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BaleDoneen Method: Medical model emphasizing dental health component in inflammation reduction

A Peer-Reviewed Publication

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BaleDoneen Method: Medical model emphasizing dental health component in inflammation reduction

EDUCATIONAL OBJECTIVES

At the conclusion of this educational activity, participants will be able to:

1. Gain an elemental understand of the BaleDoneen Method and its nine fundamental elements.
2. Understand the importance of merging medical and dental into a comprehensive approach to optimal patient care.
3. Learn pathways in which periodontal pathogens can adversely affect arterial wall integrity.
4. Understand the medical and dental connection behind disease/inflammation and significant role periodontal pathogens play in vascular destruction and vascular events.
5. Become aware of the role genetics and salivary bacterial DNA testing can play in treatment planning and individualize treatment recommendations.

ABSTRACT

The BaleDoneen Method—a medical model found to be effective in preventing heart attack, stroke, and diabetes—stresses the importance of oral health in the reduction of inflammation and bacterial burden causing vascular destruction. According to several studies conducted by the American Heart Association, periodontal disease presents Level A evidence that it is independently associated with arterial disease. This model challenges the current standard of health care, utilizing a holistic, comprehensive, preventative approach focused on a disease/inflammatory treatment paradigm to achieve optimum health. The philosophy is founded on the presence or absence of plaque (disease) in the arterial wall. By uncovering the underlying cause of inflammation—potentially initiated by the oral cavity—and providing appropriate treatment, the arterial disease process can be arrested.

The BaleDoneen Method was developed in 2003 by Bradley Bale, MD, medical director of the Heart Health program in Lubbock, Texas, and Amy Doneen, DNP, ARNP, medical director of the Heart Attack and Stroke Prevention Center in Spokane, Washington. Together, they created a program of disease prevention focused on eliminating inflammation affecting the vascular system.[1,2] Research has shown how important oral health is in maintaining overall health.[3] Their program stresses the importance of evaluating the oral cavity for underlying inflammatory conditions. This method is dynamic in nature, evolves as the science and research dictate, and strives for optimum care.

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This course was written for dentists, dental hygienists and assistants, from novice to skilled.

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PennWell designates this activity for 3 continuing educational credits.

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"This course meets the Dental Board of California's requirements for 3 units of continuing education."

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THE NINE COMPONENTS OF THE BALEDONEEN METHOD[1]

1. Education: Each patient is educated about the disease state of atherosclerosis and understands how myocardial infarctions and ischemic strokes occur.

2. Disease: Using noninvasive office-based techniques to find asymptomatic vascular disease, each patient is evaluated for the presence of atherosclerosis and monitored yearly for any changes.

3. Fire/inflammation: Biomarkers are used routinely to determine the inflammatory state of the vascular system. Endothelial markers include high-sensitivity C-reactive protein (hs-CRP), microalbumin-creatinine urine ratio, fibrinogen, and bilirubin. Lipoprotein-associated phospholipase A_2 is evaluated for intima-media activity. Patients are instructed to have these biomarkers completed every three months.

4. Root causes: Root cause or causes of atherosclerotic process is determined and managed for each person.

5. Optimum goals: Goals of therapy are set by peer-reviewed, reliable research and guidelines, with optimum targets in an attempt to minimize risk and often go beyond the values set for the standard of care. Attainment of goals is evaluated every three to six months.

6. Genetics: Genetic information is obtained to aid in the assessment of cardiovascular risk and guide therapy. Genetic testing need be completed only once: 9p21 ("heart attack gene"), APOE (lifestyle), KIF6 (encodes kinesin or statin response), 4q25 (predisposed to a-fib), CYP2C19 (metabolizer of certain antiplatelet medication), TCF7L2 (predisposed to type 2 diabetes), interleukin-1 (escalates the body's inflammatory response), haptoglobin genotype (cardiovascular danger in diabetics).

7. Individualized treatment: Tailor the treatment of individual patients based on their unique characteristics.

8. Reassess regularly: Urine and blood tests are done in quarterly intervals because inflammation can flare up suddenly.

9. Annual disease assessment: Intima-media thickness (IMT) and other tests are done yearly to determine atherosclerotic disease progression and followed over time.

ASSOCIATED CONDITIONS AND CAUSES OF INFLAMMATION

Medical and dental health-care providers should be aware of the many associated conditions and root causes of inflammation (figures 1,2).[5] Including the red flag conditions on the health history form will help identify potential associated risks of inflammation that can adversely affect patient treatment outcomes. There are several root causes of inflammation. This article will focus on insulin resistance (IR)/diabetes, periodontal disease, and endodontic lesions.

INSULIN RESISTANCE AND DIABETES

Individuals can be insulin resistant (IR), a prediabetic proatherogenic state, for many years and not know it.[6] Once a person becomes resistant to insulin, they start down the path to type 2 diabetes and increase their arterial and cardiovascular risk.[7] Several studies indicate that there is evidence that IR is present in the majority of atherosclerotic vascular disease (ASVD) and cardiovascular disease (CVD) cases, and triples the risk of stroke.[8,9] One of the major

Figure 1: BaleDoneen Method red flags.

