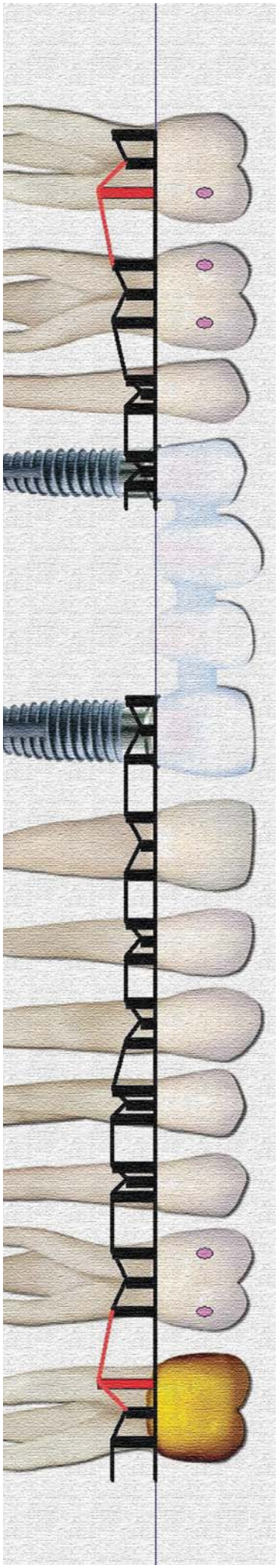

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Stress: Empowering Clinicians to Address Its Role in Periodontal Disease

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Introduction

One hundred years of experimental research have provided us with proof that there is a statistically significant association between stress and the onset, course, and outcome of certain human diseases.[1] We have long since known about the threat stress poses to health—and that stress may contribute to the onset of human opportunistic infections; however, for many dental professionals, rolling this science into everyday practice is a stretch. Few understand the mystery that weaves the deleterious effects of stress through the central nervous and immune systems to affect periodontal disease. It is a complicated biological mechanism to be sure.

The purpose of this article is to provide an overview of the research for dental professionals to better understand the progression of evidence that has accumulated over the last 5 decades to support the role stress has in increasing the risk for the onset, rate of progression, and severity of periodontal disease and its resistance to treatment. The primary objectives of this course are to:

- Give clinicians the information they need to empower them to recognize patients who are at risk for periodontal disease as a result of psychosocial stress;
- Give clinicians the information they need to recognize those patients who present with periodontal status that has significantly changed, and to assess whether psychosocial stress may be the precipitating factor; and
- Provide a compelling rationale for why patients at risk for periodontal disease due to psychosocial stress should be referred for stress-management counseling and/or to their physicians.

From Chickens with Anthrax to Periodontal Disease

In 1878, Pasteur observed that chickens normally resistant to anthrax became susceptible following immersion in cold water.[1] This discovery probably marked the beginning of research on the relationship between stress and infectious pathology. Over the last decade, the field of psychoneuroimmunology has provided more and more evidence about how the immune system communicates bidirectionally with the nervous and endocrine systems during periods of stress, evoking physiologic changes that are clinically profound. As psychiatrists, infectious-disease physicians, gastroenterologists, and endocrinologists increasingly apply this evidence to their practices, dental professionals have been reticent to do so. Evidence of the damaging effects of stress on the periodontium has been accumulating for over 50 years. Although lack of confirmed longitudinal studies prevent us from assigning the term “risk factor” to stress as it relates to periodontal disease, it is widely acknowledged that stress is a “risk indicator” for the onset, progression, and severity of periodontal disease.

Risk Factors and Risk Indicators

As with many other chronic infections, the onset and progression of periodontal disease is largely programmed by the influence of certain local and systemic risk factors that have a dramatic effect on the resistance of the host to pathologic bacteria. In periodontal disease, we call these pathogenic bacteria “periodontal pathogens” and the factors that increase the risk for periodontal disease (risk factors) include diabetes, smoking, and a genetic variable called the interleukin-1 polymorphism (IL-1 genotype). Smokers are 4 times more likely to have periodontitis than nonsmokers.[3] Diabetic patients with poor metabolic control of their glycated hemoglobin have a much greater risk for progressive bone loss compared to well-controlled patients, with an odds ratio of 11.4 to 2.2, respectively.[4] The IL-1 genotype is a specific genetic marker that identifies patients who have increased risk of developing severe periodontal disease.[2] Nonsmokers or former light smokers (< 5pk/yr) who are genotype-positive are more than 3 times more likely to have moderate-to-severe periodontal disease than individuals who are genotype negative.[5] Patients who are both genotype positive and also smoke may be 7.7 times more likely to have tooth loss than nonsmokers who are genotype negative.[6]

