and above showed that no significant increase was seen in the Healthy Eating Index (HEI) scores despite having functional dentition because an adequate nutrition was still not maintained due to poor teeth condition. The study also did not include various oral conditions that may affect oral function such as xerostomia, or dry mouth. Additional research in establishing a correlation between quality of life, general health and chewing ability was called for by the World Dental Federation.

Figure 5. Oral manifestations of Diabetes
DENTAL CARIES

Evidence has strongly supported the link between dental caries development and higher sugar consumption. The most cariogenic type of sugar is sucrose, which forms glucan and enables firm adhesion of bacteria to the teeth. Bacteria from dental plaque anaerobically metabolize dietary sugars into organic acids. Dental caries are then formed from the acid demineralizing dentin and enamel calcium hydroxyapatite. The amount of sugar and the frequency in which it is taken in are both important factors in developing dental caries. The risk of caries may also be heightened by xerostomia.

DENTAL EROSION

Dental erosion does not involve bacteria, but rather acid exposure that chemically etches away tooth structure resulting in an irreversible tooth structure loss. Examples of extrinsic dietary acids include soft drinks, fruit juices, vinegar and power drinks. Severe cases may result in complete destruction of the tooth. Dental caries development does not consider normal fruit consumption although overconsumption of natural fruits may lead to dental erosion.

Sugar sweetened beverages (or SSBs) such as fruit drinks, vitamin water, power drinks and soft drinks use high-fructose corn syrup, sucrose, fruit juice concentrate as sweeteners. SSBs have been known in the US diet as the main added sugar source, which are linked to type two diabetes and an increased metabolic syndrome risk. SSBs also act as an indicator for general health level of diets because they are associated with lower consumption of dietary fiber and higher consumption of saturated fats.

Figure 6. Dental erosion
XEROSTOMIA

People suffering from diabetes are at a greater risk of experiencing xerostomia because of the side effects of certain medications, such as tricyclics for neuropathic pain and diuretics, as well as poor control of blood glucose. Xerostomia complications are often inclusive of decreased saliva production together with complications in immune functions and hyperglycemia of the saliva which may cause oral candidiasis, burning mouth sensation which may be caused by deficiency of vitamins B12 and zinc, difficulty in lubricating, tasting, swallowing and masticating which may lead to poor intake of nutrition, or some other little known etiology. Decreased natural defense provided by phosphate ions and calcium that naturalize pH and helps mineralize tooth structure is another significant risk in dental caries development.

There are two schools of thought on this topic. One school believes that high levels of sugar in the saliva of people with uncontrolled diabetes helps bacteria thrive, which leads to the development of cavities as well as sets the stage for gum disease. Also, the fact that diabetic patients tend to eat smaller, more frequent meals throughout the day may mean there is a greater chance for bacteria to grow and lead to the development of cavities. The other school of thought is that because people with diabetes are more knowledgeable about what they eat and the need to closely monitor their sugar intake, they don't eat many foods that contain cavity-causing sugar.

The fact is that people whose diabetes is well controlled have no more tooth decay or periodontal disease than persons without diabetes. Good oral hygiene and maintenance of blood
sugar within the accepted range are the best protections against cavity formation and periodontal disease.

In the United States, periodontal diseases and diabetes mellitus are two fairly common diseases that are chronic in nature, especially in people who are tobacco users or have poor control of their metabolism. Tobacco use and diabetes have been known as two risk factors that have a significant effect on the progression and initiation of periodontitis. Attempts at managing said factors contribute significantly to the treatment and prevention of adult periodontitis. Patients with diabetes generally are more susceptible to various kinds of infections, more notably anaerobic gram-negative bacteria. These types of bacteria are nurtured further by high blood sugar levels, which in turn may possibly lead to periodontal disease. Several studies have already shown significant evidence between an increased incidence of periodontal diseases and poor glycemic control. Neurophil function impairment has also been seen in patients with diabetes.

Some of the more common oral health problems that have been associated with diabetes include taste impairment, dysfunction of the salivary gland, periodontal diseases, tooth decay, fungal infections, delayed healing of infections, lichen planus and lichenoid reactions (which is an inflammatory disease of the skin).

Uncontrolled or untreated diabetes may reduce a person’s life expectancy by 30% and possibly more. The American Diabetes Association has suggested that when the hemoglobin A₁c (HbA₁c) is at a percentage that is less than 7%, that is the period when adult patients are qualified for treatment goals. Studies have tried to establish a link between periodontal diseases and glycemic control, and the usual queries are whether it is possible to improve diabetes with healthy gums or will it otherwise worsen with periodontal diseases. A previous study has reported that subjects placed with HbA₁c > 9% are at a greater risk of up to three times more for severe and progressive loss of alveolar bone, which is a periodontal disease measure, and likewise, subjects with severe periodontal disease are at a greater risk for worsened glycemic control. Other studies have reported that a significant reduction in HbA₁c was seen in patients who have poor glycemic control who were treated for periodontal diseases. The treatment included antimicrobials (0.12% chlorhexidine rinses), tooth root cleaning and doxycycline.

Reports that periodontal disease is amenable to treatment are of clinical significance, thanks to the findings, since these may be expanded in order to study the link further in hopes of eventually
attenuating the more severe diabetes mellitus complications. A study has suggested that severe periodontal diseased patients were possibly at greater risk for angina, MI, stroke and transient ischemic attack. A supplementary study also suggest that risks for nephropathy and ischemic heart disease were greater by about 3.2-fold in patients with severe periodontal diseases than those without.
While most chapters focus on the connection between oral health and its perceived effects on systemic health, the transmission and disease development of oral cancer both primarily occur in the mouth area. Despite the area concentration, oral cancer still warrants discussion since any form of cancer in itself has a huge impact on systemic health because of the ability of cancer cells to metastasize, or spread. In the long run, oral cancer may spread to other parts of the body, such as the lungs or the brain, eventually leading to multiple organ failure and death.

Oral cancers usually develop from squamous cells that are often found in the lower third molar area or on the tongue (ventral and lateral border), soft palate, and floor of the mouth. Patient survival had been estimated to be at 50% in five years, which prompts the need for early detection and treatment. While the usual symptoms for oral cancer are white patches found in the mucosa, a number of cases are characterized by red and white lesions, most of them ulcerated, with varying discomfort levels. The patient must be asked to remove his or her dentures in order to allow

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**Figure 9.** A brief screen for oral cancer includes this eight-step examination of the inside of the mouth.
examination of tissues found underneath the prosthesis. Even if an existing ulcer is believed to stem from irritation due to dentures, it must still be as a symptom of a possible oral cancer case unless proven otherwise. Such lesions should undergo a biopsy in order to be correctly diagnosed since survival rates are usually high with cases that undergo surgical intervention.

Most oral cancer development cases also were found to occur mostly in males who are over 40 years of age, as well as heavy ethyl alcohol and tobacco users. These factors increase the chances of developing oral cancer significantly.

Oral cancer symptoms usually include:

- Thickening, lump, rough spots or eroded areas on the lip, gums and other areas within the mouth
- Velvety red, white, or a speckled combination of both colors manifesting as patches in the mouth
- Unexplained mouth bleeding
- Unexplained pain or tenderness, loss of feeling or numbness in any area of the mouth, face or neck
- Sores that are persistent that are usually found in the mouth, neck or face that bleed easily and remains even after two weeks of healing
- A feeling of having something stuck at the back of the throat or soreness
- Difficulty speaking, moving the tongue or jaw, and chewing or swallowing
- Chronic sore throat, hoarseness, or change in voice
- Pain in the ear
- Change in denture or tooth fit
- Abrupt and significant weight loss

Cytokines play an integral part in cancer. It is released as a response to inflammation and infection, which then aggravates the development and progression of cancer cells, and may likewise also cause further inflammation as well. A number of proteins are involved in cytokines, which may not only cause inflammation in systemic areas but also in the oral area. There are quite a number of varied cytokine types, with RANKL, also known as TRANCE–TNF-related activation-induced cytokine, being a TNF superfamily member. This protein was discovered to be a protein that stimulates dendritic and T-cell functions. Two groups have previously been able to clone it independently due
to its high ability for osteoclast differentiation, survival and activity. The cytokines under the TNF family have a general ability to cause oral and systemic bone loss.

**HUMAN PAPILLOMAVIRUS**

New study has found that the number of neck and head cancers that have been associated to human papillomavirus, or HPV, which is a sexually transmitted disease, has sharply increased in the past two decades. It has even been found that it has replaced tobacco as the number one cause of oral cancer.

More than 120 variations of HPV have been found over the years, with several types involving infection of different body parts other than the mouth. HPV’s are most visible as warts on the arms, legs, hands and other parts of the skin. These types are usually harmless, non-carcinogenic, common and easily treatable. Technically, genital warts are otherwise known as condylomata acuminatum, being usually associated with numbers six and 11 of HPV types.

