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DIAGNOSTICS at Your Earliest Convenience:

A Discussion of Disease Prevention

By Katrina M. Sanders, RDH, BSDH, M.Ed, RF

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ABSTRACT

In 2017, the American Academy of Periodontology (AAP) redefined the way dental professionals classify, or rather “stage,” diseases of the periodontium. With the understanding of the nature of chronic diseases, the AAP created a new set of stages and grades to better help the dental professional effectively identify and treat early and advanced disease. In the release of these stages, dental professionals have now transitioned into healthcare professionals whose clinical paradigms focus around the timely identification and effective prevention of disease.

This article discusses the clinical and histologic parameters of early gingival and periodontal diseases, common chronic systemic diseases as they relate to oral disease, and current available therapies for the management of these diseases. As a healthcare professional, learn how clearer and more concise diagnostics will align your periodontal model to more effectively prevent and treat disease while supporting patients in evidence-based care.

EDUCATIONAL OBJECTIVES

The overall goal of this course is to provide information on the 2017 American Academy of Periodontology Classifications regarding gingival diseases, stage I periodontitis, and review case selection, protocols, and common chronic systemic diseases that impact oral disease. After completing this article, the reader will be able to:

1. Describe 2017 AAP Classifications as they relate to gingival diseases and early stage I periodontitis
2. Identify appropriate case selection and protocols for managing gingival diseases and early stage I periodontitis
3. Review common chronic systemic diseases that are exacerbated by the presence of oral disease
4. Discern contraindications in treating gingival diseases and risks of substandard therapy
5. Demonstrate appropriate patient communication techniques in delivering treatment recommendations for the management of gingival diseases.

ABOUT THE AUTHOR



Katrina M. Sanders, RDH, BSDH, M.Ed, RF

Katrina M. Sanders, RDH, BSDH, M.Ed, RF is a clinical dental hygienist, author, and international speaker, Katrina’s zest for dental hygiene is infused throughout her content as she lectures on provocative topics while leaving participants with a call to action and an abundantly renewed sense of pride for their industry.

Katrina is the Clinical Liaison for AZPerio, the country’s largest periodontal practice. Katrina is a published author with DentalTown, Today’s RDH, and is a columnist and advisory board member for Modern Hygienist.

Disclosures: Katrina Sanders discloses that she is the founder, CEO, and keynote speaker for Sanders Board Preparatory . Ms. Sanders can be contacted at: www.katrinanders.com.

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Introduction

The landscape of preventive and periodontal dentistry is ever evolving and continuously changing. Today, the average life expectancy of the dental patient is increasing exponentially, more patients are maintaining a full or partial dentition, and many are living with multiple comorbidities. Chronic diseases have surpassed infectious diseases as the leading cause of death in the United States, and the incidence of newly diagnosed chronic diseases is on the rise. Collaborative research is continually identifying compelling links between systemic disease and the microorganisms present in the oral microflora.

In addition, the prevalence of diseases of the gingiva and periodontium continues to climb. In children ages 10-17 years, prevalence of gingivitis is noted to be as high as 91%.¹ In 2016, the Centers for Disease Control and Prevention performed the first study of its kind, estimating the presence of periodontitis at state and local levels across the United States. This study revealed that nearly half of all U.S. adults ages 30-79 have some form of periodontal disease² (Table 1).

With this knowledge, the Office of Disease Prevention

and Health Promotion established Healthy People 2030 Oral Health Core Objectives, which aim to significantly reduce proportions of adult tooth loss and the presence of periodontitis.³

Of greatest concern, periodontitis has been identified as the second most frequent modifiable contributor to the systemic inflammatory burden, second only to obesity.⁴ Now, more than ever, it is imperative that dental professionals understand their roles and responsibilities in the timely identification and effective management of diseases of the gingiva and periodontium and subsequent systemic disease as a manifestation of oral disease.

In 2017, more than 170 leading clinical researchers were involved in revising and improving the current classification system of identifying periodontitis, through collaboration between the European Federation of Periodontology (EFP) and the American Academy of Periodontology (AAP). This newly implemented classification system integrates a comprehensive approach to analyzing the most contemporary evidence available. As such, this classification system has integrated a staging and grading system for periodontitis, as well as an indication of severity and extent of disease, an evaluation of disease over a lifetime, and a complete approach to the overall health status of the patient.

This article discusses the clinical and histological components involved in the identification of plaque-induced gingivitis and early periodontal disease, as well as a comprehensive review of the oral-systemic link and important strategies in the evidence-based implementation and management of early disease.