Today, more clinicians are beginning to perform risk assessment for periodontal disease that identifies smoking, diabetes, and sometimes genetic predisposition in at-risk individuals. However, identifying patients at risk due to psychosocial stress is often overlooked. Perhaps this is because the evidence to support psychosocial stress as a significant periodontal disease modifier is not as well established as the risk factors of smoking, diabetes, and the IL-1 genotype—or perhaps it is because the science is still somewhat “fuzzy.” What should be clear, however, is that psychosocial stress may be a potent variable in modifying risk for periodontal disease.

A risk factor is established by longitudinal studies that confirm an association between the factor and the outcome of interest. That association must be consistent with the current understanding of the disease process.[7] Furthermore, the association must still remain after controlling for other risk factors and background characteristics.[7] Risk indicators differ from risk factors in so much as the association between the exposure event and the outcome of interest has not yet been demonstrated by longitudinal studies.[7] However, stress, like other risk indicators of periodontal disease (i.e., osteoporosis, HIV, immunocompromised conditions) are commonly accepted (putative) as variables that influence the onset, progression, and severity of periodontal disease. It is important that readers understand that, although longitudinal studies are still needed to support stress as a bona fide risk factor, the role of psychosocial stress in the etiology of periodontal disease has been proposed by many authors over the span of 5 decades.[8–12]

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Tracing the Investigation of the Relationship Between Stress and Periodontal Disease

The earliest studies linking stress to periodontal conditions were related to acute necrotizing ulcerative gingivitis (ANUG).[13] Researchers began to see that stress was related not only to increased incidence of ANUG cases in college students during examination periods, but also with chronic periodontitis. In 1974, DeMarco (Case Western Reserve) treated outpatient Vietnam veterans at the Veterans Administration Hospital and noted: "Though a definite cause and effect relationship cannot be established, it [case studies of Vietnam veterans] would lead one to believe that the alveolar bone loss may be associated with stress. These cases provide evidence to consider the naming of a disease entity entitled "Periodontal Emotional Stress Syndrome" (PESS), which results in severe vertical and horizontal bone loss with associated pocket formation, consistent involvement of the first molar teeth and a paucity of other local etiological factors." It seems DeMarco was ahead of the curve when he wrote: "Assessment of emotional stability and stress should be made in such cases. Tension and stress may produce varying deleterious effects on different parts of the body (i.e., ulcers, colitis, mental illness, etc.), and the alveolar bone does not seem to be immune. Emotional stress should be looked upon as an important contributing factor, or possibly a primary causative factor, in the pathogenesis of periodontal disease."[14]

In the 1990s case-control studies that supported a relationship between chronic periodontitis, life events, and social demographics started to emerge.[15,19,20] In his 1996 landmark editorial, Genco wrote that stress was a risk factor for periodontal disease. Genco and colleagues proposed that psychological measures of stress were associated with financial strain and should be recognized as significant risk indicators for periodontal disease in adults. In their research, they found that financial stress is significantly associated with greater attachment and alveolar bone loss after adjusting for age, gender, and smoking. Another of their findings was that salivary levels of cortisol, which depresses the immune response,[16] were higher in the test group exhibiting severe periodontitis, a high level of financial strain, and poor coping abilities. Furthermore, those patients who had inadequate stress-coping abilities had an even higher risk of having more severe attachment loss and alveolar bone loss. These findings were later corroborated by Wimmer and colleagues.[18]

