waterpik®

RISK-BASED PERIODONTAL THERAPY

Course #18-30

DISCLOSURE STATEMENT

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- This course was designed, developed, and produced by Water Pik, Inc.
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COURSE OBJECTIVE

To provide the dental team with general and oral health information and related research needed to help practitioners screen, identify and treat those with or at risk for periodontal disease.

LEARNING OUTCOMES

- Discuss new AAP/EFP periodontal classification system
- Discuss the effect the Water Flosser has on plaque biofilm and inflammation
- Identify key risk factors and clinical conditions and integrate findings in oral and general health assessments
- Detail the benefits of non-surgical and maintenance therapy
- Recommend appropriate self-care products

INTRODUCTION

Periodontal disease is one of the most common chronic diseases of adults. Newly released data from a six year national surveillance study of more than 10,000 people found the overall prevalence of periodontal disease in dentate adults age 30 and over is 42%.[1] The percentage of people affected increases as people age with periodontal disease present in nearly 60% of people 65 years and older. Total prevalence was greater in men (50.2%) than women (34.6%) and in Mexican Americans (59.7%) compared to other race and ethnicity. **(Table 1)**.

Periodontitis is a multifactorial disease. It shares many of the same common risk factors as other chronic illnesses including tobacco use and socio-economic status. Prevalence of periodontitis among current tobacco users is 62.4%.[2] Markers of economic status showed that total prevalence was greater in those with low income (59%), six or more missing teeth (68.6%), and more than 12 months since a dental visit (54.8%).[2] **(Table 1)**

Like other chronic diseases, periodontal disease and subsequent tooth loss affect both quality of life and potentially longevity. Two systematic reviews looked at the impact of having periodontal disease on quality of life.[2,3] Both found that periodontal disease was associated with a negative quality of life. Severe periodontal disease had the biggest compromising impact related to function and esthetics.[2,3] In regards to longevity, a 2017 study of 57,000 postmenopausal women found that both edentulism and periodontal disease were associated with a higher mortality rate.[4]

Table 1: Prevalence Periodontal Disease by Age and Demographics*

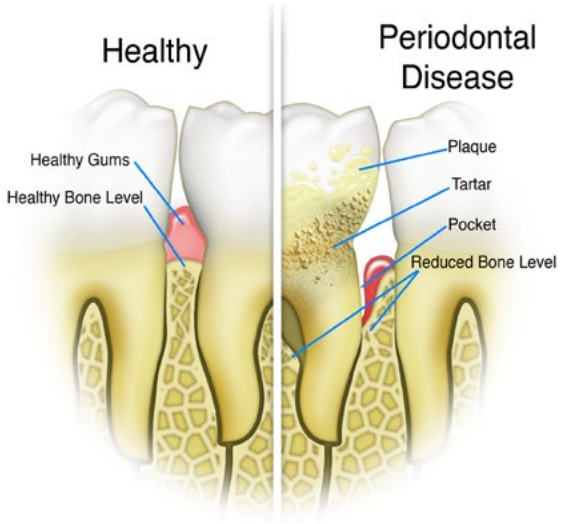
Age Group	**Mild/Moderate**	**Severe**	**Total**
Ages 30-44	25.3%	4.1%	29.5%
Ages 45-64	35.6%	10.4%	46.0%
Ages 65 & over	50.7%	9.0%	59.8%
Overall Prevalence	34.4%	7.8%	42.2%
Gender	**Mild/Moderate**	**Severe**	**Total**
Men	38.8%	11.5%	50.2%
Women	30.2%	4.3%	34.6%
Race/Ethnicity	**Mild/Moderate**	**Severe**	**Total**
Mexican Americans	46.4%	13.4%	59.7%
Other Hispanic	40.7%	7.8%	48.5%
Non-Hispanic black	42.0%	14.7%	56.6%
Non-Hispanic white	31.1%	5.9%	37%
Other races/ multiracial	36.9%	9.3%	46.2%
Smoking Status	**Mild/Moderate**	**Severe**	**Total**
Non Smoker	29.5%	4.9%	34.4%
Former Smoker	37.7%	8.0%	46.2%
Current Smoker	45.4%	16.9%	62.4%
Socioeconomics	**Mild/Moderate**	**Severe**	**Total**
Low Income	45.2%	13.8%	59.0%
Middle Income	40.0%	8.6%	48.5%
High Income	25.3%	4.5%	29.7%
Diabetes	**Mild/Moderate**	**Severe**	**Total**
Yes	49.0%	10.8%	59.9%
No	32.3%	7.5%	40.4%
Dental Floss Use	**Mild/Moderate**	**Severe**	**Total**
In Past 7 Days	32.1%	5.8%	37.9%
Not in the Past 7 Days	40.3%	12.8%	53.1%
Dental Visits	**Mild/Moderate**	**Severe**	**Total**
Last 6 mos	26.4%	3.9%	30.3%
Between 6 & 12 mos	31.6%	6.3%	37.9%
More than 12 mos	41.5%	13.3%	54.8%
Missing Teeth	**Mild/Moderate**	**Severe**	**Total**
0	20.9%	2.6%	23.5%
1-5	36.0%	7.0%	43.0%
6-27	51.5%	17.1%	68.6%

*Adapted from Eke PU et al

Most periodontal disease is preventable. Early detection, treatment, and modification of risk factors can help stop or slow the progression of the disease including reducing the risk for tooth loss. The establishment and maintenance of a healthy periodontium contributes to a better quality of life. The American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) recently launched a new classification system designed to help practitioners identify patients with periodontal diseases, especially those with early disease and predict their response to treatment.