HPV can be transmitted merely through contact of the skin. However, other forms of HPV such as the one that causes oral cancer are sexually transmitted. Some of these develop into rare but serious cancers. The most common sexually transmitted HPV’s are HPV-45, HPV-31, HPV-18 and HPV-16. These types of cancer-associated HPV’s usually show growths that appear almost invisible and are flat, in contrary to the warts caused by HPV-11 and HPV-6. Majority of cervical cancers are known to be caused by two particular types of HPV of the genital tract – HPV-18 and HPV-16, with the latter being associated with oral cancer.

In the oral cavity, the HPV-16 is manifested primarily in the posterior or back regions such as the oropharynx, or the back of the throat, the base of the tongue, the tonsillar pillars and in the tonsils. These oncogenic strings of HPV are also the main causes for squamous cell carcinomas, such as that of the anus and the penis. These HPV types are usually transmitted through genital contact, oftentimes during anal,  

![Figure 10. HPV-16 virus strain](image)
vaginal sex, or a mere genital-to-genital contact. However, the growing concern over the rapidly increasing number of sexually-transmitted HPV cases are usually that which involve oral sex.

HPV may be transmitted regardless of the gender of the partners, be it straight or same-sex. It may be transmitted even when the carrier does not showcase signs or symptoms. HPV-infected persons usually pass the disease on to a sex partner without being aware of it. A person may develop the symptoms of HPV after several years or even decades of carrying the disease since the sexual contact that allowed the transmission. Also, it is possible to be a carrier of more than one type of HPV. However rarely, a woman who has contracted genital HPV and becomes pregnant may possibly pass the disease on to her baby upon delivery.

271 samples of cancer tissue were collected from oropharyngeal cancer patients in Los Angeles, Iowa and Hawaii within the years 1984 to 2004. From 1984-1989, 16% of cases of oropharyngeal cancers, that is cancers involving the upper throat, tonsils and base of the tongue, tested positive for HPV. By 2000 to 2004, the number of cases that tested positive for HPV has risen by 72%. This meant that a 225% increase was shown HPV-related cases of oral cancer, with the initial study showing 0.8 cases out of 100,000 people during the 1980s and rising to 2.6 cases out of every 100,000 people during the 2000s. Even with the statistics of smokers steadily declining, the rate of cancers that are HPV-negative dropped by 50%.

Studies suggest that an in increase in oral sex with men have also increased the statistical risk of acquiring HPV’s in the male gender. Today, sexual behavior has vastly changed, including engaging in sex at an earlier age as well as generally having more sexual partners.

HPV has been previously known as the cervical cancer-causing virus within the female population, but with better prevention methods developed over time, such cancer rates have declined steadily in recent years. There has been speculation that by the year 2020, oral cancer may possibly become the most common cancer that is HPV-induced, eventually surpassing cervical cancer. Dr. Maura Gillison, who is a study senior hailing from the Ohio State University Comprehensive Cancer Center in Columbus, pointed out that the main concern for HPV-related cancers will eventually shift to men from women in America because of the increase of HPV-positive oropharynx cases of cancers amongst the male gender.
The silver lining is that patients with oral cancers due to HPV have better chances for survival than those stricken with cancer due to other varying causes. This is possibly because of the tumors having less damage genetically, which would make their chance of responsiveness to treatment higher. The greatest determinant of a patient’s chances for survival lies on the tumor’s HPV status, following a diagnosis of oropharynx cancer that is local-regionally advanced. Patients that are HPV-positive have approximately a 60% less chance of succumbing to cancer as compared to patients that test HPV-negative.

The HPV-16 strain is currently being targeted primarily by the vaccines Cervarix and Gardasil. Females are recommended to take the vaccines in order to ward off chances of cervical cancer. The vaccines are also recommended for males in order to protect them from anal cancer and genital warts. While these vaccines have not been proven to prevent oral cancer just yet, experts suggest that fewer HPV cases among women would probably help reduce the infection rate in men.

Figure 11. A case of HPV-positive oral cancer
Cardiovascular Diseases

Many years have been put into studying the association between cardiovascular health and oral health. Today, the possibility of a link between serious gum complications, or periodontal diseases, and the fatty deposits that build up on the artery wall linings that may lead to blood clots, eventually becoming the cause for atherosclerotic vascular disease (ASVD), is now becoming more widely accepted. More than 50 studies have been conducted in order to investigate whether there is much truth in the increase of ASVD risk due to severe periodontal diseases. Other studies have considered the effect of bacteria that reside in the gaps found around the teeth, or periodontal pockets, that pass through into the blood stream, reacting with the plaque that has built up within the arteries.

One theory is that bacteria found in periodontal diseases aggravate the immune system which then leads to inflammation. This usually means that areas in the blood vessels that may already contain plaque caused by cholesterol are aggravated further, resulting into a narrowing of the artery. Another theory is that the bacteria stemming from periodontal diseases are transmitted into the blood stream through the periodontal pockets, which then attaches itself to the arterial plaque buildup, which in turn exacerbates inflammation.

This is not to simply say that maintaining oral health will completely absolve the body from risks of cardiovascular diseases, but rather to emphasize the possible overall impact of oral health on general systemic health. A certain Professor Robin Seymour was also quick to point out that the recognition and management of gum disease does not only mean a lengthier natural teeth life expectancy but also that there are significant benefits in general health care that may be able to help in the reduction of life threatening systemic conditions.
A study conducted in 2010 included more than 11,000 Scottish people, concluding that there is an association between poor oral hygiene and higher risk levels of low-grade inflammation and cardiovascular diseases. An increase of 70% was seen in participants who didn't brush their teeth as often, as compared to those who brushed twice a day.

Several studies of observational epidemiology also resonate with the results of the aforementioned study, which confirmed that periodontal health status of low quality may be associated with increased risks for cardiovascular diseases. There is, however, a need for further studies in order to confirm the association between heart disease and poor periodontal health status, or at least prove any risk markers. Conducted by public health and epidemiology researcher Cesar de Oliveira, Mark Hamer, a senior researcher for public health and epidemiology, and Richard Watt, a dental public health professor and consultant, the study was published in the British Medical Journal, or BMJ.

Alongside a team from the Royal College of Surgeons in Ireland (or RCSI), Bristol University put together a research that concluded that bacteria from dental plaque may be responsible for triggering blood clots. Bacteria that are normally found in the mouth that contribute to plaque formation on teeth surfaces are called streptococcus gordonii. This bacteria strain may enter the blood stream through open channels in the gums, possibly causing problems through human protein fibrinogen mimicking, which may cause blood clotting. This stimulates the platelets, hence causing them to form clumps in blood vessels.

The Harvard Heart Letter tackled the association of cardiovascular health and oral health in 2007. It stated that various bacteria species that are responsible for periodontitis have been located in the heart arteries, specifically in the atherosclerotic plaque. This plaque build up may become a trigger for a heart attack. These types of oral bacteria may also damage blood vessels or trigger blood clots by toxin release, causing the immune system to react and allow for blood clots to form more easily. The exacerbation of bodily inflammation may possibly be due to oral inflammation, arteries included, where heart attack may then be triggered. Although further research must still be carried out in order to study the connection between oral problems such as periodontitis and heart disease, the Harvard Heart Letter emphasizes that maintaining good oral hygiene is still crucial, through the basics of brushing, flossing and regular trips to the dentist.
HEART HEALTH AND DENTAL TREATMENT

People with heart conditions such as endocarditis, which is a heart infection caused by bacteria that passes through the blood stream and attaches itself to damaged heart tissue or valves, may need antibiotics before undergoing dental procedures. It is important to be well aware of a patient’s cardiovascular issues, since unknowingly going into procedures may have a further negative impact on the patient’s general systemic health. The American Heart Association (AHA) released an updated guideline on antibiotic use prior to dental procedures. The revision advises the limitation of antibiotic list as compared to the older policy.

Antibiotic pre-treatment is recommended for patients who have had a history of endocarditis. It is also suggested for patients with non-natural heart valves, and patients who have undergone heart transplants and developed problems in the heart valves later on. Antibiotic pre-treatment is also recommended for patients who have had congenital heart ailments:

- Unrepaired or incompletely repaired cyanotic heart disease that includes patients with conduits or shunts
- Completely repaired heart defect using a prosthetic device or material, in which antibiotics should only be used for six months post-procedure
- Heart ailments which may have a remaining defect or is positioned next to an prosthetic device or patch area
- Antibiotics pre-dental procedure is no longer advised for people with the following:
  - Dysfunction of the heart valve which has been acquired (such as rheumatic heart disease)
  - **Bicuspid** valve disease
  - Mitral valve prolapse
  - Calcified aortic stenosis

![Endocarditis](image)

*Figure 13. A heart with endocarditis*
• Heart conditions that are congenital, such as atrial septal defect, hypertrophic cardiomyopathy, and ventricular septal defect

AHA guidelines also recommend antibiotics before dental procedures that involve gum manipulation, as well as manipulation (such as incisions) tissues that surround a root of a tooth.