Plaque-Induced Gingivitis

By definition, gingivitis is considered an inflammatory condition exacerbated by the accumulation of dental biofilm. The clinical sequelae of plaque-induced gingivitis includes a typically painless presence of gingival erythema and edema in the absence of attachment loss.⁵

As a cardinal sign of inflammation, gingival bleeding has become a clinical sign of interest, because its presence upon gentle probing typically precedes other clinical manifestations of gingivitis such as color or volume changes.⁶ Specifically, gingival bleeding upon light stimulation has been identified

TABLE 1. Top 10 states with highest periodontitis prevalence*

New Mexico	52.90%
Hawaii	51.10%
Florida	49.47%
Mississippi	49.22%
Texas	48.25%
Louisiana	48.21%
Nevada	47.84%
California	47.80%
South Carolina	47.78%
Arizona	47.73%
*Estimated prevalence for U.S. adults ages 30-79	



as perhaps the most accurate clinical sign of gingival inflammation.⁷ Of greater concern is the correlation between the presence of gingival bleeding and the subsequent migration of bacteria into the bloodstream, called *bacteremia*.

During the 2017 AAP proceedings, it was noted that bleeding on probing (BOP) is a paramount consideration in the development of disease. The AAP made a recommendation to ensure that sites with BOP be measured at every regularly scheduled appointment to allow for accurate measurement and evaluation of the status of periodontal health or to institute the timely prevention of the progression of disease.⁴

Other key indicators of plaque-induced gingivitis include erythema, edema, heat, enlargement of the gingival, and/or loss of gingival function. Some patients may present with a self-report of bleeding gingiva, tenderness upon brushing or flossing, halitosis, or swelling of the gingival tissues.

The identification of gingivitis can be defined as site-specific or as a case of gingivitis.⁸ While a site-specific identification of gingivitis can be quantified, the identification of case-based gingival disease further defines a whole-health diagnosis, and creates greater alignment with the understandings of the oral-systemic link.

Diagnosis of the earliest stages of gingivitis is considered incipient gingivitis, and at a localized level, a diagnostic term of “Periodontal Health” is used. Upon advancement of incipient gingivitis

into a more generalized and advanced state, severities of mild, moderate, and severe are utilized as diagnostic terms (Table 2).

In addition, gingival enlargement was discussed in great detail during the 2017 AAP proceedings, and notations of localized, generalized, and varying severities were defined (Table 3).⁸

Of note, medications were identified as known factors not only for gingival enlargement but also as a contributory factor to xerostomia, which can exacerbate several other diseases of the oral tissues. Figure 1 demonstrates gingival enlargement as a sequela of medications and inadequate biofilm control.

Plaque-induced gingivitis is of great interest because it is not only reversible but also preventable with a comprehensive approach. Nevertheless, plaque-induced gingivitis is considered the precursor to periodontitis and, as such, presents as an important disease in preventive dentistry.

As evidence continues to mount regarding the relationship between gingivitis and periodontitis, it becomes increasingly imperative that dental providers understand their role as

TABLE 3. Newly classified severity of gingival enlargement based on clinical evaluation

Mild enlargement	Located in the gingival papilla only
Moderate enlargement	Located in the gingival papilla and the marginal gingiva
Severe enlargement	Located in the gingival papilla, marginal gingiva, and the attached gingiva

TABLE 2. Identification of gingivitis based on clinical assessment

Incipient gingivitis periodontal health	<10% of the mouth is affected Mild erythema Broken line of bleeding Can progress to localized gingivitis (10%-30% BOP)
Mild gingivitis	BOP scores >30% Minor tissue changes
Moderate gingivitis	BOP scores >30% Erythema, edema, and enlargement
Severe gingivitis	Bleeding scores >30% Overt erythema and edema Bleeding when touched rather than probing



Figure 1. Gingival hyperplasia associated with calcium-channel blocker medications and poor control of oral hygiene. Photograph courtesy of Dr. Steven G. Reitan

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preventive experts in identifying, diagnosing, and treating the reversible disease.

2017 AAP Classifications | Staging Periodontitis

The classification of periodontitis has evolved greatly from the first documented diagnostic terms of 1977.

Since then, the AAP has continually evaluated and reclassified periodontitis to align with current research. In 2017, the AAP integrated current knowledge about the chronic and irreversible nature of diseases of the periodontium to reclassify how dental professionals identify and diagnose periodontitis^{9,10} (Tables 4, 5).

This classification system not only analyzed the histological and clinical sequela of disease, it also unveiled a new means of diagnosing the severity of disease.

