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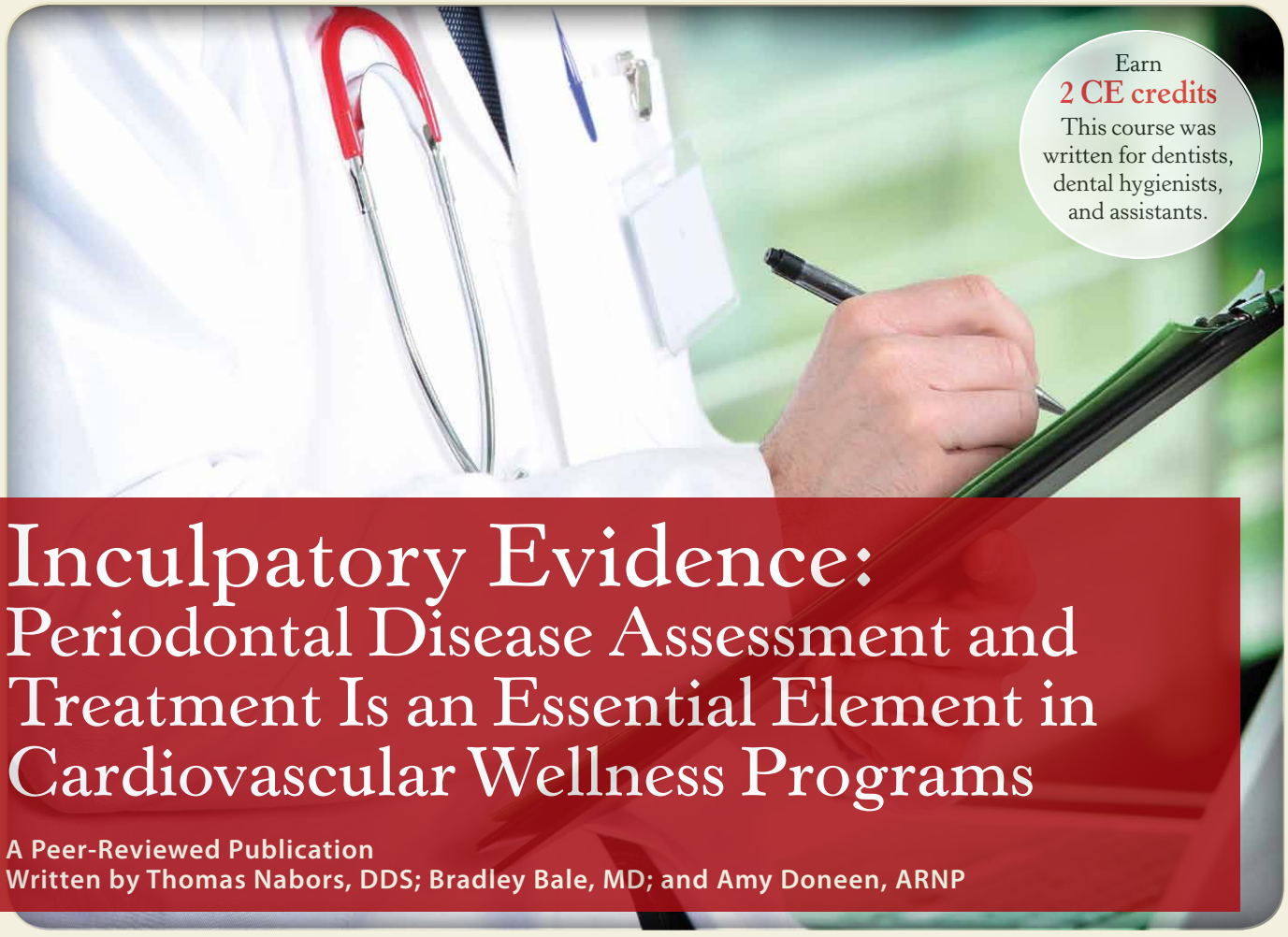

Inculpatory Evidence:
Periodontal Disease Assessment and Treatment Is an Essential Element in Cardiovascular Wellness Programs

A Peer-Reviewed Publication
Written by Thomas Nabors, DDS; Bradley Bale, MD; and Amy Doneen, ARNP

Abstract
The premature morbidity and mortality of cardiovascular disease (CVD) is claiming the lives and independence of millions of Americans. The key to CVD prevention is to determine if an atheroma is present in the vascular tree and minimize any opportunity for thrombus development through rupture or erosion. This is accomplished by mitigating the risk of any vascular inflammation, including the systemic impact of periodontal disease (PD). Evidence supports inflammation as a key player in the development and progression of atherosclerosis. In addition, inflammation is involved in destabilizing the plaque and in promoting thrombosis. A meta-analysis from the *AHA Journal* reviewed the prevalence and incidence of coronary heart disease (CHD) as being significantly increased in subjects with periodontitis and confirmed an independent association between PD and CVD with Level A evidence. Current genetic research indicates that inflammation appears to be causal for CVD, which intensifies the importance on the oral-systemic inflammatory link. Proving direct causality between oral health and vascular events proves challenging due to lack of uniformity in clinical diagnostic criteria and clinical treatment guidelines for PD. Unequivocal evidence of causality is not necessary to address PD in efforts to minimize cardiovascular (CV) risk. Any modifiable CV risk factor should be addressed in a holistic approach to lessen the impact of CVD. The plethora of evidence supporting the systemic inflammatory link with PD along with its independent association with CAD makes it imperative that efforts to enhance cardiovascular wellness incorporate PD evaluations and therapies., Guidelines should call for the assessment and treatment of PD not only for the patient's oral health, but also as one of many comprehensive measures that may help maintain CV health.

Learning Objectives:
The participants will appreciate the catastrophic economic and health expenditures associated with cardiovascular disease.