Numerous other studies during this time proposed a role for stress in the etiology of periodontal disease.[19–23] It was also during this time that another important finding was reported: Stress causes periodontal patients to be more resistant to treatment.[24] Deinzer and colleagues researched the relationship of plaque and stress on the trajectory of periodontal disease. They found that when stress and plaque are concomitantly present, stress affected periodontal health by increasing local interleukin-1ß (IL-1ß), which is the most potent osteoclast-activating factor[25] and bone formation inhibitor[26] in humans. This was especially so when oral hygiene was neglected.[27] In a subsequent study, Deinzer and colleagues found that if a patient was first exposed to stress, and then oral hygiene was neglected, the stress, may persistently alter the immunological effects of the microbial challenge to the periodontium.[28] The same researchers found that in periodontally healthy patients, IL-1ß is histologically located much further away from the alveolar bone than periodontally less healthy patients with already-existing alveolar bone loss. At inflamed sites of these patients, IL-1ß was present next to the alveolar bone. For these patients, stress-induced increases of IL-1ß might increase the risk of further bone resorption.[28] Findings from this series of studies led Deinzer and colleagues to recently note: "Stress should be included as a factor in models of patient compliance and health behavior."[29]

More recent research findings related to the role stress might play in influencing the onset, rate of progression, severity of periodontal disease, and response to therapy include:

- The frequency of moderate CAL (4–6 mm) and moderate PPD (4–6 mm) were found to be significantly associated with higher anxiety after adjusting for socioeconomic data and cigarette consumption.[30]
- Smoking and stress enhance production of inflammatory cytokines such as IL-1ß, IL-6, and IL-8, which has clinical consequences.[31]
- Stress significantly enhances the effects of nicotine on periodontal tissues.[32]
- Chronic stressors may accelerate risk of a host of age–related diseases by prematurely aging the immune response.[33]
- Lifestyle and psychosocial stress may affect the periodontal disease status of diabetic patients.[34]
- Case studies reporting exaggerated inflammatory responses to low levels of plaque, exacerbated by episodes of stress, have been reported.[35]
- In subjects affected with hereditary angio-oedema (HAO) 63% (15/24) demonstrated that mental stress was a triggering factor for an acute episode of the condition.[36]
- Psychological stress seems to adversely affect women more than men both with respect to immunological and clinical alterations.[37]

Stress and the Microbiology of Periodontal Disease

It is generally recognized that microorganisms possess the ability to recognize hormones within the host and utilize them to adapt to their surroundings. This supports the supposition that psychological stress may favor the development of many bacterial infections. To that end, the effect that stress may have on microbial compositions in the subgingival niches has more recently been researched. In 2002, researchers performed in vitro investigations to determine whether noradrenalin (norepinephrine), and adrenaline (epinephrine), which are released during human stress responses, act as environmental cues to alter the growth of 43 microorganisms found within subgingival microbial complexes.[38] The researchers found that 20 species within the subgingival biofilm significantly grew from inoculation with noradrenalin, and 27 species significantly grew when adrenaline was introduced. There was also a difference in the growth response within bacterial species and within and between microbial complexes. The researchers concluded that this variation may influence the in vivo composition of the subgingival biofilm in response to stress-induced changes in local catecholamine levels and play a significant role in the etiology and pathogenesis of periodontal diseases.

Shortly after this research was published, it was discovered that chronic psychological stress has a marked impact on the localized immune response to *P. gingivalis*.[39] The ramifications of these findings are of great significance because *P. gingivalis* is the most often cited periodontal pathogen implicated in the link between periodontal disease and cardiovascular disease.[40, 41]

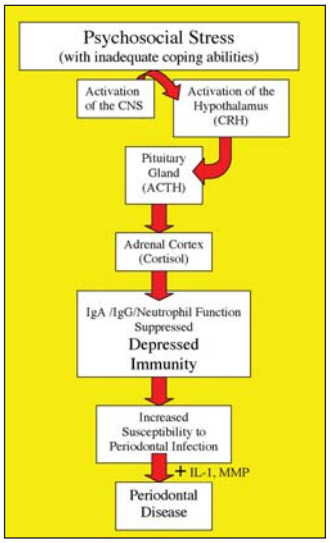


FIGURE 1

Biologic model to explain the role that stress has in influencing the onset and progression of periodontal disease. CNS= central nervous system; CRH= corticotropic-releasing hormone; ACTH= adrenocorticotropic hormone; IL-1= interleukin- 1; MMP= matrix metalloproteinase.

Adapted from Genco RJ, Ho AW, Kopman J, et al. Models to evaluate the role of stress in periodontal disease. *Ann Periodontol* 1998;3:288-302.