Previous terms of “mild periodontitis,” “moderate periodontitis,” or “severe periodontitis” were discarded for new verbiage that aims to classify the true severity, extent, and complexity of the disease in its present state. New

verbiage now classifies periodontitis as a staged disease.⁴

Utilizing staged classifications of periodontitis aligns with verbiage utilized to classify severities of other chronic diseases, including cancer, kidney disease, hypertension, and lung disease, to name a few. In addition, staging permits moving diagnostics beyond a one-dimensional approach of evaluating previous destruction alone. It permits the assessment and integration of multifactorial complexities and prognoses in identifying the severity and extent of the disease process.

2017 AAP Classifications | Grading Periodontitis

In its 2017 proceedings, the AAP identified the importance of evaluating a grade, or rather evaluation, of the progression or rate of the disease process. Previous diagnostic terms such as “rapidly progressive” or “aggressive periodontitis” were evaluated for consistency. The AAP noted that there is little consistent evidence that the disease “aggressive periodontitis” is significantly different from chronic periodontitis.¹⁰ The use of a grad-

TABLE 4. Staging of periodontitis as identified by the AAP (2017)

	Periodontitis	Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL (at site of greatest loss)	1-2 mm	3-4 mm	≥5 mm	≥5 mm
	RBL (radiographic bone loss)	Coronal third (<15%)	Coronal third (15%-33%)	Extending to middle third of root and beyond	Extending to middle third of root and beyond
	Tooth loss (due to periodontitis)	No tooth loss	No tooth loss	≥ 4 teeth	≥ 5 teeth
Complexity	Local	Maximum probing depth 4 mm Mostly horizontal bone loss	Maximum probing depth 5 mm Mostly horizontal bone loss	In addition to stage II complexity: • Probing depths ≥6 mm • Vertical bone loss 3 mm or less • Furcation involvement Class II or III • Moderate ridge defects	In addition to stage III complexity: • Need complex rehabilitation due to: • Masticatory dysfunction • Secondary occlusal trauma • Severe ridge defects • Bite collapse, drifting or flaring
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as: localized (<30% of teeth involved); generalized; or molar/incisor pattern			



TABLE 5. The three steps to effectively staging and grading a patient

Step 1: Initial case overview to assess disease	Screen: <ul style="list-style-type: none"> • Full-mouth probing depths • Full-mouth radiographs • Missing teeth Mild-to-moderate periodontitis will typically be either stage I or stage II Severe to very severe periodontitis will typically be either stage III or stage IV
Step 2: Establish stage	For mild-to-moderate periodontitis (typically stage I or stage II): <ul style="list-style-type: none"> • Confirm clinical attachment loss (CAL) • Rule out nonperiodontitis causes of CAL (eg, cervical restorations or caries, root fractures, CAL due to traumatic causes) • Determine maximum CAL or radiographic bone loss (RBL) • Confirm RBL patterns For moderate-to-severe periodontitis (typically stage III or stage IV): <ul style="list-style-type: none"> • Determine maximum CAL or RBL • Confirm RBL patterns • Assess tooth loss due to periodontitis • Evaluate case complexity factors (eg, severe CAL frequency, surgical challenges)
Step 3: Establish grade	<ul style="list-style-type: none"> • Calculate RBL (% of root length x 100) divided by age • Assess risk factors (eg, smoking, diabetes) • Measure response to SRP and plaque control • Assess expected rate of bone loss • Conduct detailed risk assessment • Account for medical and systemic inflammatory considerations

ing system has, in turn, removed diagnostic verbiage inclusive of aggressive periodontitis (Figure 2).

In response, the AAP developed a grading system in which the dental provider is able to identify the progression rate of disease, evaluate contributory risk factors, and identify an individual patient’s case of risk based on their systemic health (Table 6).

Primary criteria include evidence of mild, moderate, or rapid rate of bone destruction over a survey of five years. An evaluation of the percentage of bone loss in tandem with patient age is indicated as indirect evidence, and identifying a case phenotype based on typical or atypical host response to the presence or absence of biofilm is also included. As a grade modifier, risk factors of smoking habits and the presence and level of control of diabetes, as measured in hemoglobin A1c readings, play a role in the overall grade of the progression of periodontitis.

The use of a grading system permits an evaluation of the future risk of the progression of periodontitis, as well as a predicted responsiveness to standard therapeutic interventions of nonsurgical therapy. In addition, it highlights the importance of cotherapeutic management of

systemic disease with medical colleagues.