Periodontal disease	Psoriasis
High blood pressure	Breast cancer
HR > 75	Depression or anxiety
Smoking (nicotine use in any form)	Metabolic syndrome
Abdominal obesity	Diabetes
Insulin resistance	Gestational diabetes
High birth weight > 9 lbs.	Preeclampsia
Hodgkin's lymphoma	Polycystic ovary syndrome
Osteoporosis	Inflammatory bowel disease
Migraines with or without aura	Systemic lupus erythematosus
Autoimmune diseases	Rheumatoid arthritis
Ankylosing spondylitis	Asthma
Hypo- or hyperthyroidism	Erectile dysfunction
Gout	Retinopathy
Renal issues	Sleep issues (obstructive sleep apnea)
Kidney stones	Sjögren's syndrome
Frank's sign (ear creases)	Male-pattern baldness
Career fields (e.g., firefighters)	Fordyce granules
Gallstones	Menopause
Divorce	Working long hours

Familiarize yourself with the evidence behind each of these. They can go undetected in your patients, increasing the risk for a vascular event or diabetes and may affect dental treatment outcomes.

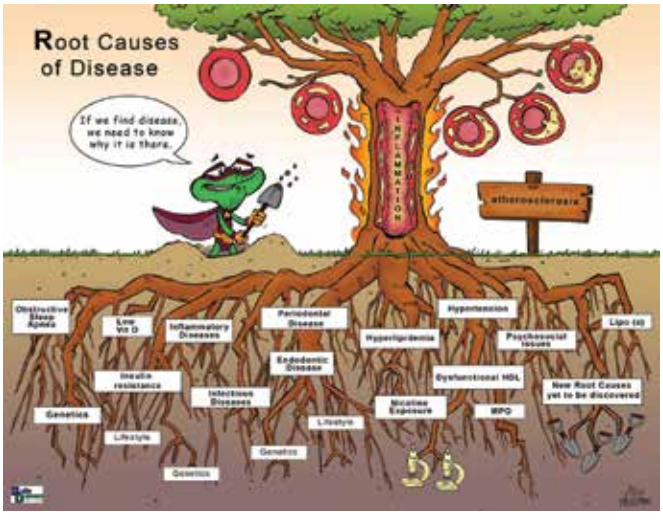


Figure 2: The science dictates the importance of identifying and treating the cause of the inflammation to arrest the arterial disease process.

contributing factors for the alarming rate of recidivism of cardiovascular events is the failure to diagnose insulin resistance.[10] On average, it takes 10 years for a person who is IR to convert to type 2 diabetes.[11] Periodontal disease worsens insulin resistance, and having insulin resistance raises the threat of developing gum disease.[12] Dental professionals are aware of the association between type 2 diabetes and periodontal disease.[13] The severity and prevalence of periodontitis is increased in people with diabetes and worse in people with poorly controlled diabetes.[14] The oral glucose tolerance test (OGTT), a gold standard test, is routinely completed in a BaleDoneen practice. The patient is given 75 gms glucose, and blood samples are taken afterward to determine how quickly it is cleared from the blood. The test is used to determine if IR is an underlying cause of inflammation.[15] Some dental professionals have incorporated HbA1c (blood test measuring average blood glucose levels over past three months) into their practices.[16] A study by Lalla et al. utilized two dental parameters of at least four missing teeth and 26% of pocket depths 5 mm or greater, coupled with an HbA1c of 5.7% or higher, and they predictably identified 92% of true diabetes cases.[17] There is not enough sensitivity or specificity to use these parameters on their own, but using them together yields great clinical value.

PERIODONTAL DISEASE IS INDEPENDENTLY ASSOCIATED WITH VASCULAR DISEASE

According to several studies conducted by the American Heart Association, periodontal disease presents Level A evidence that it is independently associated with arterial disease.[18] Inflammation and bacterial burden have been found to be causal of vascular destruction.[19] Oral infections may directly contribute to thermogenesis and thromboembolic events, providing repeated systemic vascular challenges of proinflammatory cytokines, periodontal pathogens, and lipopolysaccharides (LPS).[20] As with chronic infections elsewhere in the human body, chronic periodontal infections are associated with systemic changes to the blood and the blood-forming organs.[21] The amount of inflammation in the periodontal tissue is correlated to histological findings of inflammation in the carotid artery and aorta. The more inflamed the periodontal tissue, the more inflamed the carotid artery and aorta.[22]

PERIODONTAL PATHOGENS DRIVE ARTERIAL INFLAMMATION

There are several ways that periodontal pathogens can drive inflammation through toll-like receptors (TLRs), direct damage to the endothelium via toxins, and direct pathogenic invasion:[23–27]

- TLRs, especially TLR-4, recognize patterns on pathogens, including lipopolysaccharides of gram-negative oral pathogens. They act as a bridge that, when stimulated, causes a series of biochemical reactions to occur inside the cell, allowing nuclear factor-kappa beta (NF-**κB**) in the cytoplasm to translocate into the nucleus of the cell. This changes the genetics, resulting in the production of proinflammatory cytokines such as interleukin-1, interleukin-6, tumor necrosis factor-alpha, cell adhesion molecules by vascular endothelial cells, and enhances the release of matrix metalloproteinase by macrophages.[23]
- Direct toxic effects of bacteria such as *Aggregatibacter actinomycetemcomitans* (Aa) produce leukotoxins that are detrimental to the endothelial cells by decreasing proliferation, apoptosis (kills endothelial cells), and increasing adhesion molecules to trap white blood cells on endothelial cells.[24] Endothelial cells line every arterial wall surface and protect the intima-media from blood flow.[25] If there is an insult or change in permeability, cholesterol molecules and oxidized LDLs, including periodontal pathogens, can get into the arterial wall, forming plaque.[26]
- Finally, periodontal bacteria can directly invade the arterial wall. *Fusobacterium nucleatum* (Fn) have proteins on the outside of their surface called FadA (*Fusobacterium adhesin A*), which attach to the cadherins. Cadherins are proteins that hold the endothelial cells tightly together. When the FadA bind to the cadherins, the endothelial cells start to separate, increasing the permeability and allowing bacteria to enter into the arterial wall.[27]