Antibiotics should not be administered for the following:
• Denture placement
• Routine injections of anesthesia via non-infected tissue
• X-rays
• Adjustment or placement of removable orthodontic appliances
• Bracket placement for braces (excluding bands)
• Loss of baby teeth naturally in children
• Trauma bleeding to the mouth or lips

**MYOCARDIAL INFARCTION OR HEART ATTACK**

A heart attack can be characteristic of pain that manifests in the chest, eventually spreading to the lower jaw. Sometimes, it may start in the jaw or in the shoulder or left arm. A period of at least six months following a heart attack must be given before scheduling a dental treatment. Nitroglycerin and oxygen must be readily available during the appointment. Coordination between the dentist and the patient’s physician is advised.

It is important for the patient to disclose medication details as this may mean having to change some standard procedures during treatment. Blood-thinning drugs or anticoagulants, for example, will lessen the tendency of the blood to clot, which would mean needing to stop drug intake before certain dental procedures. However, temporarily discontinuing medication must have due approval from the patient’s physician.
HIGH BLOOD PRESSURE OR HYPERTENSION

Some anti-hypertensive drugs used for treating high blood pressure may alter the sense of taste, or what is known as dysgeusia, or cause dry mouth. The raising of a patient from a flat position to a standing or sitting position may cause the patient to faint, or what is otherwise known as orthostatic hypotension.

Anti-hypertensive drugs may also cause gum overgrowth, including calcium channel blockers, which may begin a month post-drug therapy start. Some gum overgrowth may be so extreme that the patient has difficulty chewing; in some cases, a surgical procedure is in order to remove parts of the gums.

The dentist must check the patient’s blood pressure regularly during visits in order to decide whether a non-emergency dental procedure is safe to undergo. This will depend on the level and control of the patient’s blood pressure, as well as whether any other medical condition is in existence.

Patients with high blood pressure can usually take anti-anxiety drugs without risks pre-dental procedures. These drugs include diazepam (Valium) or nitrous oxide. Local anesthesia may also be safely administered if these contain epinephrine.

Figure 15. A chart explaining the mechanics of blood pressure
CORONARY ARTERY BYPASS GRAFT (CABG)

This procedure has no oral effects. Severe pain in the chest may be felt for the first several weeks post-surgery when reclining in a dental chair as a surgical side effect. Help the patient find a comfortable position. Generally, patients who have had a history of coronary artery bypass graft do not need antibiotics in precedence of a dental procedure.

ANGINA

Pain that usually starts in the chest and sometimes spreads to the lower jaw is called Angina. Patients with angina usually take calcium channel blockers, which may cause gum overgrowth as soon as one month following the first intake of the drugs. As previously mentioned, extreme cases of gum overgrowth may need surgical removal of the gums. For patients with stable angina, very little difference in treatment may be seen, but dentists are advised to keep nitroglycerin and oxygen handy during the procedure. Non-emergency dental care is not advised for patients with unstable angina. If emergency dental care is needed, the heart must be constantly monitored. Stress may also trigger attacks of angina. The patient should not experience any anxiety brought about by dental fear.

HIGH CHOLESTEROL (HYPERLIPIDEMIA)

Having too much fat usually causes the spike in cholesterol levels. High cholesterol levels put a patient at risk for artery hardening, which may lead to a stroke or heart attack. No oral effects are usually seen in cases of hyperlipidemia. Certain drugs used to treat hyperlipidemia may cause a
feeling of being faint upon getting up from a dental chair. A thorough list of treatment drugs must be attained from the patient.

**STROKE**

A stroke may cause several long-term effects, including:

- Paralysis
- Difficulty in swallowing and speaking
- An increase or decrease of pain sensitivity
- Blurred vision
- Poor memory
- Personality changes such as behaviors connected to anxiety or depression

Some patients experience paralysis on one side of the body due to a stroke. They may need another person to help them in daily activities, which includes daily dental care. Floss holder and toothbrushes especially designed for easier handling are available. Dentures may also need to be adjusted. For paralysis of the face or tongue, rinsing of the mouth may prove to be difficult. Realizing whether there is food left in the mouth and biting of the lip or tongue unknowingly may also occur. In order to keep gums and teeth healthy, the use of a saliva substitute or fluoride gel is advised. Blood thinners are usually taken by some stroke survivors. These may need to be put on hold before undergoing dental procedures, under the approval of the patient’s physician.

**CONGESTIVE HEART FAILURE**

A number of the medicines used for treating congestive heart failure (CHF) may cause dry mouth, or xerostomia. If the patient has no complications, physical limitations or side effects due to CHF treatment usually have no need for special changes in dental procedures, aside from adjustments needed from medicines currently taken.

Patients who have had severe heart failure should not lie

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**Figure 17.** Difference between a healthy and a congested heart
down too flat on the dental chair, as this may cause fluid build-up in the lungs and affect the patient’s breathing. Lying down or sitting up in the dental chair should not be done quickly either as this may make the patient light-headed and dizzy. Some patients with a severe case of CHF may need to undergo their dental treatment in the hospital. According to the New York Heart Association, this includes class III or IV cases.

**PACEMAKER IMPLANTATION**

Pacemakers usually do not have any specific oral effects. However, it must be assured that the electromagnetic devices in the dental setting and the pacemaker do not have any interactions.

Machines such as those used for electrosurgery or ultrasound may potentially cross paths and cause problems with the pacemakers. Although the chances for interaction are very small, precaution must still be taken.

AHA released a recent statement that basically said that current observational studies support the association between atherosclerotic vascular diseases and periodontal diseases that is not readily explainable by common risk factors. A causative relationship is not supported, however, and will still need further evidence. “While establishing the causes between periodontal disease and atherosclerotic vascular disease is vital, so too is the need to investigate what effects treating periodontal disease or preventative measures would have in reducing the incidence of atherosclerotic vascular disease,” Professor Robin Seymour pointed out. Although the association between heart health and oral health cannot be proven at this time, there is still the possibility of the association being identified in the near future.
Respiratory Diseases

An association has been suggested by epidemiologic and microbiologic studies between respiratory diseases and poor oral health. Several hypotheses have suggested how the pathogenesis of respiratory infection includes oral bacteria, such as oral secretions that may have aspirated with respiratory pathogens, thus affecting organism adhesion to the respiratory epithelium. Additionally, cytokines in pharyngeal or oral aspirates or oral bacteria products may stimulate the production of cytokine from respiratory epithelial cells, which results in inducing inflammatory cells. Preliminary data present that certain oral bacteria species induce pro-inflammatory cytokine release from epithelial cell lines similar to those apparent in respiratory pathogens.

Based on reported cases of anaerobic periodontal pathogens found in lung tissues, investigations regarding the association between chronic obstructive pulmonary disease (COPD) and periodontal diseases have been carried out. In a study that included an investigation of 46 reported deadly anaerobic empyema cases, the most commonly found microorganisms that were isolated were Fusobacterium nucleatum, Peptostreptococcus Prevotella, and Bacteroides, which are all etiologic agents found in chronic periodontal diseases. These findings furthered several population-based studies examining patient risk of COPD in those suffering from periodontal diseases. One study, which was adjusted for smoking status, showed that the risk of attaining COPD was at 4.5 times greater in patients with poor oral health (that is, having high levels of calculus and biofilm) as compared to subjects with well-maintained oral hygiene.

Another study done over a long-term course suggested that bone loss at the alveolar was an isolated predictor of COPD in males. Subjects who have experienced bone loss in the oral area had an increase of 60% in risks of becoming stricken with COPD. A study conducted in nursing homes in Japan controlled the sample groups into two – one that received regular professional oral health care and one that didn’t. After following up in two years, residents who attempted to take care of
their personal oral health were at 100% more risk for contracting pneumonia and were twice as likely to die.

Respiratory pathogens have long considered the oral cavity as a potential reservoir. Infection mechanisms are potentially aspirations into the lung of pneumonia-causing oral pathogens, upper airway colonization by pulmonary pathogens or periodontal pathogen facilitation, or dental plaque colonization through respiratory pathogens that is then followed by aspiration.