As a synergistic system, the introduction of staging and grading in the diagnostics of periodontitis permits the dental provider to use clearer guidelines in identifying and treating the various stages of periodontitis.

Stage I Periodontitis | The Borderland

During the AAP proceedings, great focus was given to developing diagnostic terminology in the identification of the earliest signs of periodontal disease. Of note, the AAP identified several longitudinal studies which demonstrated that sites that progress to attachment loss previously presented with persistently greater levels of gingival inflammation.¹¹ Specifically, stage I periodontitis is termed “the borderland” between gingivitis and periodontitis and is said to present with the earliest stages of attachment loss.

As increased focus is placed on the early identification of disease, it is imperative that the dental provider is equipped with the discernible knowledge and opportunity for early diagnosis, intervention, and subsequent monitoring.

One unique change in the identification of periodontitis is an

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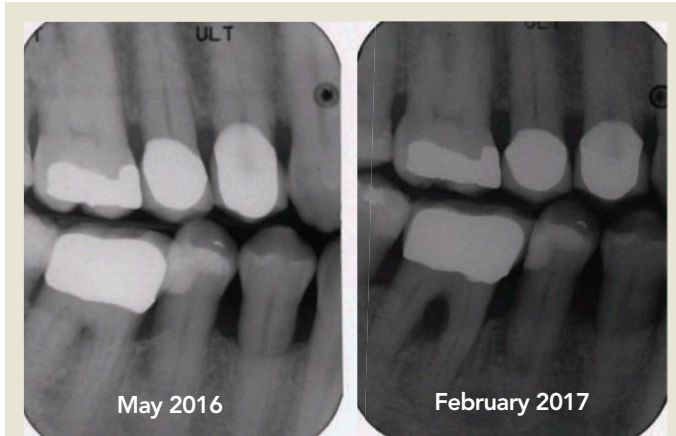


Figure 2. Example of a case that would previously be considered “aggressive periodontitis.” New grading classifications identify this case as rapid rate (grade C) periodontitis based on the progression of disease over a short period of time. Note the advanced bone loss within the buccal furcation site on tooth #30 in an otherwise healthy patient.

Radiographs courtesy of Dr. Steven G. Reitan

increased focus on the presence of clinical attachment loss (CAL) as identified through periodontal assessments. While many clinicians are trained to utilize two-dimensional radiographs to identify signs of attachment loss, it should be noted that identifying periodontitis from radiographs alone provides challenges. Specifically, bitewing and periapical radiographs frequently overlook direct buccal and lingual bone levels, and often omit bony wall defects. In addition, research currently states that bone loss can be identified on a radiograph only when approximately 30%-50% of the bone has been demineralized.¹²

Of note, stage I periodontitis is defined as radiologic bone loss of less than 15% of the coronal third, which can often be difficult, if not nearly impossible, to discern on two-dimensional radiologic images. If radiographic bone loss is able to be identified, the bone loss is horizontal in nature, and typically outlines early crestal changes and fuzziness of the bone septum, called the lamina dura.

Stage I periodontitis is defined as having an interdental CAL of 1-2 mm. Additionally, probing depths in stage I periodontitis comprise of ≥ 4 -mm periodontal pockets, which must be noted through comprehensive periodontal charting with a calibrated probe (Figure 3).

It should be noted that pockets of ≥ 4 mm that do not comprise advanced edema leading to pseudopocketing are classified as advancing from gingival pockets to periodontal pockets. As such, the presence of ≥ 4 -mm pockets in the absence of pseudopocketing indicates the earliest signs of apical migration of the junctional epithelium and, in turn, indicates attachment loss.

In addition, a patient with stage I periodontitis has not experienced tooth loss due to periodontitis.

The Oral–Systemic Link

As newly staged chronic diseases, gingivitis and periodontitis are both considered multifactorial and episodic diseases in which the periodontium is in an acute or chronic state of inflammation. As inflammatory diseases, diseases of the periodontium are characterized as systemic diseases in which there is major functional impairment of host defenses.