Gaetti-Jardim et al. studied periodontal pathogens in the coronary plaques of 44 patients who underwent coronary endarterectomies. Thirty-nine patients had periodontal disease, and 36 of 39 were positive for periodontal pathogen DNA, *Porphyromonas gingivalis* (Pg), *Aggregatibacter actinomycetemcomitans* (Aa), *Tannerella forsythia* (Tf), *Prevotella intermedia* (Pi), and *Peptostreptococcus micros* (Pm). *Porphyromonas gingivalis* (Pg), *Aggregatibacter actinomycetemcomitans* (Aa), and *Prevotella intermedia* (Pi) were detected most often and 64% of the plaques had two or more pathogens in them.[28] Additionally, another study of 42 carotid endarterectomy patients analyzed the DNA for periodontal pathogens and concluded that all samples had at least one, and many had multiple pathogens.[29] Periodontal pathogens are found in arterial plaque as shown in the following patient example.

A 72-year-old male presented with severe periodontal disease (figure 3), radiographic evidence of disease (figure 4), and a carotid duplex showed atherosclerosis/stenosis (figure 5). An endarterectomy was completed on the left carotid artery (figure 6), and plaque was surgically removed (figure 7) and dissected to determine bacterial content (figure 8). The pathogens found in the dissected carotid artery plaque and diseased periodontal tissue were the same.[30]

According to the American Heart Association, every 40 seconds someone has a stroke, and every four minutes, someone dies from one.[31] Strokes, although preventable, are the leading cause of disability.[32] The buildup of plaque and fatty streaks in the arterial wall is a silent process until the disease is more advanced or an event occurs. Periodontal disease is an important risk factor for ischemic stroke; therefore, screening for inflammation is crucial. Treating the disease has been found to lower the risk of stroke, especially among individuals ages 20–44.[33] Clinicians need to fully understand the importance of aggressive assessment and treatment to keep the oral cavity free of underlying chronic inflammation.

ORAL HEALTH ASSESSMENTS FIND DISEASE

As interrelationships develop between

Figure 3: 72-year-old man with severe periodontal disease.

Figure 4: Radiographic evidence of bone loss and periodontal disease.

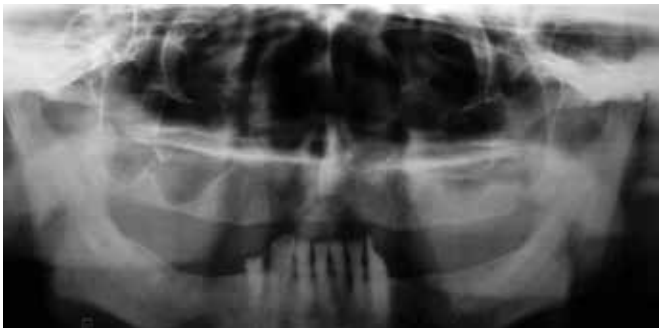


Figure 5: Carotid duplex ultrasound indicating severe atherosclerosis/stenosis of left carotid artery.

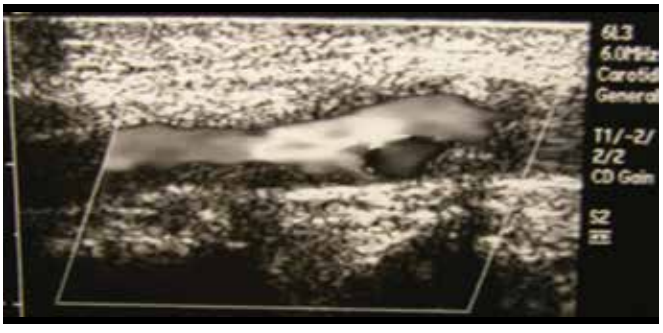


Figure 6: Post left carotid endarterectomy.

Figure 7: Specimen shows carotid plaque as cause of stenosis.

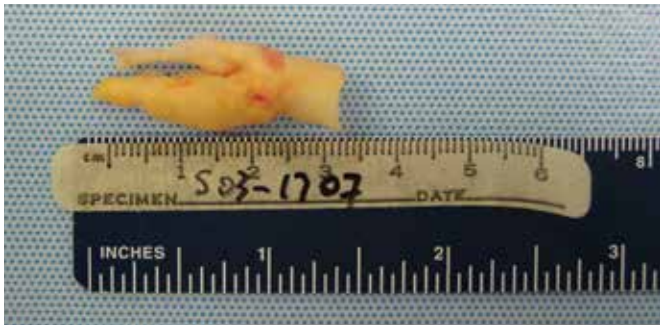
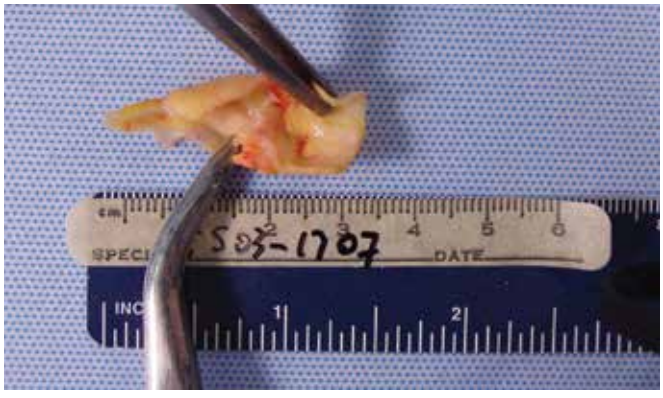


Figure 8: Dissected carotid plaque with contents same as bacterial plaque found in infected teeth.