WHAT CONSTITUTES PERIODONTAL HEALTH

Screening people for periodontal disease includes tools and measures such as radiographs, probing depth, subgingival biofilm, bleeding/inflammation and tooth mobility.[5] This provides a good indication of disease state or potential risk for disease. Conversely little has been written about how periodontal health should be defined.[5,6] From the practitioner side of the chair, it is likely about the absence of disease. Eliminating bleeding and inflammation



along with stable probing depth and good oral hygiene are common measures. Patients may have a different perspective. Tooth loss affects quality of life so measures such as the ability and confidence to eat, talk, and smile may resonate more with them than gingival bleeding.[6]

Lang and Bartold have proposed that periodontal health should be defined as a state free from inflammatory periodontal diseases. The justification is that there needs to be a common reference point for assessing periodontal disease and determining meaningful outcomes. They also note that for most of the population maintaining good periodontal health with no adverse changes is implausible for most people.[5] Therefore, they have suggested four levels of periodontal health based on the state of the periodontium and treatment outcomes.[5] **(Table 2)**

Table 2: Four Levels of Periodontal Health

Level	Periodontal Structure
1) Pristine periodontal health	Structurally sound and no inflammation
2) Well-maintained clinical periodontal health	Structurally and clinically sound
3) Periodontal disease stability	Reduced periodontium
4) Periodontal disease remission/control	Reduced periodontium/tooth loss

Pristine periodontal health is rare but possible. It means no bleeding upon probing or inflammation, normal sulcus depth and bone height. In comparison, it is more achievable for people to have good clinical periodontal health. This means that there is little to no bleeding or inflammation along with normal sulcus depth and bone height. People treated for periodontal disease are most likely to be in the disease stability or disease remission category. A characteristic of people in these categories is the presence of systemic modifying or predisposing risk factors especially use of tobacco or uncontrolled diabetes. People with stable periodontitis have minimal bleeding but may have residual or less than ideal probing depth and/or bone loss. Any predisposing or modifying risk factors are controlled. In comparison, someone whose periodontal disease is in remission or controlled may have continued bleeding/inflammation, deeper probing depths, bone loss, tooth loss, and uncontrolled risk factors.[5]

A NEW SYSTEM FOR STAGING AND GRADING CASES OF CHRONIC PERIODONTAL DISEASE

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions has resulted in a re-design of the periodontal disease classification. The American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) worked together to develop the system. It includes multi-dimensional staging and grading to allow for a more comprehensive, sophisticated, and personalized approach to the identification, treatment, and arrestment of periodontal disease. A key element of a case definition of periodontitis is having detectable interdental clinical attachment loss (CAL). Within the context of clinical care this represents interdental CAL at on non-adjacent teeth or buccal or lingual CAL ≥ 3 mm with pocketing > 3mm detectable at ≥2 teeth.[7]

The new system defines four stages of periodontitis ranging from an initial case to advanced periodontitis with extensive tooth loss and the potential for loss of dentition. Staging allows practitioners to evaluate periodontitis patients by more criteria than past bone loss. This is crucial for the early identification of periodontitis. Another benefit is that it helps practitioners determine those that may respond to a conventional treatment approach as well as patients that may need more intensive treatment.[7]

The essential criteria for staging are severity, complexity, and extent/distribution of disease. Assessment of severity includes levels of interdental CAL, radiograph bone loss (RBL), and tooth loss. Complexity takes into consideration that some cases are more difficult to manage. These include cases with probing depths deeper than six millimeters, vertical bone loss, furcation involvements and ridge defects. Extent of disease refers to the

number of teeth involved. In general a localized case would involve less than 30% of teeth and generalized greater than 30%.[7]