A number of anaerobic bacteria found in the periodontal pocket have been separated from lungs that have been infected. In long-term care facilities, elderly patients frequently experience dental plaque colonization by pulmonary pathogens. It is important to note that the destruction of connective tissues brought about by inflammatory process overreaction can be found in both emphysema and periodontal disease. The overreaction may provide the link between obstructive chronic pulmonary disease and periodontal disease, which is the fourth causative disease for death in America. These reports emphasize the need to improve oral hygiene within risk-stricken patients and those who live in chronic care homes.

**PULMONARY DISEASE EPIDEMIOLOGY**

Usually, pneumonia is classified as nosocomial or hospital-acquired pneumonia and community-acquired pneumonia (CAP). This distinction is significant because the implicated pathogens and the measures taken to prevent them are very distinct for both. CAP is a more widespread incident, with its prevalence estimated at around eight cases in every 1,000 inhabitants annually in more industrialized countries. In hospitalized patients, the mortality rate is at an estimated 7%. Accounting for around 40% to 60% of reported cases, Haemophilus influenzae and Streptococcus pneumoniae are identified as the main organisms that cause CAP. To this day, studies that have delved into the association between oral health markers and acute respiratory diseases (which also includes CAP) were not able to find sufficient evidence to link the two.
Next to urinary tract infections, nosocomial pneumonia is the most common in institutions that provide long-term care. It is responsible for roughly 10% to 15% of infections that are hospital-acquired, and around 20% to 50% of patients stricken with nosocomial pneumonia will succumb to it.

Staphylococci and gram-negative bacilli are the main etiologic agents. The frequency of anaerobic organism infection is not certain because there is difficulty in the technicality of anaerobic culture and possible contamination of anaerobic oral flora in the process of sampling. These technicalities make it difficult to ensure that anaerobic bacteria are cultured in the routine pulmonary microbiological sample analysis. One study reported that about 35% of reported cases of nosocomial infections were largely in part of obligate anaerobes. A bacterial infection that occurs in association to a condition that predisposes aspiration, such as alcoholism, Parkinson’s disease or stroke is called aspiration pneumonia. The bacterial infection is usually acquired from a hospital or through indigenous oral flora. Infections that take place during mechanical ventilation are significantly linked to anaerobic bacteria in aspiration pneumonia.
Since there has been an implication of anaerobes in pneumonia, the beginning of the 20th century has already been suspect to the role of the oral cavity in pneumonia pathogenesis. However, it wasn’t until the 1970s that efforts for extensive investigation regarding the association of pulmonary infections and anaerobic bacteria were carried out.

Pulmonary diseases that affect airflow may also implicate oral flora, with the most prevalent one being COPD or chronic obstructive pulmonary disease. This affects about 14 million people in the United States, making it the fourth cause of death in 1991, which continues to rise over the years. The primary etiological factor is smoking of tobacco, but it cannot be discounted that bacteria (which also includes oral bacteria) may possibly have a hand in the disease progression.

Since there is a high prevalence for lower respiratory tract infection, or RTI, the number of attributed pulmonary infections is still significant even if a partial percentage of the reported cases are facilitated or caused by oral flora.

**INFECTION MECHANISM**

In order for oral microorganisms to get to the lower respiratory tract, two avenues exist: aspiration and hematogenous spread. Hematogenous spread may occur after mere prophylactic procedures and is an adverse effect that is inevitable in some dental procedures. The actually journey of this infection is rare, however, dating only a few well-documented case reports to date.

For both cases, the most likely and common source of periodontal anaerobe-associated pulmonary infection was hematogenous spread. Contradictory to this, material aspiration from the upper
airway takes place in around 45% of normal and healthy subjects mid-sleep and around 70% in subjects who undergo impaired consciousness, and is probably the primary cause for nosocomial infection coupled with gastric content aspiration.

Three infection mechanisms associated with material aspiration from the upper airway can be seen. Poor oral health or periodontal disease may first result in a higher oral pathogen concentration in the saliva. These are then aspirated into the lungs, which would overwhelm the defensive immune system. Second, under certain specific conditions, facilitation of dental plaque may take place, harboring pulmonary pathogen colonies and cultivating their growth. Lastly, cultivation of pulmonary pathogens in the upper airways may be stimulated by periodontal pathogens.

**MICROBIOLOGICAL SIMILARITIES BETWEEN ORGANISMS**

Most pulmonary diseases are due largely to aerobic bacteria that may be found in non-oral disease related oral flora. On the other hand, the list of obligate or facultative anaerobes that are found in periodontal tissue destruction that have been found in infected lungs is quite great in number. For example, Fusobacterium nucleatum and Actinobacillus actinomycescomitans both underwent isolation from infected lungs while the known pulmonary pathogen, Pseudomonas aeruginosa, was isolated from cases of “refractory” periodontitis patients. In a simulating aspiration done on an animal model, Bacteroides gingivalis saw a confirmation for its pulmonary pathogenicity.

Oral commensal microorganisms like the Streptococcus intermedius may become potential pathogens when exposed under certain circumstances. Four studies have attempted to investigate the oral flora colonization by respiratory pathogens. Lindemann et al. isolated bacteria strains of P. aeruginosa taken from 14 out of 20 patients who suffered from cystic fibrosis. None of the 20 healthy patients acquired it, and the 40 samples of plaque that were taken from patients with cystic fibrosis and controls did not acquire the strains either.

In a study done within a critical care ward, however, Scannapieco et al. were able to pick a number of well-known pulmonary pathogens such as the Serratia marcescens and Klebsiella pneumoniae out of dental plaque. On the other hand, plaque samples that were collected from dental clinic patients...
showed no cultivation of the previously mentioned strains. In another study, Fourrier et al. located a high bacterial concordance between saliva, dental plaque and tracheal samples.

In a population of intensive care patients, around 40% possessed aerobic respiratory pathogen colonization in dental plaque. This colonization in the dental plaque was highly indicative of subsequent or concurrent nosocomial infection. In a recent study, colonization of plaque in chronic care home subjects was compared alongside another group of age-matched outpatients with plaque colonization within the dental clinic setting. About one quarter of samples were colonized for both groups, but the chronic care home showed a higher bacteria concentration in its subjects. Two the studies included edentulous participants.

Some species such as the Porphyromonas gingivalis and the A. actinomycetemcomitans are usually unable to detect after dental clearance. However, other pulmonary pathogens that are putative like the Prevotella spp. may be seen in edentulous patients’ oral microbiota.

In the case of inadequate denture hygiene, denture cleanliness and anaerobic bacterial count are inversely proportional. Also, the Candida albicans has been picked out from the transtracheal aspirates of pleuropulmonary-infected patients, as well as in other pulmonary samples. Nonetheless, RTI seems to be less frequent within edentulous patients.

**PERIODONTAL PATHOGENS AND MUCOSAL COLONIZATION**

The protein that layers the oral mucosa, called Fibronectin, is possibly involved in the mucosal flora ecology through equipping the oral streptococci with binding sites while preventing the adhesion of more destructive bacteria. The degrading ability of bacteria towards fibronectin seems to be the mode of competition for colonization. The process of protease of periodontal pathogens, which also includes fibronectin degradation, has been studied extensively and linked to poor oral health. Hence, fibronectin activity taking place in the cervical fluid may play a role in the development of pneumonia through adhesion of gram-negative anaerobic bacteria to the upper airway epithelium. Mylotte and Scannapieco proposed to explain the oral cavity colonization by respiratory pathogens through a model.
Sufficient evidence supporting the epidemiological indications that microorganisms and lung infections are associated is still lacking. Pneumonia occurs most frequently in the elderly, which would make the segment a most logical area of concentration for the research. However, studying pneumonia in the elderly proves to be a bit difficult because of certain specific features. Case in point, standard clinical signs that are telling of a pneumonia diagnosis (such as fever, cough and abnormal auscultation) are lacking when it comes to specificity and sensitivity. Another standard for diagnosis is chest radiography but it is hardly available. Additionally, the elderly have risk factors or comorbidities that may negatively impact the relationship between pneumonia and oral disorders. For example, mediocre functional status and poor nutritional status automatically heighten the risks for pulmonary disease, at the same time, they are associated with poor periodontal health.

Another obvious confounder to periodontal and respiratory health is smoking. It is, however, difficult to obtain an accurate history of smoking from elderly patients. Three studies have been conducted in order to investigate the association between oral health and pneumonia in elderly patients. Terpenning et al. found lower pneumonia prevalence among edentulous elderly people and a link between its development and xerostomia. A cross-sectional study of elderly patients who are frail and living in chronic care facilities investigated the residents’ medical records in order to identify RTI episodes that took place within the one-year period which preceded an oral examination. RTI prevalence was significantly greater in dentate as compared to edentulous subjects, garnering a 40% against a 27%, and was also seen to be greater in subjects with certain oral disorders as compared to those without. The presence of certain oral complications contributed with the nutritional status or the dependency degree of the subjects in order to increase RTI prevalence. Additionally,
the dentate subjects with RTI history had plaque scores that were higher than those without previous RTI episodes. The RTI difference between dentate and edentulous subjects cannot be used as an indication for whether or not the remaining teeth should be removed. Even the prevalence difference of 13% that was observed cannot be used in justifying such an act, given that edentulousness has negative impacts on quality of life and nutrition. In a recent study, cariogenic bacteria, dental decay and periodontal pathogens all surfaced as significant risk factors for pneumonia aspiration after adjusting for known risk factors.