1. The learners will synthesize the value of educating patients on the pathophysiology of arterial disease.
2. The student will understand that it is inflammation that can trigger a plaque rupture and potential thrombus that could block the flow of blood.
3. The learner will recognize that the current health-care platform is based on a risk factor paradigm rather than a disease treatment paradigm.
4. The reader will appreciate the inflammatory link between periodontal disease and vascular disease.
5. The learner will articulate the opportunity to identify silent atherosclerosis in the artery wall using carotid intima media thickness testing.
6. The student will appreciate that periodontal disease is associated with many vascular inflammatory markers such as hsCRP, fibrinogen, microalbumin, and Lp-PLA2.
7. The participant will elucidate the value of objectifying the clinical diagnosis of periodontal disease by using oral pathogen burden evaluation rather than simply relying on clinical symptoms.
8. The learner will recognize that the dental provider plays an essential role in the prevention of heart attacks and ischemic strokes.
9. The reader will understand the merit of Level A evidence that connects periodontal disease with arterial disease.

Author Profiles
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Author Disclosure
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Abstract

The premature morbidity and mortality of cardiovascular disease (CVD) is claiming the lives and independence of millions of Americans. The key to CVD prevention is to determine if an atheroma is present in the vascular tree and minimize any opportunity for thrombus development through rupture or erosion. This is accomplished by mitigating the risk of any vascular inflammation, including the systemic impact of periodontal disease (PD). Evidence supports inflammation as a key player in the development and progression of atherosclerosis. In addition, inflammation is involved in destabilizing the plaque and in promoting thrombosis. A meta-analysis from the AHA Journal reviewed the prevalence and incidence of coronary heart disease (CHD) as being significantly increased in subjects with periodontitis and confirmed an independent association between PD and CVD with Level A evidence. Current genetic research indicates that inflammation appears to be causal for CVD, which intensifies the importance on the oral-systemic inflammatory link. Proving direct causality between oral health and vascular events proves challenging due to lack of uniformity in clinical diagnostic criteria and clinical treatment guidelines for PD. Unequivocal evidence of causality is not necessary to address PD in efforts to minimize cardiovascular (CV) risk. Any modifiable CV risk factor should be addressed in a holistic approach to lessen the impact of CVD. The plethora of evidence supporting the systemic inflammatory link with PD along with its independent association with CAD makes it imperative that efforts to enhance cardiovascular wellness incorporate PD evaluations and therapies., Guidelines should call for the assessment and treatment of PD not only for the patient's oral health, but also as one of many comprehensive measures that may help maintain CV health.

Impact of CVD

Cardiovascular disease (CVD) is bankrupting our health-care system. We currently spend approximately $300 billion annually with direct and indirect costs of CVD. This figure is projected to triple in the next two decades.[1] The life expectancy for Americans is now 78 years of age. Amazingly, one-third of all cardiovascular (CV) deaths occur in individuals younger than 75 years old and more than 150,000 CV deaths are in people less than 65 years old.[2] If we continue to allocate these resources for the treatment of end-stage effects of CVD, our country will suffer further financial bankruptcy. Additionally, the premature morbidity and mortality of CVD is claiming the lives and independence of millions of Americans. Rather than continuing to feed this imbalance of resources, it is reported that we can realign financial attention to prevention efforts, which will save billions of dollars in medical expenditures. Every $1 spent in wellness programs would save $3.27 in medical costs and $2.73 in absenteeism costs. "What we spend on CVD is not sustainable. But, we can afford to prevent it." — cardiologist, Dr. William S. Weintraub.[3]

Event reality:

Understanding the mechanism from which heart attack or ischemic stroke occurs allows for an appreciation of the connection between periodontal disease (PD) and vascular events. Atherosclerotic plaque lesions develop silently within the artery wall, often with minimal luminal encroachment. When an artery wall weakens due to an influx of inflammation, the protective endothelial lining can rupture or erode, thus exposing the plaque to blood flow, creating the potential for thrombus formation. Not all plaque ruptures or erosions result in major CV events; some lead to progression of plaque volume or create microvessel thrombi that cause microvascular disease states such as silent heart attacks and silent strokes, which contribute to vascular dementia and peripheral vascular disease. Regardless of the resulting effects of a thrombus, the presence of an atheroma is essentially a condition sine qua non for an event.[6] The key to CVD prevention is to determine if an atheroma is present in the vascular tree and minimize any opportunity for thrombus development through rupture or erosion. This is accomplished by mitigating the risk of any vascular inflammation, including the systemic impact of PD.

Risk factors for heart attack and stroke:

The INTERHEART study[4] and the INTERSTROKE study[5] have helped elucidate numerous risk factors and emphasize the need for a holistic approach to maintain CV wellness. These are large scale, epidemiological trials that mitigate socioeconomic, gender, and racial barriers to determine the risk factors for both heart attacks and strokes. They provide the evidence to support that a comprehensive approach to treatment is necessary in order to reduce the morbidity and mortality associated with heart attacks and strokes. It is now clear that simply treating blood pressure and cholesterol will not eliminate costly and deadly CV events. Evidence supports inflammation as a key player in the development and progression of atherosclerosis. In addition, inflammation is

involved in destabilizing the plaque and promoting thrombosis. Inflammation is a manifestation of underlying pathology, such as obesity, stress, hypercholesterolemia, diabetes, and infection.[6] A comprehensive CV wellness program needs to evaluate each individual patient for all conditions known to be associated with CV risk or which can stimulate systemic inflammation.

Association between PD and CVD

Numerous cross-sectional epidemiological studies provide evidence of an association between periodontitis and elevated risk for CVD. Some studies have shown that periodontitis is an independent risk factor for CVD. In addition, there is evidence that specific periodontopathogenic bacteria may play a role in atherogenesis. Recent studies have focused on the systemic effect of periodontal intervention on surrogate indicators of CVD, including serum markers of inflammation, serum lipid levels, measurements of endothelial function, and hemostatic factors.[18] The systemic implications of treating these oral-biofilm infections has supportive evidence of improved glycemic control, reduction of inflammatory biomarkers, improved surrogate measures of vascular endothelial function, and reduction of risk for cardiovascular or cerebrovascular diseases.[19]

The prevalence and incidence of coronary heart disease (CHD) is significantly increased in subjects with periodontitis. A meta-analysis from the *American Heart Journal* (AHJ) reviewed the prevalence and incidence of CHD as being significantly increased in periodontitis subjects. This systematic review of eligible literature included five prospective cohort studies with a follow-up for >6 years. Also, five case-control studies and five cross-sectional studies that were eligible were included in this analysis. This meta-analysis indicated that both the prevalence and incidence of CHD are significantly increased in subjects with periodontitis.[20] The authors concluded that PD may be a risk factor for CHD.