Pathways to Explain the Role of Stress in Periodontal Disease

Unraveling the complex and multifactorial cascade of events that attempt to explain the biological mechanism associated with the stress-related periodontal disease trajectory is not so easy. Unlike animal research, research that involves human subjects imposes behavioral variables that constrain the investigation.[1] These potentially confounding variables include such things as sleep patterns, diet, smoking ,exercise, alcohol consumption, caffeine intake, licit and illicit drug use- all of which influence the function of the immune system.[1]

The mechanisms to elucidate how stress may affect periodontal disease is not known at this time; however there are 2 pathways which may explain the role that stress has in influencing the onset and progression of periodontal disease. These include the biologic model and the behavioral model.[17]

The biologic model proposes that periodontal disease may be biologically moderated through the hypothalamic-pituitary-adrenal (HPA) axis to promote the release of corticotropic-releasing hormone from the hypothalamus and glucocorticosteroid, from the adrenal cortex. **Figure 1** illustrates the following cascade of events:

1. When a potentially stressful situation is appraised as threatening the central nervous system (CNS) and hypothalamus is activated.
2. The hypothalamus induces secretion of corticotrophin-releasing hormone (CRH) which flows to the pituitary gland where it stimulates the secretion of adrenocorticotropic hormone (ACTH).

3. ACTH enters the peripheral blood flow, and induces the adrenal cortex to secrete cortisol and other steroids. Cortisol is a type of glucocorticosteroid and its increased production results in immunosuppression and reduced resistance to infection.
4. Then glucocorticosteroids (hormones made in the body that have major suppressive actions on immune and inflammatory responses), including cortisol, depress immunity.[17] This is done by suppressing IgA, which protects by preventing initial colonization of periodontal organisms, and IgG, which exerts protection by covering the periodontal bacteria with a type of coating that allows the phagocytes to bind and ingest the invading bacteria in addition to suppressing neutrophil functions.[17] All of these immune processes are important in protecting against infection by the colonization of periodontal pathogens.[17]
5. Susceptibility, is then increased, which leads to the beginning or advancement of periodontal infection, eventuating leading to destructive periodontitis.[17]

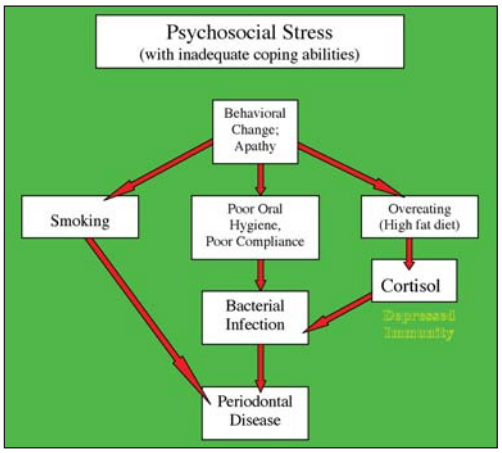


FIGURE 2
Behavioral model to explain the role that stress has in influencing the onset and progression of periodontal disease.

Source: Genco RJ, Ho AW, Kopman J, et al. Models to evaluate the role of stress in periodontal disease. *Ann Periodontol* 1998;3:288-302.

The behavioral model **(Figure 2)** suggests that psychosocial stress may precipitate behavioral changes that affect at-risk health behaviors (i.e., smoking, poor oral hygiene, poor compliance). The possibility also exists that stress leads to overeating, especially high fat diets, which can lead to immunosuppression through increased cortisol production.[16]

Future research on the role of stress in increasing risk for periodontal disease should consider the validity of both the biologic model and the behavioral model.[17]

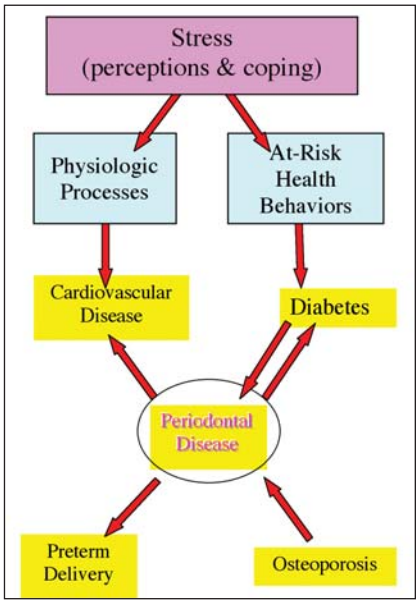


FIGURE 3
The common pathway of stress as related to periodontal disease, cardiovascular disease, diabetes, preterm delivery, and osteoporosis.

