

Comprehensive Review of Gingivitis and Periodontitis

¹Shaza Jameel Ashqar, ²Abdullah Faisal Alim, ³Hanan Abdullah Turkstani, ⁴Danyah Abdulkarim Faisal Karsan, ⁵Raji Ehsan Mohammed Kensara, ⁶Nermeen Saad Felemban

Abstract: This review was aimed to discuss the Gingivitis and Periodontitis from different perspectives, intended to review the risk factors associated with these dental diseases, diagnostic, and prevention & treatment. Comprehensive search was conducted through; PubMed, Medline, and EBSCO databases, searching literature for relevant studies discussing the Gingivitis and Periodontitis, using following Mesh terms: “Gingivitis” and “Periodontitis” AND “gum inflammation” AND “Gum loss” AND “dental inflammation”. Searching databases was restricted to English published studies up to December, 2016. Furthermore, references from different identified articles were searched for more matches articles that could be useful in this review. The prevention and also therapy of the gum diseases is based on exact medical diagnosis, decrease or removal of original representatives, risk management as well as correction of the dangerous impacts of disease. Popular and also verified risk factors or risk predictors for periodontitis in grownups consist of cigarette smoking, diabetic issues, race, *P. gingivalis*, *P. intermedia*, low education and learning, irregular oral participation as well as genetic influences. A number of various other specific periodontal germs, herpesviruses, raised age, male sex, depression, race, terrible occlusion and female osteoporosis in the presence of hefty dental calculus have been shown to be related to loss of gum support as well as can be taken into consideration to be risk indications of periodontitis.

Keywords: Gingivitis and Periodontitis, Depression, Race, Terrible Occlusion and Female Osteoporosis.

1. INTRODUCTION

Gingivitis and periodontitis represent more than 95% of all inflammatory diseases of the tissues surrounding the teeth, making up the principal source of missing teeth in adults. Gingivitis is a relatively harmless inflammation of the gum tissues, with linked blood loss and exudation ⁽¹⁾. Gingivitis might transform to periodontitis, inflammation that prolongs deep into the tissues and triggers loss of supporting connective tissue as well as alveolar bone is known as periodontitis ⁽¹⁾. Periodontitis impacts over fifty percent of the grownups in the United States ⁽²⁾ as well as could result in pain, decreased masticatory capacity, and in extreme instances oral abscesses and tooth loss. Periodontal disease has actually been linked as a possible risk factor for a number of systemic problems including cardiovascular diseases, ^(3,4) and also reduced birth weight as well as early infants ⁽⁵⁾. In the United States, periodontal diseases disproportionately impact individuals of lower socioeconomic teams, with above average levels of disease reported in Hispanic and also African American populaces ⁽²⁾. In its early stages, nonetheless, it is feasible to deal with periodontitis efficiently and prevent or regulate disease progression. Therefore, it would be better to identify and also treat this infection early, as opposed to face oral and also possible systemic problems associated with moderate or severe gum loss.

Periodontal condition has actually been classified in different methods. A classification based on infection as the primary etiology of periodontal diseases divides classifications based on gingival inflammation and gum add-on loss as well as recognizes health and wellness, gingivitis, as well as periodontitis as different entities ⁽⁶⁾. Splitting up of gingivitis from periodontitis suggests that there are distinctions in these conditions that may consist of type or intensity of infection, and/or adequacy of host reaction. Scientific attributes associated with gum loss in topics with modest to advanced periodontitis have included existence of plaque, gingival redness, hemorrhaging on penetrating, gum stealing, and

periodontal accessory loss^(7,8). The most effective sign for future periodontal accessory loss in adult periodontitis was extent of existing accessory loss, whereas in early-onset periodontitis, existence of gingivitis was strongly related to future periodontal attachment loss^(8,9). Cigarette smoking is one more vital risk factor for moderate to sophisticated periodontitis⁽⁹⁾. Therapy of periodontitis need to avoid recurrence of disease, which can be attained by numerous non-surgical and surgical therapies relying on the details therapy goal^(2,7).