COPD AND OTHER CHRONIC PULMONARY CONDITIONS

Scannapieco et al. found no sufficient evidence to support the linkage between poor periodontal health and acute respiratory diseases in the adult population of the United States. A report was however released regarding patients with COPD. An investigation by Scannapieco and Ho reported that there is sufficient evidence to link periodontal attachment loss and COPD. COPD frequency increased depending on the severity of attachment loss, with frequency diminishing for lung function as the amount of attachment loss increased.
Rheumatoid arthritis is a systemic disorder in which one’s own body attacks its own healthy tissues and cells. Rheumatoid arthritis patients are found to have inflamed membranes around the joints that release enzymes, eventually causing the bone and cartilage to wear away. In cases that are more severe, the body organs and other tissues may also be affected. Previous studies have found that rheumatoid arthritis patients are at a greater risk for fracture and bone loss.

Patients with rheumatoid arthritis find themselves at a greater risk for osteoporosis for a number of reasons. To start, the glucocorticoid medications that are often prescribed for RA may trigger bone loss. The enzyme Tissue Necrosis Factor Alpha (TNFa) has been known to cause bone loss systemically and orally, and is also known to destroy osteoclasts. This enzyme can usually only be found in patients with RA.

As earlier stated, RA can cause inflammation in the tissues and joints as well as in other organs of the body. The immune system has a tendency to attack its own bodily tissues by mistake, which leads to autoimmune diseases taking place. There are different organizations of cells and antibodies in the immune system that help fight detrimental body toxins. People who are stricken with autoimmune diseases, however, possess antibodies in the bloodstream that attack bodily tissues, in turn causing inflammation. Rheumatoid arthritis is otherwise known as rheumatoid disease because of its tendency to affect other organs in the body.

Symptoms of rheumatoid arthritis may not be apparent for some time but the disease is progressive and may
therefore result in the destruction of joints and functional disability.

Rheumatoid arthritis has a range of symptoms that go from serious to mild. The more obvious ones are apparent in the joints of the hands, wrists, knees and feet. Its association to oral health is not yet known. The disease characteristically may have varied effects on other organs such as the lungs, kidneys and tissues found around the heart.

To this day, RA has no cure, and is only kept at bay by different types of anti-inflammatory medications that are used to relieve pain and inflammation caused by rheumatoid arthritis. Aspirin is a common and effective type of anti-inflammatory. Other anti-inflammatory, non-steroidal drugs include ibuprofen and naproxophen. In order to control inflammation, it is recommended to take prednisone. However, it may cause various side effects that include an increased fluid pressure on the brain, apart from others such as infection and diabetes. Physicians usually inject steroids and cortisone in order to relieve joints quickly. Medications that are anti-rheumatic may modify diseases such as gold, methotrexate and hydroxychloroquine, which delay the progression of the disease. The use of these medications however may have severe side effects.

**RHEUMATOID ARTHRITIS AND ORAL HEALTH**

Little is still known about the effects of rheumatoid arthritis on oral health; however, the association has already been established by previous studies. Patients who suffer from rheumatoid arthritis can develop serious tooth decay and gum disease because of the swollen and inflamed joints at the wrists and hands may be unable to maintain oral hygiene to its optimum, since it may be tedious and painful. Electric toothbrushes with larger and more comfortable grips are recommended. Patients who suffer from RA may also have difficulty flossing and should be recommended interdental cleaning.

*Figure 24. An illustration showing the effects of rheumatoid arthritis*
RA usually affects the salivary gland, which in turn may cause inflammation and dryness. RA patients may also experience xerostomia or dry mouth and keratoconjunctivitis sicca or dry eye. Natural ways to treat these complications include chewing sugar-free gum, sucking on sugar-free hard candies and drinking plenty of water in order to stimulate the salivary glands. Tooth decay will take place if the saliva is not stimulated, as this is responsible for some antibacterial properties as well as cleansing. Products that are high in fluoride should be recommended, as well as lessening sugar consumption and practicing good oral hygiene daily.

Additionally, patients who are stricken with arthritis of the temporomandibular joint, which is the joint found between the mandible and the skull’s temporal bone, is one of the causes of temporomandibular joint dysfunction. There is a cartilage disk that separates the mandibular condyle from the skull. When the cartilage disk starts to degenerate, an altered occlusion and pain taking place in the mandible or discomfort in the joints may be experienced. Most temporomandibular disorder patients usually seek treatment because of pain, with the subset of main symptoms being clicking, popping and other such noises whenever the joint is set in motion. Severe arthritis of this joint may prevent the patient from opening the mouth wide enough.
Recent studies have shown that *Helicobacter pylori* (*H. pylori*) strains that have comparable virulence markers have the ability to induce various types of gastric injury and inflammation. The complete *H. pylori* genome microarray unveiled differences in the *H. pylori* strains’ ability to induce responses from epithelial cells, drawing a connection to inflammation.

*H. pylori* has previously been associated with gastric cancer and peptic ulcer development. Whether bacterium resides in the cavity as a permanent reservoir or not is still unknown, despite having proven that bacterium may be transmitted through the oral cavity. One study had set out to study this using nested polymerase chain reaction, or PCR.

Through culture technique, *H. pylori* in the oral cavity was rarely detected. However, nested PCR detected its frequent existence in the oral cavity, registering at about 35.1%, especially within periodontitis patients with the bacterium in their gastrointestinal tract, registering at about 46.4%. In a pool of subjects who have harbored *H. pylori* in the duodenum or the stomach, 91% of participants without pockets and 41.2% of participants who had periodontal pockets that were greater or equal to 4 mm were found to have *H. pylori* in their dental plaque, although a significant difference statistically was not seen. A patient with periodontal pockets continued to cultivate *H. pylori* in the oral cavity despite bacterium eradication from the duodenum and stomach. 80% of patients who have had *H. pylori* in their dental plaque also cultivated Bacteroides forsythus in their oral cavity.

In the past, peptic ulcers were believed to have been caused by acidic foods or stress, replaced by the discovery of the *H. pylori*. *H. pylori* can ruin the protective coat of the stomach, allowing the natural acids in the digestive system to damage the stomach lining which then allows for the creation of a sore. Its most common symptom is a painful burning sensation in the stomach. Treatment of peptic ulcers usually calls for bismuth subsalicylate such as Pepto-Bismol. Although temporary, bismuth subsalicylate may cause a blackening of the tongue surface as a side effect.
Patients who are recovering from an oral surgery or are plainly experiencing pain in their mouth may be prescribed non-steroidal anti-inflammatory drugs, or NSAIDs. Examples of this would be ibuprofen, aspirin, or naproxen. Although peptic ulcers are primarily caused by *H. pylori*, prolonged usage of NSAIDs may also become a source of peptic ulcers due to their hindering the stomach from protecting itself against digestive juices that are primarily acidic. Ulcers may heal by themselves after stopping NSAID intake. Some peptic ulcer drugs have been known to produce dryness of the mouth, blackening and growth of hair on the tongue, and taste change.

In a cross-sectional large-scale study of a Japanese working population, statistically significant linkages between three periodontal disease indices (actual periodontitis diagnosis, loss of five or more teeth, score of periodontal risk, etc.) and peptic ulcer were demonstrated. Out of 28,765 eligible participants who were analyzed, 397 or 1.4% had peptic ulcer. As previously mentioned, a meta-analysis led by Sridhar, Huang, and Hunt in 2002 demonstrated that both NSAID use and *H. pylori* infection increased ulcer bleeding and peptic ulcer risks, and that the effects of it were synergetic. Other factors such as genetic polymorphism and socio-economic status may possibly play roles in peptic ulcer development as well.

There have previously been four studies that have examined the association between peptic ulcers and periodontal diseases, including duodenal and stomach ulcers. Two of these studies reported a significant association statistics-wise between peptic ulcers and periodontal disease.