Source: Genco RJ, Ho AW, Kopman J, et al. Models to evaluate the role of stress in periodontal disease. *Ann Periodontol* 1998;3:288-302.

In addition, it is likely that systemic diseases associated with periodontal diseases such as diabetes, cardiovascular disease, preterm delivery, and osteoporosis may share psychosocial stress as a common risk factor **(Figure 3).**[17]

Identifying Psychosocial Stress in Our Patients

The use of a questionnaire that includes questions related to the patient's psychosocial stress (i.e., whether the patient is in bereavement, strain or failure of relationships, major work- or school-related events, periods of financial difficulty[16]) in addition to other risk indicators and risk factors, allows clinicians to intercept individuals who may be at risk for periodontal disease. When patients present with periodontal status that has significantly changed in the short term, clinicians should be aware that something within the patient's immune system has created increased susceptibility to periodontal breakdown. In patients that present with no classical periodontal disease risk factors (i.e., smoking, diabetes, or genetic predisposition), savvy clinicians will also screen for psychosocial stress. The following case fits this description.

Patient John Doe is a 58-year-old, Caucasian male who has been a patient of record for 3 years. He is married and has 3 children in high school; the whole family is diligent about coming for biannual examination and prophylaxis visits. When he was a teenager, John Doe had a traumatic injury of the mouth that caused the loss of teeth nos. 9–12. These were permanently replaced about 4 years ago with a bridge stabilized by implant replacements for teeth nos. 9 and 12. John Doe reported to be in excellent health, took no medications, and had no preexisting conditions that would have impacted treatment decisions or clinical outcomes.

Figure 4 is the periodontal chart of John Doe at his first visit to the practice. This patient presented with what most clinicians would agree as good periodontal stability.

- Minimal plaque
- Minimal bleeding upon probing (BOP) at 2%
- 3 pockets that were 4 mm
- There were no deep pockets (defined as ≥ 4 mm) with BOP
- No bone loss visible on radiographs

During visits 2, 3, 4, and 5 over the course of 2 years, little changed in the periodontal status of John Doe. Six months later, during his sixth visit, the patient was seen for examination and prophylaxis. His periodontal evaluation revealed that something dramatic had happened relative to his periodontal stability **(Figure 5)**. Some of these key clinical findings which are highlighted at the bottom of the chart in trend analysis data included:

- Minimal plaque
- Bleeding upon probing increased to 30%
- Pockets ≥ 4 mm increased to 53%
- Deep pockets (defined as ≥ 4 mm) with BOP increased to 28%
- Horizontal bone loss visible on radiographs

Nothing had changed in the medical profile of John Doe, but what had changed was his emotional health. When the patient was asked whether he had experienced any type of recent stress, the patient advised that he was going through a divorce. Also, one of his children was recently diagnosed with a serious pulmonary condition requiring on-going out patient care.

Stress comes in physical, emotional, and psychological forms. This patient's stress may have encompassed all three. It was explained to John Doe that patients with stress who had good coping ability were at less risk for periodontal breakdown than those with poor coping ability;[17] stress management counseling was recommended. The patient was somewhat resistant to this recommendation, but further discussion of the potential for serious stress-induced systemic conditions provided a compelling rationale for this patient to actively seek counseling.

Conclusion

It is widely acknowledged that individuals are not equally susceptible to periodontal disease and that periodontal disease occurs in susceptible individuals when certain pathogens colonize the periodontal niche. What we know today is that although these pathogens are necessary for degradation of the periodontal tissues, their presence alone is not sufficient to cause disease in all individuals. It is other factors, including acquired, environmental, and genetic risk factors that tip the scale from health to disease in any given individual. Psychosocial stress is considered a risk indicator for periodontal disease. It is likely that systemic diseases that are associated with periodontal disease such as diabetes, cardiovascular disease, preterm delivery, and osteoporosis may share psychosocial stress as a common risk factor.

Assessment of risk for periodontal disease is the cornerstone of contemporary periodontics. Evaluating patients' for psychosocial stress is an important component in assessing risk for periodontal disease, and may be pivotal in pointing patients in the direction of greater systemic health.