This review was aimed to discuss the Gingivitis and Periodontitis from different perspectives, intended to review the risk factors associated with these dental diseases, diagnostic, and prevention & treatment.

2. METHODOLOGY

Comprehensive search was conducted through; PubMed, Medline, and EBSCO databases, searching literature for relevant studies discussing the Gingivitis and Periodontitis, using following Mesh terms: “Gingivitis” and “Periodontitis” AND “gum inflammation” AND “Gum loss” AND “dental inflammation”. Searching databases was restricted to English published studies up to December, 2016. Furthermore, references from different identified articles were searched for more matches articles that could be useful in this review.

3. RESULTS

Risk factors:

a) Microorganisms and Periodontal Disease

The oral bacterial microbiome consists of over 700 different phylotypes, with roughly 400 types located in subgingival plaque^(10,11). The subgingival microflora in periodontitis could nurture hundreds of bacterial species however only a small number has actually been associated with the development of disease and taken into consideration etiologically important. Subgingival plaque from deepened gum pockets is controlled by gram-negative anaerobic poles and spirochetes^(12,13). Strong evidence has linked Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans to the pathogenesis of grown-up periodontitis^(14,15). On top of that, Bacteroides forsythus, Prevotella intermedia, Peptostreptococcus micros, and also Fusobacterium nucleatum have been strongly linked with the development of adult periodontitis^(14,16).

b) Tobacco as risk factor:

There is gathering proof for a greater level of gum disease amongst smokers⁽¹⁷⁾. Tobacco smoking cigarettes applies a considerable harmful result on the gum tissues as well as increases the price of gum disease progression. Risk factors consisting of tobacco cigarette smoking customize the host response to the difficulty of germs in microbial dental plaque⁽¹⁸⁾. Cigarette smokers with periodontal disease seem to reveal less indicators of professional inflammation and also gingival blood loss compared to nonsmokers⁽¹⁹⁾. That could be explained by the truth that nicotine puts in local vasoconstriction, reducing blood flow, edema, as well as medical indications of inflammation⁽²⁰⁾. Pure nicotine acetylcholine receptor has been discovered to play a crucial role in the development of nicotine associated periodontitis⁽²⁰⁾.

c) Diabetes Mellitus & Cardiovascular Diseases:

One of the essential oral indications of diabetes is gingivitis and also periodontitis. Patients with undiagnosed or inadequately regulated diabetic issues mellitus kind 1 or kind 2 go to higher risk for periodontal disease. There are lots of researches that demonstrate an association between diabetic issues as well as a boosted sensitivity to oral infections consisting of gum disease^(21,22,23,24). Periodontitis additionally advances much more rapidly in badly managed diabetics, as well as very early age of start of the disease is seen as a risk factor for more serious diseases^(23,24). Conversely, most well-controlled diabetic patients could keep gum health and will react favorably to periodontal therapy^(21,24). In spite of disparity concerning this issue in the clinical literary works, it appears that the impact of glycemic control is connected to the setting of gum therapy. Many research studies dealt with the effect of gum treatment on glycemic control of diabetes mellitus patients⁽²⁵⁾. The organic plausibility of the association between cardiovascular diseases as well as gum diseases is well researched and also it includes several of the complying with possible mechanisms: high concentrations of cholesterol and also the activity of oral bacteria when atherosclerosis or the involvement of acute-phase healthy proteins that could raise in chronic periodontitis⁽²⁶⁾. Numerous biological systems have been recommended to explain the partnership in between gum diseases and heart diseases. Therefore, periodontitis could possibly elicit a systemic inflammatory reaction as well as it is worthy of even more interest⁽²⁷⁾.

d) Genetic factors:

Studies show hereditary risk factors related to periodontitis^(28,29,30,31). McDevitt et al. show that the composite IL-1 genotype is dramatically related to the severity of adult periodontitis. They also confirmed that both IL-1 genotyping and also cigarette smoking history offer objective risk factors for gum disease in a private practice environment⁽³²⁾. Presently, there are 2 major forms of periodontitis-chronic and aggressive periodontitis. Risk for periodontitis is not shared equally by the populace. It is clear that periodontitis badly impacts a risky group standing for around 10-15% of the population, in whom the disease rapidly advances from chronic gingivitis to devastating periodontitis. This differential risk for periodontitis is consistent with heritable aspects of sensitivity, however straight proof for a differential hereditary contribution to periodontitis originates from several resources. Lots of jobs of the literary works record familial aggregation of gum diseases, but as a result of different terms, classification systems, and also lack of standard approaches of clinical evaluation, it is challenging to contrast records directly. Although gum disease nosology has transformed often times over the timeframe of these reports, many familial reports for periodontitis are for early-onset kinds now called hostile periodontitis. Records of the domestic nature of chronic kinds of periodontitis are much less frequent, although German researches of the familial nature of chronic types of periodontitis from the very early 20th century have actually been examined by^(29,32).

Diagnostic approaches:

Diagnosis is the recognition of the existence of a disease⁽³³⁾. Professional diagnosis of periodontal disease is made by the recognition of different symptoms and signs in the periodontal cells which declare a separation from health. The diagnosis of gum disease requires a firm knowledge of what comprises gum wellness. The healthy periodontium,⁽³⁴⁾ of which only the gingival tissues could be directly observed, is described as being stippled, pale pink or reefs pink, in the Caucasian (**Figure 1**), with different levels of coloring in various other races. It is snugly adjusted to the underlying cells, with a knife edge margin where it abuts the tooth, the gingival margin lies, in the lack of pathology, at the cemento-enamel junction. It shows a scalloped side setup greatest interdentally, where it constitutes the interdental papilla and also least expensive buccally and lingually. There is a gingival hole where it abuts the tooth which in health is 1 - 3 mm deep. There is an absence of hemorrhaging from the crevice on gentle probing. The gap in health will certainly show a small amount of interstitial liquid, gingival crevicular liquid⁽³⁵⁾.



Figure1: Healthy gingiva. Pale pink, stippled

Clinical periodontal examination:

Clinical examination needs to be obtained when the medical history shows a demand to learn more. A common instance is when patients offer a background of a heart murmur or joint substitutes. Present suggestions preclude periodontal probing or any kind of procedure that might induce bleeding in all patients with high or moderate risk for endocarditis unless antibiotic treatment is offered⁽³⁶⁾. According to other suggestions, patients with orthopedic pins, screws and also plates do not need antibiotic treatment, nor is it consistently needed for many dental patients with total joint substitutes⁽³⁷⁾. The gum cells are routinely taken a look at in all oral assessments. The gum assessment consists of a visual inspection of the gingiva, gum probing, and also analysis of tooth flexibility, dental plaque and also calculus. A specific kind must be made use of for tape-recording oral and also gum findings to ensure that they might be contrasted with time. Evaluations such as the Periodontal Screening and also Recording Examination™ (PSRA) that has been recommended by the American Academy of Periodontology and also the American Dental Association might be useful to the general practitioner⁽³⁸⁾. Nonetheless, it is very important to identify that throughout such screening treatments, all surface areas of the teeth must be penetrated for indications of gum disease. The gingiva ought to be visually checked out for indications of inflammation. Healthy and balanced gingiva, in the absence of considerable melanin coloring, is generally a light pink