Note: *Figures 3–8 reprinted with permission from Talib Najjar, DMD, PhD, MDS, Rutgers School of Dental Medicine, published by Medscape Drugs & Diseases (http://emedicine.medscape.com/), 2016, available at: http://emedicine.medscape.com/article/1081424-overview.*

health-care providers, more patients will be referred to dental professionals for oral evaluations and vice versa. The founders of this method encourage medical and dental health-care providers to work hand in hand to extinguish all forms of inflammation in patients.

A 35-year-old male referred by his medical provider for an oral health assessment presented with a family history of heart attack (father's dad died at age 60), stroke, type 2 diabetes (adult onset), arthritis, cancer, thyroid disorder, and smoking. The assessment included OralDNA salivary testing, Celsus One (a genetic analysis for markers of oral and systemic inflammation), cone beam 3-D imaging, oral cancer screening, periodontal assessment, and a comprehensive dental exam.

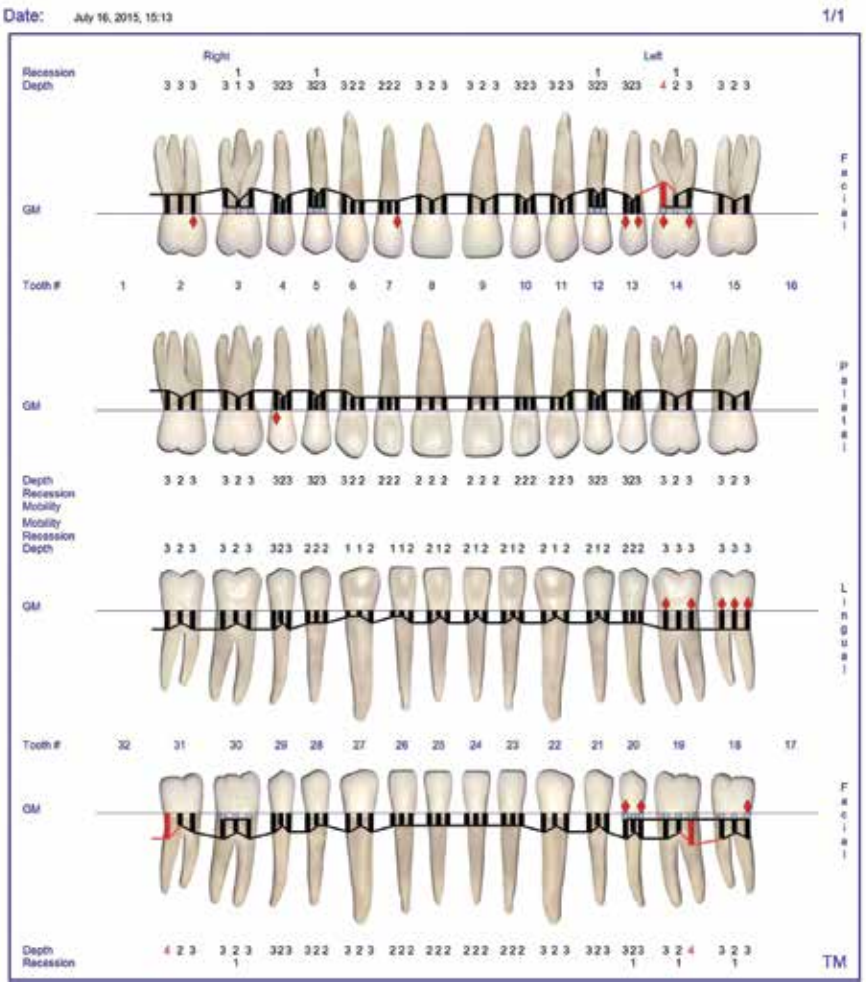
FINDINGS

The patient's periodontal charting (figure 9) shows isolated areas of bleeding and 4 mm pocketing. OralDNA testing indicated three bacteria above threshold. Two were in the high-risk category. As stated in the Gaetti-Jardim et al. studies, Pg and Tf pathogens were commonly found in dissected endarterectomy plaque.[34] The bacteria load presents a significant potential vascular risk for this patient (figure 10). The Celsus One report indicated the patient is predisposed to periodontal bacterial infections, accentuated inflammatory response to the pathogens, and intermediate risk of chronic systemic inflammation (figure 11).[35] The dental exam and x-rays showed no caries or endodontic lesions.