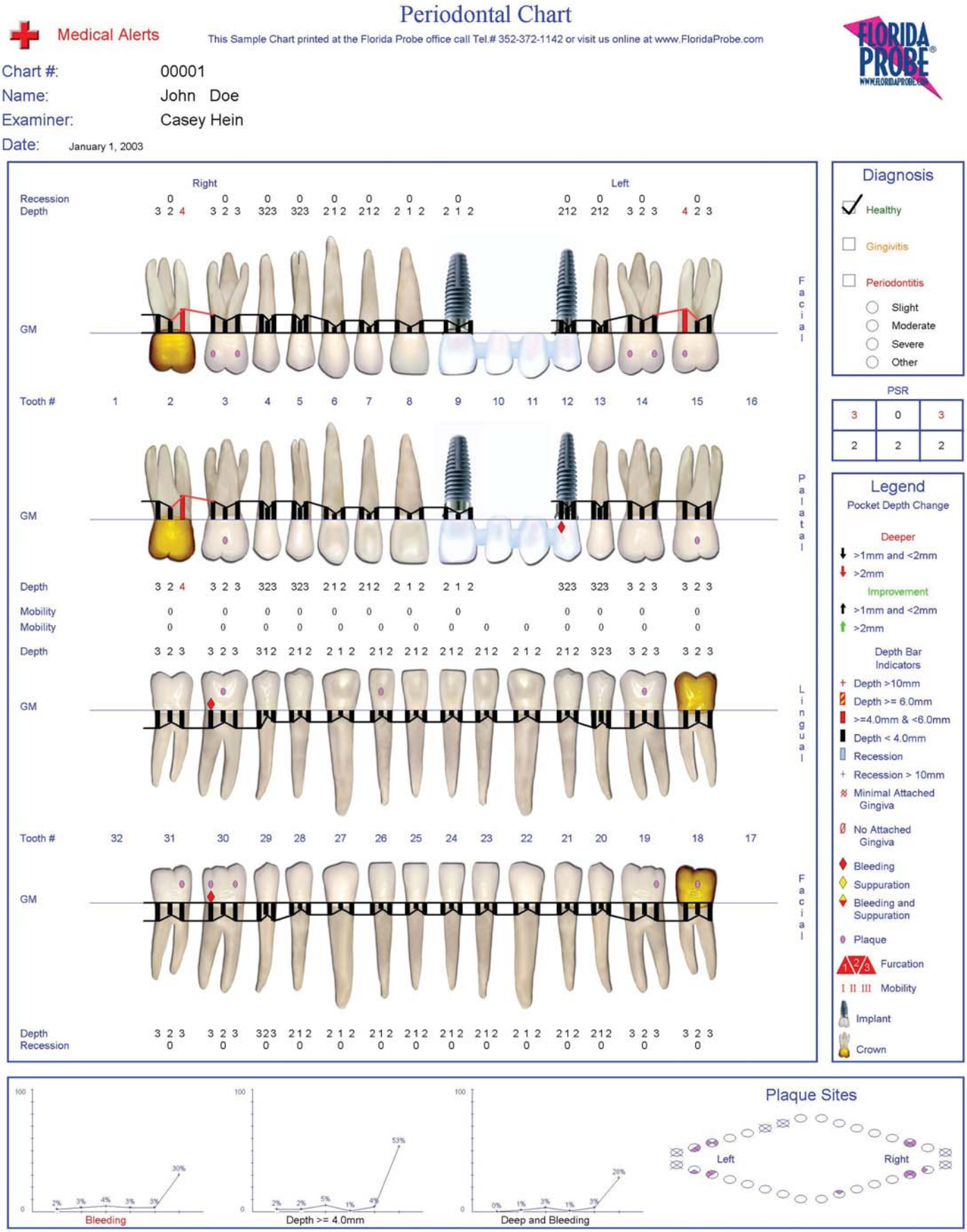
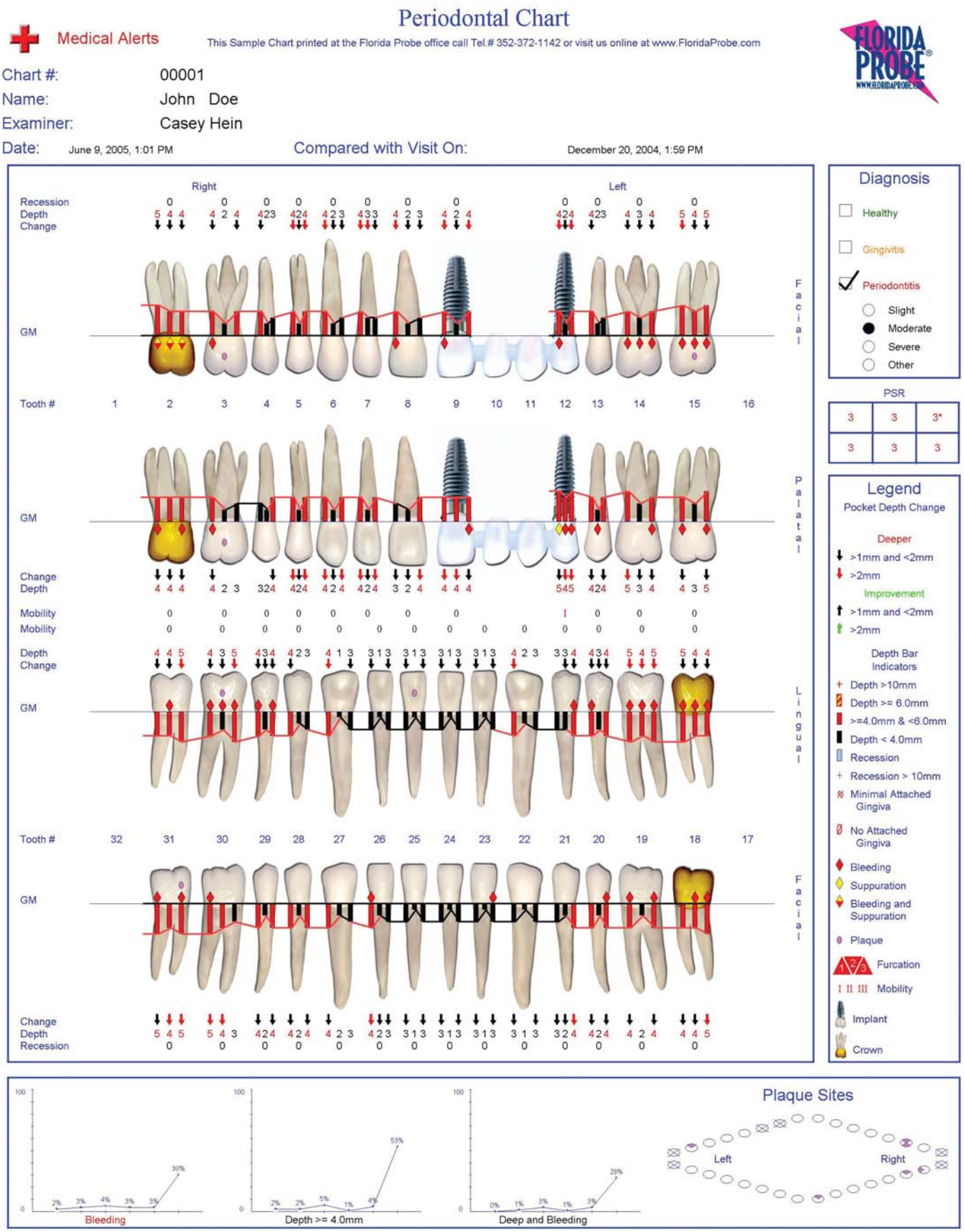