shade. Raised soreness or erythema is a medical indicator of gingival inflammation due to the enhanced gingival vascularity in feedback to regional toxic irritants such as dental plaque as well as calculus (**Figure 2**)⁽³⁸⁾. The style of the gingiva ought to be analyzed for modifications in the normal knife-edged appearance of the cost-free gingival margin and interdental papilla as it satisfies the teeth. In the absence of systemic disease or drug-associated gingival enhancement, any kind of swelling or an enlarged appearance of the low gingiva signifies inflammation. The uniformity of any type of gingival enhancement ought to be assessed with the side of a periodontal probe to establish whether it is edematous or fibrotic. An additional clinical sign of gingival inflammation is bleeding from the gingival hole when the inner facet of the gingival sulcus is delicately brushed up with the side of a periodontal probe. Any considerable lack of connected gingiva, specifically if it is connected with gingival economic crisis or a high frenum attachment, ought to be noted and also tape-recorded in the dental record⁽³⁸⁾.



Figure 2: A. Periodontal inflammation due to plaque and resubgingival calculus. B. Same patient after improving oral hygiene.

Radiographic examination:

Radiographs are utilized to confirm and extend the findings of the medical evaluation and are vital in planning implant placement to figure out the amount and character of alveolar bone along with the position of physiological structures such as the maxillary sinus and inferior alveolar canal. The presence of gingivitis, gum pockets and gingival inflammation cannot be figured out utilizing radiographs, however radiographs are necessary for identifying the degree and intensity of bone gum assistance and for finding osseous sores. Although breathtaking radiographs provide an outstanding radiographic survey of the oral structures, they do not have the resolution and detail needed for gum diagnosis. When the scientific assessment shows the existence of periodontitis, chosen periapical or bitewing radiographs should be acquired⁽³⁹⁾. When patients have scientific proof of generalized dental disease or a history of extensive dental treatment⁽³⁹⁾, a complete mouth intraoral radiographic assessment is suitable. Because the bisecting angle strategy distorts the relationship in between the alveolar crest and tooth (**Figure 9**), periapical radiographs ought to be exposed utilizing a long-cone paralleling strategy.

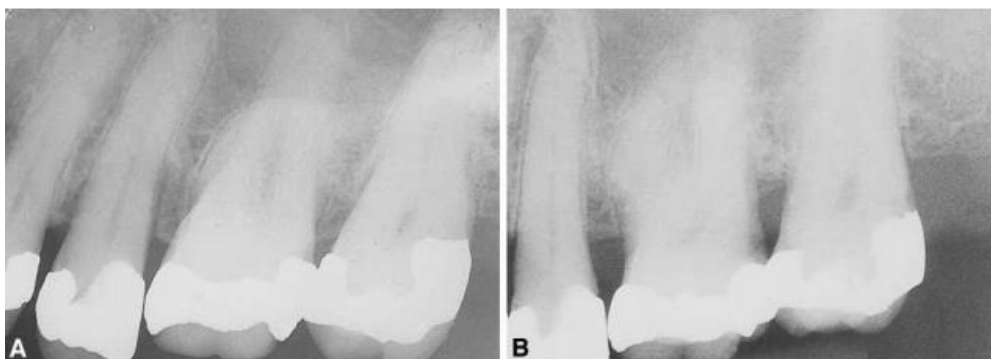


Figure 3: Radiographs (A) and a parallel long-cone method (B). Note distortion of interdental bone on bisecting angle radiograph.

o Treatment approaches of Gingivitis and Periodontitis:

Gingivitis is a reversible disease and therapy is intended primarily at getting rid of or decreasing causative factors. This allows inflammation to resolve and the gingival tissues to recover⁽⁴⁰⁾. Treatment for periodontitis typically falls into two classifications: 1) procedures developed to stop the progression of disease and 2) procedures created to restore structures destroyed by disease⁽⁴⁰⁾. Maintenance or encouraging gum treatment following active treatment is vital to accomplish an

effective outcome. Many years earlier, Ramfjord et al⁽⁴¹⁾ proposed a general plan for the treatment of the gum diseases⁽⁴¹⁾. This strategy included four phases: 1) systemic, 2) hygienic, 3) restorative and 4) maintenance or supportive care. (Table 1)⁽⁴²⁾ summarize the treatment approaches for each type of Gingivitis and Periodontitis.