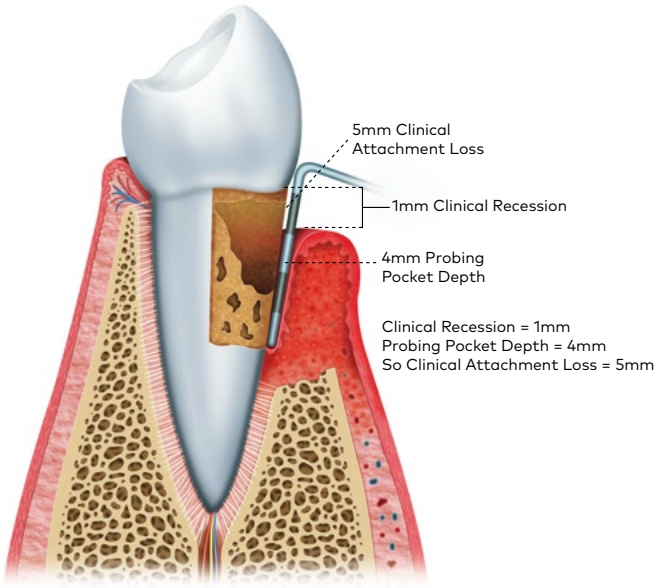
The second aspect of the classification system is grading. Grading takes into account that periodontitis no matter what stage may progress at different rates, may respond less predictably, or be influenced by general health or systemic disease. It considers risk factors such as cigarette smoking and having diabetes. A general assumption is that most cases will have a moderate or Grade B rate of progression. Heavy smoking or poor metabolic control of diabetes may elevate progression to Grade C. Conversely, if the patient does not smoke or does not have diabetes the grade progression may shift downward to a Grade A.[7]

ASSESSMENT OF CLINICAL PARAMETERS

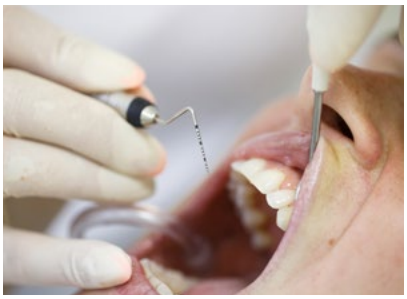
The measures used to determine the stage of periodontal disease include assessment of CAL and RBL, bleeding, and probing depth. Measurement of probing depth is part of the standard of dental hygiene care for a clinical exam.[8] However, the periodontal exam requires the more refined calculation of CAL. Probing depth measures the distance between the base of the pocket and the gingival margin. This can lead to the over or under estimate of bone loss. In comparison, CAL is the distance between the base of the pocket and a fixed point on the tooth; generally the cementoenamel junction (CEJ). A change in attachment level is a more accurate marker of periodontal destruction or improvement.[9] Determining the CAL takes a few extra steps but it not difficult. **(Table 3)**

Table 3: Determining Clinical Attachment Level[9]	
Anatomical features	Considerations for measurement
Gingival margin corresponds with the CEJ	Probing depth and CAL are the same
Gingival margin above the CEJ	Pocket depth minus the distance from the gingival margin to the CEJ = CAL
Gingival margin below the CEJ	Pocket depth plus the distance from the gingival margin to the CEJ = CAL

Probing depth is still an important consideration because it is a contributor to complexity. Deeper pockets are generally more challenging to clean for both clinicians and patients. At the same time, a deep pocket with stable probing depth and no bleeding over a long period of time likely does not have active disease. Probing has limitations. Depending upon the amount of force used, there can be great variability in measurements between practitioners. Another consideration is that different brands of probes have different tip diameters, and this can influence measurement. Patient discomfort during periodontal probing may be related to the thinness of the probe as well as the amount of pressure being used.[10]



Bleeding on probing (BOP) is the most widely used criteria to diagnose and monitor gingival inflammation. Lang et al found that sites with an incidence of BOP at four consecutive visits had a 30% chance of attachment loss. They concluded that BOP as a predictor of future bone loss is useful but limited.[11] Conversely, in a different study, Lang et al looked at the absence of bleeding on probing and its relationship to periodontal stability. They found that the lack of bleeding represented a 98% predictive value of periodontal stability and concluded the absence of bleeding is reliable predictor for the maintenance of periodontal health.[12]



It is important to consider that the absence of bleeding must be considered in the context of risk factors and systemic health and may not always denote periodontal health or disease state. Individuals who smoke may have a suppressed vascular response and consequently present with less bleeding and redness.[13] Conversely, there are several reasons a patient may have gingival bleeding. The ideal amount of pressure to use during probing is 25 N. Forces greater than this may induce bleeding thus giving a false positive reading.[10] Gingival bleeding has been found to be an adverse drug reaction in people older than 50 especially women. Antithrombotic drugs are the most common prescription drugs associated with adverse bleeding. The investigation also found enhanced bleeding with classes of drugs used to treat hypertension and edema, irregular heartbeat, and depression as well as some NSAIDS and acetaminophen.[14]

Radiographs are an essential component of a comprehensive periodontal exam. They can help identify alveolar bone loss especially in more advanced cases.[5] However, radiographic bone

level by itself does not provide enough specificity to identify early or moderate periodontitis.[7] Waiting until bone loss is present on radiographs may result in the delay of an early diagnosis and thus a more severe periodontal stage. Other considerations such as patient age, angulation of teeth or severe attrition may also influence the measurement of radiographic bone height.[5]

Tooth mobility is a common tool used for assessing periodontal health. However, tooth mobility is not a reliable indicator of periodontal status. Occlusal trauma may cause mobility in a healthy dentition. Conversely, a tooth with a reduced yet healthy periodontium may have some mobility and would not be considered to have active disease. Thus tooth mobility is not recommended as a measure of periodontal health or disease.[5]