PD as an independent risk factor for CVD

The United States Preventive Services Task Force (USPSTF) established an exhaustive review to aid in evaluating PD as an independent risk factor for incident CHD. The exhaustive search of Medline studies from 1966 to 2008 reviewed prior systematic reviews, reference lists, and consulting experts. Prospective cohort studies, Framingham risk factors, and CHD incidence in the general adult population without known CHD risk factors were reviewed and quality rated. Follow-up studies ranged from five to 21 years. These data concluded that PD is a risk factor and a marker for CHD that is independent of traditional CHD risk factors, including socioeconomic status.[21]

Carotid intima-media thickness testing — surrogate marker for CAD:

Carotid intima-media thickness (cIMT) testing is a safe, noninvasive, reliable, and reproducible test to clinically assess and monitor the atherosclerotic disease process. The appropriate-use criteria for the Society for Atherosclerotic Imaging Prevention (SAIP) has been published, and it is recommended that cIMT be used as a clinical tool for the assessment of vascular health.[25] The Atherosclerosis Risk in Communities study (ARIC)[26] demonstrated that cIMT is a surrogate marker for CHD. Most recently, cIMT was proven to add significant improvement in predicting CHD events when it was added to traditional risk factor models.[27] It is imperative to recognize that emerging evidence indicates PD by the oral pathogen burden can affect vascular health, as evidenced by cIMT.[17]

Recent articles take into consideration periodontal bacteria and their potential influence on vascular injury, including the trial by Desvarieux et al. (INVEST – The Oral Infections and Vascular Disease Epidemiology Study).[17] This review studied the influence of specific periodontal microbiota and their influence on subclinical atherosclerosis using cIMT scans in 657 dentate subjects with no history of stroke or myocardial infarction (MI) and adjusted for known risk factors including age, race, gender, education, BMI, smoking, DM, SBP, LDL, and HDL. The study investigated the relationship between specific periodontal bacteria [*Actinobacillus actinomycetemcomitans (Aa), Porphyromonas gingivalis (Pg), Tannerella forsythensis (Tf)*, and *Treponema denticola (Td.)*], total burden of causative bacteria, and relative predominance of causative bacteria. Their findings were that the overall periodontal bacterial burden was related to cIMT changes. In addition, they found those changes were clinically relevant and also related to specific causative periodontal bacteria and the dominance of etiological bacteria. The conclusion was that this data provides evidence of a direct relationship between periodontal microbiology and subclinical atherosclerosis, independent of C-reactive protein. Furthermore, the IMT changes associated with dominance of etiologic bacteria represented a 15 mm Hg increase in systolic blood pressure, a 0.04 mm increase in cIMT thickness, and was equivalent to a 10-year age difference in subjects without bulbar plaque.[17]

PD bacteria and its influence on vascular injury

The CORODENT study investigated the role of periodontal bacteria and the importance of total pathogen burden in coronary events and PD. The background included the statement that chronic inflammation from any source is associated with increased CV risk. In this controlled study of 263 subjects with confirmed CHD and 526 subjects without a history of CHD, subgingival samples of biofilm were analyzed for specific periodontal pathogens: including *Aa, Pg, Tf, Td*, and *Prevotella intermedia (Pi)*. The results found a statistically significant association between the periodontal pathogen burden or the number of *Aa* in periodontal pockets. The findings suggested an association between periodontitis and presence of CHD.[22]

In 2010, Desvarieux et al. investigated the relationship between periodontal microbiota and hypertension in 653 subjects with no history of stroke or MI (INVEST).[24] The findings were adjusted for the subjects' risk factors, which included age, race, sex, and education, BMI, smoking, DM, LDL, and HDL. The results

were that periodontal etiologic bacterial burden was positively associated with both BP and prevalent hypertension. Comparing highest and lowest tertiles of etiologic bacterial burden, SBP was 9 mm Hg higher and DBP was 5 mm Hg higher (P for linear trend, 0.001 in each case). The bacteria considered to be etiological for PD and used in this investigation included *Aa, Pg, Tf,* and *Td*. The conclusion: "Our data provide evidence of a direct relationship between the levels of these subgingival periodontal bacteria and both SBP and DBP as well as hypertension prevalence. The burden of these periodontal pathogens is positively associated with both BP and prevalent hypertension.[17]

Endotoxins and vascular injury

Endotoxins and exotoxins of gram-negative bacteria have also been the subject of investigation for their potential influence on vascular injury. *Porphyromonas gingivalis (Pg)* is a gram-negative bacteria considered to be causative for periodontitis. A specific toxin of this bacterium, known as lipopolysaccharide endotoxin (LPS), is similar to the toxins in other known bacterial diseases such as *E. coli* and *Salmonella* infections.