A chart review conducted by Molloy *et al.* back in 2004 studied 2,006 randomly selected patients from over 13,000 patients of dental clinics in the University of Minnesota. After adjusting for sex, age, and whether they smoke or not, a significantly greater number of stomach ulcers were self-reported in direct proportion to the number of missing teeth. It was also found that participants with a more severe case of alveolar bone loss have an increased likelihood of having stomach ulcer. In 2005, Abnet *et al.* found a significant linkage was also found between self-reported duodenal and peptic ulcer and tooth loss in 29,124 Finnish
subjects who smoked, after appropriate adjustments for age. In contrast, Namiot et al. found little to no significant linkage between peptic ulcer and periodontal diseases. Conducted in Poland, the study showed that the periodontal index and the quantity of natural teeth do not have significant differences in a sample population of 93 patients with peptic ulcers and 93 gender and age-matched dyspeptic controls. In the aforementioned study, the entire population of 186 subjects had the H. pylori strain, including only peptic ulcer (unrelated to NSAIDs) patients as cases. A study conducted by Khader et al. in 2003 showed a mixed outcome. Their cross-sectional study included 603 patients who attended a northern Jordan dental teaching clinic. In this study, increased clinical attachment was significantly associated with self-reported cases of peptic ulcer, after the appropriate adjustments made for plaque index, smoking, brushing, age, as well as chronic disease such as hypertension, diabetes and allergy. No significant association was, however, found between probing depth, tooth loss, or gingival recession and peptic ulcers.

Possible associations between peptic ulcers and periodontal diseases have been proposed with several mechanisms. Several researchers have pointed out that H. pylori could potentially treat the oral cavity as a reservoir, which would possibly make it vulnerable to infection and re-infection. Dowsett & Kowollik have found that H. pylori has been detected in saliva and dental plaque, and that the gastric eradication success rate has been seen as being significantly associated to bacteria prevalence in the oral cavity. In the United States, a sizable cross-sectional study was conducted, finding that periodontal pockets that measure a depth of 5mm or greater were associated with an increased risk of H. pylori seropositivity. The insignificant association between peptic ulcers and periodontal diseases carried out by Namiot et al. may possibly be due to the fact that all their participants in both controlled and non-controlled cases had the H. pylori strain. Also, it was reported that polymorphism seen in certain genes that induce inflammatory responses may have an effect on periodontal disease severity as well as gastroduodenal disease risks. Peptic ulcer and periodontal disease are both inflammatory diseases,
which makes a patient’s below-optimal capacity for handling inflammation to contribute to disease progression.

As with most research work, several limitations were present. There is speculation that the association made between peptic ulcer and age in some studies may only reflect the fact that *H. pylori* is more prevalent in older generations. Some studies also make use of a cross-sectional design that fails to take temporal relationships into account, given that both peptic ulcers and periodontal diseases are chronic diseases. Another limitation is that of data that is self-reported, making use of self-reported indices in the place of periodontal measurements and clinical dental examinations. Studies that attempted validation for self-reported periodontal disease data came back with a mixed outcome. Medical conditions such as the absence or presence of peptic ulcer, diabetes and hypertension were also data that were self-reported, save for a Japan study that claimed to be generally accurate.

However modest previous studies have been, additional efforts to analyze statistically significant linkages between the existence of peptic ulcers and certain evaluation indices for periodontal disease (actual periodontitis diagnosis, loss of five or more teeth, score of periodontal risk, etc.) have to be made. Clinical dental examination in longitudinal studies must be made in order to further crystalize the association.
Bisphosphonate-associated Osteonecrosis of the Jaw

Osteonecrosis is a type of disease wherein a reduction of blood flow to bones in joints take place. Typically, bone is replaced by bone in a healthy functioning bodily system. In the case of osteonecrosis, however, the lack of blood supply breaks down bones faster than the body can replace it. The bone is put at a risk of breaking down and eventually dying.

Osteonecrosis may be experienced in one or several bone areas, most commonly at the upper leg. Other common areas are the knees, upper thighs, ankles and shoulders. Osteonecrosis can affect both genders at any age, but it is most commonly reported in patients who are in ages thirties to sixties.

Symptoms may not be apparent at first but as the disease progresses, severe joint pains will start to arise. This may hinder the patient from moving or bending the affected joint easily. Causes for the disease are not fully known. Risk factors include alcohol abuse, injuries in the joints, long-term treatment involving steroids, and certain diseases that include cancer and arthritis. In order to diagnose osteonecrosis, doctors conduct imaging tests among other test variations. Other treatments include using crutches, limiting joint-stressing activities, surgery, medicines and electrical stimulation.

Basing from case histories, there has been evidence regarding bisphosphonate-associated osteonecrosis (BON) that occurs in the jaw. This is a chronic and often painful condition that does not respond well to resolution and treatment. Using bisphosphonates in order to inhibit activity in the osteoclast in patients who suffer from osteoporosis or metastatic cancer of the bone has been seen as a standard treatment. IV bisphosphonates such as pamidronate (Aredia) and zoledronate (Reclast and Zometa) are usually prescribed in order to keep the cancer cells from spreading and to
keep hypercalcemias in malignancies at bay. The cumulative prevalence of BON is at around 0.8%-12% in patients who use IV bisphosphonates. The bones of the jaw are put at a greater risk because the mandible has a 2.5 to 10 times higher turnover rate as compared to the tibia. Vascular bed disruption and prolonged bisphosphonate half-life are seen as contributing factors.

Jaw osteonecrosis is a rare but severe adverse occurrence that is associated with intravenous and oral therapy using bisphosphonate. A confirmed reported case of jaw-osteonecrosis that is bisphosphonate-associated is determined by exposed bone in the maxillofacial area that ceases to heal at around eight weeks post-diagnosis, given that the patient has not had any form of radiation therapy in the craniofacial region while taking bisphosphonate therapy.

In the past several years, case reports, letters and small jaw osteonecrosis case series have been published in maxillofacial surgery, dental, general medical and oncology literature. Patients who suffer from osteonecrosis of the jaw typically report jaw pain, oftentimes in the mandible rather than in the maxilla, as well as associated exposed bone.

Sixty percent (60%) of jaw osteonecrosis cases occur after dental implantation, dental extraction, root canal surgery, and other dentoalveolar surgeries, whereas all the other cases usually appear as spontaneously occurring. Ninety-four percent (94%) of the cases today have taken place in patients who were treated with IV bisphosphonates, 85% were treated for cancer using one or more IV bisphosphonates that contain nitrogen, usually once a month for continuous several years. The greatest risk for developing jaw osteonecrosis has been reported to be associated with frequent (typically on a monthly basis) infusions of IV zoledronic acid, which is used for patients with prostate or breast cancer, and multiple myeloma.

Mayo Clinic’s Department of Dental Specialties-Prosthodontics in Rochester, Minnesota’s Sreenivas Koka, D.D.S., Ph.D., explains that osteonecrosis of the jaw is usually characterized by an
intraoral lesion combined with exposed areas of whitish-yellowish hard bone. Sometimes it is associated with intraoral and extraoral sinus tracts and is characterized by a delayed healing response, with the bones staying exposed when it is supposed to be covered by mucosal or gingival tissue eight weeks post-diagnosis. Painful ulcers may also be present in the soft tissues that are adjacent to the bony areas of the lesion.

Dental radiographs do not usually provide much help in early-determined cases. Advanced cases may, however, show moth-eaten radiolucency areas with or without radiopaque bone sequestra. Development of jaw osteonecrosis may also be associated with surgical or dental trauma. For advanced cases, there may be occurrences of pathological jaw fractures or part of the maxilla or mandible may have to be removed.

Bart L. Clarke, M.D., of Mayo Clinic’s Division of Endocrinology, Diabetes, Metabolism, and Nutrition in Rochester, Minnesota, says that frequent IV infuses of bisphosphonates that contain nitrogen, cancer, and dentoalveolar trauma are all main risk factors.

However, patients who are receiving oral bisphosphonate therapy, such as risedronate, ibandronate, and alendronate, for cancer-free postmenopausal osteoporosis do not have identified risk factors as of yet due to the minimal number of published cases. Alendronate therapy was associated to most of the cases, most likely because of its wider reach as compared to the use of other oral bisphosphonates.

Dr. Clarke also points out that cases of jaw osteonecrosis that are associated with oral bisphosphonate seem to have low prevalence, estimated as 0.00038% or 3.8 cases in every million of one European database, unless jaw osteonecrosis cases have been grossly underreported. Jaw osteonecrosis that is IV bisphosphonate-associated in cancer-stricken patients appears to be more common, estimating at 0.8% to 10% in prevalence for the aforementioned study, and at 1% to 4% for general studies.
Pathophysiologic mechanisms that determine whether IV bisphosphonate treatments can cause osteonecrosis of the jaw have not been established yet. It has been hypothesized that the suppression of bone turnover that has been caused by bisphosphonate treatment may lead to fatigue damage accumulation through micro cracks, which may eventually lead to micro fractures.