TABLE 6. Grading the progression of periodontitis

		Progression		Grade A Slow	Grade B Moderate	Grade C Rapid
Primary criteria	Direct evidence	RBL or CAL	No loss over 5 years	<2 mm over 5 years	2+ mm over 5 years	
	Indirect evidence	% bone loss/age	<0.25	0.25 to 1.0	>1.0	
		Case phenotype	Heavy biofilm, low destruction	Destruction commensurate with deposit	Destruction exceeds expectations. Rapid progression/early onset	
Grade modifier	Risk factors	Smoking	Nonsmoker	<10 cigarettes/day	10+ cigarettes/day	
		Diabetes	Normal	HbA1c <7.0%	HbA1c 7.0+%	

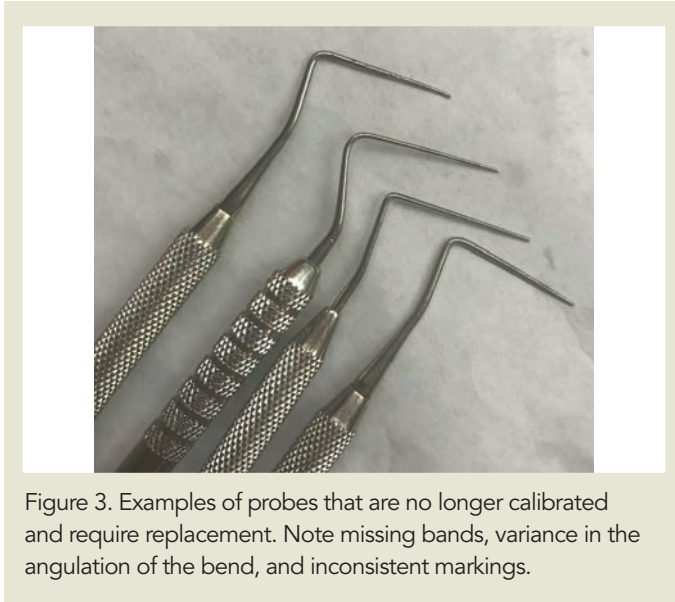


Figure 3. Examples of probes that are no longer calibrated and require replacement. Note missing bands, variance in the angulation of the bend, and inconsistent markings.

In recent years, a stronger understanding and a deeper correlation between oral diseases and various systemic diseases has been identified. The following is a compilation of the most prevalent systemic diseases that are currently being researched in tandem with diseases of the periodontium.

Diabetes

Current research has identified diabetic patients as more susceptible to periodontal disease and has in turn linked uncontrolled periodontal disease to potentially higher levels of blood glucose in diabetics.¹³ Of note, research has continually demonstrated the success of controlling hemoglobin A1c readings in diabetic patients with the activation of conventional periodontal scaling and root planing (SRP).¹⁴

Cardiovascular Disease

In the 1990s, researchers identified the earliest possible link between coronary heart disease and periodontal disease. This association led to compelling statistics in the connection between myocardial infarction and the presence of periodontal disease. It was quickly identified that patients have a 50% increased risk for heart disease and 30% increased risk of stroke if they have uncontrolled periodontal disease.¹⁵

Adverse Pregnancy Outcomes

The presence of periodontal disease in a pregnant woman has been linked to higher levels of cytokine prostaglandin E2 creating a statistically significant risk factor for premature, low-birth-weight delivery (PLBW). PLBW occurs in approximately 1 in 10 deliveries, and can result in infant mortality, long-term morbidity, or long-term neonatal intensive care stays. In 2003, a pilot study noted that performing SRP in pregnant women with periodontitis demonstrated a reduced risk of spontaneous delivery.¹⁶

Respiratory Disease

Recent hypotheses indicate that bacteria in the oral cavity are easily aspirated into the respiratory tract and can promote the exacerbation of infection by respiratory pathogens.^{17,18} Specifically, it was noted that microorganisms such as *P. gingivalis*, *E. nucleatum*, and *B. oralis* were contributory factors to respiratory diseases such as bronchitis, pneumonia, and emphysema.

Alzheimer's Disease

Early exposure to inflammatory diseases such as periodontal disease may increase the risk of the development of Alzheimer's disease later in life. The development of this conclusion resulted from an understanding between periodontitis and its effect on the elevation of C-reactive proteins, known to play a pivotal role in systemic inflammation.¹⁹

Cancer

Unique studies have identified potential links between plasma antibodies of oral bacteria and various cancers. A large European study identified increased levels of *P. gingivalis* as a twofold higher risk of pancreatic cancer.²⁰ Studies also have indicated a link between *E. nucleatum* and colorectal cancer²¹ as well as an increased risk of oral cancer in patients with greater than 1.5 mm of CAL.²²

As research continues to identify periodontal biomarkers and their systemic sequelae, the dental professional must be prepared for continued education in the greater strength of the oral-systemic link. It is in the identification of early disease that early intervention and monitoring is recommended. A prompt and proactive approach provides opportunity for the prevention or

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stability of attachment loss, the management of the oral microbiota, and the harmonious balance of oral–systemic processes.