RISK ASSESSMENT

Grading periodontal disease takes into account risk factors such as smoking, having diabetes, or poor oral hygiene. The presence of these factors singularly or together influence the rate of progression and overall prognosis of periodontitis. With smoking and diabetes, the risk is dose-dependent. Generally, the longer and heavier someone has smoked or the worse the individuals metabolic control, the more likely they are to have a severe case of periodontal disease.[13,15]

A current cigarette smoker is three to four times more likely to have periodontal disease than an individual who has never smoked.[13,15,16,17] Studies indicate that smokers are more likely to have deeper probing depths, greater attachment loss, more bone loss, and fewer teeth. There is often more calculus but less inflammation.[16] People who smoke are less likely to have regular dental visits.[18] A dose-response relationship between smoking and periodontal disease has been observed, with the heaviest smokers having the most disease severity.[13,15,16] Younger adult smokers (19–30 years) often have a higher prevalence and severity of periodontitis and tooth loss than young non-smokers.[16,17]

Smoking has been shown to impact both the vascular and cellular inflammatory response. People who smoke have been shown to have less gingival redness and less bleeding upon probing.[13] Nicotine impairs the function of neutrophils, including phagocytosis, and reduces salivary and serum IgG. Nicotine can bind to the root surface, altering fibroblast attachment while decreasing collagen and increasing collagenase production. Fibroblasts exposed to nicotine produce higher amounts of destructive pro-inflammatory mediators.[13,16]

The effect of a reduced vascular and cellular inflammatory response is a reduced healing capacity among smokers. The periodontal healing capacity of a smoker has been shown to be 28% of that of a nonsmoker and equivalent to someone 36 years older.[13] This is supported by data showing that people treated either nonsurgically or surgically for periodontal disease had less reduction in probing depth and smaller gains in clinical attachment than a nonsmoker.[13]

A 2017 paper looked at the use of recreational cannabis and its relationship to periodontal health. The investigators found that those who frequently used recreational marijuana had deeper probing depth and attachment loss versus nonfrequent users. They also had higher odds of developing severe periodontal disease.[19] Regular cigar and pipe smoking has also been shown to be detrimental to periodontal health.[20] There is limited evidence on the oral health risks from using e-cigarettes or hookahs. E-cigarette use has been associated with the use of other tobacco products in youth and young adults including cigarettes. E-cigarettes may also be used as a delivery device for cannabinoids and THC.[21]

DIABETES

A 2018 systematic review found that the presence of diabetes increases the risk or progression of periodontal disease by 86%.[22] Glycemic control is a significant risk factor as adults with the poorest control have been shown to have a greater prevalence and severity of inflammation and attachment loss.[13] A 2018 study found that that older males with type 2 diabetes were more likely to have moderate to severe periodontal disease. In those with diabetes, lower income, not being a high school graduate, and not having health insurance were also associated with having more severe periodontitis.[23] Data indicates that people with diabetes have less frequent dental visits.[24] Periodontal destruction may start early in life for children with diabetes and become more pronounced in adolescence. In children and young adults with diabetes, 13.6% of those aged 13–18 years and 39% of those aged 19–32 years have periodontitis.[25]

Periodontal disease is recognized as a complication of diabetes. Diabetes alters the immune response by impairing neutrophil function, decreasing chemotaxis, and reducing phagocytosis. At the same time, there is evidence of a hyper-responsive monocyte/macrophage phenotype leading to increased production of destructive pro-inflammatory mediators such as interleukin (IL) 1β.[13]

The evidence on response to therapy for patients with diabetes is limited. It is well-established that people with diabetes have a reduced capacity for wound healing, and it is likely this affects the outcome of periodontal therapy. It has been observed that people with well-controlled diabetes seem to have a treatment outcome similar to people without diabetes. In comparison, those with poor blood sugar control are more likely to have a less favorable treatment response.[13]

The treatment of periodontal disease has been shown to have a modest and limited impact on the improvement of glycemic control as measured by A1c. A systemic review by the Cochrane group found that periodontal therapy demonstrated a 0.29% reduction in A1c for 3-4 months post-treatment. At 6 months, the A1c had approached baseline.[26] A consensus report from the

International Diabetes Federation and the European Federation of Periodontology found that periodontal therapy accompanied by effective home care is a safe and effective means to manage periodontal disease in people with diabetes.[27]

GINGIVITIS AND BIOFILM

Gingivitis is widespread in adults and children. Biofilm accumulation is a primary cause.[10] Gingivitis can be localized or generalized. **(Table 4)** Professional removal of supra and subgingival biofilm and calculus along with good daily oral hygiene is essential for maintaining optimal periodontal health.[10]

Table 4: Gingivitis[5]

Intact Periodontium	Bleeding	Bone/attachment loss
Localized	≥10% and ≤ 30% of sites	None
Generalized	>30% of sites	
Reduced Periodontium no history of periodontitis		
Localized	≥10% and ≤ 30% of sites	Gingival recession/ crown lengthening Probing depths ≤ 3mm
Generalized	>30% of sites	
Reduced periodontium with successful treatment of periodontitis		
Localized	≥10% and ≤ 30% of sites	Probing depth ≥ 4mm
Generalized	>30% of sites	