TREATMENT

The treatment plan consisted of four quadrants of periodontal therapy for biofilm disruption and decontamination, and amoxicillin 500 mg t.i.d and metronidazole 500 mg b.i.d for 10 days per bacterial

Figure 9: Pretreatment charting indicated isolated areas of inflammation and active disease with 3–4 mm pocketing in active sites.



report recommendations. The patient bled heavily during the therapy appointments, indicating that bleeding when probing and screening can be misleading in regard to bacterial tissue invasion and host response.[36] A reevaluation was completed in six weeks, including a follow-up OralDNA saliva test (figure 12). All bacteria were below threshold and a strict three-month maintenance schedule was advised and followed. Completion of periodontal charting at the three-month hygiene appointment showed no active bleeding or inflammation (figure 13). The tissue health was maintained and stable. A yearly OralDNA salivary diagnostic test, a strict three-month maintenance schedule, and periodontal charting every six months were recommended. This protocol allows for frequent assessment to ensure a noninflammatory state is maintained.

CHRONIC ENDODONTIC LESIONS ALSO FOUND TO CONTRIBUTE TO SYSTEMIC INFLAMMATION

The BaleDoneen Method recommends that dental professionals look for asymptomatic endodontic lesions as an underlying cause of inflammation. These lesions can go undetected and cause a chronic inflammatory state. Dr. Bale and Dr. Doneen have encountered situations when a patient's inflammatory markers have spiked, and the underlying cause was identified at the dental evaluation. Subsequently, after dental treatment, inflammatory markers went back to the original levels. In 2013, Pessi et al. studied acute heart attacks and oral pathogens by analyzing the arterial blood and thrombus of a person who just had a heart attack for bacterial DNA. The DNA load was 16 times greater in the thrombi than the arterial blood. The findings showed that *Streptococcus virdens* was found 78% of the time, and periodontal pathogens 35%, which would indicate that the periodontal pathogens came out with the clot.[37] Similarly, a study utilizing panoramic and CT (x-ray based computed tomography) looking for endodontic lesions in 30 patients having had an acute heart attack found 50% had periapical abscesses. If the thrombi were positive for *Streptococcus virdens*, they were 13 times more likely to have a periapical abscess,[38] suggesting that nearly half of heart attacks are being triggered potentially by oral infections. Dissemination of oral microorganisms into the bloodstream is common, and less than one minute after an oral procedure, organisms from the infected site may have reached the heart, lungs, and peripheral blood capillaries. [39] In addition, there is evidence that up to 30 days after a dental procedure, a person can have a vascular event.[40,41] The BaleDoneen Method embraces the science behind these studies. The founders of this method have recently published an article on high-risk periodontal pathogens contributing to the pathogenesis of atherosclerosis.[42] They stress the importance of having a thorough oral health and wellness assessment to patients who have suffered a heart attack or are trying to prevent a heart attack.

SUMMARY

The BaleDoneen Method provides optimum patient treatment in eradicating arterial disease, one of the biggest health risks affecting the global population. The authors of this method advocate for medical and dental professionals to use an interdisciplinary team approach to address this issue. As integrated health-care systems evolve, the dental professional's role becomes more vital to patients' total health and well-being. Today, forward-thinking medical and dental providers are working together to offer comprehensive, science-based treatment. Merging the science and technology available—with an understanding of associated medical inflammatory conditions—will aid in the development of a comprehensive treatment plan, leading to improved overall health.

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Figure 11: The Celsus One report indicates the patient is predisposed to periodontal bacterial infections, an accentuated inflammatory response to bacterial pathogens, and intermediate risk of chronic systemic

CELSUS ONE: GENETIC ANALYSIS FOR MARKERS OF ORAL AND SYSTEMIC INFLAMMATION

Type of immunity	Gene marker	Genotype	Inflammation index
Innate	Beta-defensin 1 (DEFB1)	G/A	**Low risk**
	CD14 (CD14)	T/C	
	Toll-like receptor 4 (TLR4)	AA/CC	
Acquired	Tumor necrosis factor alpha (TNF-alpha)	C/C	**Intermediate risk**
	Interleukin 1 (IL1)	CT/CT	
	Interleukin 6 (IL6)	C/G	
	Interleukin 17A (IL17A)	G/G	
	Matrix metallopeptidase 3 (MMP3)	5A/6A	

Note: *OralDNA and Celsus One lab results, permission from patient to use. Lab information: OralDNA salivary diagnostics. OralDNA Labs. A service of Access Genetics LLC. 7499 Flying Cloud Drive, Eden Prairie, MN. Ph: (855)-ORALDNA. Fx: (952) 767-0446. www.oraldna.com.*

Figure 10: OralDNA salivary diagnostics indicate patient above threshold in two high-risk pathogens.

Result: Positive — 3 Pathogenic bacteria reported above threshold Pg Tf Cs

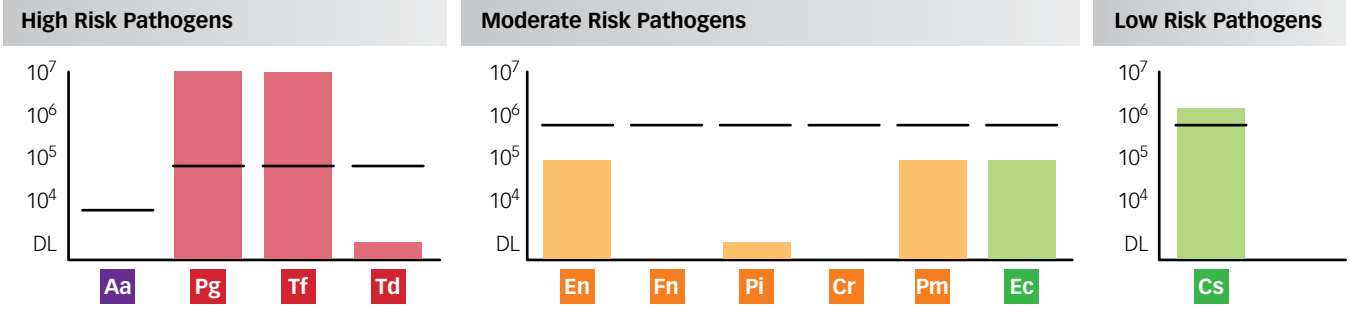
Bacterial risk: High — very strong evidence of increased risk for attachment loss