Implementation | Prophylaxis

As one of the most, if not the most, widely used Current Dental Terminology (CDT) codes within a dental hygiene program, the D1110 prophylaxis code is known by patients as one of several pseudonyms, including “regular cleaning,” “my free cleaning,” “basic cleaning,” and “the cleaning my insurance pays for,” to name a few.²³

The American Dental Association (ADA) maintains the CDT code D1110 as “removal of plaque, calculus and stains from the tooth structures in the permanent and transitional dentition. It is intended to control local irritational factors.”²³

While verbiage within the CDT code indicates it is appropriate for the removal of local factors, clinicians have used the D1110 CDT code for the management of generalized plaque biofilm and calculus deposits, moderate-to-severe gingivitis and, in some cases, undiagnosed periodontitis or periodontitis in the presence of informed refusal of active therapy for years.

In addition, the term prophylaxis denotes a preventive therapy. Previously, this was interpreted as the prevention of the advancement of disease from a reversible gingival disease to an irreversible periodontitis. However, this interpretation has been revised based on new findings.

The supportive evidence provided during the 2017 AAP proceedings acknowledges the bioburden threat between the localized and systemic concerns of oral inflammation. As such, gingivitis has been classified as a nonspecific inflammatory condition and a key contributory component to the development of periodontal and chronic systemic diseases.

It is appropriate for the dental community to consider that perhaps most D1110 cases are, in fact, incorrectly coded. In turn, incorrect coding leads to mismanagement of an active, irreversible, and perilous disease.

Implementation | Scaling in the Presence of Gingival Inflammation

In an effort to attain greater accuracy between under-coding gingival diseases as a prophylaxis and overcoding

as SRP, the ADA developed a CDT code specific to patients with moderate-to-severe gingivitis.

Scaling in the presence of generalized moderate or severe gingival inflammation is a full-mouth treatment to be performed after an oral evaluation. The CDT code D4346²³ is defined as: “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, SRP, or debridement procedures.” Figure 4 contains radiographic and clinical images of appropriate D4346 cases as defined by the ADA.

This code is considered a therapeutic procedure intended to bring the patient’s gingival condition back to a state of health. Procedurally, the dental professional must consider what is required to thoroughly and completely address the



Figure 4. Case studies of appropriate D4346 candidates as identified by the American Dental Association

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bacterial biofilm and subsequent inflammatory process. This may involve advanced therapies of oral debridement with or without local or topical anesthesia, nutritional counseling, or tongue debridement, to name a few.

In addition to early therapy to manage oral inflammation, additional therapeutic procedures should be considered, such as providing detailed oral hygiene instruction with CDT code: D1330 – oral hygiene instructions or delivering a subgingival medicinal agent with CDT code: D4921 – gingival irrigation per quadrant.²³

Finally, dental providers who are managing patients with active gingival disease should consider encouraging a shorter frequency of hygiene visits to permit more frequent and timely control of etiologic factors.

Contraindications

It should be noted that the sophistication of the oral microflora has led to a greater understanding of the importance of intentional and effective preventive or active therapy. Providers who have been or continue to perform minimal or below minimal therapeutic interventions are placing their patients at risk for continued inflammation, or worse, an exacerbation of advanced infection.

Of note, the removal of subgingival debris with traditional curets and mechanized instruments in a diseased gingival or periodontal pocket produces unintentional soft-tissue curettage. As the diseased lining of the pocket is removed, the pocket wall is left open, permitting the entrance of abrasive particles such as pumice from prophylaxis paste and potentially pathogenic microorganisms. These agents can easily become embedded in the subgingival space and are subsequently out of reach during conventional home care routines. In this example, clinicians are unintentionally creating exposure of the delicate subgingival space to advanced levels of localized factors that further inhibit the opportunity for gingival and/or periodontal healing.²⁴

As such, soft and spongy tissue, tissue that bleeds easily or spontaneously upon brushing or gentle stimulation, or patients with moderate-to-severe gingival disease are at advanced risk for further infection if they are not introduced

to more thorough or advanced therapy.

With new periodontal classifications and advanced CDT codes in place, the dental provider now has the tools available and the opportunity to initiate evidence-based protocols into clinical practice. The final section of this article discusses communication techniques to empower dental providers in creating effective and lasting educational opportunities with their patients.

Communication

Just as challenging as developing psychomotor skills necessary for managing disease is the challenge of clear and supportive patient education and communication. Patients present with unique perceptions, values, attitudes, beliefs and levels of education, or dental IQ regarding their oral and systemic health, which can pose a challenge for the dental professional. Beliefs such as “I will only do what insurance covers,” “If it doesn’t hurt, nothing is wrong,” or fears of being “sold” something unnecessary can provide immense barriers to effective patient communication.