A study in 2011 (Lei et al.) investigated the potential that *Pg* LPS was involved in the development of atherosclerosis. They specifically inquired whether this LPS may alter atherosclerotic-related gene expression in oxidized low-density lipoprotein-induced macrophages during and after the formation of foam cells, which aggregated in arterial walls to form atherosclerotic plaques. They found *Pg* LPS stimulated atherosclerosis-related gene expression in foam cells and also stimulated transcription of pro-inflammatory cytokines, adhesion molecules, and growth factors. The authors concluded that the endotoxins of *Pg* are directly involved in the development of ASVD by stimulating atherosclerosis-related gene expression. This adds more evidence to the relationship between periodontal pathogens and CVD.[23]

Recent AHA statement — Level A evidence

Recently, after an extensive search that produced 537 peer-reviewed medical and dental publications, it was concluded that periodontal disease is associated with CVD independent of known confounders. This conclusion was supported by Level A evidence.[7] This level of evidence is extremely difficult to obtain and vitally important. The USASTF states that Level A evidence means that the data improves important health outcomes and that the benefits of the treatment substantially outweigh the risk of harm. They go on to state that the net benefit of the recommendation is substantial and should be provided to eligible patients.[29] Many CV treatment guidelines are not established with Level A evidence. Because of this, clinicians were warned to use caution when embracing guidelines less than Level A. Lockhart et al. pointed out that less than 20% of the American College of Cardiology and American Heart Association guidelines were based upon Level A evidence. Many recommendations were derived from Level C evidence or simply expert opinion.[8] Furthermore, a benefit of periodontal intervention in decreasing local periodontal inflammation is also supported by Level A evidence.[7]

PD and related biomarkers for CVD

MACR

The conclusion of an independent association between PD and CVD continues to be supported by recent publications.[9] The Framingham study found UACR to be one of only two independent biomarkers for CV event risk.[10] A very recent study added to the evidence that changes even within the normal UACR range are independently associated with adverse CV outcomes. It was concluded that there is a continuous association between UACR and CV outcomes, starting as low as 4.4 mg/g.[11] PD was shown to be associated with increased microalbumin-creatinine ratios (UACR). The amount of albumin that gets into the urine is strongly influenced by the health and permeability of the endothelium. PD is known to facilitate endothelial permeability. The biochemical mechanism by which the PD pathogen *Fusobacterium nucleatum (Fn)* can produce increased endothelial permeability was recently discovered.[28] *Fn* contains a molecule FadA adhesion, which binds to vascular endothelial (VE)-cadherin, a member of the cadherin family and a cell-cell junction molecule. This causes relocation of VE-cadherin away from the endothelial cell-cell junctions and results in increased endothelial permeability.[12] It should be no surprise that *Fn*, a frequent pathogen in periodontal infections, is frequently found in carotid atheromas.[13]

Lp-PLA2

Lipoprotein-associated phospholipase A-2 (Lp-PLA2) is an enzyme that was FDA approved for coronary heart disease assessment in 2003 and stroke risk assessment in 2005. It is now emerging not only as a marker of arterial wall inflammation, but also as a direct player in the atherosclerotic disease process. A recently published study suggests Lp-PLA2 plays a key role in cholesterol plaque inflammation and vulnerability. The authors note that this supports Lp-PLA2 inhibition as a strategy for the prevention of CVD.[15] PD has been associated with elevated levels of Lp-PLA2.[14] With the above evidence, it is not surprising that a sophisticated look at the degree of inflammation utilizing F-fluorodeoxyglucose (FDG)-PET imaging showed a strong relationship between the degree of periodontal inflammation and carotid inflammation. This same study also correlated the amount of carotid inflammation found histologically from 16 endarterectomy specimens with FDG-PET periodontal inflammation and found an extremely strong relationship with an R value =0.81, p <0.001.[16] These important data support PD being independently associated with CVD. PD is a CVD risk factor.

Inflammation is causal of atherosclerosis

Current genetic research indicates that Inflammation appears to be causal for CHD.[28] Prior to this data, the hypothesis existed that persistent inflammation was a contributor to the various stages of the pathogenesis of CVD; however, causality had not been established. Sarwar's recent genetic work demonstrated via the Interleukin-6 receptor (IL-6R) signaling pathway that inflammation has a causal role in the development of CVD. Furthermore,

this research can be "used to validate and prioritize novel therapeutic opportunities to treat CVD." This research has tremendous impact on the connection between PD and CHD. The associated link between PD and CVD involves systemic inflammation. We now have the opportunity to further investigate this relationship as a possible causal role by investigating effects of PD on IL6R in randomized trials.

PD and CVD causal — obstacles to overcome

The recent AHA publication regarding PD and CVD highlighted the difficulty in proving a cause and effect relationship. One of the largest hurdles is that there is substantial variation in a clinical diagnosis of PD. It remains a research and clinical challenge to establish a causal association between the two conditions when the definition involves subjective clinical measurements. Concluding causality should demand a very objective definition of PD. A publication that helps elucidate the differences between defining PD by "clinical signs" (subjective factors) vs. defining PD by specific periodontal bacteria (objective factors) and looking for a relationship with atherosclerosis was done by Beck et al., *Circulation*, 2005.[26] Of the 15,792 subjects aged 45 to 64 in the ARIC study, 6,793 underwent a complete periodontal examination. The ARIC clinical evaluation included anthropometry including waist-to-hip ratio, BP, cognitive function, ECG, clinical chemistries, plasma lipids, medications listed, and health questionnaires. The periodontal examination included periodontal status as measured by probing depth and cementoenamel junction measures relative to the gingival margin on six sites for all teeth. They also utilized clinical attachment level by calculating the sum of probing depth and cementoenamel junction scores. A more objective assessment of PD was also used. This was done by measuring serum IgG antibody levels to 17 high-risk periodontitis organisms known as risk organisms for periodontitis. The results of this multivariable analysis indicated the subjective PD status was not significantly associated with CHD in either ever-smokers or never-smokers. In contrast, the objective assessment of PD status demonstrated high antibodies above the median to *Td, Pi, Capnocytophaga ochracea (Co), Aa,* and the combination of *A. Actinomycetemcomitans* with *Capnocytophaga ochracea* were associated with CHD among never-smokers. The odds ratio (OR) varied based on the prevalence of specific bacteria or combinations from 2.0 to 1.5.