Legend

— = pathogen load threshold*

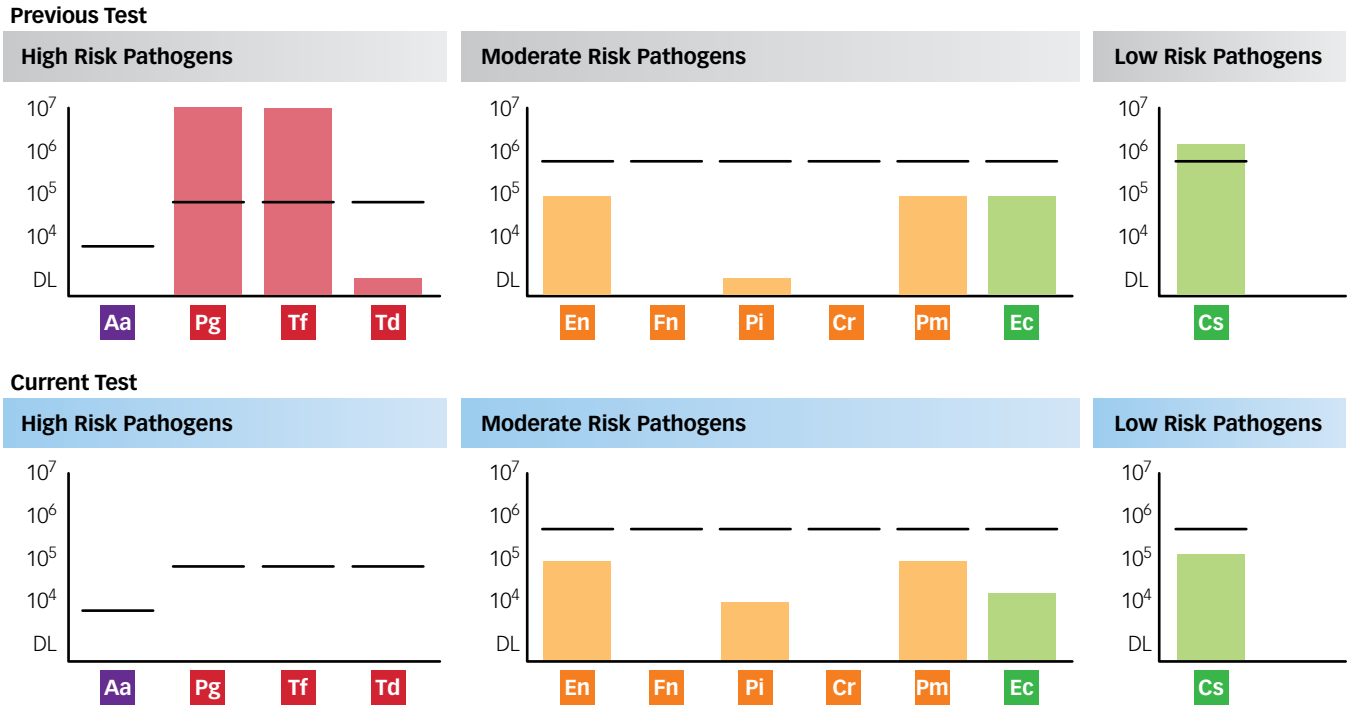
DL = detection limit

Result interpretation: Periodontal disease (PD) is caused by specific or groups of specific bacteria. Threshold levels represent the concentration above which patients are generally at increased risk for attachment loss. Bacterial levels should be considered collectively and in context with clinical signs and other risk factors.



Pathogen	Result	Clinical significance
Pg Porphyromonas gingivalis	High	Very strong association with PD: Transmittable, tissue invasive, and pathogenic at relatively low bacterial counts. Associated with agressive forms of disease.
Tf Tannerella forsythia	High	Very strong association with PD: Common pathogen associated with refractory periodontitis. Strongly related to increasing pocket depths.
Cs Capnocytophaga species (gingavalis, ochracea, sputigena)	High	Some association with PD: Frequently found in gingivitis. Often found in association with other periodontal pathogens. May increase temporarily following active therapy.

Figure 12: Posttreatment test 10/15/15 showed all bacteria were below threshold.