The following are common communication techniques to be considered when educating dental patients about their oral disease.

Terminology – As a whole-team approach, clinicians and staff members should make collaborative decisions about the terminology utilized in the office. All too often, patients hear varying terms from members of the same dental team ranging from “inflammation” to “infection,” and “disease” or “gum treatment,” “active therapy,” “scaling,” “root planing,” “deep cleaning,” and “below-the-gum treatment” to name some examples. While dental professionals are highly trained in understanding the multiple terms associated with the disease process, calibration of terms used for patient education may aid in streamlining patient understanding.

Semantics – Be mindful of terms that may devalue or diminish the education provided to the patient. Describing early disease by stating “You have a little bit of bone loss” or “My recommendation is...” can easily confuse a patient into believing their condition is not as serious as their dental professional has described. Consider verbiage such as “You have stage I

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periodontitis” or “Your prescribed treatment is...,” which deliver a stronger and more diagnostic message to the patient.

Identify the problem – Dental professionals often fail to share the multifactorial concerns of oral disease with their patients. Problem identification should begin during the medical and dental history interview and continue during risk assessment and evaluation of comprehensive periodontal, clinical, and radiologic appraisal. Integrating visual aids such as intraoral radiography and educational pamphlets provides supportive evidence for aiding patient understanding of oral diseases.

Discuss the consequences – As a generalized assessment, dental professionals understand their ethical responsibility to discuss the consequences of advanced disease with their patients. What many dental professionals fail to do, however, is tether the consequences of untreated oral disease to their patients. A comprehensive understanding of familial history can provide insight to systemic conditions of concern and a thorough understanding of etiologic factors (ie, stress, medications, nutritional status) will create compelling dialogue that is patient-specific regarding their existing disease and risk factors.

Deliver the solution – Changing the mindset of the dental provider from a production-boosting treatment plan to a patient-centered solution elevates the level of care and concern with which dental providers create resolution. Patients appreciate a solution-based approach because it clarifies a clinical need for intervention rather than a potentially unnecessary procedure that they are being “sold.”

Conclusions

As research continues to advance, as diagnostics continue to narrow for clarification, and as the overall culture of wellness focuses on the importance of inflammatory management, it is increasingly imperative that dental professionals employ strategies to ensure they are meeting the growing demands of the community.

Future trends in periodontics are supporting the dental professional in the immediate and timely identification of early oral disease and the subsequent implementation of

comprehensive intervention.

As evidence-based providers, dental professionals are encouraged to ensure that recommendations for intervention align with safe, effective, and thorough approaches to the treatment and management of oral disease.

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CEQuiz

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- The most recent research from the CDC has revealed that nearly ___ of all U.S. adults ages 30-79 have a form of periodontal disease.
 - 10%
 - one-quarter
 - one-third
 - 40%
 - half
- Periodontitis has been identified as the second most frequent modifiable contributor to the systemic inflammatory burden, second only to _____.
 - obesity
 - cardiovascular disease
 - cancer
 - diabetes
 - stroke
- The clinical sign of interest in plaque-induced gingivitis is _____.
 - inflammation
 - erythema
 - gingival bleeding
 - bone loss
 - edema
- All of the following can be clinical signs of gingivitis except _____.
 - inflammation
 - erythema
 - gingival bleeding
 - bone loss
 - edema
- Incipient gingivitis at a localized level can be described as _____.
 - mild gingivitis
 - moderate gingivitis
 - severe gingivitis
 - periodontal health
 - stable on a reduced periodontium
- Incipient gingivitis is described as _____.
 - <10% bleeding
 - enlargement
 - synonymous with stable periodontitis
 - pain
 - halitosis
- The first documented diagnostic terms from the American Academy of Periodontology are from _____.
 - 1977
 - 1986
 - 1999
 - 2006
 - 2017
- The 2017 American Academy of Periodontology Classifications include diagnostic terms of ___ and _____.
 - severity and grade
 - stage and grade
 - prognosis and grade
 - stage and prognosis
 - stage and severity
- During the 2017 American Academy of Periodontology proceedings, ___ was removed from the system as a diagnostic term.
 - periodontal health
 - peri-implantitis
 - chronic periodontitis
 - aggressive periodontitis
 - periodontitis as a manifestation of systemic disease
- Grade modifiers as risk factors are ___ and _____.
 - obesity and high blood pressure
 - genetics and obesity
 - nutrition and high blood pressure
 - smoking and plaque
 - smoking and diabetes
- A nondiabetic patient who smokes 10+ cigarettes/day would be classified as which grade of periodontitis?
 - Nonrisk
 - Grade A Slow
 - Grade B Moderate
 - Grade C Rapid
 - Grade D Advanced
- The AAP Grading evaluates each of the following as evidence for identifying the progression of disease except for one. Which is the exception?
 - radiographic bone loss
 - case phenotype
 - interleukin-1
 - smoking tendency
 - diabetic controls
- Stage I periodontitis is also termed _____.
 - gingivitis
 - stable disease
 - the borderland
 - healed periodontitis
 - refractory periodontitis
- The use of radiographs provides limitation in identifying bone loss. Current research indicates ___ of bone must be demineralized before bone loss is detected on radiographs.
 - 10%-15%
 - 15%-20%
 - 20%-25%
 - 25%-30%
 - 30%-50%
- Stage I periodontitis is defined as radiologic bone loss of less than ___ of the coronal third.
 - 15%
 - 20%
 - 25%
 - 30%
 - 35%