The conclusions were as follows: "Clinical signs of PD were not associated with CHD. The more objective assessment of PD status via systemic antibody response to specific PD bacteria was associated with CHD in ever-smokers and never-smokers. These findings indicate that the quality and quantity of the host response to oral bacteria may be an exposure more relevant to systemic atherothrombotic coronary events than clinical measures. These findings are relevant in that they indicate that the subjective clinical definition of periodontitis may not adequately represent the systemic burden of PD."[24]

Furthermore, "effective treatment" of periodontal disease must include more definitive pre- and postbiological parameters to offset the variability associated with traditional clinical measures usually associated with this term. There are several ways of obtaining this information today: subgingival microbial colonization by certain pathogens; analyzing levels of IgG or IgA for selected organisms; and identification and relative concentrations of specified pathogens through salivary DNA analysis. Studies such as INVEST[17] indicate that PD would be better defined by the concentrations or burden of various pathogens known to drive CV risk than the more subjective definitions.

Another barrier to demonstrating causation is the myriad of other known CV risk factors. A definitive study will need a control group and a PD group with statistically matched CV risk factors ranging from psychosocial to medications. In addition, those risk factors will need to be controlled in a static and controlled manner throughout the trial. This task is not impossible, but it is very difficult and will require a substantial number of subjects due to attrition and potential exclusion during the trial period. There is another hurdle to overcome when establishing causality that was not mentioned in the AHA paper. It involves the recent discovery that many plaque rupture/erosions do not result in a symptomatic event. As discussed previously in this article, there are three common scenarios. The scenarios range from a symptomatic heart attack or stroke, the "silent" damage to the heart or brain from the assault of tiny thrombi, which can embolize in the distal vessels and, lastly, a "healing" of the injury, which causes progressive enlargement of the plaque.[6] Therefore, research designed to investigate a cause and effect will have to incorporate a way to measure for the silent events and the increased plaque burden. These issues

of definition, effective treatment protocols, confounding comorbidities, and comprehensive event endpoints are not impossible to overcome, but they create challenges for a quick definitive study for PD being causal of CVD.

Why CV and PD providers need to communicate

In light of the intolerable burden of CVD, the Level A evidence that PD is independently associated with CVD, and the recently published data indicating a cause and effect relationship of inflammation to CVD, it is imperative that efforts to enhance CV wellness incorporate PD evaluations and therapies. Any modifiable CV risk factor should be addressed in a holistic approach to lessen the impact of CVD. Unequivocal evidence that PD is causal of CVD is not necessary. Guidelines should recognize the significant association between the two conditions and call for the assessment and treatment of PD not only for the patient's oral health, but also as one of many comprehensive measures that may help maintain CV health.

The medical community must value the dental role in managing the modifiable disease state of PD as it relates to CVD. The dental community should remain steadfast and stand proud of its vigilance with PD. The *American Journal of Cardiology* and *Journal of Periodontology* editors' consensus paper in 2009 called for the collaboration between medicine and dentistry to improve patient care. We are long overdue to make their joint statement a reality for our patients. There is a sense of urgency for medicine and dentistry to join together to improve the lives of our patients by reducing the morbidity and mortality associated with vascular disease. The consistent association between periodontal infections and vascular inflammation demands that dental professionals begin a mindset of systemic health management through CV wellness optimization. Knowing that the morbidity and mortality associated with vascular disease is catastrophic, the dental profession must be recognized by the medical community as true partners in cardiovascular prevention efforts. Dental professionals have a responsibility to recognize risk and celebrate that their work has systemic merit. Bridges of communication must be established between the medical and dental communities with the intention of decreasing the morbidity and mortality associated with vascular disease. Indeed, dentists play a key role in the prevention of heart attacks and ischemic strokes through appropriate screening efforts and optimization of oral health.