There is an established relationship between gingival inflammation and periodontitis. Preventing and controlling gingivitis is believed to be a primary mechanism for the prevention of periodontitis.[10] Early gingivitis has been shown to develop within two to four days of new biofilm growth. From day four to ten, the immune response heightens, including loss of connective tissue collagen. Within two to three weeks, the lesion progresses with additional connective tissue loss and fibrosis but no attachment or bone loss. In some individuals, this stage of gingivitis may be present for years without developing into periodontitis.[28]

Periodontal disease is considered a biofilm-associated disease because while biofilm/bacteria are essential for having periodontitis, biofilm alone is not enough to cause this disease.[29,20] Biofilm has been shown to account for about 20% of the direct risk of developing periodontitis.[5] Emerging research has indicated that a progressive shift in the make-up of the subgingival microflora from a decrease in beneficial bacteria to an increase in periodontal pathogens contributes the development of periodontitis. Interestingly, among patients with periodontitis there is great variation in personal oral microflora. As well, periodontal pathogens such as *p. gingivalis* are frequently present in the biofilm of periodontally healthy patients. Researchers are exploring the idea that dysbiosis, or the lack of beneficial organisms in the biofilm coupled with the presence of pathogens may an important role.[29]

The burden of periodontal pathogens triggers the chronic inflammatory response. This starts a cascade of destructive pro-inflammatory mediates, especially IL 1β. It is this chronic inflammatory process that drives connective tissue and bone loss.[31] Data show there is great variation in the severity of bone loss among patients that is not explained by the amount or type of bacteria in the biofilm. This has led to the hypothesis that some individuals may be 'high responders' to periodontal pathogens because they produce higher levels of destructive pro-inflammatory mediators. It has been demonstrated that patients with severe periodontal disease have higher levels of IL-1β in all probing depth areas, including shallow pockets. This suggests that the expression of IL-1β may be a host or genetic trait.[32]

NONSURGICAL PERIODONTAL THERAPY

A 2015 evidence-based clinical practice guideline (CPG) from the American Dental Association found scaling and root planning (SRP) the treatment of choice for the initial nonsurgical treatment of chronic periodontal disease.[33] A review of more than 72 research articles showed that SRP improves clinical attachment levels.[33] Drisko noted that SRP is both safe and effective and is still the gold standard for the removal of subgingival biofilm and calculus.[34] Heitz-Mayfield and Lang found that in pockets of 4mm- 6mm, SRP resulted in 0.4 mm more clinical attachment gain than surgical therapy. In pockets greater than 6 mm, surgical treatment demonstrated 0.2 mm more attachment gain than SRP.[35] Likewise, Suvan found nonsurgical therapy reduces inflammation and pocket depth and increases clinical attachment level.[36]

The CPG from the American Dental Association considered the benefit of systemic and local delivery agents. They found benefits from the addition of systemic subantimicrobial use of doxycycline and from traditional systemic antibiotics. However, the guideline reviewers felt the adverse effects of a traditional antibiotic outweighed the benefits. Potential risks noted were gastrointestinal effects, serious allergic reactions, and the risk of antimicrobial resistance.[33] For local delivery drugs, both doxycycline hyclate gel and minocycline microspheres had limited scientific data but were recommended based on the reviewers expert opinion.[33]

The best protocol for providing SRP has yet to be determined. Traditionally, SRP is conducted via quadrant or half mouth separated in time by one to two weeks. A novel approach called full mouth disinfection (FMD) utilized the provision of SRP within a 24-hour period to limit potential bacterial recolonization. This method has had several modifications. Comparisons between FMD and a conventional approach have found little differences in clinical outcomes.[37] Therefore, the best protocol for providing SRP may be the one that best fits the patients' lifestyle and preferences.

Clinicians have numerous choices for instrumentation including hand scalers, ultrasonic instruments, and lasers. A review by Sanz et al found that clinical outcomes are similar regardless of the type of instrument used.[37] Both Drisko and Suvan also found no differences in the efficacy of hand or powered instruments.[34,36] Drisko notes that it is likely the thoroughness of the debridement that may matter most.[34] Thoroughness requires good quality sharp instruments, adequate appointment time, visual enhancements such as loupes with a light, and appropriate pain management.