Bisphosphonates are also known as potent angiogenesis inhibitors, which may lead to a degenerated healing ability. Infection or dental trauma may increase demand for the repair of bone micro damage, which potentially leads to localized osteonecrosis, despite the lack of accurate information as to how this occurs.

Dr. Koka further explains that in describing interventions or treatment for jaw osteonecrosis, randomized clinical trials have not been published. Patients who require a significantly traumatic dental procedure, such as dental implantations, tooth extractions, or root canals, should do so prior to starting intravenous or oral bisphosphonate therapy.

Dr. Koka continues to point out that the time for withdrawing bisphosphonates before a dental surgery in order to optimize it has not been established just yet, but a number of specialists advise therapy withdrawal at around three months before surgery. Nonetheless, nothing has been proven regarding the benefits and risks of the drug holiday, since bisphosphonates tend to have a lengthened half-life of at least several years in the bones. However, it is suggested that a pre-treatment and post-treatment course of antibiotics be given from one to seven days after the surgery if dental extraction or other types of dental surgery is deemed necessary. This antibiotic course has been suggested to reduce jaw osteonecrosis risks. Retrospective analyses encouragingly indicate that dental implants in patients who are receiving bisphosphonate therapy for osteopenia or osteoporosis are successful.

Effective therapy for jaw osteonecrosis in patients who receive IV or oral bisphosphonate therapy has not been established. Several specialists suggest supportive management, which starts with further dentoalveolar trauma avoidance, hyperbaric oxygen therapy use, proper oral antibiotic rinses use, sufficient healing time, and withdrawal from IV and oral bisphosphonate therapy. In a few instances, the condition may be exacerbated through surgery in order to debride bone that has died; however, pedicled, local soft tissue flaps and debridement have been reported as a healing stimulant in certain patients.
<table>
<thead>
<tr>
<th>Study, Year (Reference)</th>
<th>Patients, n</th>
<th>Sex, n</th>
<th>Primary Diagnosis</th>
<th>Sites</th>
<th>Previous Surgical Procedure, n(%)</th>
<th>Medications</th>
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<td>Zarychanski et al., 2006</td>
<td>2005 (25) 63</td>
<td>18 45</td>
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<td>Mandible (n = 39) Maxilla (n = 23) Both (n = 1)</td>
<td>5(86)</td>
<td>Pamidronate (n = 34) Zoledronic acid (n = 9) Pamidronate and zoledronic acid (n = 13) Alendronate (n = 5) Risedronate (n = 1) Alendronate and zoledronic acid (n = 1)</td>
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<tr>
<td>Meliorati et al., 2005 (28)</td>
<td>18 4 14</td>
<td></td>
<td>Myeloma (n = 62) Breast cancer (n = 50) Prostate cancer (n = 4) Osteoporosis (n = 3)</td>
<td>Mandible (n = 81) Maxilla (n = 33) Both (n = 5)</td>
<td>55 (46)</td>
<td>Zoledronic acid (n = 48) Pamidronate and zoledronic acid (n = 36) Pamidronate (n = 32) Alendronate (n = 1)</td>
</tr>
<tr>
<td>Estilo et al., 2004 (26)</td>
<td>13 4 9</td>
<td></td>
<td>Breast cancer (n = 9) Myeloma (n = 4)</td>
<td>Mandible (n = 6) Maxilla (n = 5) Both (n = 2)</td>
<td>9(69)</td>
<td>Intravenous forms, not specified</td>
</tr>
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<td>Marx et al., 2005 (27)</td>
<td>119 NS NS</td>
<td></td>
<td>Myeloma (n = 62) Breast cancer (n = 50) Prostate cancer (n = 4) Osteoporosis (n = 3)</td>
<td>Mandible (n = 81) Maxilla (n = 33) Both (n = 5)</td>
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<td>Zoledronic acid (n = 48) Pamidronate and zoledronic acid (n = 36) Pamidronate (n = 32) Alendronate (n = 1)</td>
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<td>Bagan et al., 2006 (30)</td>
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<td>Mandible (n = 11) Maxilla (n = 1) Both (n = 8)</td>
<td>11 (55)</td>
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<tr>
<td>Pires et al., 2005 (31)</td>
<td>12 9 3</td>
<td></td>
<td>Breast cancer (n = 6) Myeloma (n = 4) Prostate cancer (n = 1) Lung cancer (n = 1)</td>
<td>Mandible (n = 8) Maxilla (n = 3) Both (n = 1)</td>
<td>8(67)</td>
<td>Pamidronate and zoledronic acid (n = 9) Pamidronate (n = 4) Zoledronic acid (n = 3)</td>
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<td>Bamias et al., 2006 (32)</td>
<td>17 10 7</td>
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<td>Myeloma (n = 11) Prostate cancer (n = 3) Breast cancer (n = 2) Other neoplasm (n = 1)</td>
<td>Mandible (n = 14) Maxilla (n = 3) Mewilla (n = 3)</td>
<td>13 (76)</td>
<td>Pamidronate and zoledronic acid (n = 9) Zoledronic acid (n = 7) Zoledronic acid and ibandronate (n = 1)</td>
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<tr>
<td>Melo and Obeid, 2005 (33)</td>
<td>11 7 4</td>
<td></td>
<td>Breast cancer (n = 3) Myeloma (n = 7) Lung cancer (n = 1)</td>
<td>Mandible (n = 8) Maxilla (n = 2) Both (n = 1)</td>
<td>9(82)</td>
<td>Zoledronic acid (n = 4) Pamidronate (n = 4) Pamidronate and zoledronic acid (n = 3)</td>
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<tr>
<td>Zarychanski et al., 2006 (34)</td>
<td>12 7 5</td>
<td></td>
<td>Myeloma (n = 10) Breast cancer (n = 1) Renal cancer (n = 1)</td>
<td>Mandible (n = 10) Maxilla (n = 1) Both (n = 1)</td>
<td>7(58)</td>
<td>Pamidronate (n = 12)</td>
</tr>
<tr>
<td>Summary of studies with fewer than 10 patients (35-54)†</td>
<td>70 38 23</td>
<td></td>
<td>Myeloma (n = 29) Breast cancer (n = 26) Prostate cancer (n = 5) Paget disease (n = 3) Osteoporosis (n = 3) Lung cancer (n = 2) Lymphoma (n = 1) Mesothelioma (n = 1)</td>
<td>Mandible (n = 30) Maxilla (n = 14) Both (n = 9) Not assigned (n = 17)</td>
<td>44(63)</td>
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</tr>
</tbody>
</table>

* NS = not stated.
† Sex was not reported for 9 patients in these studies.
Dr. Clarke suggests that it is acceptable to begin or continue intravenous or oral bisphosphonate therapy for patients with the necessary indices until further significant clinical data surfaces, unless there is a presence or development of jaw osteonecrosis. Each patient should be given the ability to decide whether to continue treatment of frequent potent IV bisphosphonate infusions, with the proper help of their physicians. Patients who are still contemplating on whether to start osteoporosis prevention treatment using IV or oral bisphosphonates must be informed of the small risk of oral bisphosphonate-associated jaw osteonecrosis and the infrequent occurrence of IV bisphosphonate-associated jaw osteonecrosis risks.

Dental evaluation must be carried out on patients before starting IV bisphosphonate treatment, followed by a regular evaluation in order to ensure optimal health. Encouraging patients with concerns regarding jaw osteonecrosis who are currently on or contemplating taking oral bisphosphonates to talk to their respective dentists is a must.
Pre-term Low Birthweight Babies

Recent studies have found that women who have periodontal diseases were over three times more likely to give birth to premature babies than expecting mothers who maintain good oral health. There’s also a 25% chance that mothers with periodontal diseases will give birth before 35 weeks. Over a thousand pregnant women with a gestation between six and 20 weeks were observed for the study, with 160 participants having periodontal disease being compared to the 872 women with optimal oral health. Results showed that participants treated for gum diseases with root planning and scaling were less likely to deliver prematurely before 35 weeks.

Dr. Nigel Carter, who is the British Dental Health Foundation’s Chief Executive, believes that this would add evidence to the association between pre-term low birthweight babies and periodontal diseases. He states that this only reinforces the belief that pregnant women should include oral health and should seek appropriate treatment during gestation to reduce the chances of a pre-term delivery. He also pointed out that hormonal changes are normal during pregnancy, entailing a need to pay closer attention to women’s dental health during this time, especially when experiencing symptoms such as the gums bleeding more easily.

Pregnancy itself does not damage the teeth. Inadequate calcium intake by the expecting mother allows the body to take calcium from the bones’ (not the teeth) in order to provide for the baby, resulting into calcium loss that also affects the teeth, eventually paving the way for certain periodontal diseases.