Diagnostics at Your Earliest Convenience: A Discussion of Disease Prevention



CE QUIZ

16. A clinical case of 1-2 mm interdental clinical attachment loss and periodontal pockets of ≥ 4 mm in the absence of pseudopocketing indicate ____.
- health
 - incipient gingivitis
 - mild gingivitis
 - apical migration of the junctional epithelium
 - early tooth loss
17. According to the 2017 AAP Classifications, a patient presenting with less than 15% radiographic bone loss and a maximum probing depth of 4mm would be classified as:
- plaque-induced gingivitis
 - Stage I periodontitis
 - Stage II periodontitis
 - Stage III periodontitis
 - Stage IV periodontitis
18. According to the 2017 AAP Classifications, teeth with Class III furcations are considered:
- plaque-induced gingivitis
 - Stage I periodontitis
 - Stage II periodontitis
 - Stage III periodontitis
 - Stage IV periodontitis
19. Ulceration or tearing of the epithelium is noted by ____ as a clinical indicator.
- deep pockets
 - edema
 - bleeding tendency
 - halitosis
 - heat
20. Patients with uncontrolled periodontal disease have a ____ increased risk for heart disease.
- 10%
 - 25%
 - 30%
 - 40%
 - 50%
21. Premature, low-birth-weight deliveries occur in approximately 1 in ____ deliveries.
- 10
 - 25
 - 50
 - 100
 - 150
22. A European study identified increased levels of ____ as a twofold higher risk of pancreatic cancer.
- S. mitis*
 - P. intermedia*
 - S. mutans*
 - P. gingivalis*
 - B. oralis*
23. The American Dental Association CDT code D1110 is appropriate for patients with ____.
- incipient gingivitis
 - moderate gingivitis
 - severe gingivitis
 - stage I periodontitis
 - stable periodontitis
24. The American Dental Association CDT code D4346 is appropriate for patients with ____.
- moderate-to-severe gingival inflammation
 - stage I periodontitis
 - stage II periodontitis
 - stage III periodontitis
 - stable periodontitis
25. Therapies to be introduced in conjunction with the D4346 code include all of the following except ____.
- nutritional counseling
 - oral hygiene instruction
 - delivery of a subgingival medicinal agent
 - prophylaxis
 - shorter recare frequencies
26. All of the following applies to soft-tissue curettage except ____.
- can be unintentional when debriding a diseased gingival or periodontal pocket
 - leaves the pocket wall open
 - permits the entrance of abrasive particles
 - permits the entrance of microorganisms
 - permits safe healing properties
27. All of the following are contraindications to minimal therapy except ____.
- incipient gingivitis
 - soft and spongy tissue
 - tissue that bleeds easily
 - spontaneous bleeding
 - moderate or severe gingival disease
28. A risk of incomplete removal of debris is ____.
- pericoronitis
 - endodontic lesions
 - stable periodontium
 - periodontal abscess
 - occlusal trauma
29. Anatomical limitations occurring in ____ can contribute to a periodontal abscess.
- occlusal table
 - incisal edges
 - furcations
 - vestibular space
 - cusps of Carabelli
30. All of the following are suggestions of effective communication strategies in patient education except ____.
- calibrate on terminology used in the office
 - be mindful of semantics when describing disease
 - integrate visual aids when identifying the problem
 - ignore etiologic factors that relate to the patient
 - deliver a solution