Flemmig stated "there are virtually no substitutes for nonsurgical periodontal care other than the extraction of teeth."[38] This is not to say that extraction is a treatment option for someone who needs nonsurgical therapy. Rather, it means that the failure to arrest periodontal disease can lead to tooth loss. SRP is a clinically proven treatment for initial nonsurgical periodontal therapy.[33] A prophylaxis is not. While patients do have choices, practitioners have an obligation to recommend evidence-based, clinically sound treatment options.[8]

PERIODONTAL MAINTENANCE THERAPY & RISK REDUCTION

Supportive periodontal maintenance therapy has been shown to limit disease progression and tooth loss.[37,39] Costa et al found that people who regularly complied with periodontal maintenance visits were less likely to have a reoccurrence of periodontitis versus those who were irregular compliers. Regular compliance was measured as not going more than 6 months between visits over a five-year span.[40] Frequent maintenance visits may also help keep the bacterial biofilm at a level compatible with good periodontal health.[5]

The modification of risk factors is essential for periodontal disease stability.[5] When patients quit smoking, the rate of bone and attachment loss slows, and evidence indicates that disease severity is intermediate to that of current and non-smokers.[13] People with periodontitis who stop smoking reduce the risk of tooth loss. Within 10-20 years of quitting the risk of tooth loss approaches that of never smokers.[17] Smoking cessation even for the period during nonsurgical or surgical treatment may improve treatment outcomes and reduce complications.[13] Dental professionals who incorporated smoking cessation benefits into the oral examination were shown to increase the rate of cessation in both cigarette smokers and users of smokeless tobacco.[53]

People with diabetes often do not realize the impact of poor metabolic control on their periodontal health. Poor metabolic control can be driven by many factors from weight management to diet and medication compliance. The American Diabetes Association recommends an A1c of <7% for most nonpregnant adults. This target has been shown to reduce the microvascular complications of diabetes such as retinopathy, neuropathy, and kidney disease.[42] To accurately assess a patient with diabetes potential response to periodontal therapy it is essential to know their A1c reading. Patients should be counseled and informed about how blood sugar control affects their periodontal health and treatment responses.

The treatment and prevention of gingivitis may help prevent the onset of periodontal disease.[10,28] While some people with gingivitis never progress to periodontitis, it is difficult to predict who will develop the more serious condition and who will not. For this reason, it is important to treat all cases of gingivitis.[28] The creation of a new treatment code, D4346, by the American Dental Association is intended for use with patients with generalized to moderate severe inflammation, with or without pseudo pockets but exhibiting no bone loss.[43] **(Box 1)**

Box 1: Definition of D4346

D4346: Scaling in the presence of moderate or severe gingival inflammation- full mouth after oral evaluation:

The removal of plaque, calculus, and stains from supra- and sub-gingival tooth surfaces when there is generalized moderate or severe gingival inflammation in the absence of periodontitis. It is indicated for patients who have swollen, inflamed gingiva, generalized suprabony pockets, and moderate to severe bleeding on probing. Should not be reported in conjunction with prophylaxis, scaling and root planning or debridement procedures.[43]

SELF-CARE

Daily oral hygiene consisting of tooth brushing and interdental cleansing is critical in preventing gingivitis, maintaining periodontal health and preventing recurrence of disease. The toothbrush is the most common and often the only means of daily self-care used by many people. What is often neglected is interdental cleaning. Many people find the use of string floss challenging, and data have shown that only a minority of people use dental floss on a daily basis.[44]

The evidence on string flossing for improving gum health is weak.[45,46] This is shocking to many dental professionals who have witnessed firsthand the benefits of flossing. Flossing does work for those who can do it correctly.[47] However, it is a skill not easily mastered by those who are not dental professionals; thus it does not work for many people.[48] Lang et al. looked at typical brushing and flossing habits of people in the Detroit area. They found that although over 95% of people reported brushing at least once a day, around 33% reported flossing daily. When the investigators looked at the number of people who could perform acceptable flossing skills, the number dropped to 22%.[48] This inability to perform flossing at a level high enough to produce a health benefit is likely the biggest factor behind the weak evidence on flossing for plaque and gingivitis reductions. When done well and regularly, flossing works. The reality is that it does not work for most people because of a lack of expertise and/or motivation.

Interdental brushes (IDB) are a common recommendation for patients with residual pockets. A 2013 Cochrane review

by Poklepovic et al. evaluated the IDB for the prevention and control of periodontal diseases and dental caries in adults. The results found insufficient evidence to determine whether an IDB reduced or increased levels of plaque when compared to flossing. Regarding gingivitis, there was low-quality evidence that the IDB provided better gingivitis reduction than flossing.[49]

IDBs come in a variety of shapes and sizes. A 2016 study with 51 participants compared conically shaped to cylindrically shaped IDBs. The results showed that conical IDBs were less effective at removing lingual approximal plaque than cylindrical IDBs.[50] A 6-week 2006 study with 120 subjects compared four interdental products: dental floss, a flosser, an IDB, and a small interdental cleaner with elastomeric fingers. The investigators found that all products performed comparably for plaque reduction and bleeding. The IDB provided a statistically significant improvement for gingivitis on the buccal versus the other products.[51]

Figure 1: Waterpik® Aquarius Water Flosser

Figure 2: Waterpik® Cordless Advanced Water Flosser®

The Waterpik® Water Flosser **(Figure 1 & 2)** has been shown to be an effective tool for reducing gingivitis and bleeding.[52-60] In a University of Nebraska study, the Water Flosser was paired with a manual or a power toothbrush, and both were compared to traditional manual brushing and flossing. Regardless of toothbrush type, the addition of a Water Flosser, once daily with plain water, to a either a manual or power brushing routine was a more effective alternative to string floss for the reduction of bleeding, gingivitis, and plaque.[53] Likewise, Rosema et al found the Water Flosser twice as effective as string floss at reducing bleeding.[54]