Prematurely born babies are at a risk for a variety of health conditions, such as hearing and eyesight problems and cerebral palsy. Studies suggest that around 18 out of 100 premature deliveries may have been
triggered by periodontal disease. Some studies claim that timely dental treatment for expecting mothers may reduce premature birth risks by over 80%. Suggestions for personal oral hygiene adjustments include the use of fluoridated toothpaste, flossing, and regular dental checkups. Dental care may also be affected by the event of pregnancy through avoidance of standard procedures such as taking x-rays in order to prevent radiation exposure.

In pregnancy, gum problems may account for periodontal diseases. Hormonal changes during pregnancy may be associated with susceptibility to gum problems such as gingivitis or gum inflammation, which is likely to occur in the second trimester. Untreated or undiagnosed periodontal diseases, characterized by gum bleeding and swelling (also referred to as pregnancy epulis), particularly during flossing and brushing, may be worsened by pregnancy, possibly resulting into tooth loss.

Pregnancy hormones tend to soften the muscles that are responsible for containing food in the stomach. Gastric reflux or vomiting that is associated with pregnancy can coat the teeth in stomach acids, and repeatedly doing so may damage the tooth enamel, increasing tooth decay risk. Brushing the teeth immediately after vomiting may damage the tooth enamel, as it is still vulnerable to scratching. Professionals advise waiting at least an hour before doing so. Retching is also another response to attempts at maintaining optimal oral health when pregnant. Snacking on foods with high sugar content may also induce tooth decay.

Periodontal disease-associated anaerobic bacterial infections that are gram-negative are oftentimes seen in women who are of childbearing age. Today, recent techniques for measuring levels of matrix metalloproteinases (or MMPs) in amniotic fluid have allowed scientists to report that certain levels of MMPs rise while others decrease in response to spontaneous labor and premature membrane rupture. These observations reinforce the theory that MMPs are critical in the events that lead up to premature labor. The results support the theory that uterus infections in some mothers and their infants warrant an immune response, which sees premature labor as a consequence. Scientists can now try to identify genetic factors that allow for the predisposition of

**Figure 32. Taking care of your oral health may directly affect the fetus**
women to premature labor, prevent this from happening, and discover new ways to prevent heightened immune response in the long run. MMPs play a significant role in the inflammatory response of periodontal diseases, shedding more evidence to the theory that good oral health may lead to a healthy full-term pregnancy.
Oral and psychological health issues are intimately bound in as one’s psychological well-being may affect oral health and oral health may, in turn, also affect the psychological well-being of a person. Little known to many, there is the existence of odontophobia, or the psychological condition that depicts an overwhelming and irrational fear of dentistry. It is believed that around 75% of adults experience varying degrees of this phobia, or ‘dental fear’, with 5%-10% clinically suffering from odontophobia. Estimates suggest about 75 percent of adults experience some degree of ‘dental fear,’ with five to ten percent suffering from odontophobia.

From childhood, various sources of dental stories may have triggered a certain degree of dental fear. Children have been known to absorb and take to heart information learned during their formation years, that general dental fear among adults may have planted fearful discontent in them.

**Figure 33.** Diagram showing the cycle between dentistry and social and psychological aspects
would usually require dentists to master a certain degree of psychological understanding and implementation and assume the role of a psychiatrist in order to help patients overcome one of their strongest primal emotions: fear. Fear is created out of the perception that one is helpless or has little or no control over a situation. These are perceptions that stem from primitive or limbic emotional system and therefore require logical and reasonable executive cognition levels in order to overcome.

Such dental fears may manifest themselves into chronic stress, which then affects the oral health through dry mouth or xerostomia, or through hyper-salivation. This may cause a disruption in the pH levels of the saliva, in turn raising the acid levels in it, eventually causing the gradual decay of the tooth. Xerostomia affects the bacterial growth in the mouth. Studies have shown that a number of medications prescribed in order to reduce stress may actually bring about xerostomia.

Agoraphobia, which is defined as the fear of leaving home, prevents patients from attaining proper oral care, not only for the routine ones but most importantly for urgent concerns such as the need for dental surgery.

Numerous unaddressed psychological issues usually result in patients subduing themselves to fewer routine dental checkups, if any at all. Patients with depressive issues or histories may neglect oral health in general – an action detrimental to oral health that may eventually result into a further vicious depressive cycle. Patients whose smiles have degenerated may suffer from low self-esteem, which in turn may cause them to limit or completely withdraw from their healthy social activities, which may also reinforce depression.

Patients with more severe psychological cases do not even understand what dental care is and why they are receiving it. Such patients would usually require a guardian to look after them and carry out even the simplest of oral health care tasks such as brushing of the teeth. Lack of understanding breeds fear, which may create continuous conflict between the patient and the guardian. Alzheimer’s and MMI (mild memory impairment), which is a stage of dementia that is pre-clinical, are some
of the more severe psychological cases that may hinder oral health. A team of neuroscientists conducted a recent study, reporting that certain dental markers exist that could possibly be indications of these two diseases. While the hypothesis is not entirely proven, there is significant evidence to show an association between dental markers and psychological diseases that is enough to render further gathering of information and study.

A theory of theirs is that inflammation caused by periodontal diseases may find their way to the brain, affecting the hippocampus in the process. Experiments carried out on rats showed that the number of teeth they had was directly proportional to the deterioration of their cerebral cortex, which was originally thought of as being associated with fewer instances of chewing, therefore reducing sensory input.
Conclusion

The idea that oral conditions can affect events elsewhere in the systemic health is not entirely new, however it has gone through numerous iterations in the past. An early publication that was cited only recently was a report done in 1891 by Miller called “The Human Mouth as a Focus of Infection.”

At the beginning of the late 1980s, an array of publications were released tackling the association between systemic conditions and periodontitis, with enthusiastic interest especially in coronary heart disease, and to a feign degree, preterm and low birth weight, as well as stroke, all of which made the dental profession stand to attention. In a way, this may be construed as a reference to the focal infection theory. However, the medical and dental profession responses were more significantly measured as compared to that of the early 20th century.

This is most probably the outcome of several factors: as statistical analysis and scientific investigations grow more sophisticated, including understanding epidemiologic study limits in determining disease causality; a significantly more enhanced understanding of the pathogenesis and etiology of systemic diseases and periodontal diseases that pave the way for a thorough assessment of the putative interactions’ biological plausibility; the availability of proven methods for treating endodontic lesions and periodontal diseases; and the recognition of the association of bacteria and certain diseases that have an uncertain etiology, such as evidence proving the etiologic role of H. pylori in gastric ulcer development, which is not an accurate analogy but a useful one anyway.

In reviewing existing data, it is crucial to differentiate between data that supports a linkage between two conditions or diseases and those that indicate a causal relationship, in order to interpret information accurately. Despite oral microorganisms from different sites potentially becoming associated with various systemic diseases, this e-book has focused on the association between periodontal disease and diabetes mellitus, oral cancer, cardiovascular diseases, respiratory diseases, rheumatoid arthritis and osteoporosis, peptic ulcers, bisphosphonate-associated osteonecrosis of the jaw, pre-term low birth weight babies and psychological well-being. As the interest in the linkage between periodontal diseases and systemic health heightens, it is safe to anticipate the time when it
is confirmed that oral health and dental care play a significant role in ensuring the overall health of patients.

Despite the lack of evidence to concretely announce an association, it is without doubt that the oral cavity is closely linked to systemic health. A significant amount of evidence points to the presence of blood inflammation as a signal for an increased risk of an array of diseases. In the long run, it is crucial that dental health care providers make use of diagnostic tests in order to identify patients who are at a greater risk for diseases apart from dental ones. Standardizing thorough patient medical history review before every dental session should be more strictly implemented in the future. Patients who have 5mm or greater periodontal pockets should be recommended root planing and scaling. Today, deep periodontal pockets may be treated using a variety of locally applied antimicrobials like Atridox, PerioChip and Arestin. In the case of patients who use tobacco, suffer from chronic periodontitis, or are immunocompromised, host modulation therapy has shown great evidence of an increased patient response towards nonsurgical periodontal treatment.

Doses of the subantimicrobial doxycycline (Periostat or SDD) may be used for definitive root planing and scaling as an adjunct. SDD use as an adjunct is supported by strong evidence towards conventional therapy when it comes to managing chronic periodontitis. When a medical concern is found, indication of a primary care physician referral may be found. Dental auxiliaries, dental hygienists, dentists, as well as other members of the dental staff must be kept up-to-date regarding the latest dental research by reading professional journals, attending conferences, and regularly checking dental and medical websites.

Research on the relationship between systemic diseases and oral health is still currently underway, and is growing in numbers by the day. Knowledge attained through received information constantly changes as attempts at researching and studying the association progresses.
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