Bibliography

1. Heidenreich PA, Trogdon JG, Khavjou OA, Butler J, Dracup K, Ezekowitz MD, Finkelstein EA, Hong Y, Johnston SC, Khera A, Lloyd-Jones DM, Nelson SA, Nichol G, Orenstein D, Wilson PW, Woo YJ; American Heart Association Advocacy Coordinating Committee, Stroke Council, Council on Cardiovascular Radiology and Intervention, Council on Clinical Cardiology, Council on Epidemiology and Prevention, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Nursing, Council on the Kidney in Cardiovascular Disease, Council on Cardiovascular Surgery and Anesthesia, and Interdisciplinary Council on Quality of Care and Outcomes Research. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. Circulation. 2011; 123:933–944.
2. Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, Bravata DM, Dai S, Ford ES, Fox CS, Fullerton HJ, Gillespie C, Hailpern SM, Heit JA, Howard VJ, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Makuc DM, Marcus GM, Marelli A, Matchar DB, Moy CS, Mozaffarian D, Mussolino ME, Nichol G, Paynter NP, Soliman EZ, Sorlie PD, Sotoodehnia N, Turan TN, Virani SS, Wong ND, Woo D, Turner MB; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics — 2012 update: a report from the American Heart Association Circulation. Jan.3, 2012; 125(1):e2-e220.
3. Weintraub WS, Daniels SR, Burke LE, Franklin BA, Goff DC, Hayman LL, Jones DL, Pandey DK, Sanchez EJ, Schram AP, Whitsel LP, on behalf of the American Heart Association Advocacy Coordinating Committee, Council on Cardiovascular Disease in the Young, Council on the Kidney in Cardiovascular Disease, Council on Epidemiology and Prevention, Council on Cardiovascular Nursing, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Clinical Cardiology, and Stroke Council. Value of primordial and primary prevention for cardiovascular disease: A policy statement from the American Heart Association. Circulation. Aug. 23, 2011; 124(8):967-990.
4. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L, on behalf of the INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. Sept. 11-17, 2004; 364(9438):937-952.
5. O'Donnell MJ, Xavier D, Liu L, Zhang H, Chin SL, Rao-Melacini P, Rangarajan S, qul Islam S, Pais P, McQueen MJ, Mondo C, Damasceno A, Lopez-Jaramillo P, Hankey GJ, Dans AL, Yusoff K, Truelsen T, Diener H-C, Sacco RL, Ryglewicz D, Czlonkowska A, Weimar C, Wang X, Yusuf S, on behalf of the INTERSTROKE investigators. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study. Lancet. Jul. 10, 2010; 376(9735):112-123.
6. Arbab-Zadeh A, Nakano M, Virmani R, Fuster V. Acute coronary events. Circulation. Mar. 6, 2012; 125:1147-1156.
7. Lockhart PB, Bolger AF, Papapanou PN, Osinbowale O, Trevisan M, Levison ME, Taubert KA, Newburger JW, Gornik HL, Gewitz MH, Wilson WR, Smith Jr. SC, Baddour LM. Periodontal disease and atherosclerotic vascular disease: does the evidence support an independent association? A scientific statement from the American Heart Association. Circulation published online April 18, 2012 http://circ.ahajournals.org/content/early/2012/04/18/CIR.0b013e31825719f3.
8. Tricoci P, Allen JM, Kramer JM, Califf RM, Smith SC. Scientific evidence underlying the ACC/AHA clinical practice guidelines. JAMA. Feb. 25, 2009; (301)8:831-841.
9. Tsioufis C, Thomopoulos C, Soldatos N, Kasiakogias A, Andrikou I, Kordalis A, Toutouzas K, Giamarelos G, Tousoulis D, Kallikazaros I, Stefanadis C. Periodontal disease severity and urinary albumin excretion in middle-aged hypertensive patients. Am J Cardiol. Jan. 2011; 107(1):52-58.
10. Wang TJ, Gona P, Larson MG, Tofler GH, Levy D, Newton-Cheh C, Jacques PF, Rifai N, Selhub J, Robins SJ, Benjamin EJ, D'Agostino RB, Vasan RS. Multiple biomarkers for the prediction of first major cardiovascular events and death. N Engl J Med Dec. 21, 2006; 355:2631-2639.
11. Estacio RO, Dale RA, Schrier R, Krantz MJ. Relation of reduction in urinary albumin excretion to 10-year cardiovascular mortality in patients with type 2 diabetes and systemic hypertension. Am J Cardiol 2012; in press.
12. Fardini Y, Wang X, Témoin S, Nithianantham S, Lee D, Shoham M, Han YW. Fusobacterium nucleatum adhesin FadA binds vascular endothelial cadherin and alters endothelial integrity. Molecular Microbiology. 2011; 82(6):1468-1480.
13. Figuero E, Sánchez-Beltrán M, Cuesta-Frechoso S, Tejerina JM, del Castro JA, Gutiérrez JM, Herrera D, Sanz M. Detection of periodontal bacteria in atheromatous plaques by nested polymerase chain reaction. Journal of Periodontology. Aug. 2011; DOI: 10.1902/jop.2011.100719.
14. Mochari H, Grbic JT, Mosca L. Usefulness of self-reported periodontal disease to identify individuals with elevated inflammatory markers at risk of cardiovascular disease. Am J Cardiol Dec. 1, 2008; 102:1509-1513.
15. Gonçalves I, Edsfeldt A, Young Ko N, Grufman H, Berg K, Björkbacka H, Nitulescu M, Persson A, Nilsson M, Prehn C, Adamski J, Nilsson J. Evidence supporting a key role of Lp-PLA2-generated lysophosphatidylcholine in human atherosclerotic plaque inflammation.

Arterioscler Thromb Vasc Biol. May 2012; 32:00-00. http://atvb.ahajournals.org/content/early/2012/04/12/ATVBAHA.112.249854
16. Fifer KM, Qadir S, Subramanian S, Vijayakumar J, Figueroa AL, Truong QA, Hoffman U, Brady TJ, Tawakol A. Positron emission tomography measurement of periodontal F-fluorodeoxyglucose uptake is associated with histologically determined carotid plaque inflammation J Am Coll Cardiol. Feb. 15, 2011; 57:971-976.
17. Desvarieux M, Demmer RT, Rundek T, Boden-Albala J, Sacco RL, Papapanou. Periodontal Microbiota and Carotid Intima-media thickness: The oral infections and vascular disease epidemiology Study (INVEST) Circulation. Feb. 8, 2005; 111(5):576.
18. Xiang Ying Ouyang, Wen Mei Xiao, Yi Chu, Shuang Ying Zhou. Influence of periodontal intervention therapy on risk of cardiovascular disease. Periodontology 2000, 2011; (56):227-257.
19. Beikler T, Flemmig TF. Systemic implications of oral biofilm-associated diseases. Periodontology 2000. 2011; (55)87-103.
20. Bahekar AA, Singh S, Saha S, Molnar J, Arora R. The prevalence and incidence of coronary heart disease is significantly increased in periodontitis: A meta-analysis: American Heart Journal (AHJ). Nov. 2007; 154(5)830-837.
21. Humphrey LL, Rongwei Fu, Buckley DI, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: A systematic review and meta-analysis. J Gen Intern Med. Sept. 2008; 23(12):2079-2086.
22. Axel S, Klein E, Khuseyinova N, Boeckh C, Muche R, Kunze M, Rothenbacher D, Pezeshki G, Hoffmeister A, Koenig W. Role of periodontal bacteria and importance of total pathogen burden in the coronary event and periodontal disease (CORODENT) Study: ARCH INTERN MED. 2006; Vol. 166, 66(5):554-559.
23. Lei L, Li H, et al. Porphyromonas gingivalis lipopolysaccharide (LPS) alters atherosclerotic-related gene expression in oxidized low-density-lipoprotein-induced macrophages and foam cells. J Periodontol Res. Mar. 21, 2011; doi: 10.1111/j.1600-0765.2011.01356.x.26.
24. Beck JD, Eke P, Heiss G, Madianos P, Couper D, Lin D, Moss E, Elter J, Offenbacher S. Periodontal disease and coronary heart disease: A reappraisal of exposure. Circulation. 2005; 112:19-24.
25. Taylor A, Doneen A, et al. SAIP Council. Appropriate use criteria for cIMT. Atherosclerosis. Oct. 19, 2010.
26. Nambi V et al. J Am Coll Cardiol. Oct. 2010; 55:1600–1607.
27. Sarwar N, Butterworth AS, et al. IL6R Genetics Consortium and Emerging Risk Factors Collaboration. Lancet Mar. 31, 2012; 379:1205–1213.
28. Tsioufis T, Thomopoulos C, Soldatos N, Kasiakogias A, Andrikou I, Kordalis A, Toutouzas K, Giamarelos G, Tousoulis D, Kallikazaros I, Stefanadis C. Periodontal disease severity and urinary albumin excretion in middle-aged hypertensive patients Am J Cardiol. Jan. 2011; 107(1):52-58.
29. http://www.uspreventiveservicetaskforce.org/uspstf/grades.htm.