The biofilm removing capabilities of the Water Flosser were evaluated in study conducted at the University of Southern California Center for Biofilms. Eight teeth were extracted from a patient with advanced periodontal disease. Pretreatment scanning electron microscopy (SEM) images of the teeth found they were colonized by a luxuriant biofilm appearing several micrometers thick **(Figure 3)**. The teeth were water flossed for 3 seconds at a medium pressure (70psi) setting. Post-SEM images found that water flossing removed up to 99.9% of plaque biofilm[61] **(Figure 4)**. The researchers concluded that the shear hydraulic forces produced by a water flosser with 1,200 pulsations per minute at medium pressure can significantly remove biofilm from tooth surfaces.[61] A single-use plaque study found that people who added a Water Flosser to manual toothbrushing removed 74% of whole mouth plaque compared to 56% for manual brushing and flossing, making the Water Flosser 29% more effective.[62]

Figure 3: Before treatment with the water flosser, Gorur et al.[5]

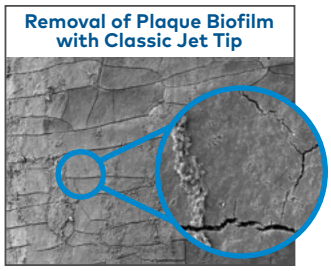


Figure 4: Tooth surface after a 3-second treatment with the Water Flosser, Gorur et al.[5]

A study by Genovesi et al. evaluated the difference between SRP followed by the local delivery of minocycline or SRP followed by daily water flossing for 30 days. The results demonstrated that both treatments effectively reduced bleeding on probing and improved pocket depth and clinical attachment at 30 days. **(Figures 5, 6, 7)** There were no statistical differences between the groups, thus showing that the Water Flosser is an effective alternative to subgingival antibiotics for periodontal maintenance patients over a 30-day period.[60]

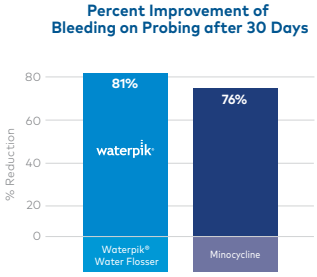


Figure 5: BOP reductions in periodontal maintenance patient, Genovesi et al.[42]

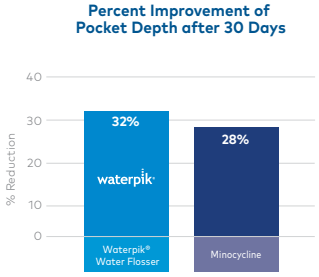


Figure 6: Probing depth improvements in periodontal maintenance patients, Genovesi et al.[42]

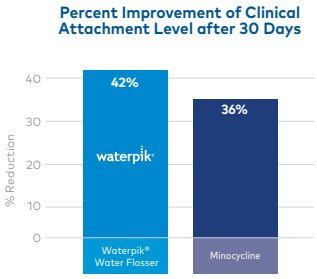


Figure 7: CAL improvement in periodontal maintenance patients, Genovesi et al.[42]

Several 6-month studies were conducted during the 1990s on periodontal maintenance patients.[56-59] Findings from these studies consistently showed that the Water Flosser improved the oral health of this demographic. Flemmig et al. found that water flossing reduced BOP by half over the 6-month time frame,[58] and Newman et al. showed that those with the most BOP had the greatest reductions.[57] In a different study, Flemmig et al. found that water flossing was more effective than rinsing with 0.12% chlorhexidine at reducing BOP.[56]

Diabetes has been shown to increase the risk for developing periodontal disease. A study at the University of Buffalo looked at how the Water Flosser benefited the periodontal health of people with diabetes. The results found that the addition of the Water Flosser to routine oral hygiene was more effective at reducing bleeding (44%) and gingival inflammation (41%) than routine oral hygiene alone.[55]

Figure 8: Waterpik® Sonic-Fusion®

There is a new entry to the self-care market that has added a water flossing function to the toothbrush handle. Waterpik® Sonic-Fusion® is a flossing toothbrush that combines the a sonic toothbrush with the efficacy of Water Flossing. **(Figure 8)** This new tool allows patients to add water flossing to toothbrushing with the touch of a button. A recent 4-week study found that the Waterpik® Sonic-Fusion® was twice as effective as string floss for removing plaque and reducing bleeding and gingivitis.[63] Sonic-Fusion® has earned the ADA Seal of Acceptance.[64]

The Water Flosser is supported by more than 70 published scientific studies and over 5 decades of use by the public. Both countertop and cordless models have earned the ADA Seal of Acceptance.[64] **(Box 2)** Despite this, skepticism about product safety and efficacy still persists.[65] Some dental professionals believe the product cannot be used at higher settings; others feel it increases probing depth or destroys the attachment.