Comparison summary
Since patient's last test on 7/22/2015:

- Total number of pathogens detected has increased from eight to five
- High-risk pathogens detected have decreased from three to zero
- Pathogens detected above threshold have decreased from three to zero
- Above-threshold high-risk pathogens have decreased from two high-risk pathogens to zero

Doctor's comparison observations:

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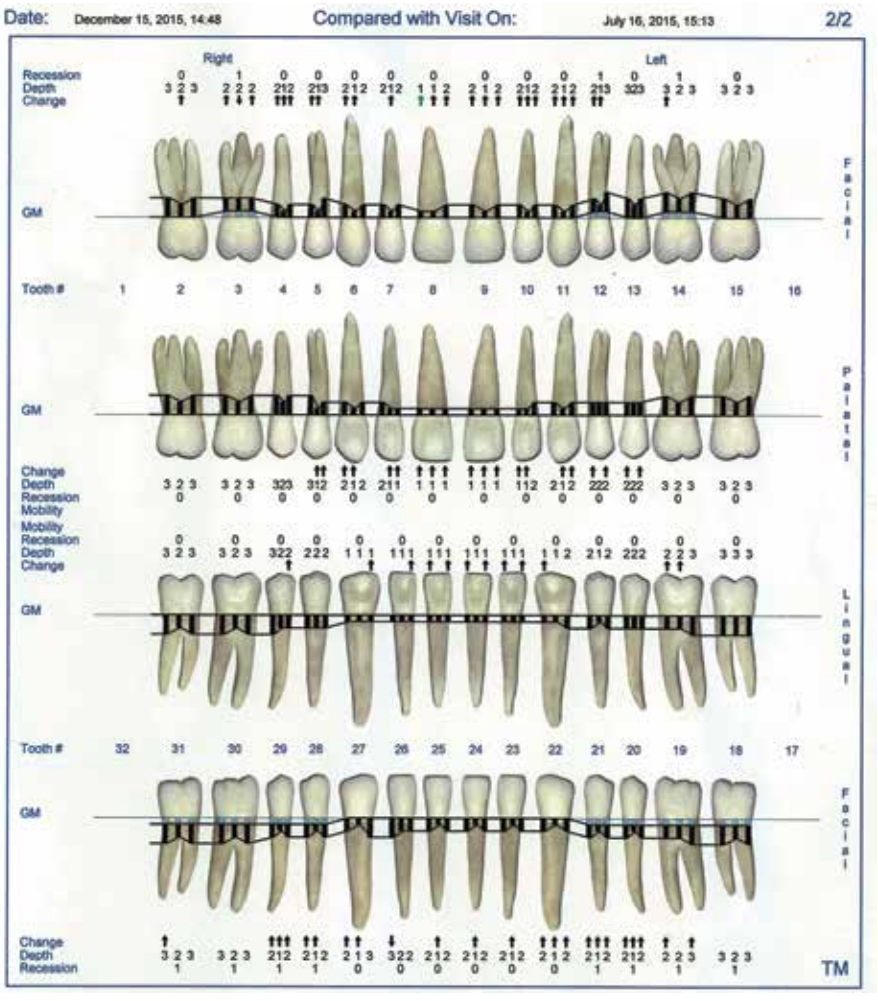
Figure 13: Periodontal charting posttreatment. Patient is in a healthy state and no inflammation indicated with BOP or instrumentation.



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KRISTON REISNOUR, BSDH, RDH, CCSH, CSOM, is an accomplished speaker, writer, and dental hygienist utilizing an encompassed 29 years of combined experience working for periodontal, cosmetic, general, and dental sleep practices across the United States. Being trained in oral systemic health, sleep dentistry, orofacial myology, ozone, laser, neuromuscular care, and non-invasive periodontal therapy (endoscope/perioscopy) has allowed her to provide optimum patient care. She is dedicated and passionate about coaching hygiene teams to a higher level of understanding of the importance their role as dental health care provider plays in integrated health. A member of ADHA, AAOSH, Speakers Bureau for the Bale/Doneen Method, Credentialed Clinical Sleep Health Educator, Certified Orofacial Myology Specialist, Air Ambassador for SomnoMed, and clinical hygienist. She can be reached at skreisnour@comcast.net

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QUESTIONS

1. The BaleDoneen Method stresses the importance of:

a. Evaluating the mouth as a source of inflammation
b. Annual eye exams
c. Reassessing for inflammation every quarter
d. A and C

2. The BaleDoneen Method is:

a. Dynamic in nature
b. Science and research based
c. Optimal care focused
d. All of the above

3. There are nine elements of the Bale-Doneen Method, which include:

a. Educate, disease, fire, root causes
b. Educate, diagnosis, generics
c. Evaluate, diagnose, root causes
d. Evaluate, fire, root causes

4. Biomarkers that determine an inflammatory state include:

a. Hs-CRP, microalbumin creatinine ratio, fibrinogen, lipoprotein associated phospholipase A2
b. Hs-CRP, microalbumin creatinine ratio, vitamin D levels
c. Microalbumin creatinine ratio, vitamin D levels, fibrinogen
d. Cholesterol levels, myeloperoxidase, hs-CRP

5. How often are inflammatory markers checked?

a. Once a year
b. Every 3 months
c. Every 4 months
d. Every 6months

6. What are some common genetic tests utilized to assess and guide cardiovascular treatment?

a. 9p21, Apo E, KIF6, 4q25
b. CYP2C19, TCF7L2, interleukin 1, haptoglobin
c. A and B
d. None of the above

7. Some red flags associated with inflammation include:

a. Migraine headaches
b. Career fields
c. Erectile dysfunction
d. All of the above

8. Root causes that can cause vascular inflammation are:

a. Obstructive sleep apnea, low vitamin D, periodontal disease
b. Lifestyle, nicotine exposure, cholesterol, Lip(a)
c. Infectious diseases, hypertension, sleep issues
d. All of the above

9. Chronic infections in the body, such as chronic periodontal disease, can be associated with:

a. Health and vitality
b. Systemic changes to the blood and blood-forming organs
c. Health issues
d. B and C