Table 1: summarize of Gingivitis types, causes, symptoms and proper management

Disease	Primary causes	Signs and symptoms	Therapy
Gingivitis (American Academy of Peridontology case type I)	Bacterial plaque, local plaque retention factors (such as faulty restorations)	Gingival redness and swelling, bleeding; does not cause loss of clinical attachment	Debridement, plaque control, correct plaque-retentive factors, supportive periodontal therapy
Acute necrotizing gingivitis	Bacterial plaque, may be associated with AIDS at any age	Pain, gingival redness, swelling, bleeding, necrosis of interproximal papilla	Debridement, gentle plaque control, antimicrobial rinse, supportive periodontal therapy
Desquamative gingival disease	Skin disease: lichen planus, pemphigus and cicatricial pemphigoid	Gingival redness; epithelial denudation; pain with trauma or on eating and brushing	Gentle plaque control, palliative and symptomatic therapy, supportive periodontal therapy
Gingivitis associated with systemic diseases (such as blood dyscrasias and Wegner's granulomatosis)	Manifestation of systemic disease in gingiva (such as leukemia, neutropenia, erythema multiforme, lupus erythematosus and Wegner's granulomatosis)	Dependent on systemic disease (such as gingival bleeding ecchymosis, redness, swelling, necrosis and pain)	Treatment of systemic disease, atraumatic plaque control, antimicrobial rinse, supportive periodontal therapy
Gingivitis associated with pregnancy	Bacterial plaque, local plaque retention factors, hormonal influence	Gingival redness and swelling, bleeding; pyogenic granuloma	Debridement and plaque control, supportive periodontal therapy; possible excision of pyogenic granuloma
Drug-induced gingival enlargement	Ca ⁺⁺ channel-blocking drugs, phenytoin, cyclosporine	Gingival enlargement	Debridement and plaque control, surgical excision, use of alternative medications, supportive periodontal therapy
Allergic reaction	Local allergens (such as mouth rinses, toothpastes, nickel restorations and acrylic)	Gingival redness and swelling	Identification and elimination of allergenic agent
Herpetic gingivostomatitis	Herpes type I virus	Pain, vesicle formation, ulceration	Palliative and symptomatic therapy, antiviral medication
Gingival disease of specific bacteria or fungal origin	<i>Neisseria gonorrhoea</i> , <i>Treponema pallidum</i> , streptococcal species, <i>Candida</i> , histoplasmosis	Varies according to infectious agent	Identification and elimination or control of infectious agent, appropriate chemotherapy

4. CONCLUSION

The prevention and also therapy of the gum diseases is based on exact medical diagnosis, decrease or removal of original representatives, risk management as well as correction of the dangerous impacts of disease. Popular and also verified risk factors or risk predictors for periodontitis in grownups consist of cigarette smoking, diabetic issues, race, P. gingivalis, P. intermedia, low education and learning, irregular oral participation as well as genetic influences. A number of various other specific periodontal germs, herpesviruses, raised age, male sex, depression, race, terrible occlusion and female osteoporosis in the presence of hefty dental calculus have been shown to be related to loss of gum support as well as can be taken into consideration to be risk indications of periodontitis. The existence of furcation involvement, tooth movement, as well as a parafunctional practice without making use of a biteguard are connected with a poorer gum prognosis complying with gum therapy. An exact medical diagnosis could just be made by an extensive analysis of information that have actually been systematically accumulated by: clinical examination as shown, professional periodontal assessment, and radiographic exam.