Author Profiles

Thomas W. Nabors, DDS, FACD Dr. Nabors received his degree from the University Of Tennessee College Of Dentistry. He served in the U.S. Navy as a dentist and rotated through all of the dental specialties including oral surgery, periodontal surgery, endodontic therapy, prosthetic dentistry and general dentistry. During his tenure in private practice, he served on the medical staff of Baptist Memorial Hospital in the Oral Surgery and Dental Division for approximately 25 years.

In 2004, he co-founded and served as CEO for Advanced Dental Diagnostics, LLC.

In 2008, he founded OralDNA® Labs: a salivary diagnostics clinical laboratory.

In 2009, Dr. Nabors worked with a dedicated team to validate saliva as a suitable molecular analyte for detection of HPV in the oral cavity.

Dr. Nabors is a frequent lecturer for both dental and medical groups on the subject of molecular genetics in the field of oral medicine: Including the role that periodontal disease contributes to systemic inflammation and this relationship to heart disease, stroke and diabetes. His lectures also include the role that HPV is associated in the development of certain forms of oropharyngeal cancer. Today, Dr. Nabors serves as an executive consultant to Quest Diagnostics and as president and founder of Integrated HealthCare Consultants, LLC. He has published numerous articles within a variety of peer reviewed publications. He is a Life Member of the American Dental Association, an associate member of the AAP, a Fellow of the American College of Dentists, a member of the Pierre Fauchard Honorary Society, and serves within numerous dental associations. He can be reached at: drtomnabors@gmail.com

Bradley F. Bale, MD, is one of the nation's leading specialists in preventing heart attacks, stroke, and diabetes. He cofounded the Bale/Doneen Method and the Heart Attack & Stroke Prevention Center. He is a principal instructor in the Bale/Doneen Method, training other medical providers across the country. He also serves as the medical director of the Heart Health Program for Grace Clinic in Lubbock, Texas. He is an assistant clinical professor at Texas Tech School of Medicine and an adjunct professor at Texas Tech School of Nursing.

Amy L. Doneen, MSN, ARNP, is the cofounder and medical director of the Heart Attack & Stroke Prevention Center in Spokane, Wash. She is also an adjunct professor at Texas Tech Health Sciences School of Nursing. An international speaker on cardiovascular disease prevention, she is the cofounder and instructor of the Bale/Doneen Method course for the prevention of heart attacks, strokes, and diabetes, as well as the chair of the Pacific Northwest Preventive Cardiovascular Nurses Association.

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Questions

1. Studies that provide evidence of the association between periodontitis and CVD include:
   a. Those that show periodontitis as an independent risk factor for CVD
   b. Those that provide evidence of periodontopathogenic bacteria in atherogenesis
   c. Those that focus on the systemic effect of periodontal intervention
   d. All of the above
2. The prevalence and incidence of coronary heart disease in subjects with periodontal disease is:
   a. Not related
   b. 100% related
   c. Significantly increased
   d. Decreased
3. The INVEST study from 2005 examined the influence of periodontal microbiota on vascular injury and carotid intima-media thickness. The findings included:
   a. Overall periodontal bacterial burden was related to CIMT changes
   b. Were clinically relevant based on IMT changes
   c. Was specific to causative bacterial burden and dominance of etiological bacteria
   d. All of the above
4. The CORODENT study concluded that:
   a. Bacteria from PD are not involved in systemic inflammation
   b. Bacteria from PD are not related to coronary events
   c. A statistically significant association between periodontal pathogen burden and CHD does exist
   d. None of the above
5. Recent literature reveals that LPS, a bacterial toxin from P. gingivalis, may increase risk for vascular injury by:
   a. Blocking WBCs
   b. Altering gene expression of macrophages
   c. Mimicking HDL
   d. Doesn't affect vascular health
6. In the ARIC trial, the results indicated that clinical parameters of PD were:
   a. Directly involved in ASVD
   b. Indirectly involved in ASVD
   c. Dependent on depth of pockets and BOP
   d. Not significantly associated with CHD
7. In the INVEST study from 2010, the relationship between periodontal microbiota and hypertension was reviewed. The findings include:
   a. Periodontal etiological bacterial burden is positively associated with hypertension
   b. SBP was elevated in the highest tertiles by 9mmHg and DPB by 5 mmHg
   c. Etiological bacteria include Aa, Pg, Tf, and Td.
   d. The data provides evidence of a direct relationship between levels of specific subgingival bacteria and both BP and DBP
   e. All of the above
8. In the ARIC trial, high levels of antibodies to specific periodontal pathogens above the medium were:
   a. Associated with CHD among never-smokers and ever-smokers
   b. Indicative that the quality and quantity of host response to oral bacteria is more relevant to coronary events than clinical measures
   c. The odds ratio of total pathogen burden showed a 100% increase in risk
   d. All of the above
9. Our country currently spends annually approximately ____ on the direct and indirect costs of cardiovascular disease (CVD)?
   a. $20 billion
   b. $98 billion
   c. $125 billion
   d. $221 billion
   e. $300 billion
10. What percent of CVD deaths occur in individuals younger than the current life expectancy age?
    a. 5%
    b. 12%
    c. 25%
    d. 33%
    e. 45%
11. Every dollar spent on wellness care would save ____ dollars in direct and indirect illness expenditures?
    a. $1.85
    b. $2.75
    c. $3.25
    d. $6.00
    e. $8.45
12. Inflammation appears causal for CVD and can be a manifestation of the following conditions:
    a. Stress
    b. Hyperlipidemia
    c. Diabetes
    d. Periodontal disease (PD)
    e. All of these conditions
13. Endothelial permeability may be impaired by the following:
    a. Nitric oxide
    b. Exercise
    c. Fusobacterium nucleatum
    d. Mediterranean diet
    e. Laughter
14. PD has been associated with carotid artery inflammation by:
    a. Empiric knowledge
    b. Calcification
    c. Carotid IMT
    d. PET imaging
    e. MRI
15. PD has evidence of being associated with the following biomarkers:
    a. Hs-CRP
    b. Fibrinogen
    c. Microalbuminuria
    d. Lp-PLA2
    e. All of these biomarkers
16. PD is objectively defined by which of the following?
    a. Pocket depth
    b. Attachment loss
    c. Salivary DNA
    d. None of these
    e. All of these
17. Overlapping CVD and PD risk factors include the following:
    a. Diabetes
    b. Smoking
    c. Hypertension
    d. a and b
    e. All of these
18. The statement that PD is independently associated with CVD arises from what level of evidence?
    a. A
    b. B
    c. C
    d. Expert opinion
    e. Anecdotal
19. Identifying and treating PD may mitigate the risk of:
    a. Halitosis
    b. Bleeding gums
    c. Tooth loss
    d. Diabetes
    e. CVD
    f. All of these
20. Programs focused on maintaining wellness should address the following issues:
    a. PD
    b. Weight
    c. Sleep
    d. Anxiety
    e. Diet
    f. All of these