FIGURE 5


Periodontal Chart
Medical Alerts
This Sample Chart printed at the Florida Probe office call Tel.# 352-372-1142 or visit us online at www.FloridaProbe.com
Chart #: 00001
Name: John Doe
Examiner: Casey Hein
Date: June 9, 2005, 1:01 PM
Compared with Visit On: December 20, 2004, 1:59 PM
Right
Left
Recession Depth Change
GM
Facial
Tooth # 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
Palatal
Change Depth
Mobility
Depth Change
Lingual
Tooth # 32 31 30 29 28 27 26 25 24 23 22 21 20 19 18 17
Change Depth Recession
Diagnosis
Healthy
Gingivitis
Periodontitis
Slight
Moderate
Severe
Other
PSR
3 3 3*
3 3 3
Legend
Pocket Depth Change
Deeper
>1mm and <2mm
>2mm
Improvement
>1mm and <2mm
>2mm
Depth Bar Indicators
Depth >10mm
Depth >= 6.0mm
>=4.0mm & <6.0mm
Depth < 4.0mm
Recession
Recession > 10mm
Minimal Attached Gingiva
No Attached Gingiva
Bleeding
Suppuration
Bleeding and Suppuration
Plaque
Furcation
I II III Mobility
Implant
Crown
Bleeding
Depth >= 4.0mm
Deep and Bleeding
Plaque Sites
Left
Right
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Continuing Education Test Questions