10. The amount of inflammation in the periodontal tissue has been found to be correlated to the histological findings in:

a. Carotid arteries
b. Aorta
c. Kidneys
d. A and B

11. Insulin resistance is:

a. A proatherogenic and proinflammatory state
b. Present in the majority of individuals with ASVD
c. Diagnosed with OGTT (oral glucose tolerance test)
d. All of the above

12. What test is given to determine if insulin resistance is an underlying cause of inflammation?

a. Hs-CRP
b. OGTT
c. A1c
d. Fibrinogen

13. According to Lalla and colleagues, what dental parameters must be met to identify diabetes with 92% accuracy?

a. Missing 4 or more teeth
b. 26% of probing depths equal or greater than 5 mm
c. A and B
d. None of the above

14. After reviewing several studies, the American Heart Association feels that periodontal disease presents what level of evidence that it is independently associated with arterial disease?

a. Level A
b. Level B
c. Level C
d. None of the above

15. Periodontal pathogens can drive inflammation by which of these modes?

a. Toxic effect
b. Toll-like receptors
c. Direct invasion
d. All of the above

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QUESTIONS

16. Actions of toll-like receptors include:

a. Recognizing patterns on pathogens
b. Causing biochemical reactions to occur within the cell
c. Affecting cell genetics
d. All of the above

17. Leukotoxins produced by gram-negative bacteria will cause what action?

a. Increase proliferation
b. Kill (apoptosis) endothelial cells
c. Decrease adhesion molecules
d. None of the above

18. *Fusobacterium nucleatum* bacteria have proteins on the outer surface called:

a. Vascular endothelial cadherin
b. Cellular proteins
c. Fadherin (fusobacterium adhesin A)
d. Endothelial proteins

19. In the Gaetti-Jardim study to detect bacteria in the coronary arterial plaque, the bacteria found most often were:

a. *Prevotella intermedia, Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans*
b. *Fusobacterium nucleatum, Prevotella intermedia, Aggregatibacter actinomycetemcomitans*
c. None of the above
d. All of the above

20. According to the American Heart Association, every ___ seconds someone has a stroke; every ___minutes someone dies from one:

a. 30 seconds; 4 minutes
b. 40 seconds; 4 minutes
c. 40 seconds; 2 minutes
d. 30 seconds; 5 minutes

21. There are 11 bacteria analyzed in a salivary DNA bacterium testing for periodontal disease. Those bacteria considered high risk for periodontal disease are:

a. Aa, Pg, Tf, Td
b. Aa, Pg, Cs, Pi
c. Aa, Pm, Ec, Pi
d. Pg, Pi, Pm, Ec

22. Although considered moderate risk on the salivary diagnostic test, which bacteria have fadherins that can attach to the vascular endothelial cadherin?

a. Pm
b. Cs
c. Fn
d. Ec

23. What test is used to obtain a genetic analysis of oral-systemic inflammation?

a. Celsus One
b. 9p21
c. 4q25
d. Apo E

24. In Pessi's study of acute heart attacks and oral pathogens, the bacterial DNA load was how much

a. greater in the thrombi than in the arterial blood?
b. 10 times
c. 5 times
d. 2 times
e. 16 times

25. Of the bacterial DNA load found in Pessi's study of acute heart attacks, what was the percentage of *Streptococcus virdans*?

a. 64%
b. 78%
c. 50%
d. 75%

26. What percentage of time were periodontal pathogens found in Pessi's study?

a. 78%
b. 50%
c. 35%
d. 25%

27. Dissemination of microorganisms into the bloodstream after a dental procedure can reach what area/areas?

a. Heart
b. Lungs
c. Peripheral blood capillaries
d. All of the above

28. There is evidence that suggests that a person can have a vascular event up to how many days after a dental procedure?

a. 7
b. 10
c. 30
d. 15

29. This medical model:

a. Challenges the current standard of health care
b. Utilizes a comprehensive preventative approach
c. Focuses on disease/inflammatory treatment paradigm
d. All of the above

30. The BaleDoneen Method advocates for:

a. Medical and dental providers to work together
b. Having oral health and wellness assessments
c. An oral health-care component for all cardiovascular programs
d. All of the above

PUBLICATION DATE:	JANUARY 2019
EXPIRATION DATE:	DECEMBER 2021

ANSWER SHEET

BaleDoneen Method: Medical model emphasizing dental health component in inflammation reduction

Name: Title: Specialty:

Address: E-mail:

City: State: ZIP: Country:

Telephone: Home ( ) Office ( )

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EDUCATIONAL OBJECTIVES

1. Gain an elemental understand of the BaleDoneen Method and its nine fundamental elements.
2. Understand the importance of merging medical and dental into a comprehensive approach to optimal patient care.
3. Learn pathways in which periodontal pathogens can adversely affect arterial wall integrity.
4. Understand the medical and dental connection behind disease/inflammation and significant role periodontal pathogens play in vascular destruction and vascular events.
5. Become aware of the role genetics and salivary bacterial DNA testing can play in treatment planning and individualize treatment recommendations.

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1. Were the individual course objectives met?

Objective #1: Yes No Objective #2: Yes No

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Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall?	5	4	3	2	1	0
3. Please rate your personal mastery of the course objectives.	5	4	3	2	1	0
4. How would you rate the objectives and educational methods?	5	4	3	2	1	0
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10. Do you feel that the references were adequate? Yes No

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_____

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_____

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3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	21.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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