ANSWER SHEET

Inculpatory Evidence: Periodontal Disease Assessment and Treatment Is an Essential Element in Cardiovascular Wellness Programs

Name: Title: Specialty:

Address: E-mail:

City: State: ZIP: Country:

Telephone: Home ( ) Office ( ) Lic. Renewal Date:

Requirements for successful completion of the course and to obtain dental continuing education credits: 1) Read the entire course. 2) Complete all information above. 3) Complete answer sheets in either pen or pencil. 4) Mark only one answer for each question. 5) A score of 70% on this test will earn you 2 CE credits. 6) Complete the Course Evaluation below. 7) Make check payable to PennWell Corp. **For Questions Call 216.398.7822**

Educational Objectives

The participants will appreciate the catastrophic economic and health expenditures associated with cardiovascular disease.

1. The learners will synthesize the value of educating patients on the pathophysiology of arterial disease.
2. The student will understand that it is inflammation that can trigger a plaque rupture and potential thrombus that could block the flow of blood.
3. The learner will recognize that the current health-care platform is based on a risk factor paradigm rather than a disease treatment paradigm.
4. The reader will appreciate the inflammatory link between periodontal disease and vascular disease.
5. The learner will articulate the opportunity to identify silent atherosclerosis in the artery wall using carotid intima media thickness testing.
6. The student will appreciate that periodontal disease is associated with many vascular inflammatory markers such as hsCRP, fibrinogen, microalbumin, and Lp-PLA2.
7. The participant will elucidate the value of objectifying the clinical diagnosis of periodontal disease by using oral pathogen burden evaluation rather than simply relying on clinical symptoms.
8. The learner will recognize that the dental provider plays an essential role in the prevention of heart attacks and ischemic strokes.
9. The reader will understand the merit of Level A evidence that connects periodontal disease with arterial disease.

Course Evaluation

1. Were the individual course objectives met?

Objective #1:	Yes	No	Objective #6:	Yes	No
Objective #2:	Yes	No	Objective #7:	Yes	No
Objective #3:	Yes	No	Objective #8:	Yes	No
Objective #4:	Yes	No	Objective #9:	Yes	No
Objective #5:	Yes	No			

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
5. How do you rate the author's grasp of the topic?	5	4	3	2	1	0
6. Please rate the instructor's effectiveness.	5	4	3	2	1	0
7. Was the overall administration of the course effective?	5	4	3	2	1	0
8. Please rate the usefulness and clinical applicability of this course.	5	4	3	2	1	0
9. Please rate the usefulness of the supplemental webliography.	5	4	3	2	1	0

10. Do you feel that the references were adequate? Yes No

11. Would you participate in a similar program on a different topic? Yes No

12. If any of the continuing education questions were unclear or ambiguous, please list them.

13. Was there any subject matter you found confusing? Please describe.

14. How long did it take you to complete this course?

15. What additional continuing dental education topics would you like to see?

If not taking online, mail completed answer sheet to

Academy of Dental Therapeutics and Stomatology,
A Division of PennWell Corp.
P.O. Box 116, Chesterland, OH 44026
or fax to: (440) 845-3447

For IMMEDIATE results,
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Answer sheets can be faxed with credit card payment to
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2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ	20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ	Ⓕ

AGD Code 018

PLEASE PHOTOCOPY ANSWER SHEET FOR ADDITIONAL PARTICIPANTS.

COURSE EVALUATION and PARTICIPANT FEEDBACK

We encourage participant feedback pertaining to all courses. Please be sure to complete the survey included with the course. Please e-mail all questions to: michellef@pennwell.com.

INSTRUCTIONS

All questions should have only one answer. Grading of this examination is done manually. Participants will receive confirmation of passing by receipt of a verification form. Verification of Participation forms will be mailed within two weeks after taking an examination.

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All participants scoring at least 70% on the examination will receive a verification form verifying 2 CE credits. The formal continuing education program of this sponsor is accepted by the AGD for Fellowship/ Mastership credit. Please contact PennWell for current term of acceptance. Participants are urged to contact their state dental boards for continuing education requirements. PennWell is a California Provider. The California Provider number is 4527. The cost for courses ranges from $29.00 to $110.00.

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Academy of General Dentistry PACE Program Approval for Continuing Education

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Completing a single continuing education course does not provide enough information to give the participant the feeling that s/he is an expert in the field related to the course topic. It is a combination of many educational courses and clinical experience that allows the participant to develop skills and expertise.

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