REFERENCES

- [1] Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. *Lancet*. 2005;366:1809–20.
- [2] Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988–1994. *J Periodontol*. 1999;70:13–29.
- [3] Beck JD, Slade G, Offenbacher S. Oral disease, cardiovascular disease and systemic inflammation. *Periodontol* 2000. 2000;23:110–120.
- [4] Mattila KJ, Asikainen S, Wolf J, Jousimies-Somer H, Valtonen V, Nieminen M. Age, dental infections, and coronary heart disease. *J Dent Res*. 2000;79:756–760.
- [5] Offenbacher S, Lieff S, Boggess KA, et al. Maternal periodontitis and prematurity. Part I: Obstetric outcome of prematurity and growth restriction. *Ann Periodontol*. 2001;6:164–174.
- [6] Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol*. 1999;4:1–6.
- [7] Haffajee AD, Socransky SS, Lindhe J, Kent RL, Okamoto H, Yoneyama T. Clinical risk indicators for periodontal attachment loss. *J Clin Periodontol*. 1991;18:117–125.
- [8] Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988–1994. *J Periodontol*. 1999;70:30–43.
- [9] Reddy MS, Geurs NC, Jeffcoat RL, Proskin H, Jeffcoat MK. Periodontal disease progression. *J Periodontol*. 2000;71:1583–1590.
- [10] Paster BJ, Olsen I, Aas JA, Dewhirst FE. The breadth of bacterial diversity in the human periodontal pocket and other oral sites. *Periodontology* 2000. 2006;42(1):80–87.
- [11] Berezow AB, Darveau RP. Microbial shift and periodontitis. *Periodontology* 2000. 2011;55(1):36–47.
- [12] Haffajee AD, Socransky SS. Microbial etiological agents of destructive periodontal diseases. *Periodontology* 2000. 1994;5:78–111.
- [13] Moore WE, Moore LV. The bacteria of periodontal diseases. *Periodontology* 2000. 1994;5:66–77.
- [14] Kou Y, Inaba H, Kato T, et al. Inflammatory responses of gingival epithelial cells stimulated with *Porphyromonas gingivalis* vesicles are inhibited by hop-associated polyphenols. *Journal of Periodontology*. 2008;79(1):174–180.
- [15] Doğan B, Kipalev AŞ, Ökte E, Sultan N, Asikainen SE. Consistent intrafamilial transmission of *Actinobacillus actinomycetemcomitans* despite clonal diversity. *Journal of Periodontology*. 2008;79(2):307–315.
- [16] Lovegrove JM. Dental plaque revisited: bacteria associated with periodontal disease. *Journal of the New Zealand Society of Periodontology*. 2004;(87):7–21.
- [17] Kubota M, Tanno-Nakanishi M, Yamada S, Okuda K, Ishihara K. Effect of smoking on subgingival microflora of patients with periodontitis in Japan. *BMC Oral Health*. 2011;11.
- [18] Vouros ID, Kalpidis CDR, Chadjipantelis T, Konstantinidis AB. Cigarette smoking associated with advanced periodontal destruction in a Greek sample population of patients with periodontal disease. *Journal of the International Academy of Periodontology*. 2009;11(4):250–257.
- [19] Zini A, Sgan-Cohen HD, Marcenes W. Socio-economic position, smoking, and plaque: a pathway to severe chronic periodontitis. *Journal of Clinical Periodontology*. 2011;38(3):229–235.
- [20] Özçaka O, Biçakci N, Pussinen P, Sorsa T, Köse T, Buduneli N. Smoking and matrix metalloproteinases, neutrophil elastase and myeloperoxidase in chronic periodontitis. *Oral Diseases*. 2011;17(1):68–76.
- [21] Campus G, Salem A, Uzzau S, Baldoni E, Tonolo G. Diabetes and periodontal disease: a case-control study. *Journal of Periodontology*. 2005;76(3):418–425.