Box 2: ADA Seal Statement on Waterpik® Water Flosser

ADA Accepted American Dental Association ®

"The ADA Council on Scientific Affairs Acceptance of the Waterpik® Water Flosser is based on its findings that the product is safe and has shown efficacy for removing plaque along the gumline and between teeth and helping to prevent and reduce gingivitis, when used as directed."

A recent study by Goyal et al. evaluated the effect of the Water Flosser on gingival and epithelial tissue at multiple pressure settings; including the highest settings at 9 and 10. One hundred and five subjects were assigned to one of three groups; 1) manual brushing and Water Flossing, 2) manual brushing and flossing, and 3) manual brushing only. For the manual brushing and water flossing group, subjects increased the pressure setting on the Water Flosser over the course of the six week study **(Table 5)** The primary outcome measured was clinical attachment levels (CAL) as assessed from the cemetoenamel junction and probing pocket depth PPD. At six-weeks, those in the Water Flosser group showed an improvement in CAL and a reduction in PPD. These changes exceeded those in the manual brushing and flossing group and the manual brushing only group. All subjects received oral examinations at baseline, two-weeks, four-weeks, and six-weeks. All subjects were negative for oral lesions, trauma or any other abnormal findings at each visit. The investigators concluded that the Water Flosser is safe to use, and the results should alleviate concerns especially regarding pressure setting that the Water Flosser may negatively impact gingival health or epithelial tissue.[66]

The findings from Goyal et al support those concluded in a 2015 literature review, which found no data to support that the Water Flosser is detrimental oral health. The review looked at a wide range of studies. It covered topics such as trauma to soft tissue, penetration of bacteria into the sulcus, probing depth and bacteremia.[66]

Table 5: Water Flosser Pressure Settings at Specific Time Points[65]

Day	Pressure
1 & 2	4
3, 4, 5	5
6, 7, 8	6
9, 10, 11	7
12, 13, 14	8
15-28	9
29-42	10

SUMMARY

Good periodontal health is a part of good general health. Early diagnosis and intervention is essential for arresting disease, limiting bone loss, and reducing the long-term risk for tooth loss. The new periodontal classification system can help practitioners identify and treat periodontal disease in its earliest stages. Professional care, risk reduction, and meticulous home care can help patients keep their teeth for a lifetime.

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POST TEST FOR COURSE #18-30:
New Paradigm: Demystifying Periodontal Disease

1. **What is the estimated prevalence of periodontal disease in people over age 30?**
   a. 30.1%
   b. 42.2%
   c. 50.0%
   d. 63.4%

2. **Having severe periodontal disease has been associated with:**
   a. Negative quality of life
   b. Negative impact on tooth function and esthetics
   c. Higher mortality rate
   d. All of the above

3. **The new periodontal disease classification system is developed around which two components:**
   a. Assessment and diagnosis
   b. Ranking and scoring
   c. Staging and grading
   d. Informing and documenting

4. **4. Which criteria is not a component of staging:**
   a. Intensity
   b. Severity
   c. Complexity
   d. Extent/ distribution of disease

5. **When the gingival margin is below the CEJ, to determine CAL you would:**
   a. Subtract the distance from the gingival margin to the CEJ from the pocket depth
   b. Add the distance from the gingival margin to the CEJ to pocket depth
   c. Use the probing depth as both probing depth and CEJ are the same
   d. None of the above

6. **Which risk factors are considered when grading periodontal disease progression?**
   a) Poor oral hygiene
   b) Smoking
   c) Diabetes
   d) All of the above

7. **The healing capacity of a smoker has been shown to be ____% of a nonsmoker?**
   a. 28%
   b. 45%
   c. 61%
   d. 73%

8. **If an individual has localized gingivitis, what is the percentage of sites affected?**
   a. > 30% and ≤ 60%
   b. > 25% and ≤ 50%
   c. >15% and ≤ 40%
   d. >10% and ≤ 30%

9. **Biofilm has been shown to account for ___ of the direct risk of developing periodontitis?**
   a. 10%
   b. 20%
   c. 30%
   d. 40%

10. **What does the American Dental Association Clinical Practice Guideline on Nonsurgical periodontal therapy promote as the 'treatment of choice'?**
    a. Full mouth debridement
    b. Prophylaxis
    c. Scaling and root planning
    d. Flap surgery

11. **The risk of tooth loss approaches that of a never smoker in someone who has not smoked for:**
    a. 1-5 years
    b. 5-10 years
    c. 10-20 years
    d. 25+ years

12. **What percentage of people have been shown to floss well enough to achieve a health benefit?**
    a. 13%
    b. 22%
    c. 33%
    d. 38%

13. **What action does the Water Flosser produce that assists in biofilm removal?**
    a. Shear hydraulic forces
    b. Hydrokinetic activity
    c. Classical mechanics
    d. None of the above.

14. **The use of the Water Flosser for 30 days post SRP had a positive improvement in:**
    a. Bleeding on probing
    b. Pocket depth
    c. Clinical attachment
    d. All of the above

15. **The Water Flosser used at the 9 & 10 setting was found to:**
    a. Increase clinical attachment
    b. Reduce pocket depth
    c. Not cause trauma or other harm
    d. All of the above.

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