Answer Sheet on Back Cover

1. Evidence of the damaging effects of stress on the periodontium:
a. has been accumulating for over 50 years
b. was first published in research in 1999
c. is just now beginning to be explored
d. does not exist

2. The difference between a risk factor and a risk indicator is that:
a. The term risk factor is used for adult diagnoses and risk indicator is used for child diagnoses
b. Risk factors have been confirmed by longitudinal studies and risk indicators have not yet been demonstrated by longitudinal studies
c. There is not as much research to support risk factors as there is risk indicators for periodontal disease
d. There is no difference; the terms may be interchanged

3. Patients who smoke are ___ times more likely to have periodontal disease than nonsmokers.
a. 2
b. 3
c. 4
d. 5

4. Diabetic patients with poor metabolic control of their glycated hemoglobin have a much greater risk for progressive bone loss compared to well-controlled diabetics with an odds ratio of:
a. 3.3 to 4.6
b. 4.2 to 5.8
c. 11.4 to 2.2
d. 5.4 to 5.9

5. Smokers who are also genetically predisposed to periodontal disease are ____ times more likely to have tooth loss than nonsmokers who are not genetically predisposed.
a. 3
b. 5
c. 6.5
d. 7.7

6. A risk indicator for periodontal disease is:
a. osteoporosis
b. HIV
c. immunocompromised conditions
d. all the above

7. Genco and colleagues found that salivary levels of cortisol were higher in patients who:
a. had severe periodontitis, poor compliance to oral hygiene, and rampant caries
b. had severe periodontitis, a high level of financial strain, and poor coping abilities
c. had healthy periodontal tissues with an intact immune system
d. had missing teeth replaced by implants

8. Deinzer and colleagues found that when stress and plaque are present at the same time, stress affected periodontal health by increasing local interleukin 1ß (IL-1ß), which is:
a. an antigen related to periodontal pathogens
b. the most potent osteoclast activating factor
c. a bone formation inhibitor
d. b and c

9. It has been discovered that chronic psychological stress has a marked impact on the localized immune response to:
a. *Streptococcus mutans*
b. *H. pylori*
c. *P. gingivalis*
d. *E. coli*

10. Variables that influence the function of the immune system and also confound research findings related to stress and periodontal disease include:
a. sleep patterns
b. diet
c. smoking
d. all the above

11. The mechanisms to explain how stress may affect periodontal disease include:
a. the biological model and the behavioral model
b. the biological model and an enzymatic mechanism
c. a behavioral model and compliance index
d. no mechanisms have yet to be proposed; no one knows

12. When a potentially stressful situation is appraised as threatening what organs and systems are activated?
a. coronary arteries and pulmonary alveoli
b. the central nervous system and hypothalamus
c. the stomach and endocrine system
d. the skeletal system and kidneys

13. Glucocorticosteroids
a. are hormones made in the body
b. suppress the immune response
c. suppress the inflammatory response
d. all the above

14. To explain the relationship between stress and periodontal disease, the behavioral model suggests that psychosocial stress may initiate behavioral changes that may lead to:
a. smoking
b. poor oral hygiene
c. overeating
d. all the above

15. The use of a patient questionnaire that includes questions relative to psychosocial stress allows clinicians to intercept patients who may be at risk for periodontal disease. Questions should include issues such as whether the patient is:
a. in bereavement
b. experiencing the strain or failure of a relationship
c. experiencing work- or school-related events
d. all the above

CONTINUING EDUCATION ANSWER SHEET

STRESS: EMPOWERING CLINICIANS TO ADDRESS ITS ROLE IN PERIODONTAL DISEASE

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ANSWER SHEET

1.	A	B	C	D	9.	A	B	C	D
2.	A	B	C	D	10.	A	B	C	D
3.	A	B	C	D	11.	A	B	C	D
4.	A	B	C	D	12.	A	B	C	D
5.	A	B	C	D	13.	A	B	C	D
6.	A	B	C	D	14.	A	B	C	D
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