- [22] Graves DT, Al-Mashat H, Liu R. Evidence that diabetes mellitus aggravates periodontal diseases and modifies the response to an oral pathogen in animal models. *Compendium of Continuing Education in Dentistry*. 2004;25(7, supplement 1):38–45.
- [23] Meng H. Association between periodontitis and diabetes mellitus. *Beijing Da Xue Xue Bao*. 2007;39(1):18–20.
- [24] Nishimura F, Soga Y, Iwamoto Y, Kudo C, Murayama Y. Periodontal disease as part of the insulin resistance syndrome in diabetic patients. *Journal of the International Academy of Periodontology*. 2005;7(1):16–20.
- [25] Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. *Annals of Periodontology*. 1998;3(1):51–61.
- [26] Al-Mubarak S, Ciancio S, Aljada A, et al. Comparative evaluation of adjunctive oral irrigation in diabetics. *Journal of Clinical Periodontology*. 2002;29(4):295–300.
- [27] Grossi SG, Skrepcinski FB, DeCaro T, et al. Treatment of periodontal disease in diabetics reduces glycated hemoglobin. *Journal of Periodontology*. 1997;68(8):713–719.
- [28] Hart TC. Genetic considerations of risk in human periodontal disease. *Current Opinion in Periodontology*. 1994:3–11.
- [29] Laine ML, Farré MA, García-González MA, et al. Risk factors in adult periodontitis: polymorphism in the interleukin-1 gene family. *Nederlands Tijdschrift voor Tandheelkunde*. 2002;109(8):303–306.
- [30] Loos BG, John RP, Laine ML. Identification of genetic risk factors for periodontitis and possible mechanisms of action. *Journal of Clinical Periodontology*. 2005;32(supplement 6):159–179.
- [31] López NJ, Jara L, Valenzuela CY. Association of interleukin-1 polymorphisms with periodontal disease. *Journal of Periodontology*. 2005;76(2):234–243.
- [32] McDevitt MJ, Wang H, Knobelmann C, et al. Interleukin-1 genetic association with periodontitis in clinical practice. *Journal of Periodontology*. 2000;71(2):156–163.
- [33] Fowler HW, Fowler FG, eds. *The Concise Oxford Dictionary*. 5th edn. Oxford: Oxford University Press, 1964.
- [34] Lindhe J, Karring T, Lang N, eds. *Clinical periodontology and implant dentistry*. 4th edn. Munksgaard: Blackwell, 2003:3–48.
- [35] Brill N, Krasse B. The passage of tissue fluid into the clinically healthy gingival pocket. *Acta Odont Scand* 1958;16:233–245.
- [36] Dajani AS, Taubert KA, Wilson W, Bolger AF, Bayer A, Ferrieri P, Gewitz MH, Shulman ST, Nouri S, Newburger JW, Hutto C, Pallasch TJ, Gage TW, Levison ME, Peter G, Zuccaro G Jr. Prevention of bacterial endocarditis: recommendations by the American Heart Association. *J Am Dent Assoc* 1997; 128: 1142–1151.
- [37] American Dental Association; American Academy of Orthopaedic Surgeons. Advisory statement. Antibiotic prophylaxis for dental patients with total joint replacements. *J Am Dent Assoc* 1997; 128: 1004–1008.
- [38] American Academy of Periodontology. *Parameters of care*. Chicago: American Academy of Periodontology, 1996.
- [39] American Dental Association. *Recommendations in radiographic practices: an update, 1988*. Council on Dental Materials, Instruments, and Equipment. *J Am Dent Assoc* 1989; 118: 115–117
- [40] Pihlstrom BL, Ammons WF. Treatment of gingivitis and periodontitis. Research, Science and Therapy Committee of the American Academy of Periodontology. *J Periodontol* 1997; 68: 1246–1253.
- [41] Ramfjord SP. A rational plan for periodontal therapy. *J Periodontol* 1953; 24: 75–87.
- [42] Ramfjord SP. Maintenance care and supportive periodontal therapy. *Quintessence Int* 1993; 24: 465–471.